Patricia Gonce Morton
Dorrie K. Fontaine

Critical Care Nursing

Tenth Edition

A Holistic Approach

Wolters Kluwer Health | Lippincott Williams & Wilkins

Critical Care Nursing

A Holistic Approach

Patricia Gonce Morton, RN, PhD, ACNP-BC, FAAN
Professor, Associate Dean for Academic Affairs
University of Maryland School of Nursing
Baltimore, Maryland
Acute Care Nurse Practitioner
Peri-Operative Services
University of Maryland Medical Center
Baltimore, Maryland

Dorrie K. Fontaine, RN, PhD, FAAN
Sadie Heath Cabaniss Professor of Nursing and Dean
University of Virginia School of Nursing
Charlottesville, Virginia
Past-President, American Association of Critical-Care Nurses (AACN)

EDITION 10

Wolters Kluwer Health | Lippincott Williams & Wilkins
Philadelphia • Baltimore • New York • London
Buenos Aires • Hong Kong • Sydney • Tokyo

Executive Editor: Chris Richardson
Senior Product Manager: Helen Kogut
Editorial Assistant: Zachary Shapiro
Design Coordinator: Joan Wendt
Illustration Coordinator: Brett MacNaughton
Manufacturing Coordinator: Karin Duffield
Prepress Vendor: SPi Global

10th edition

9 8 7 6 5 4 3 2 1

Printed in China

Library of Congress Cataloging-in-Publication Data
Critical care nursing : a holistic approach / [edited by] Patricia Gonce Morton, Dorrie K. Fontaine. — 10th ed.
p. ; cm.
Includes bibliographical references and index.
ISBN 978-1-60913-749-6
I. Morton, Patricia Gonce, 1952- II. Fontaine, Dorrie K.
[DNLM: 1. Critical Care. 2. Holistic Nursing. WY 154]
610.73'6—dc23

2012010189

LWW.com

To our former students. You have been our teachers and our source of inspiration. Thanks for all you do for critically ill patients and families.

To our husbands John and Barry. Thanks for your never ending support, love, and encouragement.

TRISH AND DORRIE

Contributors

Susan E. Anderson, RN, MSN
Senior Quality Assurance Specialist
U.S. Army Graduate Program in Anesthesia Nursing
Fort Sam Houston, Texas

Nathaniel M. Apatov, MSN, PhD, MHS, CRNA
Program Director, Nurse Anesthesia Program
Old Dominion University
Norfolk, Virginia

Sue Apple, RN, PhD
Retired
Formerly Assistant Professor
Georgetown University
Washington, District of Columbia

Richard Arbour, RN, MSN, CCRN, CNRN, CCNS, FAAN
Critical Care Nurse Specialist
Albert Einstein Medical Center
Philadelphia, Pennsylvania

Carla A. Aresco, RN, MS, CRNP
Nurse Practitioner, Shock Trauma
R Adams Cowley Shock Trauma Center
University of Maryland Medical Center
Baltimore, Maryland

Mona N. Bahouth, MSN, CRNP
Nurse Practitioner
Baltimore, Maryland

Kathryn S. Bizek, MSN, ACNS-BC, CCRN
Nurse Practitioner, Cardiac Electrophysiology
Henry Ford Heart and Vascular Institute
Henry Ford Health System
Detroit, Michigan

Nancy Blake, RN, MN, CCRN, NEA-BC
Director, PCS Critical Care Services
Children's Hospital Los Angeles
Los Angeles, California

Kay Blum, PhD, CRNP
Nurse Practitioner and Coordinator
Cardiac Risk Reduction Center
Southern Maryland Hospital Center
Clinton, Maryland

Garrett K. Chan, PhD, APRN, BC-PCM, CEN
Lead Advanced Practice Nurse
Clinical Decision Area/Emergency Department
Stanford Hospital and Clinic
Assistant Clinical Professor
Schools of Nursing and Medicine, University of California, San Francisco
San Francisco, California

Donna Charlebois, RN, MSN, ACNP-CS
Acute Care Nurse Practitioner
Interventional Cardiology
University of Virginia Health System
Charlottesville, Virginia

Dennis J. Cheek, RN, PhD, FAHA
Abell-Hanger Professor of Gerontological Nursing
Texas Christian University Harris College of Nursing and Health Sciences
Fort Worth, Texas

Mary Ciechanowski, RN, MSN, ACNS-BC, CCRN
Stroke Program Advanced Practice Nurse
Christiana Care Health Systems
Newark, Delaware

JoAnn Coleman, RN, DNP, ACNP, ANP, AOCN
Acute Care Nurse Practitioner
Gastrointestinal Surgical Oncology
Johns Hopkins Hospital
Baltimore, Maryland

Vicki J. Coombs, RN, PhD, FAHA
Senior Vice President
Spectrum Clinical Research Incorporated
Towson, Maryland

Kelley Caldwell Crusius, RN, CCRN
Staff Nurse
University Medical Center
Tucson, Arizona

Joan M. Davenport, RN, PhD
Assistant Professor and Vice-Chair
Department of Organizational Systems and Adult Health
University of Maryland School of Nursing
Baltimore, Maryland

Marla J. De Jong, RN, PhD, CCNS, Col
Dean
United States Air Force School of Aerospace Medicine
Wright-Patterson Air Force Base, Ohio

Emily Smith Des Champs, RN, MS, CCRN
Clinical Nurse Specialist, Neurology
University Medical Center
Tucson, Arizona

Nancy Kern Feeley, RN, MS, CRNP, CNN
Nephrology Adult Nurse Practitioner
Johns Hopkins University School of Medicine
Baltimore, Maryland

Charles Fisher, RN, MSN, CCRN, ACNP-BC
Acute Care Nurse Practitioner
Medical Intensive Care Unit
University of Virginia Health System
Charlottesville, Virginia

Dorrie K. Fontaine, RN, PhD, FAAN
Sadie Heath Cabaniss Professor of Nursing and Dean
University of Virginia School of Nursing
Charlottesville, Virginia
Past-President, American Association of Critical-Care Nurses (AACN)

Conrad Gordon, RN, MS, ACNP
Assistant Professor
Department of Organizational Systems and Adult Health
University of Maryland School of Nursing
Baltimore, Maryland

Christine Grady, RN, PhD
Head, Section on Human Subjects Research
Department of Bioethics
Clinical Center
National Institutes of Health
Bethesda, Maryland

Debby Greenlaw, MS, CCRN, ACNPC
Acute Care Nurse Practitioner
Providence Hospital
Columbia, South Carolina

Thomasine D. Guberski, PhD, CRNP
Director, Health Program-Nursing
Institute of Human Virology
University of Maryland School of Medicine
Baltimore, Maryland

Kimberli Haas, RN, BSN
Staff Nurse II
University Medical Center
Tucson, Arizona

John C. Hagen, MS, CCRN, CRNP-AC
Acute Care Nurse Practitioner
University of Maryland Medical Center
Baltimore, Maryland

Jillian Hamel, MS, ACNP-BC, CCNS, CCRN
Nurse Practitioner
University of Maryland Medical Center
Baltimore, Maryland

Kathy A. Hausman, RN, C, PhD
Assistant Professor
University of Maryland School of Nursing
Baltimore, Maryland

Jan M. Headley, RN, BS
Director, Clinical Marketing and Professional Education
Edwards Lifesciences LLC
Irvine, California

Janie Heath, PhD, APRN-BC, FAAN
Associate Dean for Academic Affairs
University of Virginia School of Nursing
Charlottesville, Virginia

Genell Hilton, PhD, CRNP
Nurse Practitioner, Trauma Services
San Francisco General Hospital
San Francisco, California
Adjunct Faculty
Santa Rosa Junior College
Santa Rosa, California

Janice J. Hoffman, RN, PhD, CCRN
Assistant Dean for Baccalaureate Program and Assistant Professor
University of Maryland School of Nursing
Baltimore, Maryland

Dorene M. Holcombe, RN, MS, ACNP, CCRN
Nephrology Acute Care Nurse Practitioner
Johns Hopkins University School of Medicine
Baltimore, Maryland

Elizabeth Holderness, RN, MS, ACNP-BC
Nurse Practitioner, Cardiac Surgery
University of Maryland Medical Center
Baltimore, Maryland

Christina Hurlock-Chorostecki, PhD(c), APN
Nurse Practitioner
St. Joseph Health Care, London
London, Ontario, Canada

Karen L. Johnson, RN, PhD
Director of Nursing, Research, and Evidence-Based Practice
University of Maryland Medical Center
Associate Professor
University of Maryland School of Nursing
Baltimore, Maryland

Lisa M. Johnson, RN, MS, ACNP-BC, CCRN
Major, U.S. Army Nurse Corps
Nurse Manager, Burn Intensive Care Unit
U.S. Army Medical Department
Sam Houston, Texas

Dennis W. Jones, RN, DNP, CFRN
Critical Care Flight/Transport Nurse
Johns Hopkins Hospital
Baltimore, Maryland

Kimmith M. Jones, RN, DNP, CCNS
Advanced Practice Nurse in Critical Care
Sinai Hospital of Baltimore
Baltimore, Maryland

Roberta Kaplow, RN, PhD, AOCNS, CCNS, CCRN
Clinical Nurse Specialist
Dekalb Medical
Decatur, Georgia

Jane Kapustin, PhD, CRNP, BC-ADM, FAANP, FAAN
Associate Professor
Assistant Dean for Masters and Doctor of Nursing Practice Programs
University of Maryland School of Nursing
Adult Nurse Practitioner, Joslin Diabetes Center
University of Maryland Medical Center
Baltimore, Maryland

Elizabeth Kozub, RN, MS, CCRN, CNRN
Nurse Clinician III
Sharp Memorial Hospital
San Diego, California

Sun-Ah Lee, RN, MS, CRNP
Nurse Practitioner
University of Maryland Medical System
Baltimore, Maryland

Barbara Leeper, RN, MN, CNS, M-S, CCRN, FAHA
Clinical Nurse Specialist, Cardiovascular Services
Baylor University Medical Center
Dallas, Texas

Tara Leslie, RN, BSN
RN Extender
St. Joseph Cardiology Associates
Lexington, Kentucky

Susan Luchka, RN, MSN, CCRN, ET
Director of Clinical Education
Memorial Hospital
York, Pennsylvania

Christine N. Lynch, RN, MS, ACNP, CCRN
Acute Care Flight Nurse Practitioner
Cleveland Clinic, Critical Care Transport
Cleveland, Ohio

Megan Cecere Lynn, RN, MS, MBA, FNE-A
Clinical Instructor
University of Maryland School of Nursing
Baltimore, Maryland

Cathleen R. Maiolatesi, RN, MS
Advanced Practice Nurse
Johns Hopkins Hospital
Baltimore, Maryland

E. Jane McCarthy, PhD, CRNA, FAAN
Visiting Professor
University of Maryland School of Nursing
Baltimore, Maryland

Sandra W. McLeskey, RN, PhD
Professor
University of Maryland School of Nursing
Baltimore, Maryland

Patricia C. McMullen, PhD, JD, CRNP
Dean, School of Nursing
The Catholic University of America
Washington, District of Columbia

Paul K. Merrel, RN, MSN, CCNS
Advanced Practice Nurse 2—CNS, Adult Critical Care
University of Virginia Health System
Charlottesville, Virginia

Sandra A. Mitchell, PhD, CRNP, AOCN
Nurse Scientist
National Institute of Health Clinical Center
Bethesda, Maryland

Patricia A. Moloney-Harmon, RN, MS, CCNS, FAAN
Advanced Practice Nurse
Children's Services
Sinai Hospital of Baltimore
Baltimore, Maryland

Patricia Gonce Morton, RN, PhD, ACNP-BC, FAAN
Professor and Associate Dean for Academic Affairs
University of Maryland School of Nursing
Baltimore, Maryland

Donna Mower-Wade, RN, MS, CNRN, ACNS-BC
Trauma Clinical Nurse Specialist
Christiana Care Health System
Newark, Delaware

Nancy Munro, RN, MN, CCRN, ACNP
Acute Care Nurse Practitioner
Critical Care Medicine Department
National Institutes of Health
Bethesda, Maryland

Angela C. Muzzy, RN, MSN, CCRN, CNS
Clinical Nurse Specialist, Cardiovascular Intensive Care Unit
University Medical Center
Tucson, Arizona

Colleen Krebs Norton, RN, PhD, CCRN
Associate Professor and Director of the Baccalaureate Nursing Program
Georgetown University School of Nursing and Health Studies
Washington, District of Columbia

Dulce Obias-Manno, RN, MHSA, CCDS, CEPS, FHRS
Nurse Coordinator, Cardiac Arrhythmia Device Clinic
Washington Hospital Center
Washington, District of Columbia

Mary O. Palazzo, RN, MS, FACHE
Senior Director of Transition Planning
Mercy Medical Center
Baltimore, Maryland

Archana D. Patel, MS, CRNP
Acute Care Nurse Practitioner, Thoracic Surgery
University of Maryland Medical Center
Baltimore, Maryland

Nayna C. Philipsen, RN, JD, PhD, CFE, FACCE
Director of Program Development
Coppin State University
Baltimore, Maryland

Clifford C. Pyne, RN, MS, ACNP
Nurse Practitioner—General Cardiology
Buffalo Cardiology and Pulmonary Associates
Williamsville, New York

Kim Reck, RN, MS, CRNP
Adult Nurse Practitioner
Clinical Program Manager, CRNP
Division of Cardiology
University of Maryland Medical Center
Baltimore, Maryland

Michael V. Relf, RN, PhD, CNE, ACNS-BC, AACRN, FAAN
Associate Professor and Assistant Dean for Undergraduate Education
Duke University School of Nursing
Durham, North Carolina

Kenneth J. Rempher, RN, PhD, MBA, CENP
Associate Chief Nursing Officer
University of Iowa Hospitals and Clinics
Iowa City, Iowa

Cynthia L. Renn, RN, PhD
Assistant Professor
University of Maryland School of Nursing
Baltimore, Maryland

Barbara Resnick, PhD, CRNP, FAAN, FAANP
Professor and Sonya Ziporkin Gershowitz Chair in Gerontology
University of Maryland School of Nursing
Baltimore, Maryland

Valerie K. Sabol, PhD, ACNP-BC, GNP-BC
Associate Professor and Specialty Director
Acute Care Nurse Practitioner and Critical Care Clinical Nurse Specialist Program
Duke University School of Nursing
Durham, North Carolina

Eric Schuetz, BS Pharm, CSPI
Specialist in Poison Information
Maryland Poison Center
Baltimore, Maryland

Julie Schuetz, MS, CRNP
Nurse Practitioner
Department of Hematologic Malignancies
The Sidney Kimmel Comprehensive Cancer Center
Johns Hopkins Medicine
Baltimore, Maryland

Brenda K. Shelton, RN, MS, CCRN, AOCN
Critical Care Clinical Nurse Specialist
The Sidney Kimmel Comprehensive Cancer Center at Johns Hopkins
Baltimore, Maryland

Jo Ann Hoffman Sikora, RN, MS, CRNP
Nurse Practitioner, Cardiac Surgery
University of Maryland Medical Center
Baltimore, Maryland

Kara Adams Snyder, RN, MS, CCRN, CCNS
Clinical Nurse Specialist, Surgical Trauma Critical Care
University Medical Center
Tucson, Arizona

Debbi S. Spencer, RN, MS
Chief Nurse, Joint Trauma System
U.S. Army Institute of Surgical Research
Fort Sam Houston, Texas

Allison G. Steele, RN, MSN, CRNP
Nurse Practitioner
Division of Gastroenterology and Hepatology
University Physicians Inc.
Baltimore, Maryland

Louis R. Stout, RN, MS
Lieutenant Colonel, United States Army Nurse Corps
United States Army Medical Department
Fort Lewis, Washington

Paul A. Thurman, RN, MS, ACNPC, CCNS, CCRN, CNRN
Clinical Nurse Specialist
R Adams Cowley Shock Trauma Center
University of Maryland Medical Center
Baltimore, Maryland

Sidenia S. Tribble, RN, MSN, ACNP-BC, CCRN
Acute Care Nurse Practitioner
Augusta Health
Fisherville, Virginia

Kathleen Turner, RN, DNP
Assistant Professor
Duke University School of Nursing
Durham, North Carolina

Connie M. Ulrich, RN, PhD, FAAN
Associate Professor of Nursing and Bioethics
University of Pennsylvania School of Nursing
Philadelphia, Pennsylvania

Jeffrey S. Upperman, MD
Associate Professor of Surgery
Director, Trauma Program
Children's Hospital Los Angeles
Keck School of Medicine
University of Southern California
Los Angeles, California

Mary van Soeren, RN, PhD
Director
Canadian Health Care Innovation
Guelph, Ontario, Canada

Kathryn T. Von Rueden, RN, MS, FCCM
Associate Professor
University of Maryland School of Nursing
Clinical Nurse Specialist
R Adams Cowley Shock Trauma Center
University of Maryland Medical Center
Baltimore, Maryland

Amanda S. Walther, MS, CRNP
Nurse Practitioner
Department of Cardiology
University of Maryland Medical Center
Baltimore, Maryland

Tracey Wilson, MS, CRNP
Acute Care Nurse Practitioner
Medical Intensive Care Unit
University of Maryland Medical Center
Baltimore, Maryland

Janet A. Wulf, MS, RN, CNL, CHPN
Staff Nurse
Union Memorial Hospital
Baltimore, Maryland

Karen L. Yarbrough, MS, CRNP
Acute Care Nurse Practitioner
Director, Stroke Program
University of Maryland Medical Center
Baltimore, Maryland

Elizabeth Zink, RN, MS, CCRN, CNRN
Clinical Nurse Specialist
Neurosciences Critical Care Unit
Johns Hopkins Hospital
Baltimore, Maryland

Reviewers

Sheryl Banak, RN, MSN, CNE
Faculty, RN Accelerated Program (RNA)
Baptist Health Schools Little Rock
Little Rock, Arkansas

Susan Barnason, RN, PhD, APRN-CNS, CCRN, CEN, FAHA
Professor of Nursing
University of Nebraska Medical Center
Lincoln, Nebraska

Sunshine Barron, RN, DNP
Assistant Professor
Carlow University School of Nursing
Pittsburgh, Pennsylvania

Diane Brown, RN, MSN, CCRN
Senior Instructor
The University of Akron
Akron, Ohio

Theresa Cartier, RN, MSN, CCRN
Nursing Instructor II
UPMC Shadyside Hospital School of Nursing
Pittsburgh, Pennsylvania

Patricia Connick, RegN CNCC[c]
Nursing Faculty
Durham College
Oshawa, Ontario, Canada

Dan Defeo, MSN, CFNP
Clinical Assistant Professor
West Virginia University
Morgantown, West Virginia

Frances Dunniway, DNPc, MSN
Assistant Professor
California Baptist University
Riverside, California

Kathleen Evanina, RN, PhD-C, MSN, CRNP-BC
Associate Professor
Misericordia University
Dallas, Pennsylvania

Barbara Fagan, RN, BScN, CNCC(c)
Nurse Educator
Registered Nurses Professional Development Centre
Halifax, Nova Scotia

Jackie Galea, RN, BN
Program Advisor, Critical Care, Acute Care and Emergency Nursing
Humber Institute of Technology and Advanced Learning
Toronto, Ontario, Canada

Jeanine Goodin, MSN, RN-BC, CNRN
Associate Professor of Clinical Nursing
University of Cincinnati College of Nursing
Cincinnati, Ohio

Leslie Graham, RN, MN, CNCC
Professor of Nursing
Durham College
Oshawa, Ontario

Annette Griffin, MSN, MBA
Assistant Professor of Nursing
Rhode Island College
Providence, Rhode Island

Virginia Hackett, RN, MS
Assistant Professor of Nursing
Barry University
Miami Shores, Florida

Gary Hicks, RN, MS, CEN, CPEN
Nursing Faculty
Stevenson University
Stevenson, Maryland

Trina Hill, RN, MAEd, BScN
Nursing Instructor
Saskatchewan Institute of Applied Science and Technology
Prince Albert, Saskatchewan, Canada

Sara Hoffman, RN, MSN
Assistant Professor of Nursing
University of Central Missouri
Lee's Summit, Missouri

Julie Isaacson, RN, MSN
Associate Professor of Nursing
Arkansas State University
Jonesboro, Arkansas

Dorene Konop, RN, MSN, CCRN
Nursing Instructor
UPMC Shadyside School of Nursing
Pittsburgh, Pennsylvania

Verna LaFleur, RN, PhD, MSN
Faculty, Instructor
Bowie State University
Bowie, Maryland

Jean Markie, RN, MSN, CNN, APRN-BC
Nursing Instructor
Cumberland University
Lebanon, Tennessee

Christine Markut, RN, PhD, BC
Associate Professor
Stevenson University
Stevenson, Maryland

Deborah Martin, RN, MSN, CCRN
Instructor of Adult Health
St. Luke's School of Nursing
Bethlehem, Pennsylvania

Joan Mills, RN, MN
Instructor
MacEwan University
Edmonton, Alberta

Vanessa Monroe, PhD(c), MSN
Clinical Assistant Professor
Prairie View A&M University
Prairie View, Texas

Julia Popp, RN, MSN
Professor of Nursing
Owens Community College
Toledo, Ohio

Carrie Pucino, RN, MS, CCRN
Assistant Professor
York College of Pennsylvania
York, Pennsylvania

Jennifer Robinson, RN, MSN, CCRN
Nursing Instructor
Washington Hospital School of Nursing
Washington, Pennsylvania

Becky Salmon, MS, CCRN
Associate Professor
Purdue University
Fort Wayne, Indiana

Nola Schrum, RN, MS, CCRN
Assistant Clinical Professor
Texas Woman's University
Dallas, Texas

SueEllen Schwab-Kapty, RN, MSN, BSN
Nursing Instructor
The Washington Hospital School of Nursing
Washington, Pennsylvania

Deborah Schwytzer, MSN, RN-BC, CEN
Associate Professor of Nursing
University of Cincinnati College of Nursing
Cincinnati, Ohio

Deborah Siela, RN, PhD, CCNS, ACNS-BC, CCRN, CNE, RRT
Associate Professor
Ball State University
Muncie, Indiana

Carol Smith, RN, PhD(c), MSN
Assistant Professor of Nursing
Bellarmine University
Louisville, Kentucky

Rachel Wilburn, RN, MSN
Assistant Professor
McNeese State University College of Nursing
Lake Charles, Louisiana

Shirley Woolf, RN, MSN, BSN, MA, CNE, CCRN
Clinical Assistant Professor
Indiana University School of Nursing
Indianapolis, Indiana

Preface

The practice of critical care nursing has changed dramatically since its inception in the 1960s. Critical care nurses, more than ever before, must possess an extensive body of knowledge in order to provide competent and holistic care to critically ill patients and their families. Critically ill patients are no longer found just in intensive care units. Instead, they are cared for in the emergency department, in progressive care units, in postanesthesia care units, and in the home. Today, critically ill patients are liable to be older and sicker than ever before, thus demanding extensive knowledge to meet their complex needs. Advances in nursing, medicine, and technology; the rapidly changing health care climate; and the shortage of nursing staff and faculty are other factors that have come together to effect great changes in the practice of critical care nursing.

Some of the requisite knowledge for the practice of critical care nursing can be attained through formal education and textbooks, like this one. The rest can only be gained through experience. It is our goal, with this 10th edition of *Critical Care Nursing: A Holistic Approach*, to assist readers on their journey by providing a comprehensive, up-to-date resource and reference.

As in past editions, this 10th edition promotes excellence in critical care nursing. Presenting theory and principles within the context of practical application helps the reader to gain competence and confidence in caring for critically ill patients and their families. As always, the patient as the center of the health care team's efforts is emphasized throughout. In the highly specialized and complicated technical environment of critical care, knowing how to deliver holistic care and demonstrating caring behaviors are just as important as knowing how to operate complex equipment and perform difficult procedures.

New to this 10th edition, the reader will find a special feature on genetics, more emphasis on evidence-based practice, and a more streamlined text that focuses on the most important knowledge and practices for the critical care nurse.

▲ An Overview

Critical Care Nursing: A Holistic Approach, 10th edition, consists of 13 parts. The following is a brief overview of those parts and the information they contain.

Part 1: The Concept of Holism Applied to Critical Care Nursing Practice

The six chapters that make up Part 1 introduce the student to the concept of holistic care, as it applies in critical care practice. In Chapter 1, the student is introduced to critical care nursing practice. Chapters 2 and 3 review the psychosocial effects of critical illness on the patient and the family, respectively. These chapters also describe the effect of the critical care environment on the patient and review actions the nurse can take to help reduce environment-induced stress and promote healing. Chapter 4 emphasizes the role of patient and family education in critical care. Chapter 5 focuses on strategies for relieving pain and promoting comfort. We conclude the part with Chapter 6, which concentrates on end-of-life and palliative care.

Part 2: Professional Practice Issues in Critical Care

This part consists of three chapters that are of concern to the nursing profession. In Chapters 7 and 8, ethical and legal issues are explored. Chapter 9 describes characteristics of critical care nurses, delineates aspects of nursing professionalism, and defines critical attributes of nursing excellence.

Part 3: Special Populations in Critical Care

The four chapters in this part focus on the special needs of certain groups of people who are critically ill. Chapters 10, 11, and 12 focus on the pediatric patient, the pregnant patient, and the elderly patient, respectively. Chapter 13 describes the role of the nurse in caring for the patient who is recovering from anesthesia.

Part 4: Special Situations in Critical Care

This section opens with a chapter that focuses on the care of the patient who is being transported within or between facilities as well as the role of the rapid response team. The second chapter in this section describes the role of the critical care nurse in disaster management.

Part 5: Cardiovascular System

This part, the first of eight organ system–based parts, focuses on the care of the patient with a cardiovascular disorder. Each organ system–based part begins with a chapter that reviews the anatomy and physiology of the organ system under discussion (e.g., Chapter 16). The part then continues with a chapter on patient assessment (e.g., Chapter 17), general patient management (e.g., Chapter 18), and common disorders (e.g., Chapter 19). In Part 5, heart failure and acute myocardial infarction are each given their own chapters (Chapters 20 and 21, respectively). The unit concludes with a discussion of the most recent developments in cardiac surgery (Chapter 22). Throughout the unit, the latest diagnostic

tests (such as cardiac serum markers), the newest medications for treating cardiovascular disorders, and updates on technologies (such as the left ventricular assist device, the implantable cardioverter defibrillator, and the cardiac pacemaker) are discussed.

Part 6: Respiratory System

In this part, current assessment technologies (such as end tidal carbon dioxide monitoring) and the newest modes of ventilation for patients in respiratory failure are discussed. Evidence-based treatment strategies for respiratory disorders such as pneumonia, pleural effusion, and chronic obstructive pulmonary disease are described. Chapter 27 is devoted to the latest developments in the assessment and management of the patient with acute respiratory distress syndrome (ARDS).

Part 7: Renal System

In this edition of the text, Part 7 includes a more in-depth discussion of the assessment and management of fluids, electrolytes, and acid–base balance. Updates on laboratory and diagnostic tests are included. The newest dialysis technologies and the latest drugs are discussed in Chapter 30. Chapter 31 focuses on common renal disorders, including recent developments in the care of the patient with renal failure.

Part 8: Nervous System

This part offers updates on neurological diagnostic studies and the newest approaches to treating the patient with increased intracranial pressure. The latest drugs for treating neurological disorders and the most recent developments in neurosurgery are addressed. Separate chapters are devoted to care of the patient with a head injury and spinal cord injury.

Part 9: Gastrointestinal System

In Part 9, the latest diagnostic tests for evaluating patients with gastrointestinal disorders are discussed. The management of patients with gastrointestinal disorders has been updated to include the newest drugs, the latest developments in the use of enteral and parenteral nutrition, and recent trends in the treatment of common disorders such as liver failure and hepatitis.

Part 10: Endocrine System

In this edition, Part 10 includes an assessment chapter covering multiple components of the endocrine system. The content is organized by the major gland, and for each gland addressed in the chapter, the reader is given information on the history, laboratory tests, and diagnostic tests. The most current information on the treatment of endocrine disorders, especially glycemic control and diabetic emergencies, is included in Chapter 44.

Part 11: Hematological and Immune Systems

This part continues to be a unique feature that is not included in many critical care texts. The numerous recent developments in organ and hematopoietic stem cell transplant are described in Chapter 47. Chapter 48 addresses up-to-date information on the assessment and management of critically ill patients with HIV/AIDS as well as those with oncological emergencies. The latest trends in the treatment of patients with hematological disorders such as disseminated intravascular coagulation are included in Chapter 49.

Part 12: Integumentary System

This part includes three chapters not covered in other critical care texts: the anatomy and physiology of the integumentary system, assessment of the integumentary system, and management of integumentary disorders, respectively. Evidence-based assessment and management of wounds are addressed. In addition, care of the critically ill burn patient is covered in Chapter 53.

Part 13: Multisystem Dysfunction

In Chapter 54, hypoperfusion states such as shock, systemic inflammatory response syndrome (SIRS), and multiple organ dysfunction syndrome (MODS) are discussed. The latest understanding of the pathophysiologic process is described, as well as how this knowledge guides the selection of the most recent interventions. Chapter 55 reviews care of the trauma patient, including the latest trends in the management of these complex patients. Chapter 56 reviews care of the patient with a drug overdose or poisoning, a problem that is becoming more common in the critical care setting.

Appendix

An appendix completes the textbook; it contains updated ACLS guidelines.

▲ Features

The features of the 10th edition of *Critical Care Nursing: A Holistic Approach* have been designed to assist readers with practice as well as learning.

Practice-Oriented Features

- **Considerations for the Older Patient boxes** highlight the special needs of this patient population and constitutes the largest numbers of critically ill patients.
- **Health History boxes** summarize key areas that should be covered and relevant information that may be revealed during the health history.

- **Nursing Diagnoses boxes** summarize common NANDA nursing diagnoses for particular conditions.
- **Collaborative Care Guides** describe how the health care team works together to manage a patient's illness and minimize complications. The information is presented in a tabular format, with outcomes in the first column and interventions in the second.
- **Teaching Guides** help the nurse to prepare patients and family members for procedures, assist patients and family members with understanding the illness they are dealing with, and explain postprocedure or postoperative activities.
- **Drug Therapy tables** summarize information related to the administration and monitoring of drug therapy.

Pedagogical Features

Learning Objectives, at the beginning of each chapter, help focus the reader's attention on important topics.

Clinical Applicability Challenges, at the conclusion of each chapter, consists of three to five **short-answer questions** or a **case study** followed by three to five short-answer questions.

▲ New to This Edition

- **New case study/short-answer questions** in every chapter guide the reader from knowledge to application. Discussion points for these questions are available on thePoint.
- **Evidence-Based Practice Highlights** help the reader to understand the importance of research-based practice and include excerpts from the latest AACN practice alerts as well as from recently published research and nationally published guidelines from the National Institutes of Health.
- **Spotlight on Genetics** appears in selected chapters and includes important genetic information and summaries of selected genetic disorders. These disorders may be the result of random genetic mutations, genetic mutations caused by environmental influences, or inherited mutated genes.
- **Patient Safety boxes** alert the reader to risk factors, signs and symptoms, side effects, and complications that the critical care nurse must anticipate and continuously monitor.

In order to streamline the text and to make important content readily available to users of the text, additional content to enhance learning and understanding of the chapters is available on thePoint. Visit the website to access:

- Chapter outlines
- Additional selected readings
- Chapter NCLEX-style review questions
- Answers to Chapter NCLEX-style review questions with accompanying rationales
- Internet resources

▲ Student and Instructor Resources

In addition to the chapter-specific content listed above, a wide variety of resources that support the student and instructor are also available online on thePoint.

Student Resources

Students who purchase this book have access to a wide variety of additional resources via thePoint website:

- Spanish-English audio glossary
- Concepts in Action animations
- Monographs of 100 commonly prescribed drugs
- Journal articles
- Learning objectives
- Nursing professional roles and responsibilities
- Heart and breath sounds
- Case study/short answer questions and discussion points
- Full text online

Additional study aids to help students review the basics are also available on thePoint:

- Practice and Learn video clips
- Watch and Learn video clips
- Clin Sims case study
- Clin Sim tutorial
- Dosage calculation quizzes

Instructor Resources

In addition to access to all of the student assets, instructors who adopt this text also have access to an Instructor's DVD-ROM, for those who prefer that format, as well as access to a special instructor's resource section on thePoint. The following are included on the DVD-ROM and the website:

- Test generator, featuring over a thousand questions
- Image bank, containing over 300 illustrations
- PowerPoint presentations and accompanying guided lecture notes for each chapter
- Syllabi
- Strategies for effective teaching

It is with great pleasure that we introduce these resources—the textbook and the accompanying interactive resource package—to you. One of our primary goals in creating these resources has been to promote excellence in critical care nursing practice so that nurses can help patients and families cope with the consequences of critical illness. It is our intent that these resources will provide aspiring and currently practicing critical care nurses the tools to make their optimal contribution to the care of critically ill patients and their families and to the nursing profession. We hope that we have succeeded in that goal, and we welcome feedback from our readers.

Patricia Gonce Morton, RN, PhD, ACNP-BC, FAAN
Dorrie K. Fontaine, RN, PhD, FAAN

Acknowledgments

This project required the help and cooperation of many people. First we want to thank our many colleagues who contributed to the text, either by authoring a chapter or by sharing their expertise as a reviewer. Our publisher, Lippincott Williams & Wilkins, through all editions of this book remains committed to producing the best text possible. We especially want to thank Helen Kogut, Senior Product Manager; Melanie Cann, former Senior Developmental Editor; and Elizabeth Nieginski, former Senior Executive Acquisitions Editor, for their support, commitment to excellence, and words of positive encouragement as they cheered us on to the finish line with this project. Special thanks are also due to Jacalyn Clay, former Editorial Assistant, for her assistance throughout the project.

We also wish to express our thanks to Regina Mabrey, who compiled and checked all the Internet resources that appear on thePoint website and to Lisa Vikell who assisted with all the references and additional selected readings. Their hours of work were an enormous help to us.

In addition, we wish to thank Dr. Dennis Cheek, a nationally known expert in genetics, for creating the new genetics feature, *Spotlight on Genetics*, that appears throughout the text.

And finally, we wish to express a word of thanks to our families and nursing colleagues who endured the time we took to complete this project.

Tricia and Dorrie

Contents

PART 10

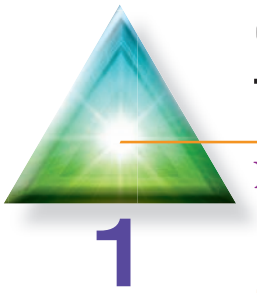
ENDOCRINE SYSTEM 955

PART 11

HEMATOLOGICAL AND IMMUNE SYSTEMS 1021

PART 1

The Concept of Holism Applied to Critical Care Nursing Practice



1

Critical Care Nursing Practice: Promoting Excellence Through Caring, Collaboration, and Evidence

Roberta Kaplow, Kathleen Turner, and Michael V. Relf

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Describe the value of certification in critical care nursing.
2. Describe the value of evidence-based practice (EBP) in caring for critically ill patients.
3. Discuss the value of collaborative practice in critical care.
4. Provide examples of how the Synergy Model can promote positive patient outcomes.
5. Discuss future issues facing critical care nursing practice.

As the health care delivery system continues to evolve and transform, so too does the discipline of nursing and the specialty of critical care nursing. Today, the care of critically ill patients occurs not only in the traditional setting of the hospital intensive care unit (ICU) but also on the progressive care unit, medical and surgical clinical units, in the subacute facility, long-term care, community, and even the patient's home. As a consequence of recent health care reform legislation coupled with an aging population and application of genomics in personalized medicine, critical care nursing practice as we know it today will continue to evolve.

Since the first critical care unit opened in the late 1960s, significant technological, procedural, and pharmacological advances have occurred, accompanied by a knowledge explosion in critical care nursing and medicine. Consequently, critical care nurses, progressive care nurses, and home health nurses of the 21st century are routinely caring for the complex, critically ill patients who just a few decades ago would not have survived a critical illness. As a result, nurses are increasingly being challenged to integrate sophisticated technologies and interventions, implement care based on contemporary evidence while simultaneously caring for the whole person by addressing the psychosocial challenges and ethical conflicts associated with critical illness and delivering care not only to the patient but also to the patient's family, as defined by the patient—not in professional isolation but in collaboration with the interdisciplinary team.

In response to the ever-changing delivery system, critical care nurses are championing the needs of the patient and the family, integrating evidence to improve standards of care, striving to maintain patient safety, engaging in interdisciplinary collaboration, and developing healthy work environments (HWEs)—all in the pursuit of quality clinical outcomes. During the past several decades, critical care nurses have experienced firsthand what nurse scientists have consistently demonstrated—critical illness is not only a physiological alteration but also a psychosocial and spiritual process as well as a threat to individuals and their families. Through specialty certification by the American Association of Critical-Care Nurses (AACN), nurses voluntarily demonstrate their knowledge of critical care nursing. Furthermore, as health care becomes increasingly technical, the need for humanization is increasingly essential. Compatible with the need for "humanized" health care is the need to provide effective evidence-based interventions instead of those steeped in tradition or based on intuition.

This chapter describes select aspects of the critical care environment. These include the value of certification, evidence-based practice (EBP), quality and safety, healthy environments, and the AACN Synergy Model for Patient Care. Each of these, when implemented, helps promote optimal outcomes for acute and critically ill patients and their families.

▲ Value of Certification

"Certification is a process by which a nongovernmental agency validates, based on predetermined standards, an individual nurse's knowledge for practice in a defined functional or clinical area of nursing."[1] A white paper, *Safeguarding the Patient and the Profession*, published by the AACN, demonstrated the value of specialty certification.[2] Certification promotes continuing excellence in the critical care nursing profession helping nurses achieve and maintain an up-to-date knowledge base essential in the practice of critical care nursing.[2–4] In addition, it validates their knowledge to patients and families, employers, and themselves.

Value to the Patient and Family

As a consequence of medical errors, adverse outcomes, and complicated regulations governing reimbursement, many consumers are wary of today's health care delivery system. Certification provides patients and families with validation that the nurses caring for them have demonstrated experience and knowledge that exceeds that which is assessed in entry-level licensure examinations.[2] Nurses who have had their knowledge validated through a certification examination make decisions with greater confidence.[2,4,5] Although "failure to rescue" a patient in trouble cannot always be avoided, experienced and knowledgeable nurses are able to recognize signs and symptoms earlier and respond accordingly. In addition, certified nurses have demonstrated commitment to continual learning. This attribute is needed to care for patients with complex, multisystem problems requiring aggressive intervention and advanced technologies.

Value to Employers and Nurses

Certification allows the employer to know that the nurses working for them have the knowledge and expertise to promote optimal patient outcomes. Nurses who become certified have demonstrated their commitment to quality since they have taken the time to become certified in a specialty area of practice. Evidence across studies demonstrates that certified nurses have higher perceptions of workplace empowerment and do not intend to leave the profession.[4] In a review of the literature examining the perceived effects of specialty nurse certification, Wade[6] identified enhanced nurse communication and coordination of care with physicians and other health care team members, and nurse–physician collaboration was perceived to be more improved by certified nurses. Both communication and collaboration are essential to quality patient outcomes. Additionally, evidence also indicates that certified nurses intend to remain employed in an area where they can apply the knowledge.[4] It has been suggested that health care organizations that support and recognize the value of certification experience decreased turnover rates and higher nurse retention rates.[4]

As health care organizations apply to achieve Magnet designation by the American Nurses Credentialing Center (ANCC), certification is one of the many important factors considered.[7] Certification is also a means for hospitals to distinguish themselves from competitors.[2] In addition, hospital administrators must demonstrate to the Joint Commission that nurses are competent to provide care—certification is a clear demonstration of knowledge competency.[2]

Certification provides nurses with a sense of professional pride and achievement.[1] A survey by the American Board of Nursing Specialties revealed that most nurses who sat for certification examinations did so for personal fulfillment and commitment to excellence in practice.[8] Certified nurses demonstrate to their employers that they are taking responsibility for their own professional development, which may give them a competitive edge when seeking promotion or new career opportunities.

Research has identified that certified nurses perceive higher empowerment, recognize the intrinsic value of certification, experience enhanced collaboration with other members of the interdisciplinary team, and perceive knowledge being enhanced and validated through certification.[4–6]

▲ Evidence-Based Practice in Critical Care Nursing

Evidence-based practice (EBP) is "the process of shared decision making between practitioner, patient, and others significant to them based on research evidence, the patient's experiences and preferences, clinical expertise or know-how, and other available robust sources of information." (p. 57)[9] EBP is essential to help optimize patient outcomes in today's dynamic health care environment. In the United States, the Institute of Medicine (IOM), the ANCC, and the Joint Commission acknowledge EBP as a crucial step in improving healthcare quality.[10] Although knowledge regarding effective nursing interventions continues to increase, practice lags behind the available evidence. Practice based on intuition or information that does not have a scientific basis is not in the best interest of patients and families and should be discouraged when care decisions are being made.

EBP is often confused with conducting research. Research is conducted to generate new knowledge. Through translation of new knowledge, EBP takes what is known and uses it to guide patient care to achieve the best possible outcomes.[11] In the translation of knowledge into clinical practice, it is essential for the critical care nurse to consider the strength or level of the evidence. First, much of health care and nursing practice has little or no evidence or research to support it. Consequently, other sources of evidence must be considered. Second, different research designs have limitations that affect generalizability of findings to populations other than those studied.[12] Baumann[13] argues that nurses and other health care professionals must critically examine the evidence that they utilize in their clinical reasoning but should not restrict their definition to what constitutes evidence to only what can be measured in a randomized controlled trial (RCT). Therefore, it is essential for critical care nurses to familiarize themselves with the levels of evidence in order to evaluate the evidence or research and determine the applicability to their patients.

The AACN was one of the first nursing organizations to develop a rating system for evidence.[12] In 2008, a work group was charged by the AACN board of directors to review the evidence-leveling system and to align the AACN hierarchy with other health care organizations.

Levels of Evidence

In EBP, the lowest levels of evidence include the opinions of authorities or expert committees. This level of evidence would result from clinical practice committees and professional organizations that may convene to discuss guidelines when higher levels of evidence are not available. The next level of evidence is a single descriptive, qualitative, or physiologic study. Continuing up the evidence hierarchy is the systematic review of descriptive, qualitative, or physiologic studies. A systematic review refers to rigorous and systematic synthesis of findings from several studies in a focused area of inquiry. Proceeding in the hierarchy, a single correlational or observational study provides the next level of evidence. To guide clinical practice, these quantitative studies lend themselves to precise measurement allowing for examination of relationships. The next level is the systematic review of correlational or observational studies. Moving toward the stronger levels of evidence is a single RCT and nonrandomized control trial (quasi-experimental). The highest level of evidence is the systematic review of RCTs, also referred to as a meta-analysis. For studies utilizing qualitative methods, a meta-synthesis is analogous to a meta-analysis.[14]

Using ventilator-associated pneumonia (VAP), as an example, a critical care nurse would identify much literature on VAP yielding editorials from experts, descriptive studies, single RCTs, and meta-analyses. Since meta-analyses, the highest level of evidence, exist for VAP, critical care nurses can revise protocols and implement interventions based on evidence to prevent VAP. To improve quality of care and to protect patient safety during critical illness, critical care nurses must be able to systematically gather, review, synthesize, and evaluate the evidence to guide practice changes and promote quality clinical outcomes.

Barriers to Implementation

Despite the value of EBP, it unfortunately takes an average of 17 to 20 years to translate research findings into clinical practice.[15] Barriers to implementation include lack of knowledge of the research process, limited access to literature, lack of confidence in ability to critique research, limited interest in scientific inquiry, limited power to change practice, time factors, and lack of organizational support and commitment including resources and availability of mentors.[10,16]

An organization's culture, climate, or environment has significant impact on the nurses' autonomy to change nursing practice and the ability to adapt to change.[10] Critical care nurses are often adept and skillful in direct patient care but when care is not based on evidence, it is difficult to optimize outcomes, utilize resources efficiently, and protect patient safety. Thus, it is essential for critical care nurses to have the required knowledge, skills, and abilities for locating, evaluating relevant evidence, and translating the evidence into practice.

Strategies to Promote Implementation

Several strategies have been proposed to help enhance incorporating evidence into clinical practice including the use of protocols, clinical pathways, algorithms, and educational interventions. Increasing a critical care nurses' awareness of available resources and educating and mentoring them to implement EBP activities are essential. Similar to building a house, an organization committed to EBP must first establish EBP as a foundation of everyday practice and create a culture of clinical inquiry and commitment to lifelong learning. Accountability and role modeling, formal and informal, must be present for a successful foundation.[17,18]

Several resources are available for critical care nurses to facilitate adoption of an EBP culture. Superb databases include PubMed, CINAHL, and MEDLINE. The Cochrane Collaboration, a website containing high-quality, independent evidence to inform health care decision making and obtain evidence-based information is available via the worldwide web. In addition, professional nursing organizations have research-based practice recommendations frequently available via their websites. For example, AACN's Practice Alerts section is available to members as well as to the public. Other EBP resources include the websites and publications from the Agency for Healthcare Research and Quality (www.ahrq.gov), Centers for Disease Control and Prevention (www.cdc.gov), Institute for Healthcare Improvement (www.ihi.org), the IOM (www.iom.edu), National Guidelines Clearinghouse (www.guidelines.gov), National Institute for Health and Clinical Excellence in the United Kingdom (www.nice.org.uk), National Library of Medicine (www.nlm.nih.gov), and American Nurses Association (www.nursiingworld.org) as well as the Society of Critical Care Medicine (www.sccm.org). Table 1-1 summarizes evidence of perceived barriers regarding the implementation of EBP and aligns strategies for those perceived barriers.

▲ Healthy Work Environments

The current nursing shortage calls for major change in the workplace. A healthy work environment (HWE) can lead to positive patient outcomes. In addition, nurses gravitate to facilities that have optimal work conditions. Conversely, unhealthy work environments play a role in health care errors, result ineffective care, waste scarce resources leading to increased costs associated with health care, and contribute to moral distress and adverse outcomes.

AACN affirms that the best way to address the nursing shortage is to focus on HWEs. After conducting an extensive literature search, AACN developed the HWE initiative based on data indicating that harmful health care working environments exist nationwide and that these environments result in medical errors, poor health care delivery, and dissatisfaction among health care providers. The HWE initiative focuses on barriers to employee and patient safety and identifies six essential standards (Box 1-1). The six elements discussed encompass the aspects that are most important as nurses strive to provide optimal care.[19] Boxes 1-2 through 1-7 list the critical elements inherent in the respective standards.

Table 1-1 Barriers to and Recommended Strategies to Optimize Evidence-Based Practice (EBP) in Critical Care

Perceived Barriers to EBP	Strategies to Overcome Perceived Barriers
Time	
• No time to read and evaluate research or implement EBP	• Schedule time for review and discussion of evidence in the form of EBP committees • Nurses need time away from clinical bedside responsibilities for EBP activities
Knowledge	
• Unaware of research • Literature not compiled in one place • Not capable of evaluating quality of research	• Invest, as a student, in the process of searching and evaluating the evidence • Be a change agent in the facility, mentoring nurses who are less familiar with the process • Commit to lifelong learning
Resources and Mentoring	
• Lack of evidence • Isolated from knowledgeable colleagues • Lack of computers, computer skills, library access, search skills • Difficulty understanding research	• Administration must recognize and commit to EBP, putting systems in place to support the clinical nurses in their role with EBP • Managers need to recognize the ability of clinical nurses, provide the necessary resources, and document the effectiveness of initiatives • Implementation of an evidence-based champion • Designated work groups
Culture	
• No authority to change practice • Other staff and disciplines not supportive • Research not generalizable to setting • Lack of value of research in practice	• A research-based needs assessment to provide an evidence-based foundation for organizational strategic planning • Performance appraisals and clinical ladder reviews require examples of EBP • Educational activities that help clinicians critically review the literature • Clinical resources available 24 h a day • Streamlined processes for practice • Incorporating the doctor of nursing practice role as leader of this process • Progress reported to a central source and shared among clinical areas and facilities

Adapted from Brown CE, Wickline MA, Ecoff L et al: Nursing practice, knowledge, attitudes and perceived barriers to evidence based practice at an academic medical center. J Adv Nurs 65(2):371–381, 2009; Ross J: Information literacy for evidence-based practice in perianesthesia nurses: Readiness for evidence based practice. J Perianesth Nurs 25(2): 64–70, 2010; and Schulman CS: Strategies for starting a successful evidence based practice program. Adv Crit Care 19(3):301–311, 2008.

Skilled Communication

As miscommunication is identified as a cause of many sentinel events in health care, skilled communication is essential to prevent these errors from occurring. A large majority of sentinel events reported to the Joint Commission from 2004 through the fourth quarter of 2010 were related to communication issues.[20]

AACN partnered with VitalSmarts, L.C., to conduct a study of conversations that do not occur in hospitals, to the detriment of patient safety and provider well-being. The "Silence Kills" study used focus groups, interviews, workplace observations, and surveys of nurses, physicians, and administrators in urban, rural, and suburban hospitals nationwide.[21] Overwhelming data indicated that poor communication and ineffective collaboration were prevalent among health care provider interactions.

In this report,[21] 53% of nurses reported concern about a peer nurse's competence but only 12% spoke with this peer about the concerns. Similarly, 34% of nurses were concerned about a physician's competence but only 12% spoke with this peer to discuss concerns. In both situations, failure to communicate skillfully and professionally can be linked to poor patient care and clinical outcomes.

A pharmacist reported letting incorrect orders "slide" if the physician was a "jerk" and the incorrect dose was not going to make the patient sicker. Another pharmacist reported filling an incorrect prescription without question because of the hostility experienced when confronting the physician in the past. Several physicians in this study stated that they worked

BOX 1-1 Essential Elements of a Healthy Work Environment (HWE)

- *Skilled Communication.* Nurses must be as proficient in communication skills as they are in clinical skills.
- *True Collaboration.* Nurses must be relentless in pursuing and fostering true collaboration.
- *Effective Decision Making.* Nurses must be valued and committed partners in making policy, directing and evaluating clinical care, and leading organizational operations.
- *Appropriate Staffing.* Staffing must ensure the effective match between patient needs and nurse competencies.
- *Meaningful Recognition.* Nurses must be recognized and must recognize others for the value each brings to the work of the organization.
- *Authentic Leadership.* Nurse leaders must fully embrace the imperative of a HWE, authentically live it, and engage others in its achievement.

From http://www.aacn.org/WD/HWE/Docs/HWEStandards.pdf.[21]

BOX 1-2 Standard 1: Critical Elements of Skilled Communication

Nurses must be as proficient in communication skills as they are in clinical skills.

- The health care organization provides team members with support for and access to education programs that develop critical communication skills including self-awareness, inquiry/dialogue, conflict management, negotiation, advocacy, and listening.
- Skilled communicators focus on finding solutions and achieving desirable outcomes.
- Skilled communicators seek to protect and advance collaborative relationships among colleagues.
- Skilled communicators invite and hear all relevant perspectives.
- Skilled communicators call on goodwill and mutual respect to build consensus and arrive at common understanding.
- Skilled communicators demonstrate congruence between words and actions, holding others accountable for doing the same.
- The health care organization establishes zero-tolerance policies and enforces them to address and eliminate abuse and disrespectful behavior in the workplace.
- The health care organization establishes formal structures and processes that ensure effective information sharing among patients, families, and the health care team.
- Skilled communicators have access to appropriate communication technologies and are proficient in their use.
- The health care organization establishes systems that require individuals and teams to formally evaluate the impact of communication on clinical, financial, and work environment outcomes.
- The health care organization includes communication as a criterion in its formal performance appraisal system, and team members demonstrate skilled communication to qualify for professional advancement.

From http://www.aacn.org/WD/HWE/Docs/HWEStandards.pdf.[21]

BOX 1-3 Standard 2: Critical Elements of True Collaboration

Nurses must be relentless in pursuing and fostering true collaboration.

- The health care organization provides team members with support for and access to education programs that develop collaboration skills.
- The health care organization creates, uses, and evaluates processes that define each team member's accountability for collaboration and how unwillingness to collaborate will be addressed.
- The health care organization creates, uses, and evaluates operational structures that ensure the decision-making authority of nurses is acknowledged and incorporated as the norm.
- The health care organization ensures unrestricted access to structured forums, such as ethics committees, and makes available the time needed to resolve disputes among all critical participants, including patients, families, and the health care team.
- Every team member embraces true collaboration as an ongoing process and invests in its development to ensure a sustained culture of collaboration.
- Every team member contributes to the achievement of common goals by giving power and respect to each person's voice, integrating individual differences, resolving competing interests, and safeguarding the essential contribution each must make in order to achieve optimal outcomes.
- Every team member acts with a high level of personal integrity.
- Team members master skilled communication, an essential element of true collaboration.
- Each team member demonstrates competence appropriate to his or her role and responsibilities.
- Nurse managers and medical directors are equal partners in modeling and fostering true collaboration.

From http://www.aacn.org/WD/HWE/Docs/HWEStandards.pdf.[21]

with peers who were incompetent, but they did not confront them. Rather, they avoided scheduling sicker patients when the incompetent physician was scheduled to work. The data further suggested that physicians were equally as unlikely to confront a peer as a nurse or other health care provider. This study also stated that 88% of physicians reported working with people with persistently poor clinical judgment, causing deleterious complications.[21]

Seventy-seven percent of nurses were concerned about the disrespect they experience in the health care environment. They reported being treated discourteously or abusively in at least 25% of their interactions. This study found a significant correlation between the frequency of being mistreated and the intent to resign from the job. The study concluded that health care providers repeatedly observe errors and dangerous levels of incompetence. Yet they do not speak up; rather, they consider leaving their respective units because of their concerns.[21]

True Collaboration

Collaboration has been defined as a "sharing of knowledge between stakeholders with joint responsibility for the outcome."[22] It permits people with varied expertise to combine efforts to accomplish the goal of helping a client, program, or organization.[23] The collaborative outcome is the integration of solutions contributed by more than one person.[24]

Many studies have been conducted regarding nurse–physician collaboration. The data suggest that a collaborative relationship was more important to nurses than physicians. However, physicians rated the quality of the collaborative relationship that existed higher than nurses did.[25–33] Ninety percent of all AACN members have reported that collaboration with physicians and administrators is among the most important elements in creating an HWE.[19]

A number of outcomes have been linked to positive nurse–physician collaborative relationships. Specifically reported are nurse and physician satisfaction,[34,35] nurse retention,[33] and attainment of optimal patient outcomes.[34–42] It has also been suggested that a positive nurse–physician relationship can result in a decrease in errors.[34] Data also suggest that a decrease in mortality and in length of stay in the ICU is linked to positive nurse–physician collaboration.[43] One researcher reported a direct relationship between nurse–physician collaboration and efficiency of health care workers.[44]

Despite the reported benefits of collaboration, it is not practiced often enough.[22] A number of barriers exist that preclude its existence in health care organizations. The lack of an agreed-upon definition and variation in how collaboration is conceptualized, the lack of time for communication, and the complexity of the skills required to ease the process are daunting.[22] Other barriers have historically included professional cultures, immaturity of nurses and physicians, coupling of unassertive nurse behavior and aggressive physician

BOX 1-4 Standard 3: Critical Elements of Effective Decision Making

Nurses must be valued and committed partners in making policy, directing and evaluating clinical care, and leading organizational operations.

- The health care organization provides team members with support for and access to ongoing education and development programs focusing on strategies that ensure collaborative decision making. Program content includes mutual goal setting, negotiation, facilitation, conflict management, systems thinking, and performance improvement.
- The health care organization clearly articulates organizational values, and team members incorporate these values when making decisions.
- The health care organization has operational structures in place that ensure the perspectives of patients, and their families are incorporated into every decision affecting patient care.
- Individual team members share accountability for effective decision making by acquiring necessary skills, mastering relevant content, assessing situations accurately, sharing fact-based information, communicating professional opinions clearly, and inquiring actively.
- The health care organization establishes systems, such as structured forums involving all departments and health care disciplines, to facilitate data-driven decisions.
- The health care organization establishes deliberate decision-making processes that ensure respect for the rights of every individual, incorporate all key perspectives, and designate clear accountability.
- The health care organization has fair and effective processes in place at all levels to objectively evaluate the results of decisions, including delayed decisions and indecision.

From http://www.aacn.org/WD/HWE/Docs/HWEStandards.pdf.[21]

BOX 1-5 Standard 4: Critical Elements of Appropriate Staffing

Staffing must ensure the effective match between patient needs and nurse competencies.

- The health care organization has staffing policies in place that are solidly grounded in ethical principles and support the professional obligation of nurses to provide high quality care.
- Nurses participate in all organizational phases of the staffing process from education and planning—including matching nurses' competencies with patients' assessed needs—through evaluation.
- The health care organization has formal processes in place to evaluate the effect of staffing decisions on patient and system outcomes. This evaluation includes analysis of when patient needs and nurse competencies are mismatched and how often contingency plans are implemented.
- The health care organization has a system in place that facilitates team members' use of staffing and outcomes data to develop more effective staffing models.
- The health care organization provides support services at every level of activity to ensure nurses can optimally focus on the priorities and requirements of patient and family care.
- The health care organization adopts technologies that increase the effectiveness of nursing care delivery. Nurses are engaged in the selection, adaptation, and evaluation of these technologies.

From http://www.aacn.org/WD/HWE/Docs/HWEStandards.pdf.[21]

BOX 1-6 Standard 5: Critical Elements of Meaningful Recognition

Nurses must be recognized and must recognize others for the value each brings to the work of the organization.

- The health care organization has a comprehensive system in place that includes formal processes and structured forums that ensure a sustainable focus on recognizing all team members for their contributions and the value they bring to the work of the organization.
- The health care organization establishes a systematic process for all team members to learn about the institution's recognition system and how to participate by recognizing the contributions of colleagues and the value they bring to the organization.
- The health care organization's recognition system reaches from the bedside to the board table, ensuring individuals receive recognition consistent with their personal definition of meaning, fulfillment, development, and advancement at every stage of their professional career.
- The health care organization's recognition system includes processes that validate that recognition is meaningful to those being acknowledged.
- Team members understand that everyone is responsible for playing an active role in the organization's recognition program and meaningfully recognizing contributions.
- The health care organization regularly and comprehensively evaluates its recognition system, ensuring effective programs that help to move the organization toward a sustainable culture of excellence that values meaningful recognition.

From http://www.aacn.org/WD/HWE/Docs/HWEStandards.pdf.[21]

behavior, challenges of human relationships and personalities, hierarchical barriers, power imbalances, decision-making processes in organizations, and role socialization.[45–51]

In the book *Internal Bleeding—The Truth Behind America's Terrifying Epidemic of Medical Mistakes*, the authors reported that despite the fact that nurses and physicians have worked closely together, "there is still little understanding and appreciation for each other's roles."[52] The question of "why is collaboration so hard to embrace when the evidence supports better outcomes when there is communication and collaboration?" still requires an answer.[52]

Effective Decision Making

Clinical decision making is an essential component of nursing responsibilities in an effort to promote quality patient outcomes. Researchers do not agree about the role of knowledge in decision making. Some have suggested that it entails application of knowledge learned in the classroom and through readings while others have proposed that clinical decision-making skills involve experiential knowledge and intuition.[53] In clinical practice, where true collaboration does not exist and the elements of a HWE are not embraced, it is difficult for nurses, who are accountable for a scope of practice, to fully participate in decisions that affect quality and safe, effective care. The HWE standards highlight that only a small percentage of physicians acknowledge nurses as part of the decision-making team.[19]

In one study, investigators found that the majority of nurses made clinical decisions related to direct patient care on a regular basis.[54] Nurses working in the ICU reported commonly making decisions in emergency situations and

BOX 1-7 Standard 6: Critical Elements of Authentic Leadership

Nurse leaders must fully embrace the imperative of a HWE, authentically live it, and engage others in its achievement.

- The health care organization provides support for and access to educational programs to ensure that nurse leaders develop and enhance knowledge and abilities in skilled communication, effective decision making, true collaboration, meaningful recognition, and ensuring resources to achieve appropriate staffing.
- Nurse leaders demonstrate an understanding of the requirements and dynamics at the point of care and within this context successfully translate the vision of a HWE.
- Nurse leaders excel at generating visible enthusiasm for achieving the standards that create and sustain HWEs.
- Nurse leaders lead the design of systems necessary to effectively implement and sustain standards for HWEs.
- The health care organization ensures that nurse leaders are appropriately positioned in their pivotal role in creating and sustaining HWEs. This includes participation in key decision-making forums, access to essential information, and the authority to make necessary decisions.
- The health care organization facilitates the efforts of nurse leaders to create and sustain a HWE by providing the necessary time and financial and human resources.
- The health care organization provides a formal comentoring program for all nurse leaders. Nurse leaders actively engage in the comentoring program.
- Nurse leaders role-model skilled communication, true collaboration, effective decision making, meaningful recognition, and authentic leadership.
- The health care organization includes the leadership contribution to creating and sustaining a HWE as a criterion in each nurse leader's performance appraisal. Nurse leaders must demonstrate sustained leadership in creating and sustaining a HWE to achieve professional advancement.
- Nurse leaders and team members mutually and objectively evaluate the impact of leadership processes and decisions on the organization's progress toward creating and sustaining a HWE.

From http://www.aacn.org/WD/HWE/Docs/HWEStandards.pdf.[21]

deciding to change patient medication. Medical and surgical nurses only made these types of decisions on an occasional basis.[54] In two review articles, the amount of clinical experience correlates with improved decision making.[55,56]

Effective clinical decision making is an essential factor that impacts delivery of quality care. Whether a nursing student or an experienced nurse, factors reported to impact nurses' clinical decision making include self-confidence, relationships with peer and other providers, organizational structure, access to supportive resources, nursing education, experience, knowledge, creative thinking ability, self-concept, work environment, and situational stressors.[55–58] It is important for nurses, organizations that employ nurses, and schools of nursing to identify barriers and augment factors that promote clinical decision making.[19,55–57,59]

To promote quality patient outcomes, nurses must utilize the nursing process, apply critical thinking, and utilize knowledge and experience to make salient clinical decisions promptly. The nurses' role is to effectively communicate with and be involved in decision making as members of the multidisciplinary team as they strive to identify and manage complex patient problems. Necessary skills include accepting the high degree of accountability and responsibility that is inherent with clinical decision making while also having the autonomy needed to guide one's own practice.

Appropriate Staffing

Appropriate staffing must consider the knowledge, skills, and abilities, collectively referred to as competencies, of staff assigned in relation to the individualized, holistic needs of the patient and family. When the needs of patients and families are matched with the competencies of the assigned nurse, optimal outcomes may be achieved.

In a landmark study, Aiken et al[60] documented an increased risk for patient mortality when the nurse/patient ratio increased in the medical–surgical setting. In this study, when the nurse/patient ratio was 1:8, the patient's risk for death was 31% higher than when the nurse/patient ratio was 1:4 or lower. After the fourth patient, each surgical patient added to a nurse's assignment resulted in a 7% increase in chance of patient death within 30 days of hospital admission and a 7% increase in failure to rescue.[59]

Researchers found significant relationships between lower nurse staffing and adverse patient outcomes. These outcomes include development of pneumonia, shock, cardiac arrest, upper gastrointestinal bleeding, and urinary tract infections. Longer hospital lengths of stay, failure to rescue, and higher 30-day mortality rates have been linked to lower nurse staffing as well. Two of the major factors that contributed to poorer staffing levels were higher patient acuity and a disparity between the number of available positions and available registered nurses (RNs) to fill them.[60–72]

In its report, the IOM acknowledged the relationship between nurse staffing and quality of care. Nurse staffing levels and knowledge and skill level have an impact on patient outcomes and safety. The Institute further acknowledged that the temporary fixes such as mandatory overtime or leaving units understaffed are ineffective and contribute to errors.[72]

Meaningful Recognition

HWEs as well as programs that effectively recognize nurses and nursing contribution to quality, safe patient care are important to retain high-performing nurses, to engage them actively in enhancing patient satisfaction, to use scarce nursing resources appropriately, and to enhance nursing accomplishment. Nurse recognition can have a significant effect on job satisfaction. Meaningful nurse recognition can be achieved in a variety of effective, inexpensive ways.[73] Because of generational, gender, and cultural differences in today's nursing workforce, nurses find workplace satisfaction through a variety of mechanisms and frequently desire more out of a job than just a big salary.[74] Staff recognition has been identified as one of the simplest, cost-effective approaches to retain experienced staff and is essential to maintain nurse morale.[75,76] Specifically, staff want to lead balanced lives, enjoy partnership with their employers, receive opportunities for personal and professional growth, be able to make a meaningful contribution through their work, and experience opportunities to socialize at work.[74–76]

There seems to be universal agreement that to retain nursing staff during a critical shortage, in addition to monetary rewards when possible, expression of recognition for excellent performance and appreciation are part of the strategy.[77–79] Nurses in one study rated recognition from patients, families, and other nurses of more value than recognition from nursing leadership and physicians. In a follow-up study, nurses continued to value recognition from patients and families most (48.9%). This was followed by recognition from other RNs (27%), administration (8.5%), immediate supervisor (7.7%), physicians (4.6%), and other coworkers (3.3%).[80]

Meaningful recognition has been found to be associated with several positive outcomes, such as job satisfaction, commitment to an organization and career, collaboration, and perceived organizational support.[81–88] Yet, nurses do not receive meaningful recognition in all situations.[89] Absence of meaningful recognition is associated with absenteeism, staff turnover, stress, burnout, and decreased quality of care. Therefore, in addition to appropriate staffing, effective decision making, and true collaboration, critical care nurses, like all nurses, value meaningful recognition which in turn improves job satisfaction and morale while reducing turnover and improving quality patient outcomes.[85–90]

Authentic Leadership

Nurses are attracted to health care systems that promote a HWE. Nursing leaders from unit manager to chief nurse executive as well as nursing peers are essential to creating and sustaining a healthy environment in today's dynamic health care climate.[19,72,90–92] It has been demonstrated that positive nursing leadership is linked to quality patient outcomes.[91] This has been corroborated by the Joint Commission, which asserts that leadership is essential to obtain high quality, safe health care and that ineffective leadership is a contributing factor when sentinel events occur.[20] Authentic leadership has been described as "the glue needed to hold together a HWE."[93] Attributes of an authentic leader include good communication skills, knowing what they stand for, inspiring others, working as part of the team, asking for and giving 100%, and establishing a vision.[94]

▲ The Synergy Model

The Synergy Model developed by the AACN has served as the foundation for certified practice since the late 1990s.[95,96] The model describes nursing practice on the basis of patients' characteristics. The underlying premises of the Synergy Model are: (1) patients' characteristics are of concern to nurses; (2) nurses' competencies are important to patients; (3) patients' characteristics drive nurses' competencies; and (4) when patients' characteristics and nurses' competencies match and synergize, outcomes for the patient are optimal.[95,97]

Eight patient characteristics and eight nurse competencies that constitute nursing practice form the basis of the model (Fig. 1-1; Boxes 1-8 and 1-9). The patient characteristics range in intensity and are expressed as level 1, 3, or 5. The level can change from 1 minute to the next. Like the patient characteristics, the nurse competencies exist on a continuum

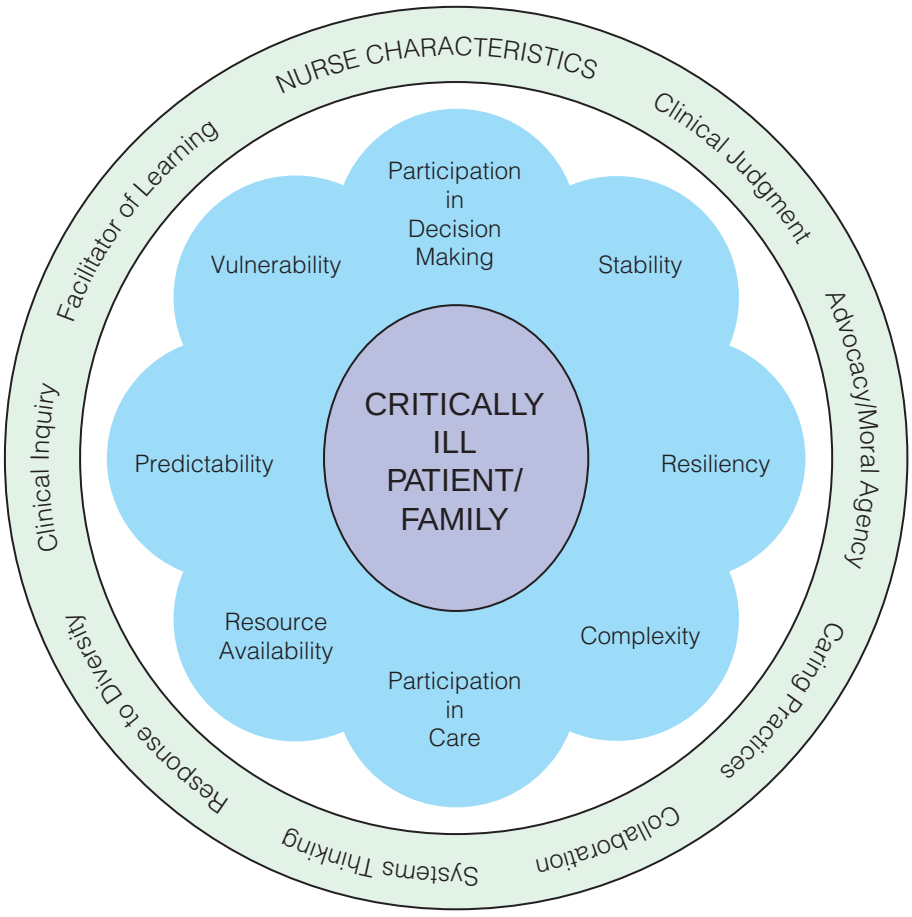


FIGURE 1-1 ▲ The relationship between the patient/family and the nurse in the Synergy Model.

BOX 1-8 Characteristics of Patients, Clinical Units, and Systems of Concern to Nurses

- **Resiliency**—the capacity to return to a restorative level of functioning using compensatory/coping mechanisms; the ability to bounce back quickly after an insult
  - *Level 1: Minimally resilient.* Unable to mount a response; failure of compensatory/coping mechanisms; minimal reserves; brittle
  - *Level 3: Moderately resilient.* Able to mount a moderate response; able to initiate some degree of compensation; moderate reserves
  - *Level 5: Highly resilient.* Able to mount and maintain a response; intact compensatory/coping mechanisms; strong reserves; endurance
- **Vulnerability**—susceptibility to actual or potential stressors that may adversely affect patient outcomes
  - *Level 1: Highly vulnerable.* Susceptible; unprotected, fragile
  - *Level 3: Moderately vulnerable.* Somewhat susceptible; somewhat protected
  - *Level 5: Minimally vulnerable.* Safe; out of the woods; protected, not fragile
- **Stability**—the ability to maintain a steady-state equilibrium
  - *Level 1: Minimally stable.* Labile; unstable; unresponsive to therapies; high risk of death
  - *Level 3: Moderately stable.* Able to maintain steady state for limited period of time; some responsiveness to therapies
  - *Level 5: Highly stable.* Constant; responsive to therapies; low risk of death
- **Complexity**—the intricate entanglement of two or more systems (eg, body, family, therapies)
  - *Level 1: Highly complex.* Intricate; complex patient/family dynamics; ambiguous/vague; atypical presentation
  - *Level 3: Moderately complex.* Moderately involved patient/family dynamics
  - *Level 5: Minimally complex.* Straightforward; routine patient/family dynamics; simple/clearcut; typical presentation
- **Resource Availability**—extent of resources (eg, technical, fiscal, personal, psychological, and social) the patient/family/community bring to the situation
  - *Level 1: Few resources.* Necessary knowledge and skills not available; necessary financial support not available; minimal personal/psychological supportive resources; few social systems resources
  - *Level 3: Moderate resources.* Limited knowledge and skills available; limited financial support available; limited personal/psychological supportive resources; limited social systems resources
  - *Level 5: Many resources.* Extensive knowledge and skills available and accessible; financial resources readily available; strong personal/psychological supportive resources; strong social systems resources
- **Participation in Care**—extent to which patient/family engages in aspects of care
  - *Level 1: No participation.* Patient and family unable or unwilling to participate in care
  - *Level 3: Moderate participation.* Patient and family need assistance in care
  - *Level 5: Full participation.* Patient and family fully able to participate in care
- **Participation in Decision Making**—extent to which patient/family engages in decision making
  - *Level 1: No participation.* Patient and family have no capacity for decision making; requires surrogacy
  - *Level 3: Moderate participation.* Patient and family have limited capacity; seek input/advice from others in decision making
  - *Level 5: Full participation.* Patient and family have capacity and make decisions
- **Predictability**—a characteristic that allows one to expect a certain course of events or course of illness
  - *Level 1: Not predictable.* Uncertain; uncommon patient population/illness; unusual or unexpected course; does not follow critical pathway, or no critical pathway developed
  - *Level 3: Moderately predictable.* Wavering; occasionally noted patient population/illness
  - *Level 5: Highly predictable.* Certain; common patient population/illness; usual and expected course; follows critical pathway

From the American Association of Critical-Care Nurses Certification Corporation.

BOX 1-9 Nurse Competencies of Concern to Patients, Clinical Units, and Systems

- **Clinical Judgment**—clinical reasoning, which includes clinical decision making, critical thinking, and a global grasp of the situation, coupled with nursing skills acquired through a process of integrating formal and informal experiential knowledge and evidence-based guidelines
  - *Level 1:* Collects basic-level data; follows algorithms, decision trees, and protocols with all populations and is uncomfortable deviating from them; matches formal knowledge with clinical events to make decisions; questions the limits of one's ability to make clinical decisions and delegates the decision making to other clinicians; includes extraneous detail
  - *Level 3:* Collects and interprets complex patient data; makes clinical judgments based on an immediate grasp of the whole picture for common or routine patient populations; recognizes patterns and trends that may predict the direction of illness; recognizes limits and seeks appropriate help; focuses on key elements of case, while sorting out extraneous details
  - *Level 5:* Synthesizes and interprets multiple, sometimes conflicting, sources of data; makes judgment based on an immediate grasp of the whole picture, unless working with new patient populations; uses past experiences to anticipate problems; helps patient and family see the "big picture"; recognizes the limits of clinical judgment and seeks multidisciplinary collaboration and consultation with comfort; recognizes and responds to the dynamic situation
- **Advocacy and Moral Agency**—working on another's behalf and representing the concerns of the patient/family and nursing staff; serving as a moral agent in identifying and helping to resolve ethical and clinical concerns within and outside the clinical setting.
  - *Level 1:* Works on behalf of patient; self-assesses personal values; aware of ethical conflicts/issues that may surface in clinical setting; makes ethical/moral decisions based on rules; represents patient when patient cannot represent self; aware of patients' rights
  - *Level 3:* Works on behalf of patient and family; considers patient values and incorporates in care, even when differing from personal values; supports colleagues in ethical and clinical issues; moral decision making can deviate from rules; demonstrates give and take with patient's family, allowing them to speak/represent themselves when possible; aware of patient and family rights
  - *Level 5:* Works on behalf of patient, family, and community; advocates from patient/family perspective, whether similar to or different from personal values; advocates ethical conflict and issues from patient/family perspective; suspends rules—patient and family drive moral decision making; empowers the patient and family to speak for/represent themselves; achieves mutuality within patient/professional relationships

(*continued on page 10*)

BOX 1-9 Nurse Competencies of Concern to Patients, Clinical Units, and Systems (continued)

- **Caring Practices**—nursing activities that create a compassionate, supportive, and therapeutic environment for patients and staff, with the aim of promoting comfort and healing and preventing unnecessary suffering. Includes, but is not limited to, vigilance, engagement, and responsiveness of caregivers, including family and health care personnel
  - *Level 1:* Focuses on the usual and customary needs of the patient; no anticipation of future needs; bases care on standards and protocols; maintains a safe physical environment; acknowledges death as a potential outcome
  - *Level 3:* Responds to subtle patient and family changes; engages with the patient as a unique patient in a compassionate manner; recognizes and tailors caring practices to the individuality of patient and family; domesticates the patient's and family's environment; recognizes that death may be an acceptable outcome
  - *Level 5:* Has astute awareness and anticipates patient and family changes and needs; is fully engaged with and sensing how to stand alongside the patient, family, and community; caring practices follow the patient and family lead; anticipates hazards and avoids them, and promotes safety throughout patient's and family's transitions along the health care continuum; orchestrates the process that ensures patient's/family's comfort and concerns surrounding issues of death and dying are met
- **Collaboration**—working with others (eg, patients, families, health care providers) in a way that promotes/encourages each person's contributions toward achieving optimal/realistic patient/family goals. Involves intradisciplinary and interdisciplinary work with colleagues and community
  - *Level 1:* Willing to be taught, coached, and/or mentored; participates in team meetings and discussions regarding patient care and/or practice issues; open to various team members' contributions
  - *Level 3:* Seeks opportunities to be taught, coached, and/or mentored; elicits others' advice and perspectives; initiates and participates in team meetings and discussions regarding patient care and/or practice issues; recognizes and suggests various team members' participation
  - *Level 5:* Seeks opportunities to teach, coach, and mentor and to be taught, coached, and mentored; facilitates active involvement and complementary contributions of others in team meetings and discussions regarding patient care and/or practice issues; involves/recruits diverse resources when appropriate to optimize patient outcomes
- **Systems Thinking**—body of knowledge and tools that allow the nurse to manage whatever environmental and system resources exist for the patient/family and staff, within or across health care and non–health care systems
  - *Level 1:* Uses a limited array of strategies; limited outlook—sees the pieces or components; does not recognize negotiation as an alternative; sees patient and family within the isolated environment of the unit; sees self as key resource
  - *Level 3:* Develops strategies based on needs and strengths of patient/family; able to make connections within components; sees opportunity to negotiate but may not have strategies; developing a view of the patient/family transition process; recognizes how to obtain resources beyond self
  - *Level 5:* Develops, integrates, and applies a variety of strategies that are driven by the needs and strengths of the patient/family; global or holistic outlook—sees the whole rather than the pieces; knows when and how to negotiate and navigate through the system on behalf of patients and families; anticipates needs of patients and families as they move through the health care system; utilizes untapped and alternative resources as necessary
- **Response to Diversity**—the sensitivity to recognize, appreciate, and incorporate differences into the provision of care. Differences may include, but are not limited to, cultural differences, spiritual beliefs, gender, race, ethnicity, lifestyle, socioeconomic status, age, and values.
  - *Level 1:* Assesses cultural diversity; provides care based on own belief system; learns the culture of the health care environment
  - *Level 3:* Inquires about cultural differences and considers their impact on care; accommodates personal and professional differences in the plan of care; helps patient/family understand the culture of the health care system
  - *Level 5:* Responds to, anticipates, and integrates cultural differences into patient/family care; appreciates and incorporates differences, including alternative therapies, into care; tailors health care culture, to the extent possible, to meet the diverse needs and strengths of the patient/family
- **Facilitation of Learning**—the ability to facilitate learning for patients/families, nursing staff, other members of the health care team, and community. Includes both formal and informal facilitation of learning
  - *Level 1:* Follows planned educational programs; sees patient/family education as a separate task from delivery of care; provides data without seeking to assess patient's readiness or understanding; has limited knowledge of the totality of the educational needs; focuses on a nurse's perspective; sees the patient as a passive recipient
  - *Level 3:* Adapts planned educational programs; begins to recognize and integrate different ways of teaching into delivery of care; incorporates patient's understanding into practice; sees the overlapping of educational plans from different health care providers' perspectives; begins to see the patient as having input into goals; begins to see individualism
  - *Level 5:* Creatively modifies or develops patient/family education programs; integrates patient/family education throughout delivery of care; evaluates patient's understanding by observing behavior changes related to learning; is able to collaborate and incorporate all health care providers' and educational plans into the patient/family educational program; sets patient-driven goals for education; sees patient/family as having choices and consequences that are negotiated in relation to education
- **Clinical Inquiry (Innovator/Evaluator)**—the ongoing process of questioning and evaluating practice and providing informed practice. Creating practice changes through research utilization and experiential learning
  - *Level 1:* Follows standards and guidelines; implements clinical changes and research-based practices developed by others; recognizes the need for further learning to improve patient care; recognizes obvious changing patient situation (eg, deterioration, crisis); needs and seeks help to identify patient problem
  - *Level 3:* Questions appropriateness of policies and guidelines; questions current practice; seeks advice, resources, or information to improve patient care; begins to compare and contrast possible alternatives
  - *Level 5:* Improves, deviates from, or individualizes standards and guidelines for particular patient situations or populations; questions and/or evaluates current practice based on patients' responses, review of the literature, research, and education/learning; acquires knowledge and skills needed to address questions arising in practice and improve patient care. (The domains of clinical judgment and clinical inquiry converge at the expert level; they cannot be separated.)

From http://www.certcorp.org/certcorp/certcorp.nsf/vwdoc/SynModel.

and are expressed as level 1, 3, or 5. The level can vary based on level of expertise in a given clinical situation.

The Synergy Model is also used to determine outcomes. Outcomes are evaluated based on those derived from the patient, nurse, and health care system. Patient-derived outcomes may include functional change, trust, satisfaction, comfort, and quality of life. Nurse-derived outcomes may include physiological changes, absence of complications, and extent to which treatment objectives are attained. Health care system–derived outcomes may include recidivism, costs, and resource utilization.

Since its development, the Synergy Model has been used in a variety of clinical settings as a basis for nursing clinical advancement, determining staffing ratios, preceptorship, and as a foundation for advanced practice nursing.[98–103] For example, the Clarian Health System in Indianapolis, Indiana, uses the model to facilitate organizational change.[104] Here, job descriptions are based on the eight nurse characteristics. At Baylor Hospital in Dallas, Texas, the model is the foundation for evaluation of nursing orientation and is the basis for the nursing advancement program. The model also has an effect in other areas, such as shift report, curriculum design, and nurse manager job analysis.

Case reports in the literature illustrate the use of the Synergy Model in promoting optimal patient outcomes.[105–108] Use of the Synergy Model has also been described as a framework that can be used as a basis for work to be done by facilities seeking Magnet designation.[109,110]

▲ Future Challenges in Critical Care Nursing

Because health care, nursing, and the world are dynamic, critical care nursing must continue to evolve. As the United States becomes increasingly diverse, nursing must increase its ability to deliver evidence-based care that is culturally congruent and relevant. The growing U.S. Hispanic population makes it increasingly essential for health care systems to have a multilingual, culturally diverse workforce. This requires not only the need to recruit and retain diverse professionals but also the need to expand the skill set of today's already experienced critical care nurses.

As new and emerging infectious diseases present, critical care nurses must be prepared to identify, manage, and treat unknown threats. Similarly, Hurricane Katrina clearly demonstrated the impact that natural disasters have on a nation, health outcomes, and the nursing workforce. Furthermore, in the era after September 11, critical care units must be prepared to handle any actual or potential bioterrorism threat.

Finally, as critical care continues to evolve and become increasingly technologically sophisticated, critical care nurses must continue to expand their repertoire of skills and evidence-based interventions to address not only the physiological needs but also the psychosocial, spiritual, ethical, and advocacy needs of patients and families. With implementation of advances in technology, the critical care unit will continue to require caring, competent, and knowledgeable nurses who can foster collaboration, navigate complex delivery and reimbursement systems, and facilitate patient/family learning while responding to diverse communities that are vulnerable with complex needs. These exciting challenges await critical care nurses—challenges that they will overcome with commitment, dedication, and grace!

▲ Clinical Applicability Challenges

SHORT ANSWER QUESTIONS

1. Describe a time in your workplace where you received meaningful recognition.
2. Describe some of the barriers in your workplace to implementing practice changes based on evidence.
3. Describe a clinical situation that exemplifies high levels of competence with the nurse competency response to diversity from the AACN Synergy Model for Patient Care.

References

1. American Association of Critical-Care Nurses Certification Corporation: What is certification? Retrieved January 5, 2011, from http://www.aacn.org/WD/Certifications/Content/consumer- whatiscert.pcms?menu=Certification&lastmenu=
2. American Association of Critical-Care Nurses and AACN Certification Corporation: Safeguarding the patient and the profession: The value of critical care nurse certification—executive summary. Retrieved January 5, 2011, from http://www.aacn.org/WD/Certifications/Docs/certwhitepaper.pdf
3. Watts M: Certification and clinical ladder as the impetus for professional development. Crit Care Nurs Q 33:52–59, 2010
4. American Association of Critical-Care Nurses: Linkages between Certification and Outcomes for Patients, Systems or Nurses. Retrieved January 5, 2011, from http://www.aacn.org/WD/Certifications/Docs/researchvalidatingcertification.pdf
5. Kendall-Gallagher D, Blegen MA: Competence and certification of registered nurses and safety of patients. Am J Crit Care 18:106–113, 2009
6. Wade CH: Perceived effects of specialty nurse certification: A review of the literature. AORN J 89(1):183–512, 2009.
7. American Nurses Credentialing Center: Forces of magnestism. Retrieved January 5, 2011 from http://www.nursecredentialing.org/Magnet/ProgramOverview/ForcesofMagnetism.aspx
8. Niebuhr B, Biel M: The value of specialty nursing certification. Nurs Outlook 55(4):176–181, 2007
9. Sigma Theta Tau International Research and Scholarly Advisory Committee: Sigma Theta Tau International Position Statement on Evidence Based Practice February 2007, Summary. Worldviews Evid Based Nurs 5:57–59, 2008
10. Brown CE, Wickline MA, Ecoff L et al: Nursing practice, knowledge, attitudes and perceived barriers to evidence based practice at an academic medical center. J Adv Nurs 65(2):371–381, 2009
11. Cullen L, Adams S: What is evidence based practice? J Perianesth Nurs 25(3):171–173, 2010
12. Armola RR, Bourgault AM, Halm MA, et al; 2008–2009 Evidence-Based Practice Resource Work Group of the American Association of Critical-Care Nurses: Upgrading the American Association of Critical Care Nurses' evidence-leveling hierarchy. Am J Crit Care 18(5): 405–409, 2009
13. Baumann SL: The limitations of evidence based practice. Nurs Sci Q 23(3):226–230, 2010
14. Polit DF, Beck CT: Nursing Research: Generating and Assessing Evidence for Nursing Practice,8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008
15. Kitson A: Knowledge translation and guidelines: A transfer, translation or transformation process? Int J Evid Based Healthc 7(2):124–139, 2009
16. Ross J: Information literacy for evidence-based practice in perianesthesia nurses: Readiness for evidence based practice. J Perianesth Nurs 25(2):64–70, 2010
17. Hockenberry M, Wilson D, Barrera P: Implementing evidence-based nursing practice in a pediatric hospital. Pediatr Nurs 32(4):371–377, 2006
18. Soltero P, Pugh K, Carnacho L: Evidence-based practice: Journal club can bring research and education to the bedside to promote best nursing practices. J Pediatr Nurs 23 (2):e22–e23, 2008
19. Schulman CS: Strategies for starting a successful evidence based practice program. Adv Crit Care 19(3):301–311, 2008
20. American Association of Critical Care Nurses: AACN Standards for Establishing and Sustaining Health Work Environments: A Journey to Excellence. 2005. Retrieved July 7, 2011, from http://www.aacn.org/WD/HWE/Docs/HWEStandards.pdf
21. The Joint Commission: Sentinel Event Data Root Causes by Event Type, 2004-Fourth Quarter 2010. Retrieved July 7, 2011 from http://www.jointcommission.org/assets/1/18/SE_RootCausesEventType2004_4Q2010.pdf
22. Maxfield D, Grenny J, McMillan R, et al: Silence kills: The seven crucial conversations for healthcare. 2005. Retrieved July 7, 2011 from http://www.aacn.org/WD/Practice/Docs/PublicPolicy/SilenceKills.pdf
23. Lindeke LL, Sieckert AM: Nurse-physician workplace collaboration. Online J Issues Nurs 10(1):4, 2005
24. Dougherty M: Psychological consultation and collaboration in school and community settings, 5th ed. Belmont, CA: Brooks/Cole, 2009, p 13
25. Gardner DB: Ten lessons in collaboration. Online J Issues Nurs 10(1):2, 2005
26. Ferrand E, Lemaine F, Regnier B, et al: Discrepancies between perceptions by physicians and nursing staff of intensive care unit end-of-life decisions. Am J Respir Crit Care Med 167(10):1310–1315, 2003
27. Scott J, Sochalski J, Aiken L: Review of magnet hospital research findings and implications for professional nursing practice. J Nurs Admin 29(1):9–19, 2003
28. McMahon EM, Hoffman K, McGee GW: Physician-nurse relationships in clinical settings: A review and critique of the literature 1966–1992. Med Care Rev 51(1):83–112, 1994
29. Baggs JG, Ryan SA, Phelps CG, et al: The association between interdisciplinary collaboration and patient outcomes in a medical intensive care unit. Heart Lung 32(1):18–24, 1992
30. Sterchi LS: Perceptions that affect physician-nurse collaboration in the perioperative setting. AORN J 86(1):45–57, 2007
31. Thomson S: Nurse-physician collaboration: A comparison of the attitudes of nurses and physicians in the medical-surgical patient care setting. MedSurg Nurs 16(2):87–91, 2007
32. Hojat M, Nasca T, Cohen M, et al: Attitudes toward physician-nurse collaboration: A cross-cultural study of male and female physicians in the United States and Mexico. Nurs Res 50(2):123–128, 2001
33. Hojat M, Gonnella J, Nasca T, et al: Comparisons of Americans, Italian and Mexican physicians and nurses on the total and factor scores of the Jefferson Scale of Attitudes toward physician-nurse collaborative relationships. Int J Nurs Stud 40:425–435, 2003
34. Rosenstein A: Nurse-physician relationships: Impact on nurse satisfaction and retention. Am J Nurs 102(1):26–34, 2001
35. Vazirani S, Hays RD, Shapiro MF, et al: Effect of a multidisciplinary intervention on communication and collaboration among physicians and nurses. Am J Crit Care 14(1):71–77, 2005
36. Kramer M, Schmalenberg C: Securing "good" nurse-physician relationships. Nurs Manage 34(7):34–38, 2003
37. Baggs J, Schmitt M, Mushlin AI, et al: Association between nurse-physician collaboration and patient outcomes in three intensive care units. Crit Care Med 27(9):1991–1998, 1999
38. Reader TW, Flin R, Mearns K, et al: Interdisciplinary communication in the intensive care unit. Br J Anaesth 91(3):347–352, 2007
39. McCaffrey RG, Hayes R, Stuart W, et al: A program to improve communication and collaboration between nurses and medical residents. J Contin Educ Nurs 41(4):172–178, 2010
40. Chang W-Y, Ma J-C, Chin H-T: Job satisfaction and perceptions of quality of patient care, collaboration and teamwork in acute care hospitals. J Adv Nurs 65(9):46–55, 2001
41. McGrail KA, Morse DS, Glessner T, et al: "What I found there"[1] Qualitative analysis of physician-nurse collaboration stories. J Gen Intern Med 24(2):198–204, 2008
42. Stein-Parbury J, Liaschenko J: Understanding collaboration between nurses and physicians as knowledge at work. Am J Crit Care 16:470–477, 2007
43. Schmalenberg C, Kramer M: Nurse-physician relationships in hospitals. 20,000 nurses tell their stories. Crit Care Nurse 29:74–83, 2009
44. Miller PA: Nurse-physician collaboration in an intensive care unit. Am J Crit Care 10(5):341–350, 2001
45. Aiken LH: Evidence-based management: Key to hospital workforce stability. J Health Adm Educ Special issue:117–125, 2001
46. D'Amour D, Ferrada-Videla M, San Martin Rodriguez L, et al: The conceptual basis for interprofessional collaboration: Core concepts and theoretical frameworks. J Interprof Care 19(Suppl 1):116–131, 2005
47. Hall P: Interpersonal teamwork: Professional cultures as barriers. J Interprof Care 19(Suppl 1): 188–196, 2005
48. Evans JA: The role of the nurse manager in creating an environment for collaborative practice. Holist Nurs Pract 8(3):22–31, 1994

49. Grumbach K, Bodenheimer T: Can health care teams improve primary care practice? JAMA 291:1246–1251, 2004
50. Tellis-Nayak M, Tellis-Nayak V: Games that professionals play: The social psychology of physician-nurse interaction. Soc Sci Med 18(12):1063–1069, 1984
51. Cashman SB, Reidy P, Cody K, et al: Developing and measuring progress toward collaborative, integrated, interdisciplinary health care teams. J Interprof Care 18(2):183–196, 2004
52. Orchard CA, Curran V, Kabene S: Creating a culture of interdisciplinary collaborative professional practice. Med Educ Online 10:11–24, 2005. Available at http://www.med-ed-online.org
53. Wachter RM, Shojania K: Internal Bleeding—The Truth Behind America's Terrifying Epidemic of Medical Mistakes. New York: Rugged Land, LLC, 2004
54. Rashotte J, Carnevale FA: Medical and nursing clinical decision making: A comparative epistemological analysis. Nurs Philos 5(2):160–174, 2004
55. Bakalis NA, Watson R: Nurses' decision-making in clinical practice. Nurs Standard 19(23):33–39, 2005
56. Banning M. A review of clinical decision making: models and current research. J Clin Nurs 17(2):187–195, 2007
57. Cranley L, Doran DM, Tourangeau AE, et al: Nurses' uncertainty in decision-making: A literature review. Worldviews Evid Based Nurs 6 (1):3–15, 2009
58. White A: Clinical decision making among fourth-year nursing students: An interpretative study. J Nurs Educ 42(3):113–120, 2003
59. Hagbaghery MA, Salsali M, Ahmadi F: The factors facilitating and inhibiting effective clinical decision-making in nursing: A qualitative study. BMC Nurs 3(1):2, 2004
60. Aiken LH, Clarke SP, Sloane DM, et al: Hospital nurse staffing and patient mortality, nurse burnout, and job dissatisfaction. JAMA 288:1987–1993, 2002
61. Stanton MA: Hospital nurse staffing and quality of care. Retrieved April 15, 2010 from http://www.ahrq.gov/research/nursestaffing/nursestaff.htm
62. Hickam DH, Severance S, Feldman A, et al: The effect of health care working conditions on patient safety. Evidence report/Technology Asst Number 74. AHRQ Publication NO 03-E031. Rockville, MD: Agency for Healthcare Research and Quality, 2003
63. Cho SH, Ketefian S, Barkauskas VH, et al: The effects of nurse staffing on adverse outcomes, morbidity, mortality, and medical costs. Nurs Res 52(2):71–79, 2003
64. Estabrooks CA, Midodzi WK, Cummings GC, et al: Determining the impact of hospital nursing characteristics on 30-day mortality among patients in Alberta acute care hospitals. Nurs Res 54(2):74–84, 2005
65. McGillis HL, Doran D, Dink GH: Nurse staffing models, nursing hours and patient safety outcomes. J Nurs Adm 34:41–45, 2004
66. Needleman J, Buerhaus P, Mattke S, et al: Nurse-staffing levels and the quality of care in hospitals. N Engl J Med 346:1715–1722, 2002
67. Thungjaroenkul P, Cummings GC, Embleton A: The impact of nurse staffing on hospital costs and patient length of stay: A systematic review. Nurs Econ 25(5):255–265, 2007
68. Kalisch BJ: Missed nursing care: A qualitative study. J Nurs Care Q 21(4):306–313, 2006
69. Vahey DC, Aiden LH, Sloane DM, et al: Nurse burnout and patient satisfaction. Med Care 42(Suppl 2):1157–1166, 2004
70. Kennedy MS: Low nurse staffing linked to neonatal infection. Am J Nurs 106(12):22, 2006
71. Kane RL, Tatyana A, Shamliyar MD, et al: The association of registered nurse staffing levels and patient outcomes. Systematic review and meta-analysis. Med Care 45(12):1195–1204, 2007
72. Potter P, Barr N, McSweeney M, et al: Identifying nurse staffing and patient outcome relationships: A guide for change in care delivery. Nurs Econ 21(4):158–166, 2003
73. Institute of Medicine: Keeping Patients Safe: Transforming the Work Environment of Nurses. Washington, DC: National Academies Press, 2003
74. Freed DH: Fifty-two effective, inexpensive ways to reward and recognize hospital employees. Healthc Manage 18(1):20–28, 1999
75. Izzo JB, Withers P: Winning employee-retention strategies for today's healthcare organizations. Healthc Financ Manage 56(6):52–57, 2002
76. Bryant-Hampton L, Walton AM, Tracy C, et al: Recognition: A key retention strategy for the mature nurse. J Nurs Admin 40(3):121–123, 2010
77. Goode CJ, Ibarra V, Blegan MA: What kind of recognition do staff nurses want? Am J Nurs 93(5):64–68, 1993
78. Collins SK, Collins KS: Employee retention: An issue of survival in healthcare. Radiol Manage 26(4):52–55, 2004
79. Lamberth B, Comello RJ: Identifying elements of job satisfaction to improve retention rates in healthcare. Radiol Manage 27(3):34–38, 2005
80. Randolph DS: Predicting the effect of extrinsic and intrinsic job satisfaction factors on recruitment and retention of rehabilitation professionals. J Healthc Manage 50(1):49–60, 2005
81. Ulrich BT, Lavandero R, Hart KA, et al: Critical care nurses' work environments 2008: A follow-up report. Crit Care Nurse 29(2):93–102, 2009
82. Qaseem B, Shea J, Connor SR, et al: How well are we supporting hospice staff? Initial results of the survey of team attitudes and relationships (STAR) validation study. J Pain Symptom Manage 34:350–358, 2007
83. Tourangeau AE, Cranley LA: Nurse intention to remain employed: Understanding and strengthening determinants. J Adv Nurs 55:497–507, 2006
84. Chan E, Morrison P: Factors influencing the retention and turnover intentions of registered nurses in a Singapore hospital. Nurs Health Science 2:113–121, 2000
85. Hausknecht JP, Rodda J, Howard MJ: Targeted employee retention: Performance-based and job-related differences in reported reasons for staying. Hum Resour Manage 48:269–288, 2009
86. Hayes LJ, O'Brien-Passas L, Duffield C, et al: Nurse turnover: A literature review. Int J Nurs Stud 43:237–263, 2006
87. Lacey SR, Teasley SL, Henion JS, et al: Enhancing the work environment of staff nurses using targeted interventions of support. J Nurs Adm 38:336–340, 2008
88. Hurst KL, Croker PA, Bell SK: How about a lollipop? A peer recognition program. Nurs Manage 25:68–72, 1994
89. Lu H, While AE, Barriball LK: Job satisfaction among nurses: A literature review. Int J Nurs Stud 42:211–227, 2005
90. Bylone M: Nurses week: Is this what they mean by meaningful recognition? AACN Adv Crit Care 19:121–124, 2008
91. Sourdif J: Predictor of nurses' intention to stay at work in a university health center. Nurs Health Sci 6:59–68, 2004
92. Wong CA, Cummings GG: The relationship between nursing leadership and patient outcomes: A systematic review. J Nurs Manage 15(5):508–521, 2007
93. McGillis HL: Quality Work Environments for Nurse and Patient Safety. Sudbury, MA: Jones & Bartlett, 2005
94. McCauley K: President's note: All we needed was the glue. AACN News 22:2, 2005
95. Brodie D: Leadership—The 6 characteristics of authentic leaders. Retrieved April 15, 2010 from http://exmearticles.com/?leadership–the-6-characteristics-of-authentic-leaders&id1334017
96. American Association of Critical-Care Nurses Certification Corporation: The AACN Synergy Model for Patient Care. Retrieved July 7, 2011 from http://www.aacn.org/wd/certifications/content/synmodel.pcms?mid=2890&menu=
97. Reed KD, Cline M, Kerfoot KM: Implementation of the synergy model in critical care. In Kaplow R, Hardin SR (eds): Critical Care Nursing: Synergy for Optimal Outcomes. Sudbury, MA: Jones & Bartlett, 2007
98. Hardin SR, Kaplow R: Synergy for Clinical Excellence: The AACN Synergy Model For Patient Care. Sudbury, MA: Jones & Bartlett, 2005
99. Czerwinski S, Blastic L: The Synergy Model: Building a clinical advancement program. Crit Care Nurse 19(4):72–77, 1999
100. Hartigan RC: Establishing criteria for 1:1 staffing ratios. Crit Care Nurse 20(2):112–116, 2000
101. Kaplow R: Applying the Synergy Model to nursing education. Crit Care Nurse 22(3):77–81, 2002

102. Maloney-Harmon PA. The Synergy Model: Contemporary practice for the clinical nurse specialist. Crit Care Nurse 19(2):101–104, 1999
103. Collopy KS. Advanced practice nurses guiding families through systems. Crit Care Nurse 19(5):80–85, 1999
104. Alspach G: Extending the Synergy Model to preceptorship. Crit Care Nurse 26(2):10–13, 2006
105. Kerfoot K: Multihospital system adapts AACN Synergy Model—In Our Unit—Clarian Health Partners chooses patient care model. Crit Care Nurse 23(5):88, 2003
106. Hardin SR, Hussey L: AACN Synergy Model for patient care. Case study for a CHF patient. Crit Care Nurse 23(1):73–76, 2003
107. Graham-Garcia J, George-Gay B, Heater D, et al: Application of the Synergy Model with the surgical care of smokers. Crit Care Nurs Clin North Am 18(1):29–38, 2006
108. Kuriakose A: Using the Synergy Model as best practice in endotracheal tube suctioning of critically ill patients. Dimens Crit Care Nurs 27(1):10–15, 2008
109. Smith AR: Using the Synergy Model to provide spiritual nursing care in critical care settings. Crit Care Nurse 26(4):41–47, 2006
110. Kaplow R, Reed KD: AACN Synergy Model for patient care. Nursing model as a force of magnetism. Nurs Econ 26(1):17–25, 2008

2 The Patient's Experience with Critical Illness

Kathryn S. Bizek and Dorrie K. Fontaine

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Explore relationships among stress, response to illness, and anxiety.
2. Explore the role of the nurse in controlling environmental stressors to promote healing.
3. Compare and contrast techniques that the patient and family can learn in an effort to manage stress and anxiety.
4. Describe strategies to promote sleep in critically ill patients.
5. Develop nursing interventions that foster the ability of patients to draw strength from their personal spirituality.
6. Discuss alternatives to the use of physical restraint in the intensive care unit.

The patient's experience in an intensive care unit (ICU) has lasting meaning for the patient and his or her family members and significant others. Although actual painful memories are blurred by drugs and the mind's need to forget, attitudes that are highly charged with feelings about the nature of the experience survive. These attitudes shape the person's beliefs about nurses, physicians, health care, and the vulnerability of life itself.

This chapter describes specific measures that nurses use to support patients in managing the stressors associated with critical illness and injury. It is the caring and emotional support given by the nurse that will be remembered and valued.

▲ Perception of Critical Illness

Admission to an ICU may signal a threat to the life and well-being of the patient who is admitted. Critical care nurses perceive the unit as a place where fragile lives are vigilantly scrutinized, cared for, and preserved. However, patients and their families frequently perceive admission to critical care as a sign of impending death, based on their own past experiences or the experiences of others. Understanding what critical care means to patients may help nurses care for their patients. However, effective communication with critically ill patients is often challenging and frustrating.[1–3] Barriers to communication may relate to the patients' physiological status; the existence of endotracheal tubes, which inhibit verbal communication; medications; or other conditions that alter cognitive function.

About 30% to 100% of former ICU patients could recall part or all of their stay in the ICU. Although many of the patients recalled feelings that were negative, they also recalled neutral and positive experiences. Negative experiences were related to fear, anxiety, sleep disturbance, cognitive impairment, and pain or discomfort. Positive experiences were related to feelings of being safe and secure. Often, these positive feelings were attributed to the care provided by nurses. The need to feel safe and the need for information were predominant themes in other research studies.[2,4–6] Nurses' technical competence and effective interpersonal skills were cited by patients as promoting their sense of security and trust.

▲ Stress

Stress exists when an organism is faced with any stimulus that causes disequilibrium between psychological and physiological functioning. Patients admitted to the ICU are subject to multiple physical, psychological, and environmental stressors. Stimulation of the body's stress response involves activation of the hypothalamic–pituitary–adrenal axis. The resultant increase in catecholamine, glucocorticoid, and mineralocorticoid levels leads to a cascade of physiological responses.[7]

Acute Stress Response

Critical injury or illness can initiate the first phase of the stress response. This phase is characterized by the body's efforts to survive and involves the stimulation of the sympathetic nervous system and activation of multiple neuroendocrine responses. This "ebb phase" results in increased heart rate and contractility, vasoconstriction, and increase in blood pressure. Blood flow is redirected to vital organs. Pain sensations are temporarily attenuated. Body temperature and nutrient consumption fall.

Table 2-1 Major Stress-Related Hormones and Effects

Stress Hormones	Source	Major Effects
Adrenocorticotropic hormone	Anterior pituitary	Stimulates adrenal cortex to release cortisol
Catecholamines Epinephrine Norepinephrine	Adrenal medulla and the sympathetic nervous system	Increases overall strength, blood flow to vital organs, glyconeogenesis Increases myocardial contractility (inotropic effect), heart rate (chronotropic effect), venous return to the heart and cardiac output Constricts smooth muscle in all blood vessels, increases blood pressure, dilates pupils, inhibits gastrointestinal activity
Cortisol	Adrenal cortex (following stimulation by adrenocorticotropic hormone from the anterior pituitary)	Gluconeogenesis; hyperglycemia; decreases protein synthesis, immunoglobulin synthesis, number of lymphocytes, and leukocytes (at inflammatory site); promotes muscle and lymphoid tissue catabolism; delays healing; suppresses cell-mediated immune response
Antidiuretic hormone	Posterior pituitary	Increases water retention
Aldosterone	Adrenal cortex	Increases sodium and water retention
Growth hormone (somatotropin)	Anterior pituitary	Increases immune function; levels are increased during stress
Prolactin	Anterior pituitary	β-Cell activation and differentiation; levels are reduced during stress
Testosterone	Testis	Regulates male secondary characteristics; levels are decreased during chronic stress
Endorphins	Anterior pituitary	Endogenous opiates, elevated during stress, downregulate pathways to the stress response
Enkephalins	Adrenal medulla	Endogenous opiates, elevated during stress, downregulate pathways to the stress response

From Lusk B, Lash AA: The stress response, psychoneuroimmunology, and stress among ICU patients. Dimens Crit Care Nurs 24(1):25–31, 2003, with permission.

A sensation of thirst may be prominent. Other physiological effects include increase in minute ventilation and respiratory rate, hyperglycemia, insulin resistance, and coagulopathies. This initial phase is deeply catabolic as protein stores are mobilized to respond to the threat and begin to repair the injury. If this phase is prolonged, it can result in impaired oxygen and nutrients delivery to tissues secondary to alterations in microcirculatory blood flow. Table 2-1 summarizes the effects of hormones released in response to major stress.[8–18]

The second phase of the stress response, or the "flow phase," is a hyperdynamic state that results as the body compensates for the oxygen deprivation. This phase is also characterized by multiple hormonal influences. Pain and discomfort are now prominent. Movement is minimized to conserve metabolic costs. Prolonged activation of the stress response can lead to immunosuppression, hypoperfusion, tissue hypoxia, and eventual death. Treatment is directed at eliminating the stressors and providing supportive care in the form of nutrition, oxygenation, pain management, anxiety control, and specific measures related to the cause of illness or injury.[15,19–21]

Environmental Stressors in the Intensive Care Unit

The ICU is a stressful environment for patients and care givers. Walk into any ICU and you will find the following common physical features: blinking monitors, ventilators, intravenous (IV) pumps, noise from equipment and the many practitioners talking at the bedside, bright lights, and a hurried pace in a crowded space. Intra-aortic balloon pumps, extracorporeal membrane oxygenation machines, and multiple sophisticated technologies are commonplace. Critical care nursing was invented to flourish in this setting, where the most acutely ill and injured receive concentrated nursing care to enhance survival. In the early ICUs of the 1950s, nurses were confronted daily with pain, suffering, and death, while caring for patients in a confined though open space.[22] These ICUs were often a few beds carved out of existing wards in older hospitals that brought the sickest patients to one area. The most distinctive feature of the first ICUs was this concentration of nursing care, and the specialty of intensive care nursing was born. The design of ICUs has changed over the decades and provides a rationale for why care needs for the patient and family have also evolved. The concept of healing environments in hospitals emerged as one where the environment can make a difference in how quickly the patient recovers.[23]

Table 2-2 summarizes the key design features of ICUs from the 1950s to an envisioned future. Common to all these designs is the notion of close observation and rapid intervention. Meeting patient needs through continuous monitoring is the hallmark of all critical care. However, the close monitoring has led to patient complaints of

Table 2-2 Intensive Care Unit Designs

	First Generation (1950s)	Second Generation (1970s)	Third Generation (1980s–Present)	Fourth Generation—Future
Characteristics	Open unit/ward. No partitions except curtains or screens. Nurses' station/desk in center or at the foot of beds. Unit lighting control often on one switch.	Individual rooms or walled cubicles. Rooms often on either side of a hall containing an open nursing station or surrounding an open nursing station on three to four sides (square configuration). Central monitoring. Some units without external patient room windows (increased incidence of delirium). Patient room lighting with separate switch(es) from nursing station. Calendars and clock in patient rooms.	Individual rooms. Folding or sliding glass doors. Rooms often arranged on a semicircle or circle with the nursing station in the center. Some units configured with decentralized nursing stations. Patient room windows with external views/lighting. Increased control of patient room lighting levels.	Individual rooms. Folding or sliding glass doors with privacy curtains/blinds. Circular/pod-shaped floor plan. Increased noise reduction design. Patient windows with a view of outdoors (natural or contrived). Patient-controlled lighting—artificial and natural. Planned areas for family in patient rooms. Increased use of color and texture in wall, floor, and ceiling coverings.
Advantages	Increased nurses' proximity to patients	Increased patient privacy. Better control of lighting, noise, and infection.	Increased nursing access during high-intensity activities.	Nursing access and availability of high-tech care in a more homelike environment.
Disadvantages	Lack of privacy. Inability to control noise or light. Infection control issues.	Less direct patient access/ observation. Less than optimal control of noise and lighting.	Glass doors reduce patient privacy.	

From Fontaine DK, Prinkey Briggs L, Pope-Smith B: Designing humanistic critical care environments. Crit Care Nurs Q 24(3):21–34, 2001, with permission.

noise, lighting with no day–night distinction, and frequent interruptions of sleep and rest. Intensive care beds were often so close to each other that patients could hear everything happening to the critically ill patient in the next bed. Lack of privacy and fears related to overheard procedures and conversations in the unit created undue anxiety and the potential for physiological instability in vulnerable patients.

The evolution of ICUs has demonstrated increasing use of the precepts of family-focused care. Early units typically had no space for family to visit, and visits were not encouraged. Emphasis today is on how the design of the ICU can best meet the needs of patients and families as a unit, despite the important life-sustaining technology. The shift to family-focused care is a good example of how the structure and function of ICUs has changed. Signs that welcome family and visitors to the ICU often suggest the philosophy of the hospital and the culture of the unit. The more welcoming the ICU is to visitors, the more likely the environment is to offer a healing culture of care and support. Does the sign on the door read "Stop, Do Not Enter" or "Welcome to the ICU"?

Patients experience a positive outcome in an environment that incorporates natural light, elements of nature, soothing colors, meaningful and varied stimuli, peaceful sounds, and pleasant views.[24] In fact, research demonstrates less pain medication is needed and a faster recovery may occur when careful attention is given to providing a soothing environment. Hospitals that combine creative design elements with an emphasis on family-focused care are the leaders in creating healing spaces for recovery.

Noise

Despite third-generation unit design and architecture, the problems of noise and bright lighting have remained a challenge. Beds surrounded by noisy machines and equipment are intimidating to patients, family, and novice nurses in critical care. Noise is an environmental hazard that creates discomfort in a patient. Consequences of noisy environments include disrupted sleep, impaired wound healing, and activation of the sympathetic nervous system. Moderate noise levels may produce vasoconstriction. Hyperarousal related to noise can occur over many days to even weeks for patients with prolonged ICU stays.

Patient complaints include listening to banging noises, alarms going off at all times, water sounds (such as the bubbling of chest tubes), and doors opening and closing. Sources of noise include equipment, alarms, telephones, televisions, ventilators, and staff conversations. Health care providers are often unaware of the loudness of their conversations and the irritation they may create in the minds of patients. People differ in their perceptions of noise as irritating; therefore, nurses should perform an objective assessment of the environment.

Noise is measured in decibels using a logarithmic scale. An increase of 10 decibels makes a sound seem twice as loud. Sleep occurs best below 35 decibels. The Environmental Protection Agency recommends unit noise be less than

45 decibels during the day and 35 decibels at night. Numerous studies measuring noise levels in the ICU demonstrate consistent elevations as high as 80 to 90 decibels. Background noise can be above 50 decibels day and night.[25] New technology can be an additional source of noise, although several manufacturers attempt to provide equipment that lowers the total unit volume of sound.

Decades of studies consistently point to noise as a key aspect of the ICU environment. Noise was measured in two ICUs using a sound meter placed at the head of a patient's bed.[26] More than 50% of the noise in the environment was attributed to human behavior, with a mean sound level in the medical ICU of 84 decibels. Television and talking were some of the most frequent disruptive sounds for patients. Another study investigated the perceptions of 203 patients who filled out a questionnaire on discharge from the ICU and found that noise from talking and alarms was the most disruptive to sleep.[27] Sound peaks greater than 80 decibels are common in ICUs and are directly related to arousals from sleep.[28] Noise levels in ICUs have remained fairly unchanged despite the evolution of unit design. Newer thinking suggests that noise is not the only culprit in limiting sleep in the ICU, although it remains an important one.[29]

Lights and Color

Light is a powerful *zeitgeber*, or environmental synchronizer, that assists in entraining sleep by promoting the normal circadian cycle of sleep and wakefulness. Many critical care settings could benefit from more natural lighting and lights that are lowered during normal sleep times. In addition to natural lighting, providing a soothing view for the patient to look on instead of the ceiling or a hospital curtain may foster recovery. A classic study found that when a patient had a view of natural scenery and the outdoors, as opposed to viewing a brick wall, less pain medication was used and the hospital stay was shorter.[30] Other studies have demonstrated that impaired cognition occurs more often in windowless units than in those with windows.

In the hospital setting, artificial light is provided by fluorescent bulbs and tubes. If unshielded, this harsh light leads to visual fatigue and headaches. Glare reflected off environmental surfaces, such as glass, shiny metal, mirrors, and enameled or polished finishes, is troublesome to patients, especially elderly patients. Bright lights may be left on for many hours in ICUs, even when no direct patient care is being performed. Lack of control over artificial lighting is a source of frustration to critical care patients.

Interruptions in normal light–dark patterns can disrupt normal physiological processes. For example, artificial light exposure for as little as 20 minutes during a normal sleep cycle caused a drop in melatonin levels.[31] In addition, constant lighting and high-intensity light can lead to a complete disruption of the normal melatonin concentration rhythm. Melatonin secreted at night synchronizes the sleep–wake and dark–light cycles.[32] Patients with sepsis in an ICU are likely to have disrupted melatonin secretion not linked to the normal circadian pattern.[33] Therapy with melatonin can potentially increase sleep duration in critically ill patients.

The ideal ICU environment has windows with natural views, soothing artwork, and calm colors. The nurse and other health care providers have access to work and computer stations with glass soundproof partitions that permit proximity to the patient (for easy observation) while shielding the patient from noise. Equipment is selected for its low noise level. Stress created by unnecessary noise and light is diminished for the good of the patients, family, and staff. This vision may already be a reality in some institutions. For example, muted colors of beige, blue, and green were used to design a holistic nursing unit in a Minnesota Hospital.[34] Art on the walls depicts many different cultures and the peacefulness of nature. The goal of a more peaceful, healing ICU environment is possible to attain.

▲ Anxiety

For many, anxiety is an emotional state of apprehension in response to a real or perceived threat. Typically, the threat is associated with motor tension, increased sympathetic activity, and hypervigilance.

Causes of Anxiety

Any stressor that threatens a person's sense of wholeness, containment, security, and control can cause anxiety. Illness and injury are such stressors. Other common causes of anxiety include feelings of increased vulnerability and decreased security, which occurs when patients admitted to ICUs perceive a loss of control, a sense of isolation, and fear of death or loss of functionality. Anxiety, pain, and fear can all initiate or perpetuate the stress response. Left untreated or undertreated, anxiety can contribute to the morbidity and mortality of critically ill patients.

Anxiety occurs when people experience the following:

- Threat of helplessness
- Loss of control
- Sense of loss of function and self-esteem
- Failure of former defenses
- Sense of isolation
- Fear of dying

Assessment of Anxiety

Assessment of anxiety is challenging in the critical care population because of the severity of illness, barriers to communication, and altered cognitive states. However, most critical care nurses believe that assessment of anxiety is important. Multiple-item, self-report scales of anxiety may be used, but they have specific drawbacks in critical care areas, especially for patients receiving mechanical ventilation because of communication barriers.[1–3] According to many critical care nurses, the top five physiological and behavioral indicators of anxiety are agitated behavior, increased blood pressure, increased heart rate, verbalization of anxiety, and restlessness. Monitoring of these patient parameters is useful, but there is still a need for a reliable and comprehensive anxiety assessment tool.[10,16] Examples of nursing diagnoses associated with critical illness and injury can be seen in Box 2-1.

BOX 2-1 EXAMPLES OF NURSING DIAGNOSES

For the Patient With Critical Illness or Injury

- Grieving
- Anxiety
- Disturbed Body Image
- Impaired Verbal Communication
- Ineffective Coping
- Ineffective Denial
- Fear
- Hopelessness
- Risk for Loneliness
- Powerlessness
- Situational Low Self-Esteem
- Sleep Deprivation
- Spiritual Distress
- Readiness for Enhanced Spiritual Well-Being

▲ Nursing Interventions

In caring for the critically ill patient, the nurse helps the patient manage a multitude of stressors. Stress management includes not only physical and environmental stressors but also psychological stressors. This complex and labor-intensive process requires advanced assessment skills, adept manipulation of a variety of highly technological treatment strategies, and creativity in care and compassion.[35–38]

Creating a Healing Environment

Florence Nightingale, the founder of modern nursing, often wrote about the nurse's role in creating an environment to allow healing to occur. She emphasized holism in nursing—that is, caring for the whole person. In today's technological age, critical care nurses are challenged to create an environment of healing. These environments must allow critically ill patients to have their psychological needs as well as physical needs met. Manipulating the milieu may involve timing interventions to allow adequate sleep and rest, providing pain-relieving medication, playing music, or teaching deep-breathing exercises. As previously discussed, the physical environment of the ICU can be altered to create a more healing and restful environment.

Promoting Rest and Sleep

Sleep Assessment

Promotion of sleep and rest for critically ill patients begins with an understanding of sleep, the major environmental disruptions, and a sleep assessment. Sleep involves two very distinct types of brain activity: rapid eye movement (REM) sleep and non-REM sleep. These sleep stages are described in Box 2-2. Healthy adults progress through sleep stages in a specific order, from a light stage to a deeper stage, in 90-minute cycles. REM sleep increases later in the normal nighttime sleep patterns of most people, with morning naps containing primarily REM sleep. Specific sleep stages have a circadian rhythm and are controlled by brainstem mechanisms.[39]

BOX 2-2 Stages and Characteristics of Sleep

Stage 1	Transitional stage between wakefulness and sleep
	Relaxed state where person is somewhat aware of surroundings
	Involuntary muscle jerking that may waken the person
	Normally lasts only minutes
	Easily aroused
	Constitutes only about 5% of total sleep
Stage 2	Beginning of sleep
	Arousal occurs with relative ease
	Constitutes 50% to 55% of sleep
Stage 3	Depth of sleep increased and arousal increasingly difficult
	Constitutes about 10% of sleep
Stage 4	Greatest depth of sleep (*delta sleep*)
	Arousal from sleep difficult
	Physiological changes in the body—slow brain waves on electroencephalogram; decreased pulse and respiratory rates; decreased blood pressure; relaxed muscles; slow metabolism and low body temperature
	Constitutes about 10% of sleep
Stage REM	Sleep with vivid dreaming (rapid eye movement [REM])
	REM, fluctuating heart and respiratory rates, fluctuating blood pressure
	Skeletal muscle tone lost
	Most difficult to arouse
	Duration of REM sleep increased with each cycle and averages 20 minutes
	Constitutes about 20% to 25% of sleep

Adapted from Taylor C, Lillis C, LeMone P: Fundamentals of Nursing: The Art and Science of Nursing Care, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008.

Although sleep patterns are very individual, most patients can tell when they feel rested and have had a "good night's sleep." Unfortunately, this is a rare occurrence in the hospital. Sleep, once thought to be a quiescent state, actually involves physiological activation while the brain and body rejuvenate themselves. Sleep is often appreciated only when it has been "lost" and is typically taken for granted by health care providers, who often do not make sleep a priority for patients. Often in the ICU, sleep is severely fragmented and nonconsolidated,[40] with those receiving mechanical ventilation experiencing some of the worst sleep disruption.[41]

Sleep deprivation in patients in ICUs can have cumulative effects and lead to altered cognition, confusion, impaired wound healing, and the inability to wean from the ventilator because of muscle fatigue and carbon dioxide retention. The clinical significance is not fully appreciated because the relationship between poor sleep and recovery is unknown. However, promotion of sleep for patients is not only a humanistic intervention but may also be a life-sustaining one.

Sleep in patients with critical illness is greatly disrupted.[42] Many drugs interrupt sleep in critically ill patients.[43] Over four decades, researchers have noted that patients in ICUs have frequent awakenings, little to no REM sleep, shorter total sleep time than at home, and perceived poor quality of sleep.[29,44,45] Care interventions, including unnecessary baths between the hours of 2:00 and 5:00 AM, have disturbed the sleep of critically ill patients on a routine

basis.[46] A poor sleep pattern is characteristic of all age groups, from elderly to pediatric patients.[47] The impact of sleep disruption on the clinical outcome of ICU patients is not fully known. However, patients often report that sleep disruption is one of the most unpleasant aspects of their illness.

The patient's own report of sleep quality is the best measure of sleep adequacy, although this is inherently difficult when the patient is receiving mechanical ventilation. Similar to pain assessment, only the person can make the assessment: "I slept well" or "I didn't sleep at all." Monitoring brain waves by polysomnography is the gold standard for measuring patient sleep but is not feasible as a standard measure in the ICU.[43] If self-report of sleep is unobtainable, systematic observation of patients by nurses has been somewhat valid and reliable.[48] In addition, a visual analog scale is recommended for select patients at high risk for sleep disruption owing to extended stay in the ICU.[49] Wrist actigraphy is used as a research tool to continuously monitor activity and rest but may overestimate sleep in sedentary and elderly people.[43]

Promoting Sleep

Despite four decades of research into reasons why patients do not sleep in the ICU, little is done to facilitate what patients often rate as their number one priority after pain relief: sleep. Box 2-3 outlines strategies that are most often recommended to promote sleep. The challenging environment dictates that the nurse first is sensitive to the patient's needs and attuned to the environment and then has the tools and resources to implement sleep promotion. An old idea is the 5-minute backrub. The concept of using back massage to ease patients to sleep seems intuitive; however, until recently, it has never been systematically studied. In a study of 69 patients in an ICU, a 5-minute slow back massage (or *effleurage*) promoted increased sleep by 1 hour, compared with a control group.[50] If back massage were a hypnotic medication, it would be routinely ordered for ICU patients. The effective backrub was not the cold application of lotion and a quick one-handed massage while holding the patient on his side with the other hand, but rather a soothing, slow-stroke massage, in which the nurse first became centered and truly present with the patient.

The role of the nurse as a gatekeeper to protect patient sleep time will be more difficult to fulfill as patient:nurse ratios escalate but must remain a priority. According to the 2012 clinical practice guidelines for sedatives and analgesics in the critically ill adult, developed by a multidisciplinary team of physicians, pharmacists, and nurses, sleep promotion should include optimization of the patient's environment through the use of strategies to control light and noise, to cluster patient care activities, and to decrease stimuli at night in order to protect the sleep cycle.[51] One intervention is to implement a sleep protocol that institutionalizes the importance of sleep,[52] blocks sleep times, and truly controls the environment. In several studies, having a "quiet time" helped improve the opportunity for sleep.[53,54] Earplugs and eyemasks can be considered[55] as well as staff behavior modification programs for limiting noise.[56]

Another innovation in sleep promotion, which decreased noise and promoted sleep, involved moving all routine chest radiographs from 3:00 AM to 10:00 PM.[57] Although this increased the workload of the radiology department on the evening shift, patient and nurse satisfaction rose dramatically. All health care providers need to be aware of the importance of restorative sleep and the impact on a patient's well-being and work toward a "sleep-friendly ICU."[58] Sleep of the health care provider is also an important aspect of the healing dyad in the ICU. Nurses who work nights are routinely sleep deprived and may have young children to care for at home or school to attend when the next day begins. The growing interest in patient safety makes the work patterns of nurses, including 12-hour shifts and overtime, a focus of study.[59] Nurses' vigilance to patient needs is threatened with longer work hours and increased risk for error. Antidotes for working at night include scheduling of shifts to phase-advance the sleep cycle (ie, going from days to evenings to nights), eating healthy snacks, using bright lights during a shift away from patient rooms, and obtaining regular exercise.[59] Compassionate caring includes the nurse caring for self to meet better the demands of patients, families, and colleagues.

BOX 2-3 NURSING INTERVENTIONS

For Promoting Sleep

- Provide large clocks and calendars.
- Block sleep times.
- Provide a quiet time.
- Have the patient use earplugs.
- Assess sleep time and quality of sleep by asking the patient when possible.
- Provide opportunity for music therapy.
- Provide a 5-minute backrub before sleep.
- Consider using white noise or ocean sounds.
- Eliminate pain.
- Position patient for comfort with pillows.
- Stop the practice of bathing patients in the middle of the night for the convenience of the nursing staff.
- Titrate environmental stimuli: turn down lights, turn down alarms, and decrease noise from television and talking.
- Evaluate the need for nursing care interruptions.
- At bedtime, provide information to lower anxiety. Do a review of the day and remind patient of progress made toward recovery, then add what to expect for the next day.
- Institute "PM Care" back to basics, brushing teeth, and washing face before "bedtime."
- Allow family to be with the patient.
- Use relaxation techniques and guided imagery.
- Ensure patient privacy: close door or pull curtains.
- Post sign at designated times: "Patient Sleeping."

Fostering Trust

Almost every nurse in critical care can relate stories of special bonds that formed with individual patients and families. They can describe special situations where a trusting relationship developed and they made a difference in the patient's recovery or even dignified death. In contrast, research has shown that when patients mistrust their care givers, they are more anxious and more vigilant of staff behaviors and lack the feeling of safety and security. The goals, then, are to display a confident, caring attitude, demonstrate technical competence, and develop effective communication techniques that will foster the development of a trusting relationship. Communication can be especially difficult with mechanically ventilated and intubated patients. Use of nonverbal

signals, writing pads, or commercial communication boards can help make communication of basic needs easier.[60]

Providing Information

Besides the need to feel safe, critically ill patients identify the need for information as having a high priority.[38,61,62] This need to know involves all aspects of care. Patients need to know what is happening at the moment. They also need to know what will happen to them, how they are doing, and what they can expect. Many patients also need frequent explanations of what happened to them. These explanations reorient them, sort out sequences of events, and help them distinguish real events from dreams or hallucinations. Anxiety can be greatly relieved with simple explanations. Consider the patient, for example, who was being weaned from the ventilator who just needed reassurance that if he did not breathe, the machine would do it for him. Families, too, have identified the need for information as a high priority. This is followed closely by the need to have hope. Most families identify physicians as the primary source for information. It is important for nurses to be mindful of patient confidentiality issues when speaking to family members. Nurses should have the patient's permission before giving confidential medical information to family members. If that is not possible because of the patient's condition, a family spokesperson should be identified as the person who may receive confidential information. This information should be recorded in the patient's medical record.

Allowing Control

Nursing measures that reinforce a sense of control help increase the patient's autonomy and reduce the overpowering sense of a loss of control. The nurse can help the patient exert more control over his or her environment in the following ways:

- Providing order and predictability in routines
- Using anticipatory guidance
- Allowing the patient to make choices whenever possible
- Involving the patient in decision making
- Providing information and explanation for procedures

Providing order and predictability allows the patient to anticipate and prepare for what is to follow. Perhaps it creates only a mirage of control, but anticipatory guidance keeps the patient from being caught off guard and allows the mustering of coping mechanisms. Allowing small choices when the patient is willing and ready increases the patient's feeling of control over the environment. Would the patient prefer to lie on his or her right or left side? In which arm should the IV line be placed? What height is preferred for the head of the bed? Does the patient want to cough now or in 20 minutes after pain medication? Any decisions that afford the patient a certain amount of control and predictability are important. These small choices may also help the patient accept lack of control during procedures that involve little choice.

Practicing Cultural Sensitivity

Interventions for individual patients must be contextually based and culturally sensitive. Transcultural nursing refers to a formal area of study and practice that focuses on providing care that is compatible with the cultural beliefs, values, and lifestyles of people. A cultural assessment includes the patient's usual response to illness as well as his or her cultural norms, beliefs, and world views. Because individual responses and values may vary within the same culture, the patient should be recognized as an individual person within the cultural context. Exploring the meaning of the critical event with the patient, family members, and significant others may give clues to the patient's perception of what is happening. In addition, the nurse may ask if there is a particular ethnic or religious group with which the patient identifies and if there is anything the nurses may do to provide care that is sensitive to individual values or norms while the patient is hospitalized. Awareness and acceptance are the heart of cultural competence. Incorporating complementary therapies that are culturally based may have a role in a person's treatment plan. Careful exploration of traditional or complementary therapies needs to be performed before implementation to avoid any harm from interaction of therapies. This is especially true with use of herbal therapies or nutritional supplements that may have multiple ingredients with unknown or undesired side effects or interactions.

Presencing and Reassurance

Presence, or just "being there," can in itself be a meaningful strategy for alleviating distress or anxiety in the critically ill patient. *Presencing* is the therapeutic use of self, adopting a caring attitude, and paying attention to a person's needs. However, this presence implies more than just physical presence. It means giving one's full attention to the person, focusing on the person, and practicing active listening. When a nurse uses presence, the focus is not on a task or outside thoughts. Energy and attention are directed at the patient and his or her needs or feelings. This means one makes a conscious effort to use all of one's capacity, including eyes, voice, energy, and touch, in a more intentionally healing way. Reassurance can be provided to the patient in the form of presencing and caring touch. Reassurance can also be verbal. Verbal reassurance can be effective for patients if it provides realistic encouragement or clarifies misconceptions. However, verbal reassurance is not valuable if it prevents a patient from expressing his or her emotions or stifles the need for further dialogue. Reassurance is intended to reduce fear and anxiety and evoke a calmer, more passive response. It is best directed at patients expressing unrealistic or exaggerated fears.

Cognitive Techniques

Techniques that have evolved from cognitive theories of learning may help anxious patients and their families. They can be initiated by the patient and do not depend on complex insight or understanding of one's own psychological makeup. They can also be used to reduce anxiety in a way that avoids probing into the patient's personal life. Furthermore, the patient's friends and family members can be taught these techniques to help them and the patient reduce tension.

Internal Dialogue

Highly anxious people are most likely giving themselves messages that increase or perpetuate their anxiety. These messages

are conveyed in one's continuously running "self-talk," or internal dialogue. The patient in the ICU may be silently saying things such as, "I can't stand it in here. I've got to get out." Another unexpressed thought might be, "I can't handle this pain." By asking the patient to share aloud what is going on in this internal dialogue, the nurse can bring to awareness the messages that are distracting the patient from rest and relaxation. Substitute messages should be suggested to the patient. It is important to ask the patient to substitute rather than delete messages because the internal dialogue is continuously operating and will not turn off, even if the patient wills it to do so. Therefore, asking the patient to substitute constructive, reassuring comments is more likely to help the patient significantly reduce his or her tension level. Comments such as, "I'll handle this pain just 1 minute at a time" or "I've been in tough spots before, and I am capable of making it through this one!" automatically reduce anxiety and help the patient shape coping behaviors accordingly. Any message that enhances the patient's confidence, sense of control, and hope and puts him or her in a positive, active role, rather than the passive role of victim, increases the patient's sense of coping and well-being.

The nurse helps the patient develop self-dialogue messages that increase:

- Confidence
- Sense of control
- Ability to cope
- Optimism
- Hope

External Dialogue

A similar method can be applied to the patient's external conversation with other people. By simply requiring patients to speak accurately about themselves to others, the same goals can be accomplished. For example, patients who exclaim, "I can't do anything for myself!" should be asked to identify the things that they are able to do, such as lifting their own bodies, turning to one side, making a nurse feel good with a rewarding smile, or helping the family understand what is happening. Even the smallest movement in the weakest of patients should be acknowledged and claimed by the patient. This technique is useful in helping patients correct their own misconceptions of themselves and the way others see them. This reduces patients' sense of helplessness and therefore their anxiety.

Cognitive Reappraisal

This technique asks the patient to identify a particular stressor and then modify his or her response to that stressor. In other words, the patient reframes his or her perception of the stressor in a more positive light so that the stimulus is no longer viewed as threatening. The patient is given permission to take personal control of responses to the stimulus. This technique may be combined with guided imagery and relaxation training.

Guided Imagery and Relaxation Training

Guided imagery and relaxation training are two useful techniques that can be taught to the patient to help reduce tension. The nurse can encourage the patient to imagine either being in a very pleasant place or taking part in a very pleasant experience. The patient should be instructed to focus and linger on the sensations that are experienced. For example, asking the patient questions such as, "What colors do you see?", "What sounds are present?", "How does the air smell?", "How does your skin feel?", or "Is there a breeze in the air?" helps increase the intensity of the fantasy and thereby promotes relaxation through mental escape.

Guided imagery also can be used to help reduce unpleasant feelings of depression, anxiety, and hostility. Patients who must relearn life-sustaining tasks, such as walking and feeding themselves, can use imagery to prepare mentally to meet the challenge successfully. In these instances, patients should be taught to visualize themselves moving through the task and successfully completing it. If this method seems trivial or silly to the patients, they can be reminded that this method demands concentration and skill and is commonly used by athletes to improve their performance and to prepare themselves mentally before an important event. Guided imagery is a way of purposefully diverting or focusing the patients' thoughts and has been shown to empower patients, improving their satisfaction and well-being.[63,64] The nurse can also use techniques that induce deep muscle relaxation to help the patient decrease anxiety. Deep muscle relaxation may reduce or eliminate the use of tranquilizing and sedating drugs. In progressive relaxation, the patient is first directed to find as comfortable a position as possible and then to take several deep breaths and let them out slowly. Next, the patient is asked to clench a fist or curl toes as tightly as possible, to hold the position for a few seconds, and then to let go while focusing on the sensations of the releasing muscles. The patient should practice this technique, beginning with the toes and moving upward through other parts of the body—the feet, calves, thighs, abdomen, chest, and so on. This procedure is done slowly while the patient gives nonverbal signals (eg, lifting a finger) to indicate when each new muscle mass has reached a state of relaxation. Extra time and attention should be given to the back, shoulders, neck, scalp, and forehead because many people experience physical tension in these areas.

Once a state of relaxation is achieved, the nurse can suggest that the patient fantasize or sleep as deeply as the patient chooses. The patient must be allowed to select and control the depth of relaxation and sleep, especially if the fear of death is prominent in the patient's mind. A moderately dark room and a soft voice facilitate relaxation. Asking the patient to relax is frequently nonproductive compared with directing the patient to release a muscle mass actively, let go of tension, or imagine tension draining through the body and sinking deeply into the mattress. Again, the patient is assisted to take an active rather than passive role by the nurse's careful use of language. In addition, a number of commercially available recordings can be used to assist in guided imagery and relaxation

Deep Breathing

When acutely anxious, the patient's breathing patterns may change, and the patient may hold his or her breath. This could be physically and psychologically detrimental. Teaching

diaphragmatic breathing, also called abdominal breathing, to the patient may be useful as both a distraction and a coping mechanism. Diaphragmatic breathing can be taught easily and quickly to the preoperative patient or to a patient experiencing acute fear or anxiety. The patient may be asked to place a hand on the abdomen, inhale deeply through the nose, hold briefly, and exhale through pursed lips. The goal is to have the patient push out his own hand to demonstrate the deep breath. The nurse may demonstrate the technique and perform it along with the patient until the patient is comfortable with the technique and is in control. The mechanically ventilated patient may be able to modify this technique by concentrating on breathing and on pushing out the hand. However, a mechanically ventilated patient experiencing severe agitation may not be able to respond to this technique.

Music Therapy

Music therapy has been used in the critical care environment as a strategy to reduce anxiety, provide distraction, and promote relaxation, rest, and sleep.[65–68] The patient receives a choice of specially recorded audiotapes and a set of headphones. Usually, music sessions are 20 to 90 minutes long, once or twice daily. Music selections may vary by individual taste, but the most commonly used selections have a tempo of 60 to 70 beats; a simple, direct musical rhythm; and a low-pitched sound with primarily a string composition. Most patients prefer music that is familiar to them. Many ICUs maintain a CD library with a variety of genres to satisfy patient choices. Patients or family members are also encouraged to bring in their own MP3 players with the patient's favorite music selections already programmed. This intervention has proved effective for relaxing mechanically ventilated patients. Some hospitals have used loudspeakers to play music overhead in their ICUs, emergency rooms, and postanesthesia units to promote a healing environment.

Humor

A good belly laugh produces positive physiological and psychological effects. Laughter can increase the level of endorphins, the body's natural pain relievers, which are released into the bloodstream. Laughter can relieve tension and anxiety and relax muscles. Humor is a universal emotion that can help patients cope with stressful experiences. The use of humor by nurses in critical care, which can be spontaneous or planned, can help reduce procedural anxiety or provide distraction. Once again, the humor must be compatible with the context in which it is offered and with the person's cultural perspective. Many nurses report using humor cautiously after they have established a rapport with the individual. Nurses also report that they are able to take cues from the patient and visitors regarding the appropriate use of humor. Patients have reported that nurses who have a good sense of humor are more approachable and easier to talk with. Humor that is lighthearted, witty, and, of course, timed just right, is the most well received by adults.

Humor therapy has been used successfully in a variety of treatment settings, including pediatrics, surgery, oncology, and palliative care. In an effort to incorporate the positive effects of humor into health care settings, some institutions have developed humor resource rooms or mobile humor carts. These provide patients with a variety of lighthearted reading materials, videotapes, and audiotapes. Also included on the cart may be games, puzzles, and magic tricks. Some nurses have created their own portable therapeutic humor kits, comic strips, jokes, or humorous stories to which their patients can relate.

Use of humor by patients may help them reframe their anxiety and channel their energy toward feeling better. Some patients link humor with spirituality, noting that humor helped them cope better with serious illness and develop a closer relationship with God. In addition, appropriate use of humor can relieve stress among critical care nurses who work in complex, challenging environments with significant economic pressures.

Massage, Aromatherapy, and Therapeutic Touch

Massage is the purposeful stroking and kneading of muscles with the goal of providing comfort and promoting relaxation.[69–72] Nurses have traditionally used effleurage for backrubs for patient comfort. Effleurage uses slow, rhythmic strokes from distal to proximal areas of long muscles such as the back or extremities. Consistent, firm, yet flexible hand pressure is applied with all parts of the hand to conform to body contours. Lotion may be used to decrease friction and add moisture. Massage has been effective at reducing anxiety and promoting relaxation. Patient selection is an important consideration when electing massage as a therapeutic intervention. Patients who are hemodynamically unstable, for example, would not be appropriate candidates. In addition, nurses require additional training in massage therapy to effectively incorporate more advanced massage techniques such as pétrissage or pressure points into plans of care for critically ill patients.

Massage can be combined with aromatherapy, in which massage is carried out with scented oils or lotions. Some scents have been associated with specific beneficial effects. For example, lavender oil and other floral scents are said to be relaxing, citrus oils to be positive mood enhancers, and peppermint oils to be promoters of mental stimulation. Aromatherapy can also be accomplished with use of scented bath water or unlit scented candles placed in the room.

Therapeutic touch is a set of techniques in which the practitioner's hands move over a patient in a systematic way to rebalance the patient's energy fields. An important component of therapeutic touch is compassionate intent on the part of the healer. Therapeutic touch as a complementary therapy has been used successfully in acute care settings to decrease anxiety and promote a sense of well-being. It is a foundational technique of healing touch. Healing touch involves a number of full-body and localized techniques to balance energy fields and promote healing. Implementation of healing touch therapy involves a formal educational program for healers, and its potential benefits are under active investigation.

Meridian Therapy

Complementary and alternative medicine describes an array of nontraditional healing approaches. Meridian therapy refers to therapies that involve an acupoint, such

as acupuncture, acupressure, and the activation of specific sites with electrical stimulation and low-intensity laser.[73–76] Meridian therapy originates from traditional Chinese medicine. Meridians are complex energy pathways that integrate into intricate patterns. These pathways contain sensitive energy points that are amenable to stimulation to relieve blockages that affect various physiological functions. Research has demonstrated the effectiveness of meridian therapy for pain relief, postoperative nausea, and other functions. Currently, research is underway to validate acupoint sites. Meridian therapy should be performed only by professionals with specialized training.

Animal-Assisted Therapy

The human–animal bond has been well documented. Pet ownership has been linked to higher levels of self-esteem and physical health. Pet therapy (or, more broadly, animal-assisted therapy) has had measurable benefits for schoolchildren and residents of nursing homes. More recently, this concept has been introduced to the acute and critical care settings with positive results. Some hospitals have developed guidelines for pet visitation; a patient's leashed pet may be brought to the hospital to visit with the patient. This type of program has been well received by patients and staff. However, it does require coordination between staff and family members. Pets must in good health, have up-to-date vaccinations, and be well-behaved in unfamiliar environments. The handler must be familiar with the pet and agree to follow hospital guidelines regarding time limits (generally 20 to 30 minutes per visit). A private patient room or visiting room is required. It is recommended that pets be leashed and wear a "shirt," which reduces shedding and identifies the pet. In some hospitals, a formal program exists in which volunteer owner–dog teams visit patients in the hospital on a variety of units. In addition, one hospital reported patients' delight in having fish aquariums placed in their rooms while they were awaiting heart transplantation.

Fostering Spirituality and Healing

Caring in nursing includes recognition and support of the spiritual nature of human beings. Spirituality refers to the realm of invisible and intangible factors that influence our thoughts and behaviors. This includes religious beliefs and extends beyond them. When people sense power and influence outside of time and physical existence, they are said to be experiencing the metaphysical aspects of spirituality.

Spirituality, which includes one's system of beliefs and values, can be defined as the *means or* manner by which persons seek meaning in their lives and experience transcendence-connectedness to that which is beyond the self.[77–79] Intuition and knowledge from unknown sources and origins of unconditional love and belonging typically are viewed as spiritual power. A sense of universal connection, personal empowerment, and reverence for life also pertains to the existence of spirituality. These elements also may be viewed as benefits of spirituality. Spirituality includes the following:

- Religion
- Beliefs and values
- Intuition
- Knowledge from the unknown
- Unconditional love
- A sense of belonging
- A sense of connection with the universe
- Reverence for life
- Personal empowerment

Critical care patients and their families frequently find strength in prayer, which is used by people of many faiths. Research on prayer and health has demonstrated prayer to be a powerful tool to help patients cope with difficult situations, chronic illnesses, and impending death.

Nursing goals related to spirituality include the recognition and promotion of patients' spiritual sources of strength. By allowing and supporting patients to share their beliefs about the universe without disagreement, nurses help patients recognize and draw on their own sources of spiritual courage. Recognition of the unique spiritual nature of each patient is thought to assist personal empowerment and healing.

Nurses who find their own spiritual values in religion must acknowledge and respect that nonreligious people may also be spiritual and experience spirituality as a life force. Regardless of personal views, the nurse is obligated to assess patients' spiritual belief systems and assist them to recognize and draw on the values and beliefs already in existence for them.

Furthermore, critical illness may deepen or challenge existing spirituality. Patients have reported deeper faith after coping with critical illness. During these times, it may be useful for the nurse or family to call on a spiritual or religious leader, hospital chaplain, or pastoral care representative to help the patient make meaningful use of the critical illness experience. Patients may also gain support from members of their congregation or family. It is important for nurses to assess and recognize the spiritual nature of their patients, to allow time for spiritual and religious practices, and to make referrals when needed. Referrals may be made to the hospital chaplain or to a clergy person of the patient's choice.

▲ Restraints in Critical Care

Restraints in critical care include any drug or device that is used to restrict the patient's mobility and normal access to his or her body. Physical restraints may include limb restraints, mittens with ties, vests or waist restraints, geriatric chairs, and side rails. Side rails are considered a restraint if used to limit the ability of the patient to get out of bed rather than to help him or her sit or stand up.

Chemical Restraint

Chemical restraints refer to pharmacological agents that are given to patients as discipline or to limit disruptive behavior. Medications that have been used for behavior control include, but are not limited to, psychotropic drugs such as haloperidol, sedative agents such as benzodiazepines (eg, lorazepam, midazolam), or the anticholinergic antihistamine, diphenhydramine. This definition does not apply to medications that are given to treat a medical condition. The use of sedative, analgesic, and anxiolytic medications is an important adjunct in the care of the critically ill patient.

BOX 2-4 Summary of Care Standards Regarding Physical Restraints

Initiating Restraints

- Restraints require the order of a licensed independent practitioner who must personally see and evaluate the patient within specified time periods.
- Restraints are used only as an emergency measure or after treatment alternatives have failed. (Treatment alternatives and patient responses are documented.)
- Restraints are instituted by staff members who are trained and competent to use restraints safely. (A comprehensive training and monitoring program must be in place.)
- Restraint orders must be time limited. (A patient must not be placed in a restraint for longer than 24 hours, with reassessment and documentation of continued need for restraint at more frequent intervals.)
- Patients and families are informed about the reason/rationale for the use of the restraint.

Monitoring Patients in Restraints

- The patient's rights, dignity, and well-being are to be protected.
- The patient will be assessed every 15 minutes by trained and competent staff.
- The assessment and documentation must include evaluation of adequate nutrition, hydration, hygiene, elimination, vital signs, circulation, range of motion, injury due to the restraint, physical and psychological comfort, and readiness for discontinuance of the restraint.

Care must be taken to provide adequate comfort for patients experiencing life-threatening illnesses and a variety of noxious interventions. It is desirable to use the least amount of medication as feasible to achieve the goals of patient care because all medications have potential side effects and adverse reactions. Patients must be continually assessed for adequacy of comfort. Behaviors that seem to indicate pain may actually indicate a change in the patient's physiological status. Agitation, for example, may be a sign of hypoxemia. Caution must be exercised when using as-needed (PRN) medications to reduce pain and promote comfort. Without consistency in assessment, goal setting, and administration, PRN dosing may inadvertently lead to overmedication or undermedication in the critically ill patient. In addition, these medications can have rebound effects if abruptly withdrawn. Weaning a patient from analgesic or sedative medication may be as important as weaning a patient from a mechanical ventilator. Many ICUs incorporate assessment tools for patient comfort on their daily flow sheets.

Physical Restraints

Historically, physical restraints have been used for patients in critical care to prevent potentially serious disruptions in patient care through accidental dislodgment of endotracheal tubes or lifesaving IV lines and other invasive therapies. Other reasons that have been cited for use of restraints include fall prevention, behavior management, and avoidance of liability suits resulting from patient injury. However, research related to restraint use, especially in elderly patients, has demonstrated that these reasons, although well intentioned, are seldom valid.[80–84] Patients who are restrained have been shown to have more serious injuries secondary to falls as they "fight" the device that limits their freedom. In addition, there are reportedly a greater number of lawsuits related to improper restraint use than to injuries sustained when restraints were not used. Critically ill, intubated patients have been known to self-extubate despite the use of soft wrist restraints.

The forced immobilization that results from restraining a patient can prolong a patient's hospitalization by contributing to skin alterations, loss of muscle tone, impaired circulation, nerve damage, and pneumonia. Restraints have been implicated in accelerating patients' levels of agitation, resulting in injuries, such as fractures or strangulation.

Standards on physical restraint use are published and monitored by the Joint Commission and the Centers for Medicare and Medicaid Services. A summary of these standards is given in Box 2-4. These standards may be viewed on the websites of the respective agencies. Many hospitals have revised their policies, procedures, and documentation of the use of restraints to comply with the most recent revision of these standards. Clinical practice guidelines have been published by the Society of Critical Care Medicine.

BOX 2-5 Alternatives to Physical Restraints

Modifications to Patient Environment

- Keep the bed in the lowest position.
- Minimize the use of side rails to what is needed for positioning.
- Optimize room lighting.
- Activate bed and chair exit alarms where available.
- Remove unnecessary furniture or equipment.
- Ensure that the bed wheels are locked.
- Position the call light within easy reach.

Modifications to Therapy

- Frequently assess the need for treatments and discontinue lines and catheters at earliest opportunity.
- Toilet patients frequently.
- Disguise treatments, if possible (eg, keep intravenous [IV] solution bags behind patient's field of vision, apply loose stockinette or long-sleeved gown over IV sites).
- Meet physical and comfort needs (eg, skin care, pain management, positioning wedges, hypoxemia management).
- When possible, guide the patient's hand through exploration of the device or tube, and explain the purpose, route, and alarms of the device or tube.
- Mobilize the patient as much as possible (eg, consider physical therapy consult, need for cane or walker, reclining chairs, or bedside commode).

Involvement of the Patient and Family in Care

- Allow patient choices and control when possible.
- Family members or volunteers can provide company and diversionary activities.
- Consider solitary diversionary activities (eg, music, videos or television, books on tape).
- Ensure that the patient has needed glasses and hearing aids.

Therapeutic Use of Self

- Use calm, reassuring tones.
- Introduce yourself and let the patient know he or she is safe.
- Find acceptable means of communicating with intubated or nonverbal patients.
- Reorient patients frequently by explaining treatments, devices, care plans, activities, and unfamiliar sounds, noises, or alarms.

Alternatives to Restraints

What, then, is the well-meaning nurse to do when a patient is experiencing confusion or delirium and is pulling at his or her lifesaving devices and tubes? Remember that physical restraint is the last resort, to be used only when the patient is a danger to himself or others and when other methods have failed. Restraints may actually potentiate the dangerous behavior. Rather, the nurse should attempt to identify what the patient is feeling or experiencing. What is the meaning behind the behavior? Is the patient cold? Does the patient itch? Is the patient in pain? Does the patient know where he or she is and why he or she is there? Sometimes addressing the patient's needs or concerns and reorienting the patient is all that is needed to calm him or her. Other interventions may include modifying the patient's environment, providing diversionary activities, allowing the patient more control or choices, and promoting adequate sleep and rest (Box 2-5, p. 25). Some hospitals have instituted restraint protocols and decision trees to help nurses with assessment and care of patients in restraints.

▲ Clinical Applicability Challenges

SHORT ANSWER QUESTIONS

1. Anxiolytic medication has been used to assist the intubated patient cope with the stressor of being on a mechanical ventilator. Please describe a nonpharmacological intervention that may be employed in this situation.
2. An elderly Arab-American female recovering from emergency bowel surgery has indicated that she only wants female nurses to care for her. How should this request be handled?

References

1. Happ MB, Garrett K, Thomas DD, et al: Nurse-patient communication interactions in the intensive care unit. Am J Crit Care 20(2): e28–e40, 2011
2. Khalaila R, Zbidat W, Anwar K, et al: Communication difficulties and psychoemotional distress in patients receiving mechanical ventilation. Am J Crit Care 20(6):470–479, 2011
3. Samuelson KA, Corrigan I: A nurse-led intensive care after-care programme-development, experiences and preliminary evaluation. Nurs Crit Care 14(5):243–263, 2009
4. Field K, Prinjha S, Rowan K: One patient amongst many: A qualitative analysis of intensive care unit patients' experiences of transferring to the general ward. Crit Care 12(1):R21, 2008
5. Hinkle JL, Fitzpatrick E, Oskrochi GR: Identifying the perception of needs of family members visiting and nurses working in the intensive care unit. J Neuroscience Nurs 41(2):85–91, 2009
6. McAdam JL, Dracup KA, White DB, et al: Symptom experiences of family members of intensive care unit patients at high risk for dying. Crit Care Med 38(4):1078–1085, 2010
7. Bauer ME, Jeckel CM, Luz C: The role of stress factors during aging of the immune system. Ann N Y Acad Sci 1153:139–152, 2009
8. Costa-Pinto FA, Palermo-Neto J: Neuroimmune interactions in stress. Neuroimmunomodulation 17(3):196–199, 2010
9. Dhabhar FS: Enhancing versus suppressive effects of stress on immune function: Implications for immunoprotection and immunopathology. Neuroimmunomodulation 16(5):300–317, 2009
10. Dragos D, Tanasescu MD: The effect of stress on the defense systems. J Med Life 3(1):10–18, 2010
11. Gouin JP, Kiecolt-Glaser JK: The impact of psychological stress on wound healing: Methods and mechanisms. Immunol Allergy Clin North Am 31(1):81–93, 2011
12. Heffner KL: Neuroendocrine effects of stress on immunity in the elderly: Implications for inflammatory disease. Immunol Allergy Clin North Am 31(1):95–108, 2011
13. Ho RC, Neo LF, Chau AN, et al: Research on psychoneuroimmunology: Does stress influence immunity and cause coronary artery disease? Ann Acad Med 39(3):191–196, 2010
14. Lowry SF: The stressed host response to infection: The disruptive signals and rhythms of systemic inflammation. Surg Clin North Am 89(2): 311–326, 2009
15. Lusk B, Lash AA: The stress response: Psychoneuroimmunology and stress among ICU patients. Dimens Crit Care Nurs 24(1):25–31, 2005
16. Marques AH, Silverman MN, Sternberg EM: Evaluation of stress systems by applying noninvasive methodologies: Measurements of neuroimmune biomarkers in the sweat, heart rate variability and salivary cortisol. Neuroimmunomodulation 17(3):205–208, 2010
17. Segerstrom SC: Resources, stress, and immunity: An ecological perspective on human psychoneroimmunology. Ann Behav Med 40(1): 114–125, 2010
18. Stojanovich L: Stress and autoimmunity. Autoimmun Rev 9(5):A271–A276, 2010.
19. Buchman TG: Stress and the biology of the responses. In: Albert RK, Slutsky AS, Ranieri VM, et al (eds): Clinical Critical Care Medicine. Philadelphia, PA: Mosby Elsevier, 2006
20. Caine RM: Psychological influences in critical care: Perspectives from psychoneuroimmunology. Crit Care Nurse 23(2):60–70, 2003
21. Hadley JS, Hinds CJ: Anabolic strategies in critical illness. Curr Opin Pharmacol 2:700–707, 2002
22. Fairman J, Lynaugh JE: Critical care nursing: A history. Philadelphia, PA: University of Pennsylvania Press, 1998
23. Day L: Healing environments and the limits of empirical evidence. Am J Crit Care 16:86–89, 2007
24. Molter N: Environmental design and strategies to promote healing. In: Molter N (ed): AACN Protocols for Practice: Creating Healing Environments, 2nd ed. Sudbury, MA: Jones and Bartlett Publishers, 2007, pp 1–28.
25. Christensen M: Noise levels in a general intensive care unit: A descriptive study. Crit Care Nurse 12:188–197, 2007
26. Kahn DM, Cook TE, Carlisle CC, et al: Identification and modification of environmental noise in an ICU setting. Chest 114:535–540, 1998

27. Freedman NS, Kotzer N, Schwab RJ: Patient perception of sleep quality and etiology of sleep disruption in the intensive care unit. Am J Respir Crit Care Med 159(4 Pt 1):1155–1162, 1999
28. Aaron JN, Carlisle CC, Carskadon MA, et al: Environmental noise as a cause of sleep disruption in an intermediate respiratory care unit. Sleep 19:707–710, 1996
29. Gabor JY, Cooper AB, Crombach SA, et al: Contribution of the intensive care unit environment to sleep disruption in mechanically ventilated patients and healthy subjects. Am J Respir Crit Care Med 167:706–715, 2003
30. Ulrich RS: View through a window may influence recovery from surgery. Science 224:420–421, 1984
31. Vinall PE: Design technology: What you need to know about circadian rhythms in healthcare design. J Healthc Design 9:141–144, 1997
32. Cajochen C, Krauchi K, Wirz-Justice A: Role of melatonin in the regulation of human circadian rhythms and sleep. J Neuroendocrinol 15: 432–437, 2003
33. Mundigler G, Delle-Karth G, Koreny M, et al: Impaired circadian rhythm of melatonin secretion in sedated critically ill patients with severe sepsis. Crit Care Med 30:536–540, 2002
34. Horrigan B: Region's hospital opens holistic nursing unit. Altern Ther Health Med 6(4):92–93, 2000
35. Mundy CA: Assessment of family needs in neonatal intensive care units. Am J Crit Care 19(2):156–163, 2010
36. Papathanassoglou ED: Psychological support and outcomes for ICU patients. Nurs Crit Care 15(3):118–128, 2010
37. Prachar TL, Mahanes D, Arceneaux A, et al: Recognizing the needs of family members of neuroscience patients in an intensive care setting. J Neuroscience Nurs 42(5):274–279, 2010
38. Puntillo KA, Aria S, Chohen NH, et al: Symptoms experienced by intensive care unit patients at high risk of dying. Crit Care Med 38:2155–2160, 2010
39. Collop N, Salas RE, Delayo M, et al: Normal sleep and circadian processes. Crit Care Clin 24:449–460, 2008
40. Weinhouse GL, Schwab R: Sleep in the critically ill patient. Sleep 29:707–716, 2006
41. Ozsancak A, D'Ambrosio C, Garpestad E, et al: Sleep and mechanical ventilation. Crit Care Clin 24:517–531, 2008
42. Fontana CJ, Pittiglio LI: Sleep deprivation among critical care patients. Crit Care Nurs Q 33(1):75–81, 2010
43. Bourne RS, Mills GH: Sleep disruption in critically ill patients—pharmacological considerations. Anaesthesia 59:374–384, 2004
44. Parthasarathy S, Tobin MJ: Sleep in the intensive care unit. Intensive Care Med 30:197–206, 2004
45. Elliott R, McKinley S, Cistulli P: The quality and duration of sleep in the intensive care setting: An integrative review. Int J Nurs Stud 48:384–400, 2011
46. Tamburri LM, DiBrienza R, Zozula R, et al: Nocturnal care interactions with patients in critical care units. Am J Crit Care 13:102–115, 2004
47. Cureton-Lane RA, Fontaine DK: Sleep in the pediatric ICU: An empirical investigation. Am J Crit Care 6:56–63, 1997
48. Edwards GB, Schuring LM: Pilot study: Validating staff nurses' observations of sleep and wake states among critically ill patients using polysomnography. Am J Crit Care 2:125–131, 1993
49. Richardson SJ: A comparison of tools for the assessment of sleep pattern disturbance in critically ill adults. Dimens Crit Care Nurs 16:226–239, 1997
50. Richards KC: Effect of a back massage and relaxation intervention on sleep in critically ill patients. Am J Crit Care 7:228–299, 1998
51. Barr J, et al: Clinical practice guidelines for the management of pain, agitation and delirium in adult patients in the intensive care unit. Crit Care Med (in press)
52. Edwards GB, Schuring LM: Sleep protocol: A research-based practice change. Crit Care Nurse 13(2):84–88, 1993
53. Olson DM, Borel CO, Laskowitz DT, et al: Quiet time: A nursing intervention to promote sleep in neurocritical care units. Am J Crit Care 10:74–78, 2001
54. Dennis CM, Lee R, Woodard EK, et al: Benefits of quiet time for neurointensive care patients. J Neurosci Nurs 4:217–224, 2010
55. Richardson A, Allsop M, Coghill E, et al: Earplugs and eye masks: Do they improve critical care patients' sleep? Nurs Crit Care 12:278–286, 2007
56. Monsen MG, Edell-Gustafsson UM: Noise and sleep disturbance factors before and after implementation of a behavioural modification programme. Intensive Crit Care Nurs 21:208–219, 2005
57. Cmiel CA, Karr DM, Gasser DM, et al: Noise control: A nursing team's approach to sleep promotion. Am J Nurs 104(2):40–48, 2004
58. Sareli AE, Schwab RJ: The sleep-friendly ICU. Crit Care Clin 24: 613–626, 2008
59. Scott LD, Rogers AE, Hwang WT, et al: Effects of critical care nurses' work hours on vigilance and patients' safety. Am J Crit Care 15:30–37, 2006
60. Samuelson KA: Adult intensive care patients' perception of endotracheal tube-related discomforts: A prospective evaluation. Heart Lung 40(1):49–44, 2011
61. Sturdivant L, Warrren NA: Perceived met and unmet needs of family members of patients in the pediatric intensive care unit. Crit Care Nurs Q 32(2):149–158, 2009
62. Whalin I, Ek AC, Idvall E: Empowerment in intensive care: patient experiences compared to next of kin and staff beliefs. Inten Crit Care Nurs 25(6):332–340, 2009
63. Gonzales EA, Ledesma RJ, McAllister DJ, et al: Effects of guided imagery on postoperative outcomes in patients undergoing same-day surgical procedures: A randomized, single-blind study. AANA J 78:181–182, 2010
64. Kline WH, Turnbull A, Labruna VE, et al: Enhancing pain management in the PICU by teaching guided mental imagery: A quality-improvement project. J Pediatr Psychol 35(1):25–31, 2010
65. Austin D: The psychophysiological effects of music therapy in intensive care units. Paediatr Nurs 22(3):14–20, 2010
66. Cooke M, Chaboyer W, Schulter P, et al: The effect of music on discomfort experienced by intensive care unit patients during turning: A randomized cross-over study. Int J Nurs Pract 16(2):125–131, 2010
67. Fredriksson AC, Hellstrom ML, Nilsson U: Patients' perception of music therapy in a postanesthesia care unit: A randomized crossover trial. Intensive Crit Care Nurs 25(4):208–213, 2009
68. Hunter BC, Oliva R, Sahler OJ, et al: Music therapy as an adjunctive treatment in the management of stress for patients being weaned from mechanical ventilation. J Music Ther 47(3):198–219, 2010
69. Henricson M, Ersson A, Maatta S, et al: The outcome of tactile touch on stress parameters in intensive care: A randomized controlled trial. Complement Ther Clin Pract 14(4):244–254, 2008
70. Lee D, Higgins PA: Adjunctive therapies for the chronically critically ill. Adv Crit Care 21(1):92–106, 2010
71. Teixeira MZ, Leal SM, Ceschin VM: Homeopathic practice in intensive care units: Objective semiology, symptom selection and a series of sepsis cases. Homeopathy 97:206–213, 2008
72. Raab A: Aromatherapy in the intensive care unit: An overview. Connect: The World Crit Care Nurs 7(2):127–130, 2010
73. Lindquist R, Sendelbach S, Windenburg DC, et al: Challenges of implementing a feasibility study of acupuncture in acute and critical care settings. Adv Crit Care 19(2):202–210, 2008
74. Nayak S, Wenstone R, Jones A, et al: Surface electrostimulation of acupuncture points for sedation critically ill patients in the intensive care unit—a pilot study. Acupuncture Med 26(1):1–7, 2008
75. Pfab F, Winhard M, Nowak-Machen M, et al: Acupuncture in critically ill patients improves delayed gastric emptying: A randomized controlled trial. Anesth Analg 112(1):150–155, 2011
76. Yang L, Yang J, Wang Q, et al: Cardioprotective effects of electroacupuncture pretreatment on patients undergoing heart valve replacement surgery: A randomized controlled trial. Ann Thorac Surg 89:781–786, 2010
77. Baumhover N, Hughes L: Spirituality and support for family presence during invasive procedures and resuscitations in adults. Am J Crit Care 18(4):357–366, 2009
78. Dunn LL, Handley MC, Dunkin JW: The provision of spiritual care by registered nurses on a maternal-infant unit. J Holist Nurs 27(1):31–33, 2009
79. Weiland SA: Integrating spirituality into critical care: An APN perspective using Roy's adaptation model. Crit Care Nurs Q 33(3):282–291, 2010
80. Chang LY, Wang KW, Chao YF: Influence of physical restraint on unplanned extubation of adult intensive care patients: A case-control study. Am J Crit Care 17(5):408–415, 2008
81. Hine K: The use of physical restraint in critical care. Nurs Crit Care 12(1):6–11, 2007

82. Mion LC, Minnick AF, Leipzig R, et al: Patient-initiated device removal in intensive care units: A national prevalence study. Crit Care Med 35(12):2714–2720, 2007
83. Mion LC: Physical restraint in critical care settings: Will they go away? Geriatr Nurs 29(6):421–423, 2008
84. Vasilevskis EE, Ely EW, Speroff T, et al: Reducing iatrogenic risks: ICU-acquired delirium and weakness-crossing the quality chasm. Chest 138:1224–1233, 2010

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

3 The Family's Experience with Critical Illness

Colleen Krebs Norton

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Describe the impact of a critical illness and the critical care environment on the family.
2. Describe methods to assess the needs of individual family members.
3. Describe nursing behaviors that help families cope with crisis.
4. Discuss palliative care issues in the critical care environment that have an impact on the family.
5. Define the components and application of the Critical Care Family Assistance Program.
6. List items to include in a plan of care that reflect the needs of the family.

Nurses who strive to deliver consistent quality holistic critical care need to recognize the importance of assessing and caring for patients and their families. The interaction between the family/support system, the nurse, and the patient in the critical care environment, and the needs that result from this interaction, remain a challenge and responsibility of the contemporary critical care nurse.

The *Oxford English Dictionary* defines family as "a group of persons consisting of the parents and their children whether actually living together or not; in a wider sense, the unity formed by those who are nearly connected by blood and affinity." For the purpose of this chapter, *family* is defined as any people who share intimate and routine day-to-day living with the critically ill patient. Anyone who is a significant part of the patient's normal lifestyle is considered a family member. The term family describes the people whose social homeostasis and well-being are altered by the patient's entrance into the arena of critical illness or injury. To provide family centered care, a philosophy that acknowledges that patients are part of a larger "whole" or family is essential to provide the best possible care to the patient and family.[1]

This chapter addresses the family in crisis, stressors in the critical care environment, family assessment, coping mechanisms, and the nursing process. The evolution of the Critical Care Family Assistance Program (CCFAP), family presence during resuscitation in the intensive care unit, and palliative care are also discussed.

▲ Stress, Critical Illness, and the Impact on the Family

A critical illness is a sudden, unexpected, and often life-threatening occurrence for both the patient and the family that threatens the steady state of internal equilibrium usually maintained in the family unit. It can be an acute illness or trauma, an acute exacerbation of a chronic illness, or an acute episode of a previously unknown problem. A family member's entrance as a participant in the life–death sick role of a loved one threatens the well-being of the family and can trigger a stress response in both the patient and the family. Family members of patients in the critical care unit (CCU) may experience stress, disorganization, and helplessness, which may ultimately result in difficulty in mobilizing appropriate coping resources, thus leading to anxiety.[2] The family enters this unplanned situation with its unexpected outcomes and often is forced into the role of decision maker. The balance of strengths and limitations within the family, including the degree of cohesion within the family and conflict resolution strategies, often determines how much stress can be confronted.[3] The astute critical care nurse recognizes that the fear and anxiety demonstrated by the patient and family is an expected consequence of activation of the stress response, a somewhat protective, adaptive mechanism initiated by the neuroendocrine system in response to stressors. The stress response of family members varies.

Stress Syndrome

Studied initially by Hans Selye in 1956,[4] *stress* has been defined as a specific syndrome that was nonspecifically induced. Selye also discussed the role of stressors, the stimuli that produced tension and could contribute to disequilibrium. Stressors can be physiological (trauma, biochemical, or environmental) and psychological (emotional, vocational, social, or cultural). The critical care environment is rich in both physiological and psychosocial stressors that threaten the state of well-being of the patient and family.

In response to a stressor, the fight-or-flight mechanism is activated, releasing the catecholamines norepinephrine and epinephrine through the sympathetic nervous system. These hormones are responsible for the increased heart rate, increased blood pressure, and vasoconstriction that make up the physiological response to the *alarm stage*, the initial stage of the general adaptation syndrome to stress described by Selye. The alarm stage is followed by the *stage of resistance*, which attempts to maintain the body's resistance to stress. According to Selye's theory, all people move through the first two stages many times and become adapted to the stressors encountered during ordinary life. If the person is unsuccessful at adaptation, or if the stressor is too great or prolonged, alarm and resistance are followed by the *stage of exhaustion*, which can lead to death, the result of a wearing down of the human body. It is a challenge to the critical care nurse to assist both the patient and family through this stress response, resulting in an adaptation to the critical care environment. Helping the patient's family resolve the crisis response and facilitate successful coping by creating a safe passage is an identified competency of the contemporary critical care nurse.[5] Family members expect nurses to intervene and meet their needs, and they have high expectations for family centered care.[6]

After the initial fear and anxiety over the critical illness and possible death of the family member, other considerations affect the family, including a shift in responsibilities and role performance, unfamiliarity with the routines of the CCU, and a lack of knowledge concerning the course and outcome of the disease. These issues can develop and persist over the duration of the patient's stay in the CCU.

Contributions to the family unit previously attributed to the patient are added to the responsibilities of others. Financial concerns are usually major, and daily activities that were of little consequence before become important and difficult to manage. Chores, such as balancing a checkbook, contributing to car pools, and shopping for groceries, can become critically significant if left undone. This consequence of the critical illness requires adding the responsibilities of the patient to the responsibilities of others.

The social role that the patient plays in the family is absent during the critical illness. Comforter, organizer, mediator, lover, friend, and disciplinarian are examples of important roles in family functioning that may be, under normal circumstances, fulfilled by the patient. When that role function is unfulfilled, havoc and grief may ensue.

The circumstances surrounding the nature of the patient's illness can also be a stressor for the family. In a sudden, unexpected event, such as a blunt trauma or an acute myocardial infarction, the life of the family can be brought to a halt in a matter of minutes. Having little or no time to prepare for such an event, the family is overwhelmed with a massive amount of unmanageable stress and can be thrown into crisis. The hospital CCU, in most instances an unknown entity, becomes the center of the life of the family. When allowed to visit in the CCU, the family observes sophisticated, intimidating equipment that causes additional fear. Such stress often can manifest itself as anger toward the care giver. The care giver, absorbed with the physical care of the patient, frequently has limited or inadequate time to respond to family members' emotional needs, unrealistic goals, and expectations of the health care staff.[7]

In other instances, the critical event is an acute exacerbation of a chronic but life-threatening illness. Such an episode brings with it a different set of stressors, reminding the family of difficult and painful times in the past when they have faced similar circumstances. Prolonged critical illness can present emotional difficulties for the family, which may increase the likelihood of crisis.

Coping Mechanisms

Coping mechanisms can be defined as a person's response to a change in the environment; they can be healthy or unhealthy. The critical care nurse, as care giver to both the patient and family, should be aware of the use of coping mechanisms by the family as a means of maintaining equilibrium. A sense of fear, panic, shock, or disbelief is sometimes followed by irrational acts, demanding behavior, withdrawal, perseveration, or fainting. The family attempts to obtain some sense of control over the situation, often demonstrated by refusing to leave the bedside or, alternatively, by minimizing the severity of the illness through denial. Reactions to crisis are difficult to categorize because they depend on the different coping styles, personalities, and stress management techniques of the family. The nurse must be able to interpret the feeling that a person in crisis is experiencing, particularly when that person cannot identify the problem or the feeling to himself or herself or to others. The following are four generalizations about crisis:

- Whether people emerge stronger or weaker as a result of a crisis is based not so much on their character as on the quality of help they receive during a crisis state.
- People are more open to suggestions and help during an actual crisis.
- With the onset of a crisis, old memories of past crises may be evoked. If maladaptive behavior was used to deal with previous situations, the same type of behavior may be repeated in the face of a new crisis. If adaptive behavior was used, the impact of the crisis may be lessened.
- The primary way to survive a crisis is to be aware of it.

▲ The Family and the Nursing Process

Nursing Assessment

In 2010, the Institute of Medicine recommended that health care delivery systems become patient centered rather than disease or clinician centered and that an assessment be made of the patient's preferences and beliefs.[8] In the CCU, this translates into an increase in family involvement. Nursing assessment by the critical care nurse involves primarily, but not exclusively, an appraisal of the patient. It also includes an assessment of the members of the family. The family remains the most important social component to consider. Family centered care is described as an expansion of total patient care and includes the family in the planning and implementation phases of the nursing process.[9] Family centered care is advocated by both the Joint Commission and Nursing Practice Standards.[10]

The nursing assessment serves as a database and an identification of strengths and concerns on which care of the patient and family can be supported. It includes not only physiological data but also psychological, social, environmental, cultural, economic, and spiritual reactions. It involves an assessment and validation of verbal and nonverbal behavior and requires clinical expertise. A thorough nursing assessment guides the formulation of nursing diagnoses. The standards of the American Association of Critical-Care Nurses (AACN) emphasize and support the importance of an assessment of the family and the continual involvement of the family in implementing the plan of care and participating, to the extent they are able, in decision making about the nursing care.[11]

An important part of the family assessment is a history of the family. Who does the patient include in the description of his or her family? Although all patients belong to a family, the family might not include or be restricted to blood relatives. Who are the people most upset about the patient's illness? Is there a formal or informal leader identified by the group? This becomes important when communicating with the family in decision making, as well as in legal matters, such as obtaining consent and withdrawing life support. What is the coping style of the family? Does the family have a history of dealing with a critical illness? What are the relationships between the members of the family? How close is the family? Do the family members identify any unresolved issues? The family history can aid the nurse in interpreting how the family is coping with stress, how their coping mechanisms will affect the patient, and how they are adapting to the patient's illness.

Four intrinsic elements of an assessment of the family are:[12]

1. Providing a human, caring presence
2. Acknowledging multiple perceptions
3. Respecting diversity
4. Valuing each person in the context of the family

Numerous assessment tools are available to aid the nurse in determining the needs and problems the family faces. One of the initial assessment tools was developed by Molter in 1979.[13] This method includes a 45-item needs assessment tool, which became an instrument to describe the needs of critical care family members. Leske modified the tool used by Molter by adding an open-ended item and calling it the Critical Care Family Needs Inventory (CCFNI).[14] The CCFNI has been used widely during the past two decades to identify the needs of family members in the CCU. The CCFNI, after analysis, was found to contain five distinct subscales: support, comfort, information, proximity, and assurance.[15] Mendonca and Warren[2] used the CCFNI to assess the needs of family members and the importance of these needs in the first 18 to 24 hours after admission of the adult patient to the CCU. The family remains the most important social context to assess and consider when determining interventions to influence patient outcomes in a positive way.[14]

Nursing research using these tools reveals consistency in which areas are important and should be addressed with family members. These areas include but are not limited to:

1. Family satisfaction with care given
2. Explanations that the family can understand
3. The need for close proximity to the patient
4. Honest information about the patient's condition
5. An understanding of why things are done
6. Delivery of care by staff members who are courteous and show interest in how the family is doing
7. Assurance that someone will notify the family of any changes

The tools also suggest assessing how comfortable the family is in the waiting room and inquiring about what things could be made better for them. Identified needs included physical needs (eg, having comfortable furniture, having the waiting room near the patient, and having a bathroom nearby) as well as emotional needs, such as a place to be alone in the hospital and the opportunity to discuss negative feelings. In addition, families of critically ill patients have other needs, which should be addressed frequently. These needs include:[15]

- To feel that there is hope
- To feel that hospital personnel care about the patient
- To know the prognosis
- To receive information about the patient at least once a day
- To see the patient frequently

In summary, recent research has shown that the top needs of families of critically ill patients are the need for information, the need for support from hospital staff, and the need for hope.[16]

Although the needs perceived by the family may differ from those perceived by the nurse, strong communication skills, as well as an atmosphere of concern and caring, help the nurse gather the subjective and objective assessment data and formulate the appropriate nursing diagnoses for the family. Examples of nursing diagnoses appropriate to the family members of a critically ill patient are given in Box 3-1. The nursing diagnosis guides both the nurse and the family in establishing mutual goals.

BOX 3-1 EXAMPLES OF NURSING DIAGNOSES

For the Family With Critical Illness or Injury

- Acute Confusion
- Anxiety
- Risk for Caregiver Role Strain
- Readiness for Enhanced Communication
- Decisional Conflict
- Defensive Coping
- Deficient Knowledge
- Disturbed Sleep Pattern
- Dysfunctional Family Process
- Fatigue
- Fear
- Grieving
- Hopelessness
- Ineffective Health Maintenance
- Ineffective Denial
- Ineffective Role Performance
- Interrupted Family Processes
- Impaired Memory
- Impaired Parenting
- Risk for Loneliness
- Powerlessness
- Spiritual Distress
- Risk for Spiritual Distress

Nursing Interventions

The time spent by the critical care nurse with the family is often limited because of the crucial physiological and psychosocial needs of the patient. Therefore, it is important to make every interaction with the family as useful and therapeutic as possible. Categories of nurse caring behaviors as perceived by the families of critical care patients include informing, enhancing, touching, and spiriting. Interaction between the nurse and family has the potential to develop a bond as a result of these caring behaviors.[17] Nursing interventions should address cognitive, affective, and behavioral domains[18] and should be designed to:

- Help the family learn from the crisis experience and move toward adaptation
- Regain a state of equilibrium
- Experience the normal (but painful) feelings associated with the crisis, to avoid delayed depression, and allow for future emotional growth

A novel idea in the practice of family centered care is to consider inviting a family member on daily rounds with the team in the CCU. An advantage of having a family member on rounds includes reduced stress in families because members have current information and a representative who can ask questions. A disadvantage could be that care is complex; a family member may not be well prepared to handle the information cognitively or emotionally. However, a recent study demonstrated widespread family member satisfaction with the invitation to join patient care rounds in a trauma patient population.[19] This example of including family members on rounds is just one of several ideas to improve the family's experience in critical care. Asking family members to assist in providing some aspects of basic care has also been studied, but not all family members are willing participants, and thus the requests must be considered on an individual basis.[20,21] The use of family visiting kits has also been explored, which includes activities that could be performed at the bedside (hand massage), directions on personal care activities (applying lip balm around an endotracheal tube), cognitive recovery tools, (such as dominoes and plating cards), and personal care items for the families themselves (toiletries and a log for questions).[10]

Suggestions for nursing interventions with the family in crisis are outlined in Box 3-2. Considerations for the older patient are presented in Box 3-3.

BOX 3-2 NURSING INTERVENTIONS

For Care of the Family in Crisis

- Guide the family in defining the current problem.
- Help the family identify its strengths and sources of support.
- Prepare the family for the critical care environment, especially regarding equipment and purposes of the equipment.
- Speak openly to the patient and the family about the critical illness.
- Demonstrate a concern about the current crisis and an ability to help with the initial relationship.
- Be realistic and honest about the situation, taking care not to give false reassurance.
- Convey feelings of hope and confidence in the family's ability to deal with the situation.
- Try to perceive the feelings that the crisis evokes in the family.
- Help the family identify and focus on feelings.
- Assist the family to determine the goals and steps to take in facing the crisis.
- Provide opportunities for the patient and the family to make choices and avoid powerlessness and hopelessness.
- Assist the family in finding ways to communicate with the patient.
- Encourage the family to help with the care of the patient.
- Discuss all issues as they relate to the patient's uniqueness; avoid generalizations.
- Help the family to set short-term goals so that progress and positive changes can be seen.
- Ensure that the family receives information about all significant changes in the patient's condition.
- Advocate the adjustment of visiting hours to accommodate the needs of the family as permitted by the situation in the unit.
- Determine whether there is space available in the hospital near the unit where the family can be alone and have privacy.
- Recognize the patient's and family's spirituality, and suggest the assistance of a spiritual advisor if there is a need.

Visitation Advocacy

The concept of open visiting hours is seen as another method of enhancing family centered care. Policies regarding the use and provision of visiting hours should be evaluated periodically. Research demonstrates that novel approaches to visitation, such as allowing children who are accompanied by an adult to visit a relative in the CCU and the use of animal-assisted therapy in the CCU, can have positive effects on the patient, including increased feelings of happiness and calmness and reduced feelings of loneliness.[22]

Visiting hours in CCUs have been restricted for many years, with the rationale that rest, quiet, and an undisturbed environment were all therapeutic nursing interventions. Families often interpreted these restrictions as denying access to their loved ones. As early as 1978, Dracup and Breu reported that satisfying the needs of the families of patients was improved by relaxing a policy of restricted visiting hours and initiating set communication with patients' spouses.[23]

BOX 3-3 CONSIDERATIONS FOR THE OLDER PATIENT

Providing Care for the Critically Ill Older Patient

- Respect the dignity, intelligence, privacy, and maturity of the patient at all times.
- Maintain the patient's right to make decisions as long as possible.
- Avoid the use of paternalism in patient care.
- Integrate the physiological and cognitive changes of aging with the assessment and care of the patient.
- Allow the family to share in the care of their family member.
- Provide active participation and a sense of control for the patient and family.
- Make sure that the patient remains the focus of care and that interventions are performed for the good of the patient.
- Assess the impact that medical and nursing interventions have on quality of life and sense of well-being.
- Determine family burdens resulting from the critical illness.

Increased visiting time was seen as a strategy to improve coping skills and strengthen the relationship between nurses and the family members of patients.[24] Over time, many units began the policy of unrestricted visiting hours. Such interventions were designed to strengthen the relationship between the family and health care provider as well as foster adaptation to the crisis on the part of the family. Research demonstrates that patients have increased satisfaction and decreased anxiety with family around, that families are satisfied with the experience, that nurse/patient/family communication is enhanced, and that patients receive increased support, which improves their well-being.[25] Unrestricted visiting policies in CCUs have been linked to a decrease in septic complications; a decrease in the amount of cardiovascular complications, specifically shock and pulmonary edema; and an improvement in the physiological presentation of blood pressure, heart rate, and intracranial pressure.[26] See Evidence-Based Practice Highlight 3-1.

Critical care nurses must take the responsibility for revising visiting hours to meet the needs of patients and families. When choosing a less-restrictive policy, the physical layout of the unit must be considered. Smaller units may be less appropriate for unrestricted visiting hours with unlimited visitors. The focus should be on what proves to be best for the patient, not for the nurse. The effectiveness of changes in visiting hours must also be evaluated. Additional nursing research is needed to determine the effect of changes in visiting hours on the needs of patients and families.

The nurse must prepare family members for the initial visit to the CCU because it can be an overwhelming environment. The functions of monitors, intravenous drips, ventilators, and other technologies, as well as the meaning of alarms, should always be explained before and during the family visits to avoid causing anxiety and the potential of becoming a barrier between nurse/patient/family.

The names, roles, and responsibilities of all members of the interdisciplinary health care team should be identified to both the patient and family. The nurse, by example, can demonstrate the value of communication and touch to the family. Encouraging family members to provide direct care to the

EVIDENCE-BASED PRACTICE HIGHLIGHT 3-1
Family Visitation in the Adult ICU

△ Expected Practice

- Facilitate unrestricted access of hospitalized patients to a chosen support person (eg, family member, friend, or trusted individual) who is integral to the provision of emotional and social support 24 hours a day, according to patient preference, unless the support person infringes on the rights of others and their safety, or it is medically or therapeutically contraindicated.[1] (Level D)
- Ensure that the facility/unit has an approved written practice document (ie, policy, procedure, or standard of care) for allowing the patient's designated support person—who may or may not be the patient's surrogate decision maker or legally authorized representative—to be at the bedside during the course of the patient's stay, according to the patient's wishes.[1–6] (Level D)
- Evaluate policies to ensure that they prohibit discrimination based on age, race, ethnicity, religion, culture, language, physical or mental disability, socioeconomic status, sex, sexual orientation, and/or gender identity or expression.[1–6] (Level D)
- Ensure that there is an approved written practice document (ie, policy, procedure, or standard of care) for limiting visitors whose presence infringes on the rights of others and their safety or are medically or therapeutically contraindicated to support staff in negotiating visiting privileges.[6] (Level D)

△ Supporting Evidence

- In practice, 78% of ICU nurses in adult critical care units prefer unrestricted policies[7–13]; yet, studies show that 70% of hospital ICU policies restrict family visitation.[3,7–9,13,14] This variability creates conflict between nurses and confusion in families.[10,15]
- Some ICU nurses believe that family visitation increases physiologic stress in the patient and interferes with the provision of care,[16] is mentally exhausting to patients and families,[11,15–19] and contributes to increased infection[8,19]; however, the evidence does not support these beliefs.[8,9,20–29]
- Evidence does suggest that for patients, flexible visitation decreases anxiety,[17,20,21] confusion, and agitation,[22] reduces cardiovascular complications,[20] decreases length of ICU stay,[31] makes the patient feel more secure,[32] increases patient satisfaction,[14,20,29,32–34] and increases quality and safety.[20,24–26,30,35,36]
- For family members, evidence suggests that unrestricted visitation increases family satisfaction,[7,11,17,20,29,36–38] decreases family member anxiety,[7,17,29,36,39,40] promotes better communication,[11,14,17,24,29,41] contributes to better understanding of the patient,[11,32,36] allows more opportunities for patient/family teaching as the family becomes more involved in care,[11] and is not associated with longer family visits.[36]
- Finally, evidence suggests that some nurses in adult ICUs restrict children's visits based on the intuition that children will be harmed by what they see or based on a concern that they would be uncontrollable. These biases are not grounded in evidence or based on the patient's or the child's actual needs.[8,42–44] Yet, when allowed to visit relatives in the ICU, properly prepared children have less negative behavior and fewer emotional changes than those who did not visit.[45–48] It is recommended that they be allowed to visit unless they carry contagious illnesses.[49]

AACN Levels of Evidence

Level A Meta-analysis of quantitative studies or metasynthesis of qualitative studies with results that consistently support a specific action, intervention, or treatment
Level B Well-designed, controlled studies with results that consistently support a specific action, intervention, or treatment
Level C Qualitative studies, descriptive or correlational studies, integrative reviews, systematic reviews, or randomized controlled trials with inconsistent results
Level D Peer-reviewed professional and organizational standards with the support of clinical study recommendations
Level E Multiple case reports, theory-based evidence from expert opinions, or peer-reviewed professional organizational standards without clinical studies to support recommendations
Level M Manufacturer's recommendations only

Excerpted from American Association of Critical-Care Nurses Practice Alert. Available online at http://aacn.org. All references cited in this alert are available with the associated resources related to this chapter. Visit: http://thepoint.lww.com

patient, if they are interested, can help decrease anxiety and provide the family with some control. Direct care activities for the family to perform may include brushing teeth, combing hair, helping with a meal, providing skin care, or giving a bath.

Allowing children to visit a CCU may require special arrangements on the part of the staff. Visits should include short, simple explanations to the child concerning the patient's condition. Answering the child's questions in developmental terms that he or she can understand helps reduce possible fears. The person who is escorting the child into the CCU should be aware that invasive monitoring and other equipment might upset a youngster. If a visit from the child is not possible, arrangements can be made for a telephone visit.

In addition to unrestricted visiting hours, family presence during invasive procedures and resuscitation should be addressed. Positive and compelling evidence has emerged in the research on family presence and the emergency department (ED), necessitating that family presence in adult CCUs be investigated. Although differences exist between the CCU and the ED in terms of appropriateness, terminal weaning, organ donation, and planned invasive procedures seem suitable for the family to witness. When studied, a significant positive relationship was found between spirituality and support for family presence among physicians, physician assistants, and nurses.[27,28] Families believe it is their right to be present, but nurse perceptions continue to vary widely. A common theme that emerges is the need for individuality and uniqueness in each situation.

Use of the Nurse–Family Relationship

Initiating nursing interventions and establishing a meaningful relationship with the family tends to be easier during crisis than at other times. People in crisis are highly receptive to an interested, caring, and empathetic helper. When first meeting the patient's family, the nurse must demonstrate the desire and ability to help. Help that is specific to the family's needs at that time demonstrates the nurse's interest in their comfort and well-being. Deciding on a family representative who is to be notified of the patient's status and validating that contact person's telephone number can be an overwhelming family decision. Assisting the family to determine immediate priorities is essential in the early phase of crisis intervention. The existence of advanced directives, a living will, and power of attorney should be determined. In the absence of these documents, support and methods to obtain them should be given to the family.

With this type of timely involvement, the family will begin to trust and depend on the nurse's judgment. This process then allows family members to believe the nurse when the nurse conveys feelings of hope and confidence in the family's ability to cope with whatever is ahead. It is important to avoid giving false reassurance; rather, the reality of the situation should be expressed in a kind, supportive fashion.

Problem Solving With the Family

As the relationship between the nurse and the family develops, the nurse begins to understand the dynamics of the problem facing the family. Problem solving with the family takes into consideration items such as:

- The meaning the family has attached to the event
- Other crises with which the family may be coping
- The adaptive and maladaptive coping behaviors previously used in time of stress
- The normal support systems of the family, which might include friends, neighbors, clergy, and colleagues

Using the information collected and recorded in the assessment base enables the nurse to help the family deal with the stress. Areas to include in interventions are defining the problem, identifying support, focusing on feelings, and identifying steps.

Defining the Problem

A vital part of the problem-solving process is to help the family clearly state the immediate problem. Often people are overwhelmed and immobilized by the anxiety or panic caused by acute stress. Being able to state the problem and acknowledge the difficulty or threat it poses reduces the family's anxiety by helping family members realize that they have achieved some sort of an understanding of what is happening. Defining the problem is a way of delimiting its parameters. Simply asking the family members their understanding of the problem and the greatest concern for them at this time helps in problem definition. In addition, the family's response helps the nurse to clarify his or her understanding of what the family needs.

Defining and redefining problems can and should occur many times before the problem is solved. Stating the problem clearly helps the family assign priorities and direct needed actions. Goal-directed activities help decrease anxiety.

Identifying Support

Under high levels of stress, some people expect themselves to react differently. Rather than turning to the resources they use daily, they become reluctant to involve them. Asking people to identify the person to whom they usually turn when they are upset, and encouraging them to seek assistance from that person now, helps direct the family back to the normal mechanisms for handling stressful issues. Few families are truly without resources; rather, they only have failed to recognize and call on them.

Defining and redefining the problem may also help put the problem in a different light. It is possible in time to view tragedy as a challenge and the unknown as an adventure. The process of helping the family view a problem from a different perspective is called reframing.

The nurse can also help the family call on its own inherent strengths. What is it as a family that they do best? How have they handled stress before? Encouraging the family members to capitalize on their strengths as a family unit is well worth the effort.

Focusing on Feelings

A problem-solving technique emphasizing choices and alternatives helps the family achieve a sense of control over part of their lives. It also reminds them, and clarifies for them, that they are ultimately responsible for dealing with the event and that they will live with the consequences of their decisions.

Helping the family focus on feelings is extremely important to avoid delayed grief and protracted depression in the future. The reflection of feelings or active listening is necessary

throughout the duration of the crisis. Valuing the expression of feelings might help the family avoid the use of unhealthy coping mechanisms, such as alcohol or excessive sleep. In difficult and sad times, the critical care nurse can promise the family, with some certainty, that things will become easier with the passage of time. Adaptation takes time. Providing valid assurance (eg, that a patient will eventually be weaned from a ventilator, or that tube feeding is a temporary measure) can give the family a sense of trust in the care giver.

During the difficult days of the critical illness, the family may become dependent on the judgment of professionals. The family may have some difficulty identifying the appropriate areas in which to accept the judgments of others. It is important that the nurse acknowledges the family's feelings and recognizes the complexity of the problem, while emphasizing the responsibility each member of the family has for his or her feelings, actions, and decisions. Encouraging family members to focus on things they can change helps to give them a sense of control. For example, if the patient is experiencing pain, the family can be encouraged to advocate for the patient by requesting that the physician evaluate the patient's pain control.

Identifying Steps

Once the problem has been defined and the family begins goal-directed activities, the nurse may help further by asking the family members to identify the steps that they must take. Such anticipatory guidance may help reduce the family's anxiety. However, the nurse must recognize moments when direction is vital to health and safety. It is often necessary to direct families, for example, to return home to rest. This can be explained by stating that by maintaining their own health, family members will, at a later date, be as helpful to the patient as possible. To make each interaction more meaningful and therapeutic, the nurse should focus on the crisis situation and avoid involvement in long-term chronic problems.

Interdisciplinary Management

The health care providers who most often meet the needs of family members are generally thought to be nurses and physicians. Additional help comes in the form of written materials, other family members, the patient, and other hospital sources. In some cases, nurse-coached hospital volunteer programs have proved effective.[29] These programs consist of an in-service program taught by nurses and followed by the assignment of a nurse mentor to the volunteer. Some families benefit by a referral to a mental health clinical specialist, a social worker, a psychologist, or a chaplain. Other interdisciplinary teams can include pharmacists, family care specialists, and therapists.[9] A nurse can best encourage the family to accept help from others by acknowledging the difficulty and complexity of the problem and providing several names and phone numbers. It may be appropriate for the nurse to set up the first meeting, with follow-up meetings coordinated between the family and the consultant. Many CCUs have such resources on a 24-hour on-call basis to ensure prompt interventions. An objective professional with experience in critical illness and its impact on the family can be an excellent resource. Family members of former patients are also being used as a method of sharing experiences and providing information to assist with the critical care experience.[29]

A new concept for improving family centered care in critical care is the Patient-Family Advisory Council.[30] An advisory council composed not just of nurses and other health care providers but also of past patients and family members represents the needs of family members. Driven by the consumer movement, the concept of the advisory council has the potential to provide new insights leading to improved family care. In one institution, clinical nurse specialists served as the facilitators, and the council worked on assessing how well the unit was doing in several categories with plans for improvement.[31]

▲ Palliative Care Issues in Critical Care

According to the World Health Organization, palliative care is an "approach that improves the quality of life of patients and their families facing the problem associated with life-threatening illness, through the prevention and relief of suffering by means of early identification and impeccable assessment and treatment of pain and other problems, physical, psychosocial, and spiritual."[32] Important components of palliative care are the inclusion of the family in decision making and the provision of patient care. Families are faced with complex palliative care decisions that must be made in the unfamiliar environment of the CCU. These decisions can be made easier with the involvement of the nurse in facilitating and guiding the decision-making process. The focus of palliative nursing care should be the entire family. This is often difficult in acute care units with time and space constraints. Nurses' personal issues, such as previous experiences with the death of their own family members, have the potential to either enhance or threaten assessment and intervention. Family meetings with the critical care nurse and the palliative care team help to ensure that the wishes and concerns of the family are heard.[33]

Caring for a patient's family at any point during the dying process encompasses three major areas: access, information and support, and involvement in care giving activities. Family members of dying loved ones should be allowed more liberal access in both visiting hours and number of visitors allowed. Ensuring that a family can be with their critically ill loved one will be a source of comfort. Information has been identified as a crucial component in the family's coping, and support in the form of the nurse's caring behaviors is influential in shaping the critical care experience for both the patient and family. Honesty and truth telling are important skills in this emotionally charged time. Finally, family involvement in care giving, in tasks as simple as being physically present to those as complex as assisting with postmortem care, may help families work through their grief. Facilitation of family involvement is a practical nursing intervention. See Chapter 6 for further discussion of end-of-life and palliative care issues in the critical care setting.

▲ The Critical Care Family Assistance Program

Several hundred research studies during the past 20 years have focused on the environmental and social issues of anxious families awaiting the outcome of a family member's stay in the CCU, demonstrating that attending to the needs of

these family members cannot be ignored.[33] Additionally, a crucial initiative of the AACN, established in 1969, focuses on establishing respectful, healing, and humane nursing care environments. As a result of collaboration between the Chest Foundation and Eli Lilly and Company Foundation, the Critical Care Family Assistance Program (CCFAP) was developed as an example of a renewed awakening to the concepts of family centered care.[1] The objectives of the CCFAP are:[34]

- To better prepare a multidisciplinary team to meet the needs of families
- To increase families' satisfaction with the care and treatment of critically ill patients
- To improve families' comprehension of, and satisfaction with, the information provided by care givers
- To identify common formats for providing information and financial resources
- To improve a hospital's ability to respond to family needs within a structured feedback model
- To increase the medical team's knowledge and understanding of the CCFAP model
- To increase knowledge about the CCFAP and foster dissemination of the information within the medical and lay community
- To compare and contrast specific levels of family needs across various models of care

Needs assessments completed at the original CCFAP sites validate the findings of decades of research. Commonly noted gaps in supporting the families of patients included:

- Discrepancies in the viewpoint with regard to the sharing of information
- The need of the family to involve more family members in decision making
- A lack of resources and services offered during this time of crisis

Because of shortened lengths of stay and nursing shortages, family members are increasingly more active participants in the care of their loved ones. Family centered care focuses on better integration of the family into the care planning process. Engaging family members early and encouraging them to work in partnership with the nursing staff can make a difference. Box 3-4 lists the components of the CCFAP model. It is hoped that with expanded use of this model, quality of critical care delivered will increase, whereas cost of critical care delivery will decrease.

BOX 3-4 Components of the Critical Care Family Assistance Program (CCFAP)

Communication
Example: "ICU Navigators," weekly group family sessions that provide information about equipment, medical procedures, and assertiveness skills

Environmental Changes
Examples: Expanding the waiting area, brightening the look of the room, new and more comfortable furniture

Educational Materials
Example: Publications that are up-to-date and written in nontechnical language

Information Kiosk
Examples: Electronic messaging system, Internet access, CCFAP family satisfaction surveys

Hospitality Programs
Examples: Hotel discounts, meals for families

Other Services
Examples: Music therapy, pet therapy

▲ Cultural Issues Related to Critical Illness

Nursing interventions for the critically ill patient include recognition and appreciation of the cultural uniqueness of each person. In today's diverse society, culture affects the nursing care of patients in many ways—from pain control and visitation expectations to care of the body after death. Critical care nurses must recognize the uniqueness of each person and the ways in which that uniqueness affects the care of the patient and the needs of the family.

Health care providers in Western medicine often address a critical illness as a disease process and focus on the physical symptoms, the pathology of organ function, or injury to a body part. The patient and family, having a different cultural perspective, may view the illness in a more psychophysiological manner, focusing on the physical, psychological, personal, and cultural ramifications of the illness. In other cultures, the patient's critical illness may be viewed as a curse or disharmony in the universe. Culture serves as an important influence in the patient's attitudes about approach to suffering, beliefs about life-prolonging treatments, palliative care, and advanced directives and health care proxies.[35]

Cultural competence is a reflection of one's attitudes, knowledge base, acquired skills, and behavior. Although it is unrealistic to expect that the nurse should know the customs and beliefs of all critically ill patients he or she cares for, it is not unreasonable to expect some degree of cultural competence. Glass et al[36] make the following suggestions:

- Be aware of one's own ethnocentrism.
- Assess the family's beliefs about illness and treatment.
- Consistently convey respect.
- Request that the family and patient act as guides for cultural preferences.
- Ask for the patient's personal preference.
- Respect cultural differences regarding personal space and touch.
- Note appropriate complementary and alternative medical practices, and allow their use if possible.
- Incorporate the patient's cultural healing practices into the plan of care.
- Be sensitive to the need for a translator.

Cultural characteristics, such as language, values, behavioral norms, diet, and attitudes toward disease prevention, death and dying, and management of illness, vary from culture to culture. Critical illness may be viewed by the family from a religious or spiritual perspective. Astuteness and sensitivity on the part of the critical care nurse ensure that the beliefs of the highly technologic, illness-focused health care system will not clash with cultural beliefs in folk medicine, rituals, religious healing, and medicine men.

▲ Clinical Applicability Challenges

SHORT ANSWER QUESTIONS

1. Mrs. J. is a critically ill patient with multiple trauma who has been admitted to your unit. She is not expected to survive her injuries. Mrs. J.'s husband, her two children ages 6 and 10, and her parents have just arrived on your unit. Formulate a plan of care that reflects sensitivity to the issues that will assist the patient's family in dealing with the probable death of their loved one.

2. Mr. E. is a 73-year-old man admitted to your unit after resuscitation. He has a long-standing history of cardiac disease and collapsed at home after what has been interpreted as a life-threatening rhythm disturbance. Although he received cardiopulmonary resuscitation, he was unconscious for 10 minutes before the rescue team arrived. His older daughters are fighting over the maintenance of ventilatory support. Discuss how you would help his daughters at this difficult time.

3. Mr. and Mrs. P. are the parents of a critically ill child on your unit. They are Indian and speak very little English. Describe the criteria you would use to assess their degree of stress and anxiety. How could you be certain that the cultural needs of the family are met while the child is a patient in your unit?

References

1. Henneman EA, Cardin S: Family-centered critical care: A practical approach to making it happen. Crit Care Nurse 22(6):12–19, 2002
2. Mendonca D, Warren N: Perceived and unmet needs of critically ill family members. Crit Care Nurs Q 21(1):58–67, 1998
3. Leon A, Knapp S: Involving family systems in critical care nursing. Dimens Crit Care Nurs 27(6):255–262, 2008
4. Selye H: The Stress of Life. New York, NY: McGraw-Hill, 1956
5. Curley M: Critical Care Nursing of Infants and Children. New York, NY: Elsevier Science, 1996
6. Fox-Wasylyshyn S, El-Masri M, Williamson K: Family perceptions of nurses' roles toward family members of critically ill patients: A descriptive study. Heart Lung 34:335–344, 2005
7. Warren NA: Critical care family members' satisfaction with bereavement experiences. Crit Care Nurs Q 25:54–60, 2002
8. Institute of Medicine, National Academy of Sciences. Retrieved May 30, 2010 from http://iomwww@nas.edu
9. Nelson D, Polst G: An interdisciplinary team approach to evidence based improvement in family centered care. Crit Care Nurs Q 31(2):110–118, 2008
10. Davidson J, Powers K, Hedayat K, et al: Clinical practice guidelines for support of the family in the patient-centered intensive care unit: American College of Critical Care Medicine Task Force. Crit Care Med 35(2):605–622, 2007
11. American Association of Critical-Care Nurses: Standards for Acute and Critical Care Nursing Practice, 3rd ed. Aliso Viejo, CA: AACCN, 2010
12. Hartrick G, Lindsey AE, Hills M: Family nursing assessment: Meeting the challenge of health promotion. J Adv Nurs 20:85–91, 1994
13. Molter NC: Needs of relatives of critically ill patients. Heart Lung 8:332–339, 1979
14. Leske J: Internal psychometric properties of the Critical Care Family Needs Inventory. Heart Lung 20:236–244, 1991
15. Kosco M, Warren NA: Critical care nurses' perceptions of family needs as met. Crit Care Nurs Q 23:60–72, 2002
16. Holden J, Harrison L, Johnson M: Families, nurses and intensive care patients: A review of the literature. J Clin Nurs 11:140–148, 2002
17. Frey S, Warren N. Perceived needs of critical care family members: A phenomenological discourse. Crit Care Nurse Q 30(2):181–188, 2007.
18. Naebel B, Fothergill-Bourbonnais F, Dunning J: Family assessment tools: A review of the literature from 1978–1997. Heart Lung 29:196–209, 2000
19. Schiller WR, Anderson BF: Family as a member of the Trauma Rounds: A strategy for maximized communication. J Trauma Nurs 10(4):93–101, 2003
20. Azoulay E, Pouchard F, Chevret S, et al: Family participation in care to the critically ill: Opinions of families and staff. Intensive Care Med 29:1498–1504, 2003
21. Eldredge D: Helping at the bedside: Spouses' preferences for helping critically ill patients. Res Nurs Health 27:307–321, 2003
22. Gavaghan S, Carroll D: Families of critically ill patients and the effect of nursing interventions. Dimens Crit Care Nurs 21(2):64–71, 2002
23. Dracup KA, Breu CS: Using nursing research to meet the needs of grieving spouses. Nurs Res 27:212–216, 1978
24. Stillwell SB: Importance of visiting needs as perceived by family members of patients in the intensive care unit. Heart Lung 13:238–242, 1984
25. Duran C, Oman K, Abel J, et al: Attitudes towards and beliefs about family presence: A survey of health care providers, patients' families, and patients. Am J Crit Care 16:270–279, 2007
26. Fumagalli S, Boncinelli L, LoNostro A, et al: Reduced circulatory complications with unrestricted visiting policy in the intensive care unit. Circulation 113:946–952, 2006
27. Baumhover N, Hughes L. Spirituality and support for family presence during invasive procedures and resuscitation in adults. Am J Crit Care 18(4):357–366, 2009
28. Meyers TA, Eichhorn DJ, Guzzetta CE, et al: Family presence during invasive procedures and resuscitation: The experience of family members, nurses and physicians. Am J Nurs 100:32–34, 2000
29. Appleyard M, Gavaghan S, Gonzalez C, et al: Nurse-coached interventions for the families of patients in critical care units. Crit Care Nurse 20(3):40–48, 2000
30. Sacco T, Stapleton M, Ingersoll G. Support groups facilitated by families of former patients creating family inclusive critical care units. Crit Care Nurse 29(3):36–45, 2009
31. Halm MA, Sabo J, Rudiger M: The patient-family advisory council: Keeping a pulse on our customers. Crit Care Nurse 26(5):58–67, 2006
32. World Health Organization. Palliative Care. Retrieved May 30, 2010, from www.who.int/cancer/palliative/definition

33. Davies B: Supporting families in palliative care. In Ferrell B, Coyle N (eds): Textbook of Palliative Nursing. Oxford, UK: Oxford University Press, 2001
34. Lederer M, Goode T, Dowling J: Origins and development: The critical care family assistance program. Chest 128(3):65S–75S, 2005
35. Davidson JE, Powers K, Hedayat KM, et al: American College of Critical Care Medicine Task Force 2004–2005, Society of Critical Care Medicine. Clinical practice guidelines for support of the family in the patient-centered intensive care unit: American College of Critical Care Medicine Task Force 2004–2005. Crit Care Med 35:605–622, 2007
36. Glass E, Cluxton D, Rancour P: Principles of patient and family assessment. In Ferrell B, Coyle N (eds): Textbook of Palliative Nursing. Oxford, UK: Oxford University Press, 2001

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

4 Patient and Family Education in Critical Care

Mary O. Palazzo

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Describe the barriers to learning that are unique to the critical care setting.
2. Describe and differentiate between the concepts of education and learning.
3. Identify the three domains of learning.
4. Identify the six principles of adult learning.
5. Describe the assessment of learning in the critical care environment.

In the critical care setting, it is always a challenge to meet the educational needs of patients and families because of the life-threatening nature of critical illness. Patient and family education is a vital component of nursing care. The nurse must deal with the anxiety and fear that is associated with a diagnosis of critical illness, while trying to teach difficult concepts in an environment that is poorly suited to learning.

Health care is no longer solely defined in terms of sound clinical decision making; now, it also encompasses prudent use of resources and financial accountability. In March of 2010, the American people witnessed the passage of "The Affordable Care Act," deemed by many to be the broadest health care reform legislation in U.S. history. A key element of this reform shifts the payment structure for hospitals and health care providers from a traditional fee-for-service model to an incentive model. Reimbursement for care will be based on hospital performance against several clinical outcome measures. Quality outcomes related to myocardial infarction, congestive heart failure, pneumonia, surgical care, health care–associated infections, and patient satisfaction will be required. Many of the quality indicators include patient and family education components. Reforms to the health care infrastructure necessitate clear evidence of nursing involvement in patient and family instruction and quantification of nursing care.

Hospital admissions and length of stay have also decreased with patients being discharged home, sometimes before they are ready to learn. Today, it is not unusual for a patient to be discharged home from an intensive care unit (ICU), placing even greater responsibility on the patient and family to provide for high-intensity care at home. The critical care nurse not only manages the hemodynamic instability that often accompanies critical illness but also prepares the patient and family for the likelihood of early discharge from the hospital.

At the same time, hospitals are facing an ongoing shortage of experienced critical care nurses. ICUs that were once reserved for the most experienced nurses are now training grounds for the newly graduated nurse. The novice nurse must focus on learning how to manage the myriad of technological devices used to support the critically ill patient while understanding the pathophysiology of multisystem illness. For the new nurse, it may be very difficult to move beyond the essential nursing tasks that are an integral part of patient care to address the educational needs of the patient and family. In addition, the 12-hour nursing work schedule creates a highly variable pattern; often, nurses work only 2 or 3 days per week. The resulting staff inconstancy provides limited opportunity for care continuity, relationship building, or follow-up related to patient and family learning needs.

These are just a few examples of the realities of health care. The fragmentation of patient care across the health care system presents many obstacles and barriers to patient education. The purpose of this chapter is to assist students and nurses in developing the skills and tools needed to meet the challenge of patient and family education in the presence of critical illness. Nurses who understand the learning barriers that are unique to the critical care setting are better prepared to address the learning needs of patients and families.

▲ Barriers to Learning

Critical Illness and Stress

Typically, the patient and family enter an ICU quite unexpectedly because of a life-threatening event. The onset of illness signals the beginning of a physical and emotional crisis for all involved. Altered metabolic responses, sepsis, exposure to general anesthesia, organ failure, use of cardiopulmonary bypass, episodes of hypoxia, and marked sleep deprivation are common conditions of the critically ill. Each of these factors

can compromise mental acuity and decrease a person's learning capacity and recall. In addition, combating a severe illness consumes most of the patient's energy, leaving him or her with a limited ability to learn.

The patient experiences not only the physical effects related to the disease process but also emotional and spiritual distress. When facing serious illness, patients express feelings of powerlessness, profound terror, and fear of death. In a qualitative study by Lof,[1] ICU patients recounted unpleasant emotions, horrifying unreal experiences, and feelings of imminent death as they lost consciousness from anesthetics. The vigilant critical care nurse who recognizes a patient's fear and anxiety and offers the patient guidance through the unfamiliar course of illness, treatment, and recovery can support the patient's need for safety. For example, patients frequently report that the process of ventilator weaning is difficult to endure. Many experience feelings of "air hunger" and heightened anxiety when the ventilator support is reduced and the patient assumes more of the work of breathing. The nurse who provides clear explanations and reassurances during this period can greatly enhance the patient's probability for successful extubation. Other examples of nursing interventions with patient education include the preparation of the patient for an invasive procedure or treatment, or through discussions regarding an impending transfer to another level of care.

However, in the critical care setting, it is not unusual for the focus of education to quickly shift away from the patient and be redirected to meet the learning needs of the family members. An emotional and physical toll is exacted on the family members of critically ill patients, with stress levels peaking within 72 hours of admission to the unit.[2] The descriptive study by Halm et al[2] demonstrates changes in the sleeping and eating patterns of family members, as well as an increased use of cigarettes, over-the-counter medications, alcohol, and prescription drugs, while coping with the crisis of a critically ill patient. Stress can also manifest in hypervigilant behaviors such as repetitive questions, frequent telephone calls, and numerous visits. Early research by Molter using the Critical Care Family Needs Inventory (CCFNI) identified that the family of the critically ill patient has needs for support, comfort, information, proximity to the patient, and assurance.[3] Subsequent research by Burr using the CCFNI tool validated the same results and identified another family need: the need to maintain vigil or watch over the patient in the event that something happens.[4] Critical care nurses often witness this "hovering" behavior of family members, especially during the admission phase of care. Research studies such as these have provided evidence of family needs of the critically ill patient and have provoked substantive changes to hospital visitation policies. Many critical care units now offer "open visitation," or unrestricted visiting hours. This paradigm shift creates greater opportunity for the critical care nurse to educate the family members and helps them to feel more comfortable in this stress-filled circumstance. The critical care nurse can encourage the family to participate in the basic care of a loved one. Simply getting ice chips, providing mouth care, or combing hair may assist the families in feeling that they are doing something to help the patient in an otherwise "helpless" situation.[4] The following case study demonstrates how the interventions of the critical care nurse are used to support the educational and emotional needs of a family in crisis.

CASE STUDY

A Patient and Family in Crisis

The electronic doors to the cardiovascular intensive care unit (CVICU) swing open, and Vernon and Barbara enter their 43-year-old daughter's room. Sherrill has just returned to the CVICU after a mitral valve replacement and a tricuspid valve repair. Unfortunately, the valve replacement was complicated, and she had a prolonged course on cardiopulmonary bypass. Her heart was quite weak before the surgery, and now she requires an intra-aortic balloon pump and multiple medications to support her cardiac output. She has also developed a coagulopathy related to the extended time on cardiopulmonary bypass and continues to bleed. Her vital signs are stable, but she requires a continuous infusion of blood products to keep up with the blood loss from her chest tubes.

Vernon and Barbara are shocked by their daughter's appearance. Her pale, edematous face and lifeless expression are certainly not what they expected to see. There is little about their daughter that resembles the person they know and love. Equipment surrounds her bed and supports nearly every bodily function. Visibly shaken, they look at each other with tears in their eyes, wondering how a seemingly routine valve operation could turn out this way.

Kate, the critical care nurse caring for Sherrill, greets Vernon and Barbara and begins to talk about their daughter's appearance. She carefully explains the purpose of all the bedside equipment as she cares for their daughter. Kate tells the worried parents what she is doing and why she is performing each procedure. It is the nurse's calm and caring approach that engenders a sense of hope for their daughter's eventual recovery. The parents begin to relax a little and start to ask questions about what to expect in the next few hours.

This brief example illustrates the unpredictability of illness and just how quickly a crisis can develop. In this scenario, the bedside nurse initiates family education through an informal discussion of the patient's status. The initial interaction with family members is fundamental for the care delivery team and the family to establish frequent and open communication about the patient.[5] In the course of talking with the parents, the nurse is continually assessing their learning needs and developing an understanding of their coping mechanisms. By allowing the family to spend time with the patient at the bedside, the nurse enables them to meet their need to keep watch over the patient. Moreover, consistent and accurate information about their loved one is enabling the family to deal with the crisis phase of illness. Research has demonstrated that up-to-date information is the highest priority for family members who are coping with critical illness.[6] The critical care nurse teaches families about the pathophysiology of the illness, the diagnostic studies that are performed, the equipment that is used to support the patient, and the treatment plan that is under way. The primary education goal for most families is to learn all that they can about their loved one.

Prolonged Illness and Stress

Frequently, the period of illness extends well beyond the initial crisis phase and creates additional burdens for the patient and the family. The critically ill patient may experience a slow and unpredictable course with periods of organ system compromise or failure over time. Recovery is tedious and is

measured in small changes that occur over days and weeks. Families are forced to balance their home and work schedules with time spent at the hospital, often evoking feelings of guilt and anxiety. Over time, it may become increasingly difficult for the family to obtain information and patient status reports from the health care team. Often, physician schedules are unpredictable and do not mesh with family visits. This further underscores the vital role that the critical care nurse plays as a link to the family. With protracted critical illness, many families struggle to keep the lines of communication open to the extended family, creating opportunities for conflict and misinformation.

As a patient and family advocate, the nurse provides accurate information and shares the plan of care with the family. Additional interventions such as a patient care or ethics conference may be arranged by the critical care nurse to give the family an opportunity to discuss the case with the entire health care team. Inviting key family members to participate in the patient care conference can be an effective strategy for the health care team. Once everyone is assembled, the conference should begin with open-ended questions to allow the family members to express in their own words their understanding of the patient situation and the current plan of care. It allows the health care team to clarify misunderstandings and to provide additional opportunity for consensus decision making.[7] Patient conferences offer a therapeutic method of shared decision making between the health care team and the patient's family.

As the patient's condition improves and plans for transfer to progressive care or medical–surgical floor status are discussed with the health care team, the critical care nurse must prepare the patient and family for the eventual discharge from the unit. This milestone in recovery is typically viewed by the patient and family in one of two ways. If the patient and family believe that the patient's condition has improved sufficiently and that the intensity of critical care is no longer necessary, then this step is viewed in a positive light. However, should they believe that the depth of nursing support and level of monitoring on the accepting unit are inadequate to meet the needs of the patient, there may be resistance to the transfer process. When the critical care nurse spends time educating the patient and family about the unit routine, staffing patterns, and visiting hours before making the transfer, it helps mitigate some of the negative feelings and anxiety associated with the change.[8]

Once the transfer has been made, it is important that the receiving nurse further assist the patient and family with "settling" into the new routine. The nurse should begin by acknowledging the normal anxiety that accompanies the transfer process. He or she should establish trust and allay fears by explaining the changeover in care in transitioning from ICU to progressive care status, emphasizing that the transition is a positive stage in the recovery process. The nurse should also reassure the patient and family that even though the intensity of treatment has changed, the staff are trained to anticipate each patient's recovery needs and will respond appropriately to changes in the patient's status. Once the patient and family's initial anxiety diminishes, the nurse can begin to set new self-care goals and expectations based on his or her assessment. Clearly, nursing plays a vital role in helping the patient and family cope with the crisis of critical illness and the transitions in care by providing education from admission to discharge.

Environmental Stress

Ringing telephones, chiming call lights and pagers, overhead announcements, equipment alarms, staff conversations, banging automatic doors, and pneumatic tubes are just a few examples of the sounds that fill the air of a typical ICU. It is easy for nurses to become desensitized to these familiar noises because they are an integral part of the work environment. However, taking a quick moment to listen to the background sounds from a patient's bedside reminds a person how stressful noise can be. Patients and families are not accustomed to the normal sounds of an ICU. Yet, as difficult as it may be, nurses ask patients and families to learn in this setting.

A typical ICU setting is hardly an optimal learning environment. Ideally, a quiet moment is spent with the patient and family using comfortable chairs arranged to optimize discussion and with audiovisual aids, if possible. However, common measures can help reduce the environmental stress and enhance the success of learning. The simple act of closing the door to the patient's room or placing a comfortable chair at the bedside can reduce the background noise sufficiently and enhance the learner's attention span. Reducing the alarm volumes on bedside equipment while the nurse is talking with the patient or family helps minimize the number of interruptions and may improve the learner's ability to focus on the topic of a teaching session.

Ensuring privacy while sensitive or confidential information is being exchanged can markedly reduce the anxiety of a patient or family member. Often, strangers witness the emotional outbursts and intimate interactions of families who are agonizing over the illness of a loved one. Health care providers are not always mindful of their surroundings when discussing confidential details of a patient's case. Critical care nurses can direct health care team members and families to a quiet room away from the general waiting area to afford privacy when discussing specific patient information.

This regard for the patient also applies to teaching rounds or patient care rounds that are held in the halls of an ICU. Patients should be treated with respect, and they often wish to be included in bedside presentations. Open visitation in the ICU requires that the health care team be mindful of visitors who may be at the patient's bedside and to make sure that the patient has given permission for health care issues to be discussed in the presence of visitors before proceeding with rounds. A concerted effort to ensure patient privacy is a responsibility of all health care team members, but often it is the nurse who knows the patient's wishes and the family dynamics and is in the best position to direct the team. At the beginning of rounds, health care team members should make appropriate introductions and give clear explanations of the medical jargon used in the course of conversation.

Cultural and Language Barriers

As the U.S. population changes, the patients and families whom nurses care for in hospitals and critical care settings are becoming increasingly diverse. Currently, Asians and Hispanics are the two largest immigrant groups in our country.

The U.S. Census Bureau projections for 2050 estimate that the Hispanic population will double and the Asian population will increase by 79%.[9] Beliefs about health and illness are deeply rooted in culture. How a patient or family member responds to the diagnosis or a proposed treatment and education may be strongly influenced by his or her values and culture.[10] Although the nursing literature readily acknowledges the importance of providing culturally sensitive patient care, in practice there is little evidence of cultural awareness in the nurse's daily assessments and interactions with patients and families. Culturally competent nursing care is patient centered and focuses on asking the right questions to understand the patient's point of view.[11] Galanti describes an effective method of questioning patients to ascertain information that is culturally sensitive. She uses the mnemonic, the*4 C's of Culture*, with the first C standing for *Call*. This question attempts to identify what the problem is by asking, "What do you call your problem?" The second C is for *Cause*. The latter is intended to understand what the patient believes to be the source of the problem. The third C is for *Cope*. In this instance, the focus of the health care provider shifts to how the patient is managing the problem, or what other treatments have been prescribed to date. The final C is for *Concern*, such as, "What concerns do you have about your condition or the recommended treatment?"[11] This open-ended method of seeking information can also be used when speaking with family members, as culturally competent nursing considers the family structure and gender role as it relates to the patient. For example, in the Asian culture, important health care decisions should be discussed with the family. In many other cultures, the oldest male makes all the important decisions and this would include health care treatment decisions.

Successful education of culturally diverse patients and families requires more than just basic knowledge regarding ethnic groups. Critical care nurses must recognize their own individual biases and examine their personal values and beliefs about health and nursing care. Many of our health beliefs are based on commonly held Euro-American values such as belief in individualism, autonomy, independence, clock time, physical fitness, and beauty.[11] The imposition of these Euro-American values on other cultures may impede communication between the nurse and patient and hinder the education process. Although critical care nurses do not have the time to complete a thorough cultural assessment, several key pieces of information should be obtained. This information is outlined in Box 4-1.

BOX 4-1 Key Pieces of Information to Obtain as Part of the Cultural Assessment

- Does the patient live in an ethnic community?
- When medical decisions are made, who should be consulted before making the decision?
- Primary and secondary languages (speaking and reading ability)
- Religious practices
- Health and illness beliefs and practices
- Family expectations to remain with a hospitalized family member
- Communication practices (verbal and nonverbal)
- How decisions are made in the context of the patient and family.

Adapted from Galanti GA: Caring for Patients from Different Cultures, 4th ed. Philadelphia, PA: University of Pennsylvania Press, 2008, pp 93–108.

Language barriers also pose a major obstacle to patient and family education, especially in the stressful critical care environment. Every effort should be made to provide an interpreter to translate information for the non–English-speaking patient and family. Although it is convenient for health care providers to rely solely on a family member or friend to translate complex medical information and terminology that is likely to be unfamiliar, it may be difficult for the family member or friend to keep personal bias from entering the context of the conversation. In many cultures, decision making is assumed by the eldest member of the family, and asking a child to interpret medical information disrupts the social order of the family.[11] In addition, the information that is exchanged between the health care provider and the patient may be personal or embarrassing to either the patient or the family member who is pressed into service. Box 4-2 offers some suggestions for communicating with a patient and his or her family through a trained interpreter. Written instructions should also be translated and reviewed in the presence of an interpreter so that any questions can be addressed immediately. Printed instructions in several languages should be readily available for use in the ICU.

BOX 4–2 TEACHING GUIDE *Guidelines for Communication Using a Trained Interpreter*

- Before the session, meet with the interpreter to give background information and explain the purpose of the session.
- If possible, have the interpreter meet with the patient or family to determine their educational level, health care beliefs, and health care attitudes to plan the depth of information needed.
- Speak in short units of speech and avoid long explanations and use of medical jargon, abbreviations, and colloquialisms.
- Pause at regular intervals so that the interpreter has time to interpret and convey the information.
- Some concepts have no linguistic or conceptual equivalent in other languages and may require the interpreter to paint a picture, and this takes additional time and effort.
- When communicating to the patient or family, look directly toward the person and not at the interpreter. Watch the patient's and family members' body language and nonverbal communication response.
- Have the interpreter ask questions to alert about potential cultural misunderstandings that may arise.
- Be patient. Interpreted interviews take a long time to complete and may become tiresome for the patient.
- Have the patient and family members validate the information given to them through the interpreter, to make sure that they understand the instructions or message that has been given.
- Debrief with the interpreter after the session is complete.

Adapted from CCHCP: The Cross Cultural Health Care Program: Guidelines for Providing Health Care Services Through an Interpreter. August 22, 2010: Available at http://www.xculture.org./BTGintroMedInterp.php

Sensory Barriers

Effective education for deaf and hearing-impaired patients and families necessitates planning and additional resources. The Americans with Disabilities Act prohibits discrimination against people with disabilities, including those who are deaf or hearing impaired.[12] Under the law, these disabled people must be able to communicate with hospital staff, and the medical facility must be ready to meet that requirement. Deaf or hearing-impaired patients or family members should indicate their preferred mode of communication such as sign language, written notes, lip reading, oral interpreters, or other assistive devices.[12] To ensure that deaf or hearing-impaired patients or family members can communicate effectively, an oral interpreter should be used in the critical care setting for the discussion of treatment options; informed consent for procedures, blood administration, or surgery; and discharge instructions. These types of decisions typically require extended discussions and free-flowing communication that is best supported with an interpreter.

▲ Education and Learning

Many times the terms *education* and *learning* are used interchangeably, but there is a difference between these two concepts. *Education* is an activity, initiated by one or more persons, that is designed to effect changes in the knowledge, skill, and attitudes of people, groups, or communities.[13] Education places more emphasis on the person facilitating the learning, whereas *learning* is a phenomenon of internal change. The learner experiences a flash of insight that results in behavioral changes.[13] The focus shifts from the role of the educator to that of the person who experiences the change.

Three Domains of Learning

Three domains of human behavior or learning to consider when developing an education plan are the cognitive domain, the affective domain, and the psychomotor domain. Keeping these domains in mind while assessing and developing a teaching plan can assist the nurse in selecting suitable teaching methods.

The *cognitive domain* of learning involves the development of insight or understanding that provides a basis or guideline for behavior.[14] In this domain, knowledge expands, and teaching–learning material is organized from simple to complex. Learning is enhanced when information builds on previous knowledge. Therefore, the basic ideas should be well introduced before attempting to teach the hard-to-remember facts. As an example, cognitive learning occurs when a family member learns to assess wound healing. The critical care nurse provides basic information about the normal healing process and the appearance of a healthy incision. Once the family member understands how a healed incision should look, the nurse can explain the signs and symptoms of infection and when to call the physician. Once prepared, the family member should then be able to apply the learned principles to provide appropriate home care for the patient.

The *affective domain* pervades all spheres of learning because it encompasses the patient's values, attitudes, and feelings.[14] Attempting to modify an attitude or emotional response requires a safe and trusting relationship between the patient and the nurse. When formulating a teaching plan, the nurse should take a nonthreatening approach to assessing what the patient considers important enough to learn. A helpful teaching strategy may be the interactive group learning that is typical of a smoking cessation class. In this situation, the teacher demonstrates behaviors that the learner wants to imitate and provides positive feedback to the participants to encourage them to stop smoking cigarettes. If the learning experiences are satisfying and the patient associates positive feelings with the experience, it may help influence the change in behavior.

The *psychomotor domain* involves motor skills that are composed of an ordered sequence of movement that must be learned.[14] To learn a particular skill, the patient must have a neuromuscular system that is able to perform the skill and the capacity to form a mental image of the act.[14] A mental image is created when the learner watches a demonstration while the teacher points out the relevant steps that are required to successfully complete the task. The nurse may use a written, step-by-step guide as a reference while demonstrating the skill and allowing the patient to ask questions. Learning to inject insulin is an example of psychomotor learning. It takes practice for the patient or family member to become proficient at performing the task. The thought of learning a new skill is intimidating to many adults; therefore, it is important that the nurse provide praise and encouragement with each teaching session.

Teaching methods that are based on the three domains of learning are displayed in Figure 4-1.

Adult Learning Principles

The principles of adult learning are based on multiple learning theories that originate in many different disciplines, such as developmental psychology, sociology, philosophy, and education. Adult learning is a relatively new field (about 50 years old), with the fundamental principles grounded in childhood learning and education. A new conceptual framework known as the andragogical model emerged from research studies that identified some of the unique characteristics of adult learners.[13] The core principles of the andragogical model of adult learning are as follows:

1. *The need to know.* Adults need to understand why they need to learn something before they are willing to commit the energy and time to learn it. It is important for the learner to understand and be aware of the "need to know." To raise the learner's level of awareness, the facilitator may need to use real or simulated experiences to help the learner discover the lack of knowledge.
2. *The learner's self-concept.* Adults are self-directed and responsible for their own decision making. In general, adults resent the feeling that others are making choices for them. Adult educators need to create learning situations that are more self-directed and independent.

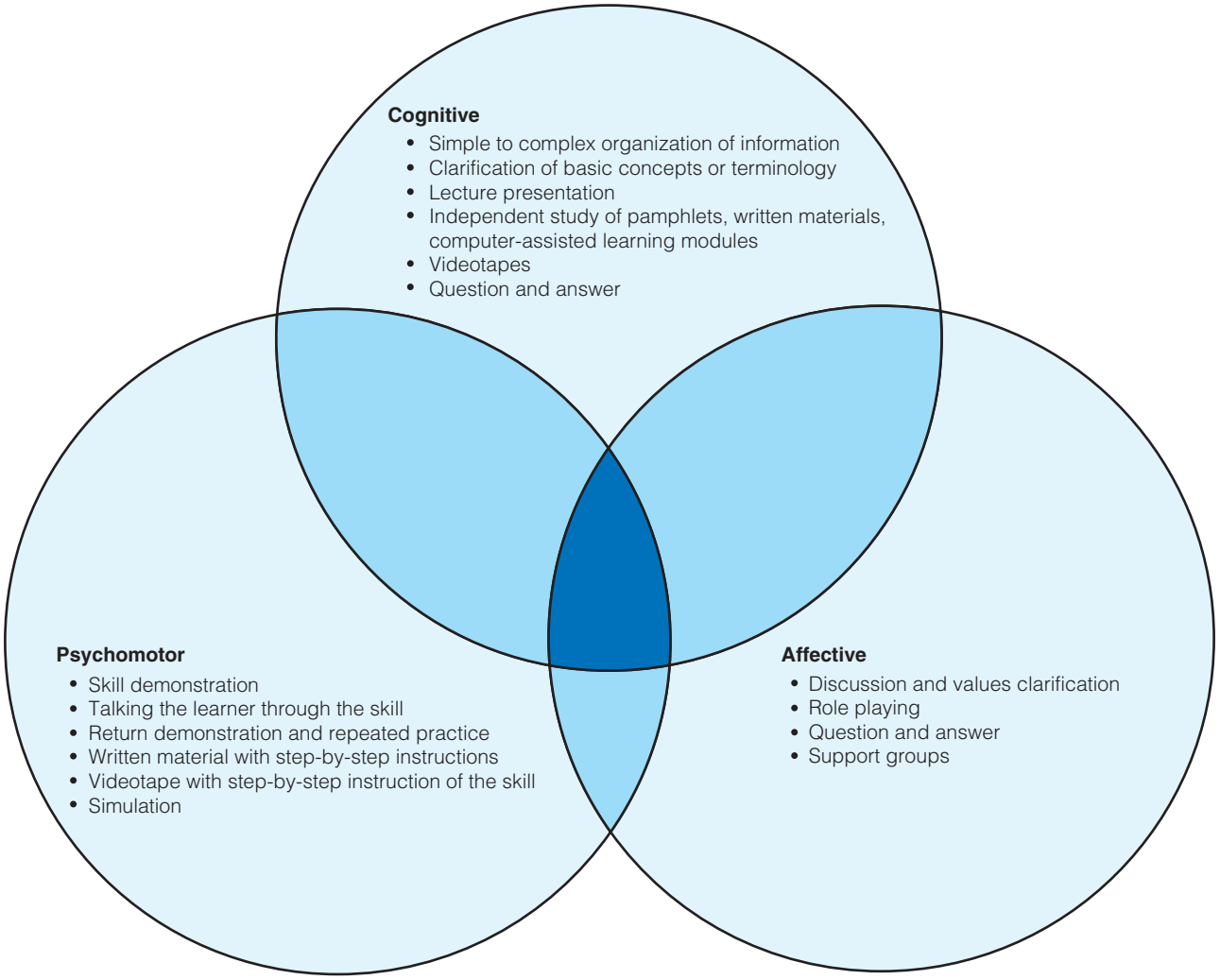


FIGURE 4-1 ▲ Teaching methods based on the domains of learning.

3. *The learner's life experience.* Adults have lived longer and accumulated more life experiences than children. Life experience defines and shapes adult beliefs, values, and attitudes. Adult education methods emphasize experiential techniques such as case method, simulation, and problem-solving exercises. In addition, adults learn well from their peers, making group learning an effective teaching method.
4. *Readiness to learn.* Adults are ready to learn the things they need to know. The information should be applicable to real-life situations.
5. *Orientation to learning.* Adults are motivated to learn if the information will help them to perform useful tasks or to deal with problems in their life.
6. *Motivation to learn.* Adults are more motivated by internal forces such as improved quality of life, increased job satisfaction, and improved self-esteem. External factors such as job promotion or increased salary are less likely to sustain learning.[13]

An example of how the critical care nurse might use adult learning principles in the practice setting is presented in the following scenario.

CASE STUDY

A Patient Who Is Motivated to Learn

Mr. Murray underwent a coronary artery bypass graft procedure 2 days ago. He questions the nurse about a breakfast tray that contains scrambled eggs and ham, while explaining that the eggs contain too much cholesterol and that he has been told to avoid all high-cholesterol foods. The nurse replies that the scrambled egg is an egg-substitute product that is actually part of a heart-healthy diet. Mr. Murray is demonstrating his readiness to learn and is attempting to apply the new knowledge to alter his eating habits. His learning motivation stems from an intrinsic desire to change that is now focused on his overall quality of life and improving his health. His question affords the nurse an opening to discuss other heart-healthy activities and lifestyle changes that will help the patient achieve wellness in his recovery.

The critically ill patient and family are highly motivated to learn because a life-threatening event has triggered an intense need for information. Successful teaching plans should incorporate adult learning principles and relevant information that readily apply to real life and assist with recovery from critical illness.

▲ The Process of Adult Education

The process of patient and family education entails more than just providing an educational brochure or turning on an instructional videotape; it is an interactive process based on a therapeutic relationship. The fundamentals used in patient and family education include assessment, diagnosis, goals, intervention, and evaluation.[14] Frequently, the nursing process is used informally by critical care nurses because teaching is so highly integrated into routine nursing care and family interactions. Just as the bedside nurse uses clinical judgment to recognize and treat the hemodynamic instability that often accompanies critical illness, he or she also diagnoses and intervenes to meet the learning needs of the patient and family. As nurses advance in practice, learning assessment becomes more refined and focuses on meeting the educational goals. Each learning session enhances the knowledge of the patient and family and offers the nurse a chance to evaluate the success or failure of what he or she has taught.

Assessing Learning Needs in a Time of Crisis

The critical care nurse must be very sensitive to the heightened anxiety that accompanies an admission to the ICU. Anxiety markedly reduces the ability of the patient and family to concentrate. Therefore, the nurse should avoid long explanations or tedious questions. The first step in the assessment process is to get to know the patient and family. This often begins with a simple introduction. Taking a few minutes to learn the family names and their relationship to the patient signifies respect and begins to build a therapeutic and trusting relationship. It gives the nurse a chance to orient the patient and family to the ICU as well as to teach them about some of the equipment used in the care of the patient.

Assessment of the learning needs can be a formal or informal process. In the critical care setting, it is preferable to use an informal and open-ended dialogue between the nurse and the family to establish the "need to know." Use of open-ended questions such as "What is your understanding of your mother's condition?" or "What did the physician tell you about the surgery?" gives the nurse a starting point for teaching the family. It also validates whether the patient or family member clearly understands previous explanations given by other members of the health care team.

Informal assessment often provides the nurse with a baseline evaluation of literacy and the person's level of education. Literacy assessment can be very difficult and requires sensitivity because most adults with a reading difficulty spend a lifetime hiding it.[14] Nonthreatening questions such as, "Do you prefer to learn new information by reading or watching a program on television?" may give the nurse a clue about a patient or family member's level of literacy. With about 20% of the U.S. population considered to be functionally illiterate, it is very likely that the educational brochures or the operative consent forms that are given to patients or families are beyond their reading level.[14] Every day, critical care nurses assume that the consent forms are clearly understood when they are returned signed and unquestioned. Written educational material should always be in the active voice and targeted for a 7th- to 8th-grade reading level.[15] In addition, the nurse should verbally review any written material with the patient or family in case they are unable to read the document and are too embarrassed to admit it.

Further assessment may reveal that a patient or family has a low level of health literacy. This term describes "the degree to which individuals have the capacity to obtain, process, and understand basic health information and services needed to make appropriate health decisions."[16] Within the context of critical care, patients and families with low health literacy often struggle with the urgency and the abstract concepts that health care decisions demand.[17] For example, a physician explains the risks and benefits of a tracheostomy for a patient who has been intubated for 3 weeks and cannot be weaned from mechanical ventilation. To fully comprehend the assignment of risk, it is necessary to have a basic understanding of the concept of percentages as applied to the probability of complications. In this situation, a family member may have difficulty making an informed decision because the risks and benefits are not meaningful, and they are fearful of "cutting a hole into a loved one's neck." Such an emotional reaction should signal to the critical care nurse the need to bridge the knowledge gap between the physician's explanation and the family's negative perception of the procedure. To accomplish this, it helps to use more concrete methods such as the phrase "most people experience few complications following this procedure" to illustrate the risk for complications instead of using percentages to quantify the risk. In addition, using familiar words, showing a picture of a tracheostomy, and giving family members time to talk about the procedure may dispel further misconceptions and reduce their anxiety, thus enabling them to reach a decision about the surgery.

Assessment is a dynamic and ongoing process, providing the critical care nurse with many opportunities to assist patients and families to cope with the stress and anxiety associated with critical illness while meeting their learning needs. It also entails knowing when the patient or family is unable to learn. For example, patients who are experiencing pain are not able to focus on learning a new skill such as insulin administration without first having adequate pain control. A family member who has just learned that a loved one has suffered a cardiac arrest is not likely to be able to assimilate the intricate details of myocardial ischemia. Setting unrealistic educational goals hinders learning and frustrates both the nurse and the learner. The teaching plan must be continually evaluated. If the plan is ineffective, poorly timed, or not meeting the learner's needs, it should be altered.

Intervention: Effective Teaching Strategies in Critical Care

Learning Opportunities

Life-threatening illness often stimulates changes in unhealthy behavior patterns, only then igniting a patient's interest in learning. Much of the learning required of a patient who is recovering from critical illness involves behavior changes that require alterations in lifestyle. Smoking cessation, dietary restrictions, and activity limitations are the types of

lifestyle changes that patients frequently struggle to achieve and maintain. Learning opportunities often present in the course of routine patient care. Therefore, the nurse should be ready to incorporate teaching while providing care. For example, the nurse can review postoperative incision care in a brief teaching session while performing skin assessment. The review can include the signs and symptoms of infection, proper wound cleansing, and a description of a healthy healing incision. Teaching pertinent information about the indications or side effects of medications, while giving them to the patient, is another way to reinforce learning. Both of these examples highlight the importance of focusing on a single concept, especially considering the limited attention span that is typical of a patient who is recovering from a critical illness. Learning is best accomplished when the message is consistent and the knowledge progresses from simple to more complex concepts.

The Family Connection

Often the critical care nurse recognizes the limitations of the patient's ability to comprehend information and then turns to the family to provide instruction. Most patients forget 80% of the information they receive, and nearly half of what they remember is incorrect.[18] It is likely that retention of information by critically ill patients is much worse. Therefore, family participation in teaching sessions helps ensure the success of the teaching plan. In addition, providing written materials that patients can review after discharge from the hospital helps bridge the retention gap. Guidelines for developing printed materials that are appropriate for use with older patients are presented in Box 4-3.

Another effective teaching strategy for the adult learner is group learning. For example, postoperative cardiac surgical patients can benefit from a class on posthospital care. A group teaching session allows patients the chance to share common experiences and concerns about recovery with each other. Including families in the group may stimulate questions and allow them to express concerns about potential complications and fears about taking care of a loved one at home. Many times, families are fearful of caring for a loved one who has recovered from a life-threatening illness. They are afraid that they will miss an important symptom or that something will go wrong and their loved one will become ill again or even die. The critical care nurse should acknowledge these feelings and provide the family with emotional support while giving the tools and information to ensure safe care at home. The family interaction also provides the nurse with an assessment of potential home health care needs. A newly learned skill such as a complex dressing change or administration of an IV medication requires both the patient and a family member to learn and demonstrate the basic steps of the procedure to ensure that adequate learning has occurred before the patient leaves the hospital. It may become apparent that a home health care consultation is indicated for further teaching, and reinforcement of newly learned skills may be needed to facilitate a safe transition from the hospital to home.

Hospitals have expanded education tools to include computer-based educational libraries and other audiovisual systems that can be used to supplement individual instruction. The content can be especially helpful for patients and families who are learning a new skill, such as intermittent urinary catheterization. Some education systems are quite sophisticated, linking the health care provider to the patient by "prescribing" specific education content. The computer software can be interactive with the learner through a series of questions and answers that can assist the nurse with validating the learning success of the patient or family member. In addition, electronic interfaces to the education system can provide basic documentation of the teaching session in the medical record. This deliberate and interactive process between the health care provider, the nurse, and the patient and family helps to facilitate education. Such checks and balances are designed to ensure that the patient and family are exposed to basic information before they are discharged from the hospital. Electronic patient education libraries also provide consistent information and are less dependent on the teaching skills of a particular nurse to get the information across to the patient and family. Moreover, these patient education libraries are often available in multiple languages and can reduce the need for an interpreter for each learning session. Regardless of the level of sophistication of electronic learning tools, an interpreted session remains an important tool to ensure that the patient and family have learned the required information and that questions are answered thoroughly.

BOX 4-3 CONSIDERATIONS FOR THE OLDER PATIENT

Guidelines for Printed Educational Materials

- Font should be 12 points or greater.
- Serif type is preferred over sans-serif type.
- Avoid script or stylized types.
- Use boldface headings.
- Avoid using all uppercase letters for body type.
- Use specific language and avoid generalizations.
- Use "calls to action" to highlight important points.
- Use four to five lines of text broken up with white space.
- Avoid paper with a glossy finish because the glare makes reading difficult. Use matte-finish paper instead.
- Enhance legibility by using black ink printed on white or off-white paper.
- Avoid printing over a designed or customized background.

Evaluating the Learning Process

Evaluation is a measurement of the critical learning elements established in the teaching plan. It provides evidence about patient accomplishments or skills that may need further development.[19] Evaluation also reinforces correct behavior on the part of learners and helps teachers determine the adequacy of their instruction.[19] Questions provide the teacher and learner with immediate feedback and validate the learner's grasp of the information presented. The nurse should avoid using leading questions to achieve a desired answer. True evaluation is based on the learner's responses, which indicate whether additional reinforcement of the key concepts is needed. Direct observation of newly learned skills or procedures should also be part of the evaluation. Because adults do not want to appear awkward or clumsy when performing a task, it is important to have a relaxed, positive learning environment where the teacher and student have a good rapport before asking a patient or family member to

demonstrate a new task or skill. The learner should be able to successfully answer or perform 94% of all the critical elements outlined in the teaching plan.[19] Often the success or failure of patient and family education influences the discharge plans. Patients who are unable reliably to perform new tasks need supervision and further practice to learn the new skill. Therefore, adequate evaluation of the learning process is an essential component of the health care continuum.

There are many ways to develop a patient and family teaching plan. Given a homogeneous population, standardized patient teaching plans and records can be used. Plans should include information that is essential for most patients but can also be flexible enough to accommodate individual needs. Teaching plans include nursing diagnoses, outcome criteria, and interventions. Box 4-4 provides a sample teaching plan for a patient who has experienced a myocardial infarction.

BOX 4–4 *COLLABORATIVE CARE GUIDE for Educating a Patient About Myocardial Infarction*

Mr. Mitchell is a 54-year-old married African-American man who was admitted to the hospital after experiencing chest pain while at work. He had mild elevation in his cardiac enzyme levels and was taken to the cardiac catheterization laboratory within a few hours of the onset of the chest pain. The interventional cardiologist found two arteries 80% occluded. He was able to perform an angioplasty, place a stent in the arteries, and successfully restore the blood flow to the affected myocardium. The morning after his procedure, Mr. Mitchell spoke with the cardiac rehabilitation nurse, who reviewed his cardiac risk factors. He is 30-pound overweight with mild hypertension, his cholesterol level is 250 mg/dL, and he smokes 2 packs of cigarettes per day. After this procedure, Mr. Mitchell is anxious to learn ways to reduce his risk for myocardial infarction.

Nursing Diagnoses	Outcomes	Interventions
Deficient Knowledge	Patient will be able to state content presented.	• Plan teaching sessions for a period of time with minimal interruptions. • Include patient's wife and family in teaching sessions. • Provide written information to reinforce the verbal information. • Review the diagnosis of myocardial infarction and the therapies used to prevent further damage to the heart muscle. • Review cardiac risk factors for this patient and identify those risk factors that he can control. • Consult the dietitian for weight reduction and meal planning. • Discuss the sodium content of foods related in order to control his hypertension. • Discuss the target cholesterol level for this patient and the medications and dietary changes needed to reduce the risk for myocardial infarction. • Discuss tobacco use related to myocardial oxygen demand and the vasoactive effects of nicotine. • Offer information about smoking cessation programs and medical options available to assist him with stopping smoking. • Refer the patient to a formal cardiac rehabilitation program after discharge from the hospital.
Readiness for Enhanced Knowledge	Patient will participate in goal setting for weight loss, tobacco cessation, and achievement of cholesterol and blood pressure targets. The patient will participate in an exercise program, lose weight, stop smoking, and decrease cholesterol according to his personal goals.	• Plan and set goals with the patient for weight loss, cessation of tobacco use, and target cholesterol levels. • Have the patient identify appropriate menu selections and portion control to achieve weight loss. • Have the patient identify the triggers for tobacco use and steps that he might find useful to reduce or stop smoking. • Refer the patient to a weight-loss and a smoking-cessation support group. • Have the patient identify appropriate exercises after myocardial infarction.
Ineffective Coping	Patient will begin to demonstrate effective coping mechanisms.	• Discuss the patient's feelings about multiple lifestyle changes and the diagnosis of myocardial infarction. • Discuss the patient's feelings about participating in both a weight-loss and smoking-cessation program. • Mobilize the patient's resources for support. • Help the patient to design a chart to map his progress with weight loss, smoking cessation, and blood pressure and cholesterol target goals. • Acknowledge all questions and concerns that the patient expresses as meaningful.

The nursing diagnosis assists the nurse to identify the appropriate content to teach. It also helps formulate the outcomes that are used to evaluate the progress of the patient and family and the effectiveness of the teaching plan. The outcomes should be stated in measurable terms, and the teaching should be outlined in a logical sequence. Each nursing intervention should include the content, method, and media used for teaching. In addition, the patient's barriers to learning should be addressed, and the nursing interventions are aimed at meeting those personal needs. In critical care, families are often included in the teaching plans because of the limited learning ability of the patient.

▲ The Standards of Patient and Family Education

There is great emphasis on patient and family education that stems from the Joint Commission patient care standards. These standards serve to promote overall patient care quality in health care organizations. Hospitals voluntarily participate in Joint Commission surveys to ensure that the patient care provided meets or exceeds the criteria set forth in the standards. Some examples of Joint Commission standards related to patient and family education include the following:

- "The hospital provides, coordinates, and evaluates patient education and training based on each patient's needs and abilities."
- "The hospital performs a learning needs assessment which includes cultural and religious beliefs, emotional barriers, desire and motivation to learn, physical or cognitive limitations, and barriers to communication."[20]

The goal of these educational standards is to guide hospitals to create an environment in which both the patient and the health care team members are responsible for teaching and learning. The medical record should reflect an interdisciplinary approach toward patient education throughout the hospital stay. The initial nursing history should include an assessment of the patient's understanding of the current health issues, preferred learning methods, family support, and any social or cultural beliefs that will impact learning. The Joint Commission recommended educational topics are detailed in Box 4-5. Teaching documentation should indicate the learning topic and how it relates to the patient's health issues. In addition, if home health care or community resources are necessary to support the safe transition of the patient from the hospital to home, the medical record should reflect coordination of these services prior to discharge.

Finally, the teaching record should also reflect an evaluation of how well the patient and family absorbed the information. The details of teaching documentation are outlined in Box 4-6. A sample patient education record is shown in Figure 4-2.

How do these standards affect patient education in the critical care setting? It may be difficult for critical care nurses to think in terms of teaching plans and interdisciplinary learning because critically ill patients have so much instability and require great vigilance just to maintain physiological function. However, remember that much of the patient teaching is informal and may not be clearly visible at first glance. Nurses are taught to explain each procedure, medication, intervention, or diagnostic test to the patient beforehand. This is patient education. For example, the nurse who explains that the bag of liquid medication she is hanging is an antibiotic that is given through the intravenous line to fight the patient's abdominal wound infection is teaching. Yet, many nurses would not recognize this action as patient education, and they would not document it in the teaching record. Nonetheless, this type of informal instruction meets the Joint Commission standard for patient education. Critical care nurses teach patients and families routinely, but often the patient education record is left blank because "there isn't enough time to teach." If critical care nurses would only remember to note each informal teaching session, the patient's educational records would be filled with entries after just 1 day in the unit.

BOX 4-5 The Joint Commission–Recommended Topics for Patient and Family Education

- Explanation of the plan of care
- Basic health practices and safety
- Safe and effective use of medications
- Nutrition interventions
- Pain management
- Safe and effective use of medical equipment
- Techniques that help the patient reach maximum independence
- Fall reduction strategies

Adapted from 2010 Comprehensive Accreditation Manual for Hospitals. Edition Release 2.5. Accessed August 29, 2010 http://e-dition.jcrinc.com/Standard.aspx

BOX 4-6 Components of Teaching Documentation

- Participants (Who was taught?)
- Content (What was taught?)
- Date and time (When was it taught?)
- Patient status (What was the patient's condition at the time?)
- Evaluation of learning (How well was the information absorbed?)
- Teaching methods (How was the patient taught?)
- Follow-up and learning evaluation (If teaching was incomplete, what was the reason? What additional education needs does the patient have?)

KEY

Barriers to Learning	**Learners**	**Tools/Method**	**Level of Learning (LOL)**
1. No barrier 2. Language/Communication/Literacy 3. Cultural/Religious Practices 4. Cognitive/Sensory Impairment 5. Severity of illness/pain 6. Motivation 7. Physical limitation	P = patient F = family/significant other O = other	C = Class/group D = Demonstration A = Audiovisual L = Literature T = Translator TV = Video/ed channel M = Model 1:1 One to one	8. Needs further reinforcement 9. Demonstrates partial skill or knowledge 10. Demonstrates skill with minimal assistance 11. Demonstrates competent skill and knowledge

EDUCATION	Content	Date	Barrier	Learner	Tool	LOL	Initials
Outcome Criteria **Discharge Plan of Care** • Identifies disease process • Cause • Signs and symptoms • Risk factors • Prevention							
Medications/ Food and Drug Interactions • Identifies purpose of medications • States side effects of medications • Demonstrates administration of medications • Reviews food–drug interactions							
Activity • Verbalizes activity restrictions after discharge • Identifies need for assistive apparatus as needed • Identifies safety precautions							
Equipment • States purpose • Demonstrates correct use of equipment • Identifies safety measures							
Treatments • States the purpose of the treatment • Demonstrates correct technique • Identifies findings that should be reported to health care provider							
Follow-up Care and Community Resources							

FIGURE 4-2 ▲ Example of a patient education record. (Adapted from Georgetown University Hospital: Interdisciplinary Patient Education. Washington, DC: Author, 2010.)

▲ Clinical Applicability Challenges

SHORT ANSWER QUESTIONS

1. From the *Case Study: A Patient and Family in Crisis* on page 40, discuss the strategies used by the nurse to effectively manage the admission phase for the patient's family.
2. Discuss nursing interventions that have effectively reduced patient and family anxiety in a clinical situation that you have encountered.
3. Discuss when patient and family anxiety would preclude all learning.

References

1. Lof L, Berggren L, Ahlstrom G: ICU patients' recall of emotional reactions in the trajectory from falling critically ill to hospital discharge: Follow-ups after 3 and 12 months. Intensive Crit Care Nurs 24: 108–121, 2008
2. Halm MA, Titler MG, Kleiber C, et al: Behavioral responses of family members during critical illness. Clin Nurs Res 2:414–437, 1993
3. Molter NC: Needs of relatives of critically ill patients: A descriptive study. Heart Lung 8(2):332–339, 1979
4. Burr G. Contextualizing critical care family needs through triangulation: An Australian study. Intensive Crit Care Nurs 14(4):161–169, 1998
5. Casarini K, Gorayeb R, Filho A: Coping by relatives of critical care patients. Heart Lung 38 (3):217–227, 2009
6. Verhaeghe S, Defloor T, Van Zuuren F, et al: The needs and experiences of family members of adult patients in an intensive care unit: A review of the literature. J Clin Nurs 12:501–509, 2005
7. Davidson JE, Powers K, Kamyar M, et al: Clinical practice guidelines for support of the family in the patient-centered intensive care unit: American College of Critical Care Medicine Task Force 2004–2005. Crit Care Med 35(2):605–622, 2007
8. Pattison N: Psychological implications of admission to critical care. Br J Nurs 14(13):708–714, 2005
9. Ortman JM, Guarneri CE: United States population projections 2000–2050. U.S. Census Bureau: August 28, 2010. Available at http://www.census.gov/population/www/projections/2009projections.html
10. Ersek M, Kagawa-Singer M, Barnes D, et al: Multicultural considerations in the use of advance directives. Oncol Nurs Forum 25:1683–1690, 1998
11. Galanti GA: Caring for Patients from Different Cultures, 4th ed. Philadelphia, PA: University of Pennsylvania Press, 2008, pp 2–40
12. National Association of the Deaf Law Center: Obligations of Hospitals and Nursing Homes to Provide Interpreters and Auxiliary Aids for the Deaf and Hard of Hearing patients: August 22, 2010. Available at www.deaf-talk.com/compliance.html
13. Knowles MS, Holton EF, Swanson RA: The Adult Learner, 5th ed. Houston, TX: Gulf Publishing, 1998, pp 35–72
14. Redman BK: The Practice of Patient Education: A Case Study Approach, 10th ed. St. Louis, MO: Mosby Elsevier, 2007, pp 1–26
15. Robbins M: The $73 billion hidden problem of the health care industry. Employee Benefit News. June 1, 2008. Available at http://ebn.benefitnews.com
16. Glassman P: Health literacy: National Network of Libraries of Medicine nnlm.gov. September 5, 2010. Available at http://nnlm.gov/outreach/consumer/hlthlit.html
17. Riley JB, Cloonan P, Norton C: Low health literacy: A challenge to critical care. Crit Care Nurs Q 29(2):174–178, 2006
18. Kessels RPC: Patients' memory for medical information. J Royal Soc Med 96:219–222, 2003
19. Redman BK: The Practice of Patient Education: A Case Study Approach, 10th ed. St. Louis, MO: Mosby Elsevier, 2007, pp 56–73
20. The Joint Commission: Comprehensive Manual: CAMH for Hospitals: The Official Handbook. Joint Commission Resources. 2010, e-dition release 2.5. Available at http://e-dition.jcrinc.com/Standard.aspx

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

5 Relieving Pain and Providing Comfort

Cynthia L. Renn and Tara Leslie

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Differentiate between acute and chronic pain
2. Identify factors that exacerbate the experience of pain in the critically ill
3. Prepare patients for the common sources of procedural pain in intensive care
4. Compare and contrast tolerance, physical dependence, and addiction
5. Discuss national guidelines and standards for pain management
6. Identify appropriate analgesics for high-risk critically ill patients
7. Describe nonpharmacological interventions for alleviating pain and anxiety

Pain is one of the greatest stressors and most common symptoms for critically ill patients.[1,2] In addition to being a coexisting symptom of critical illness, patients experience increased levels of pain during many critical care interventions and routine procedures.[3,4] Even though pain management has become a national priority in recent years, pain continues to be misunderstood, poorly assessed, and undertreated in intensive care units (ICUs) and many other health care settings.[4,5] Uncontrolled pain triggers physical and emotional stress responses, inhibits healing, increases the risk of other complications, and increases the length of ICU stay. Critical care nurses need a clear understanding of concepts related to pain assessment and management to achieve effective pain control. For an overview of the physiological processing of pain, refer to Chapter 32. Chapter 5 provides an overview of key concepts related to managing acute pain and comfort in the critically ill adult patient.

▲ Pain Defined

Pain is a complex, subjective phenomenon. It is a protective mechanism, causing one either to withdraw from or to avoid the source of pain and seek assistance or treatment. The International Association for the Study of Pain defined pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage."[6] McCaffery provides an operational definition of pain that considers the subjectiveness and individuality of the pain experience. This definition is based on the premise that the individual experiencing the pain is the true authority: "Pain is whatever the experiencing person says it is, existing whenever he or she says it does."[7]

Not all pain is the same. The various types of pain are categorized based on the duration (acute or chronic) and source (somatic, visceral, or nerve) of the pain. It is essential that the type(s) of pain a patient is experiencing be properly identified so that the most efficacious management strategies can be implemented. The type of pain that most ICU patients experience is classified as *acute*. Acute pain is a physiological response to an identifiable cause, is generally time-limited, and typically responds well to opioid and nonopioid therapies. For example, the pain experienced during endotracheal suctioning or a dressing change is expected to end when the treatment is completed. Similarly, pain at an incision or area of injury is expected to cease once healing has occurred. Often, patients in the critical care setting will experience pain from more than one source. For example, a postoperative patient will likely have somatic pain stemming from the site of the incision, visceral pain from organs that were manipulated, and possibly, nerve pain from nerves that were cut or damaged during the surgical procedure.

In contrast, *chronic* pain is caused by physiological mechanisms that are less well understood. Chronic pain differs from acute pain in terms of etiology and expected duration. Chronic pain extends past the usual course of an acute illness or injury for an indefinite period of time, usually exceeding 3 to 6 months. It is difficult to treat, responds poorly to routine pain management strategies, and adversely affects the quality of life for the individual.[8] It is essential for the critical care nurse to be aware that, in addition to acute pain associated with the illness or injury leading to hospitalization, ICU patients will frequently have a concomitant chronic pain condition that must be properly managed as well.

▲ Pain in the Critically Ill

Acute critical illness, regardless of the etiology, is painful. Consider the most common illnesses or injuries treated in the ICU: myocardial infarction, thoracic surgery and neurosurgery, multiple trauma, and extensive burns. All of these conditions are associated with severe pain. Therefore, nearly all ICU patients will experience moderate to severe acute pain.[9] Moreover, ICU patients with chronic critical illnesses are likely to experience a combination of both acute pain (due to a new illness, injury or procedure) and the chronic pain stemming from the chronic illness. Further, chronic pain in a critically ill patient is often associated with additional symptoms, such as respiratory distress, fatigue, and cognitive impairment, that must be managed.[10] Previously, it was thought that critically ill patients were unable to remember their painful experiences because of the acute nature of the illness or injury. More recent research, however, demonstrates that ICU patients do remember painful experiences, and they frequently describe their pain as being moderate to severe in intensity.[4]

In addition to the patient's illness-, injury- or procedure-induced pain, the critical care nurse must also be cognizant of the many factors inherent to an ICU admission and the ICU environment that will increase the patient's pain experience (see Box 5-1). Each of these factors alone will exert a significant negative effect on the patient's pain and, when they are experienced in combination, they act synergistically to further increase pain. For example, pain and anxiety act in a cyclical fashion to exacerbate each other.

BOX 5-1 Factors Contributing to Pain and Discomfort in the Critically Ill

Physical
Symptoms of critical illness (eg, angina, ischemia, dyspnea)
Wounds—posttrauma, postoperative, postprocedural, or penetrating tubes and catheters
Sleep disturbance and deprivation
Immobility, inability to move to a comfortable position due to tubes, monitors, restraints
Temperature extremes associated with critical illness and the environment—fever, hypothermia

Psychosocial
Anxiety and depression
Impaired communication, inability to report and describe pain
Fear of pain, disability, or death
Separation from family and significant others
Boredom or lack of pleasant distractions
Sleep deprivation, delirium, or altered sensorium

Intensive Care Unit (ICU) Environment or Routine
Continuous noise from equipment and staff
Continuous or unnatural patterns of light
Awakening and physical manipulation every 1 to 2 hours for vital signs or positioning
Continuous or frequent invasive, painful procedures
Competing priorities in care—unstable vital signs, bleeding, dysrhythmias, poor ventilation—may take precedence over pain management

▲ Procedural Pain

Efforts to provide pain relief and comfort measures are complicated by the fact that critical care nurses must continuously perform procedures and treatments that cause pain to the patient. Procedures like chest tube insertion and removal, endotracheal suctioning, and wound débridement are obviously painful. Less obvious are the simple procedures, such as turning and positioning, that can also cause considerable pain for critically ill patients.

Several studies have investigated which routine nursing procedures were reported to be painful or not painful by conscious oriented patients.[4,11] The investigators documented the patients' responses to six procedures that are frequently performed on critically ill patients: position changes, tracheal suctioning, deep line removal, deep breathing and coughing exercises, dressing changes, and drain removal. The findings of this study are summarized as the least painful to the most painful procedures in Table 5-1.[4] Studies also found that procedural pain is often described by patients as different from other types of pain; descriptors included "intense," "episodic," "severe," and "tiring."[12,13] It was also found that 50% of patients undergoing drain removal, 38.5% of patients doing deep breath and coughing exercises, and 32.6% of patients undergoing position changes received preemptive analgesia with morphine within an hour prior to their procedure, while only 12.5% of the patients undergoing tracheal suctioning, reported by patients to be one of the more painful procedures, received prophylactic morphine.[4] Approximately 79% of patients undergoing a painful procedure reported inadequate pain management with only 22.1% receiving preemptive analgesia prior to the procedure, 84.1% receiving an analgesic medication during the procedure, and only 7.3% receiving analgesic pain medication postprocedure.[14] Critical care nurses must be attuned to the pain the patient is experiencing before the procedure and anticipate the pain that occurs as a result of the procedure in order to provide the best interventions to help the patient during the procedure.

Before performing procedures known to be associated with pain, patients should be premedicated (preemptive analgesia), and the procedure should be performed only after the medication has taken effect. During procedures, intravenous (IV) opioids, such as morphine or fentanyl, are

Table 5-1 Patients' Self-Report of Painful Procedures*

Procedure	Level of Pain
Simple dressing change	Least painful
Central line removal	Painful
Position changes/turning	Painful
Tracheal suctioning	More painful
Deep breathing and coughing exercises	More painful
Drain removal	Most painful

*Ranked from least to most popular.

From Siffleet J, Young J, Nikoletti S, et al: Patients' self-report of procedural pain in the intensive care unit. J Clin Nurs 16(11):2142–2148, 2007.

usually used for analgesia. The IV bolus dose of morphine is individualized and depends on the age, weight, pain intensity, and type of procedure. The patient's response must be monitored during the procedure with additional doses given as needed for breakthrough pain. Anxiolytic medications, such as midazolam or propofol, can be given to relieve anxiety during the procedure; however, these agents should *only* be used as *adjuncts*, because they simply provide sedation and no analgesia to relieve the pain associated with the procedure. In addition to providing preemptive analgesia and anxiolytic medications before the procedure, the nurse should educate patients about the procedure to help them prepare for anticipated pain and discomfort. Further, the nurse can use interventions, such as imagery, distraction, and family support during procedures, to supplement the effect of the analgesic and anxiolytic medications.

▲ Consequences of Pain

Pain produces many harmful effects that impact negatively on the function of all body systems, inhibit wound healing, and slow recovery from critical illness. The autonomic nervous system responds to pain by causing vasoconstriction and increased heart rate and contractility. Additionally, pulse, blood pressure, and cardiac output all increase, leading to concomitant increases in myocardial workload and oxygen use, both of which can cause or exacerbate myocardial ischemia in an already compromised critically ill person. Patients in pain are also hesitant to move, cough, or breathe deeply because the associated movements increase pain. This decrease in movement manifests as pain-induced respiratory alterations that include splinting, decreased respiratory effort, and reduced pulmonary volume and flow. These pain-induced respiratory alterations can then lead to pulmonary complications, such as atelectasis and pneumonia. In the gastrointestinal system, undertreated pain can cause decreased gastric emptying and intestinal motility, which can result in impaired function and ileus.

Unrelieved pain also negatively affects the musculoskeletal system by causing muscle contractions, spasms, and rigidity and by suppressing immune function, which predisposes the patient to pneumonia, wound infections, and sepsis. The negative effects of uncontrolled pain on the course of a critical illness are clear. It is vital for the critical care nurse to understand that the impact of uncontrolled pain during a critical illness can extend beyond the time of recovery from the critical illness. Evidence shows that patients who have a high level of uncontrolled pain during an acute hospitalization are at increased risk for delayed recovery and the development of chronic pain syndromes after discharge.[15]

Patients who are pain free have better outcomes than those stressed by unrelieved pain. In a classic study, patients whose pain was controlled with epidural anesthesia and epidural analgesia had shorter ICU stays, shorter hospital stays, and half as many complications as patients receiving standard anesthesia and analgesia.[16] The benefits of effective pain relief are summarized in Table 5-2.

Table 5-2 Benefits of Effective Pain Relief

System	Benefit
Cardiovascular	Decreased pulse rate, blood pressure, and myocardial workload
Respiratory	Enhanced respiration, oxygenation, ability to perform deep breathing and coughing exercises, and decreased incidence of pulmonary complications
Neurological	Decreased anxiety and mental confusion, enhanced sleep
Gastrointestinal/ Nutritional	Enhanced gastric emptying, promotion of positive nitrogen balance, increased appetite
Musculoskeletal	Earlier ambulation, reducing complications of immobility
Economic	Decreased length of stay, decreased costs, enhanced patient satisfaction with care

▲ Barriers to Effective Pain Control

Pain continues to be undertreated in many settings, even though the negative consequences of uncontrolled pain and the benefits of pain relief have been well documented. Often pain relief is relegated to a low priority due to the life-threatening nature of the patient's illness and the other life-saving interventions that are required. Critical care nurses are often concerned that analgesic administration may create problems, such as hemodynamic and respiratory compromise, oversedation, or drug addiction.[17]

The fear of addiction is one of the greatest concerns and impediments associated with analgesia and pain control. This fear causes anxiety for patients and their families as well as health care providers. Critical care nurses must have a clear understanding of the differences between, and implications of, addiction, tolerance, and dependence. Patients who require long-term analgesic medication for pain control can develop tolerance or physical dependence. However, these scenarios should not be confused with addiction, which is characterized by behaviors, such as impaired control, compulsive use, and continued use despite serious negative physical and/or social consequences.[18] See Table 5-3 for clarification of these concepts.

Patients with a history of opioid addiction and tolerance provide a unique challenge to nurses trying to manage pain when these patients present with a critical illness. This particular patient population may require much larger doses of pain medication to produce an adequate level of pain control than a patient not addicted to opioids. Research shows that patients with opioid addiction often require 30% to 50% more opioid pain medication than they were taking preoperatively. Continuous epidural delivery and patient-controlled analgesia (PCA) are often beneficial in these patients due to the continuous infusion of opioid medication. Opioid withdrawal is another significant concern when caring for an opioid-addicted patient. The onset of withdrawal can occur between 6 and 48 hours after the last use, depending on the drug.[19] Symptoms of withdrawal may include flu-like symptoms

Table 5-3 Tolerance, Physical Dependence, and Addiction

Condition	Definition	Implication
Tolerance	A state of adaptation in which exposure to a drug induces changes that result in a diminution of one or more of the drug's effects over time.	Increase dose by 50% and assess effect. Tolerance to side effects, such as respiratory depression, will increase as the dose requirement increases.
Physical dependence	A state of adaptation that is manifested by a drug class–specific withdrawal syndrome that can be produced by abrupt cessation, rapid dose reduction, decreasing blood level of the drug, and/or administration of an antagonist.	Gradually taper opioid dosage to discontinuation to avoid withdrawal symptoms.
Addiction	A primary, chronic, neurobiological disease, with genetic, psychosocial, and environmental factors influencing its development and manifestations. It is characterized by behaviors that include one or more of the following: impaired control over drug use, compulsive use, continued use despite harm, and craving.	Rarely seen in critical care patients, unless patient is admitted for drug overdose or other sequelae of illicit drug use

Definitions from American Pain Society: Definitions related to the use of opioids for the treatment of pain. Available at: http://www.ampainsoc.org/advocacy/opioids2.htm. Accessed April 1, 2010

(nausea, vomiting, diarrhea, headaches, muscle/joint/joint pain, fever), tachycardia, dilated pupils, runny nose, lacrimation, piloerection (hair standing on end), yawning, restlessness, sweating, irritability, anxiety, and hypertension. Although opioid withdrawal does not tend to be fatal, underlying medical conditions (hypertension or recent myocardial infarction) may increase the patient's risk for death.[20] Important to note is that withdrawal can also occur in patients who do not have a history of opioid drug abuse, but who have been treated for an extended period of time with opioids to control pain. In this patient population, it is vital to taper (incrementally decrease) the use of the opioid and avoid an abrupt stoppage of the drug.

Pain treatment should not be restricted in patients with a history of opioid abuse; rather, it should be more aggressive. The period of critical illness in these patients is not the appropriate time for drug rehabilitation attempts, and the symptoms associated with opioid withdrawal can exacerbate conditions related to the critical illness. The critical care nurse must focus on providing all necessary measures to allow a full recovery from the illness, which can then be followed by a proper referral for drug-dependence rehabilitation. However, though the incidence is fairly small, the nurse should be alert to the possibility of a patient feigning symptoms to seek more than required opioid pain medication. In all cases, the nursing care for substance-abusing patients should be focused on forming a nonjudgmental, trusting relationship between the nurse and the patient.[19]

▲ Resources to Promote Effective Pain Control

In recent decades, government agencies, professional organizations, health care institutions, and pain management experts have focused attention on improving pain management across the United States. These efforts have produced abundant resources to support nurses in their efforts to provide effective pain management.

Clinical Practice Guidelines

Early in the 1990s, the Agency for Health Care Policy and Research (AHCPR), now known as the Agency for Healthcare Research and Quality (AHRQ), introduced the concept of clinical practice guidelines. These guidelines were intended to serve as nationwide standards of care for specific clinical problems. This concept arose from the recognition that in the midst of a rapidly expanding body of health care research and literature, there were still wide variations in opinions and practice patterns regarding the best interventions for common clinical problems. The AHCPR convened multidisciplinary panels of national experts to review the research, provide expert opinions, summarize the current knowledge, and make recommendations for practice for each targeted clinical problem. Acute pain management was the topic of the first guideline that was published and disseminated by this agency.[21]

Over the next few years, several national agencies, including the American Association of Critical-Care Nurses, the American College of Cardiology, and the Society of Critical Care Medicine (SCCM), assembled their own panels of experts to develop clinical practice guidelines for their target populations. In 1996, the AHRQ discontinued its support for producing clinical practice guideline documents and instead entered into a collaborative relationship with the American Medical Association and the American Association of Health Plans to sponsor the web-based National Guideline Clearinghouse.[21] This Web site currently contains more than 2,500 practice guidelines developed by a variety of organizations. Via these various mechanisms, pain management guidelines have been disseminated throughout the United States and have served as a catalyst for several improvements in pain management. These guidelines are also used as legal documents representing the national standard of care for pain management in medical liability cases.

One practice guideline that is particularly useful to critical care nurses and physicians is the guideline published jointly

Table 5-4 National Standards and Guidelines Related to Pain Management

Agency or Source and Standard or Guideline	Content Highlights
Society of Critical Care Medicine and the American Society of Health-System Pharmacists Clinical Practice Guidelines for the Sustained Use of Sedatives and Analgesics in the Critically Ill Adult[22]	Developed by a national panel of experts in critical care medicine, nursing, and pharmacy. Includes summaries and recommendations of recent research related to analgesia and sedation specifically in the critically ill population. Contains 28 explicit recommendations targeted to the critically ill, including the following: • Patient report is the most reliable standard for pain assessment. • Scheduled doses or continuous infusions of opioids are preferred over PRN or "as needed" regimens. • Fentanyl, hydromorphone, and morphine are the drugs of choice for intravenous (IV) doses of opioid analgesia. • Fentanyl is preferred for rapid onset of analgesia in acutely distressed patients. • Sedation of agitated patients should be provided only after providing adequate analgesia. • Lorazepam is recommended for sedation of most patients via intermittent IV or continuous infusion • Midazolam or diazepam should be used for rapid sedation of acutely agitated patients • Haloperidol (Haldol) is the preferred agent for treatment of delirium (pp 140–141)[22]
American Geriatric Society The Management of Persistent Pain in Older Adults[23] Pharmacological Management of Persistent Pain in Older Persons[24]	This guideline, originally published in 1998, was revised in 2002 and 2009 by an interdisciplinary panel of geriatric experts. Major recommendations include the following: • All older persons should be screened for persistent pain on admission to any health care facility. • The verbal 0 to 10 scale is a good first choice for assessment of pain intensity; however, other scales such as word descriptor scales or pain thermometers may be more appropriate for some older patients. • For patients with cognitive impairment, assessment of behaviors and family observations are essential. • Opioid analgesic drugs are effective, with a low potential for addiction, and may have fewer long-term risks than other analgesic drugs.[23] • Acetaminophen should be the first drug to consider in the treatment of mild to moderate musculoskeletal pain. • Patients with neuropathic pain and other types of refractory persistent pain may be candidates for adjuvant analgesics. • Patients with moderate to severe pain, or continuous pain on a daily basis, that diminishes quality of life or causes functional impairment should be considered for opioid therapy.[24]
American College of Cardiology/American Heart Association Task Force on Practice Guidelines[21]	This joint task force has published multiple practice guidelines that are relevant to painful conditions experienced by critically ill patients, including: • Management of patients with chronic stable angina • Management of patients with unstable angina • Management of patients with peripheral arterial disease • Management of patients with ST-elevation myocardial infarction • Guideline update for coronary artery bypass graft surgery These guidelines are available from the National Guideline Clearinghouse at http://www.guideline.gov/[21]

by the SCCM and the American Society of Health-System Pharmacists on *Sustained Use of Sedatives and Analgesics in the Critically Ill Adult*.[22] Table 5-4 includes information about this and other important guidelines and standards for pain management.[21–24]

Internet Resources

The Internet is one of the most important sources of information and resources for pain management. Table 5-5 lists websites containing pain management information that may be useful to critical care nurses, patients, and families.

▲ Pain Assessment

The failure of staff to routinely assess pain and pain relief is one of the most common reasons for unrelieved pain in hospitalized patients.[25] Assessment of pain is as important as any method of treatment and is the only way to determine the presence and severity of pain. The patient's pain must be assessed at regular intervals to determine the effectiveness of therapy, the presence of side effects, the need for dose adjustment or the need for supplemental doses to offset procedural pain. Pain should be reassessed at an appropriate interval after pain medications or other interventions have

Table 5-5 Internet Resources

Website	Resources Provided
American Chronic Pain Association www.theacpa.org	Offers information and support for people with chronic pain
American Pain Foundation www.painfoundation.org	Resource center for people with pain, their families, friends, care givers, the media, legislators, and the general public
American Society of Pain Management Nurses www.aspmn.org	Information about society membership, conferences, resources, guidelines, and position statements
City of Hope Pain/Palliative Care Resource Center http://prc.coh.org	Resources to assist others in improving pain management and end-of-life care; a source for assessment tools, patient education materials, quality assurance materials, end-of-life resources, and research instruments
National Guideline Clearinghouse www.guidelines.gov	Multiple evidence-based clinical practice guidelines for pain and various other clinical problems; sponsored by the Agency for Healthcare Research and Quality

been administered, for example, 30 minutes after an IV dose of morphine. However, in the critical care setting, a number of conditions may exist that can make assessment of the patient's pain and subsequent treatments difficult. These conditions include the following:

- Acuity of the patient's condition
- Altered levels of consciousness
- Inability to communicate pain
- Restricted or limited movement
- Endotracheal intubation

When any of the above-mentioned conditions are present, a common misconception among health care professionals is that they are the most qualified to determine the presence and severity of the patient's pain, and unfortunately, the absence of physical signs or behaviors is often incorrectly interpreted as the absence of pain. Thus, the patient may suffer unnecessarily. The critical care nurse must make every effort to elicit a self-report from the patient to perform an effective pain assessment. Behavioral observation and changes in physiological parameters can be helpful but should be considered along with the patient's self-report. In those instances when the patient cannot self-report due to sedation or cognitive impairment, an objective tool for assessing the noncommunicative patient should be used.

Pain assessment is an ongoing process. In addition to the initial pain assessment, assessment after pain management interventions and prior to procedures is essential. After pharmacological therapy, pain reassessment should correspond to the time of onset or peak effect of the drug administered and the time the analgesic effect is expected to dissipate.

BOX 5-2 EXAMPLES OF NURSING DIAGNOSES

For the Patient in Pain
- Acute Pain
- Anxiety
- Chronic Pain
- Fear
- Powerlessness
- Ineffective Coping
- Impaired Physical Mobility

Response to therapy is best measured as a change from the patient's baseline pain level. Occasionally, there may be discrepancies between the patient's self-report and behavioral and physiological manifestations. For example, one patient may report pain as 2 out of 10, while being tachycardic, diaphoretic, and splinting with respirations. Another patient may give a self-report of 8 out of 10 while smiling. These discrepancies can be due to the use of diversionary activities, coping skills, beliefs about pain, cultural background, fears of becoming addicted, or fears of being bothersome to the nursing staff. When these situations occur, they should be discussed with the patient. Any misconceptions or knowledge deficits should be addressed and the pain treated according to the patient's self-report. Box 5-2 lists common nursing diagnoses for the patient in pain.

Patient Self-Report

Because pain is a subjective experience, the patient's self-report is considered to be the gold standard and forms the foundation of the pain assessment (remember McCaffery's operational definition). However, family members and care givers can be used as proxies for the patient self-report in situations, such as critical illness, where significant communication barriers can be present.[26] A self-report and/or proxy assessment of pain should be obtained not only when the patient is resting, but also during routine activities and procedures such as coughing, deep breathing, and turning. Critical care nurses are frequently more attuned to objective indicators of pain than to the patient's self-report. If the patient can communicate, the ICU nurse must accept the patient's description of pain as valid. In the conscious and coherent patient, behavioral cues or physiological indicators should *never* take precedence over the patient's self-report of pain. Behavioral and physiological manifestations of pain are extremely variable and may be minimal or absent, despite the presence of significant pain.

During the pain assessment, the nurse should elicit a specific verbal description of the quality of the patient's pain, such as burning, crushing, stabbing, dull, or sharp whenever possible. The location, duration, and exacerbating/alleviating measures should also be established in the assessment process. Often, this descriptive information helps to determine the cause of the pain and the best treatment strategies to employ. All of the information gathered during the pain assessment must be clearly documented in the patient record with a quotation from the patient's self-reported description included.

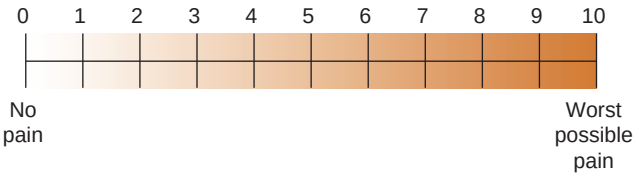


FIGURE 5-1 ▲ Numeric rating scale for pain assessment.

Pain scales and rating instruments based on the patient's self-report provide a simple, but consistent, measure of pain trends over time. It is vital that the same pain scale is used for every pain assessment to produce a valid comparison with previous and subsequent measurements. Numeric rating scales and visual analog scales are used to measure pain intensity. With these scales, the patient chooses a number, word, or a point on a line that best describes the amount of pain he or she is experiencing. The SCCM clinical practice guideline suggests that the numeric rating scale is the preferred type of scale for use in critical care units.[22] With this type of scale, the patient is asked to rate the pain with 0 being no pain and 10 being the worst possible pain imaginable. See Figure 5-1 for an example.

Pictures or word boards can also facilitate communication about the patient's pain. The board should include questions, such as "Do you have pain?" "Where is the pain located?" "How bad is your pain?" and "What helps your pain?" Developing a simple system of eye movements ("blink once for yes and twice for no") or finger movements can be effective for the patient who cannot speak or move his hands.[26]

If the patient is unable to use any of the above methods to verbalize or indicate that he or she is in pain, this can lead to a dilemma when attempting the pain assessment and planning the subsequent treatment. In this situation, it is appropriate to observe for the behavioral cues or physiological indicators discussed in the next section. However, the absence of physiological indicators or behavioral cues should *never* be interpreted as absence of pain. If the procedure, surgery, or condition is believed to be associated with pain, the presence of pain should be assumed and treated appropriately.

Observation

Recent research has demonstrated that ICU nurses can rely on behavioral and physiological indicators of pain in critically ill patients who cannot provide a verbal self-report.[5,26] Nonverbal behaviors, such as guarding, withdrawal, and avoidance of movement, protect the patient from painful stimuli. Attempts by the patient to seek relief, such as touching or rubbing the affected area and changing positions, are palliative behaviors. Crying, moaning, or screaming are affective behaviors and reflect an emotional response to pain. Facial expressions, such as frowning, grimacing, clenching of the teeth, tight closure of the eyes, and tears, can indicate pain. If one or more of these behaviors are present in the nonverbal/noncommunicative patient, it should be assumed that the patient has pain and the proper treatment should be administered.

Patients who are alert and oriented but who are unable to speak may use eye or facial expressions or movement of hands or legs to communicate their pain. Restlessness, agitation, and muscular bracing may be indicators of pain in the nonverbal patient. Since nonverbal cues can be difficult to interpret as indicators of pain, input from family members or other care givers is often helpful in interpreting specific behavioral manifestations of pain based on their knowledge of the patient's behavior before hospitalization.

Physiological Parameters

Critical care nurses are skilled in assessing the patient's physical status in terms of changes in blood pressure, heart rate, or respirations. Therefore, the observation of the physiological effects of pain will assist in pain assessment. Unfortunately, the physiological response to pain is highly individualized. Vital signs, such as heart rate, blood pressure, and respirations, may increase or decrease in the presence of pain.[5] The potential for physiological changes can also be masked by medications that are administered to manage the critical illness, for example, medications that slow the heart rate or decrease blood pressure. When physiological changes do occur in critically ill patients, it can be difficult to attribute these physiological changes specifically to pain rather than other causes. Further, a sudden unexpected increase in the severity and intensity of a patient's pain, with or without associated physiological signs, may be an indication that a life-threatening complication has developed and must be evaluated immediately.

▲ Pain Intervention

The nurse plays a key role in providing pain relief. While pharmacological intervention is the most commonly used strategy, nursing management of pain also includes physical, cognitive, and behavioral measures. Further, in addition to administering medications or providing alternative therapies, the nurse's role involves measuring the patient's response to those therapies. Because pain may diminish or the pain pattern may change, therapy adjustments may be needed before improvements are seen. General guidelines for nursing interventions are listed in Box 5-3.

BOX 5-3 NURSING INTERVENTION GUIDELINES

For Pain Management

- Perform systematic pain assessments in all critically ill patients.
- Reassess hourly the need for rescue doses of analgesic.
- If the patient is experiencing a condition or procedure that is thought to be painful, provide preemptive analgesic medication.
- Remember that critically ill patients who are unconscious, sedated, or receiving neuromuscular blockade are at high risk for undertreatment of pain.
- Prevent pain by treating it in advance.
- If the patient has frequent or continuous pain, give analgesics by continuous intravenous infusion or around the clock, rather than PRN.

From Puntillo K: Part 1: Managing pain in the ICU patient. Critical Care Nurse 27(1):8–10, 2007.

Pharmacological Interventions

In general, the ideal method of analgesia should allow adequate serum drug levels to be achieved and maintained. Therefore, analgesic medications should always be administered on a regular schedule around the clock and not on an "as needed" (PRN) basis.[22] The efficacy of analgesia depends on the presence of an adequate and consistent serum drug level. Regardless of the method being used, scheduled opioid doses or a continuous infusion are preferred over "as needed" (PRN) administration.[22] The traditional PRN analgesic order is a major barrier to effective pain control in all patient populations. The PRN order suggests that the nurse should administer a dose of analgesic only when the patient requests it and only after a certain time interval has elapsed since the previous dose. Invariably, a delay occurs between the time of the request and the time the medication is actually administered. In some cases, this delay can be up to an hour. The PRN order poses another problem when the patient is asleep. As serum drug levels decrease, the patient is suddenly awakened by severe pain, and a greater amount of the drug is needed to achieve adequate serum levels. PRN analgesic medication doses should be reserved for breakthrough pain or when the patient has recovered from the critical illness, is predominantly pain free, and continuous analgesia is no longer required.

Analgesic medications should be titrated based on the patient's response, and the drug should be eliminated when analgesia is no longer needed. Most clinicians agree that when using a numerical scale for assessment, pain medications should be titrated according to the following goals:

- The patient's reported pain score is less than his or her own predetermined pain management goal (eg, 3 on a scale of 1 to 10).
- Adequate respiration is maintained.

Nonopioid Analgesics

Ideally, analgesic regimens should include a nonopioid drug, even if the pain is severe enough to also require an opioid.[2] In many patient populations, nonsteroidal anti-inflammatory drugs (NSAIDS) are the preferred choice for the nonopioid component of analgesic therapy. NSAIDS decrease pain by inhibiting the synthesis of inflammatory mediators (prostaglandin, histamine, and bradykinin) at the site of injury and effectively relieve pain without causing sedation, respiratory depression, or problems with bowel or bladder function. When NSAIDs are used in combination with opioids, the opioid dose can often be reduced and still produce effective analgesia. This decreases the incidence of opioid-related side effects.

Many NSAIDS are supplied only in oral forms, which is not sufficient for critically ill patients whose oral intake is restricted. Ketorolac (Toradol) is available in parenteral form, but it can cause renal impairment if administration exceeds 5 days; therefore, it must be used with caution in patients with renal insufficiency or those receiving dialysis. Indomethacin (Indocin) is available in suppository form and can be combined with opioids to provide effective pain relief.[25]

NSAIDS have been widely used in the non-ICU settings, but the potential benefits of NSAIDS compared to their potential risks have not been studied in critically ill patients. When administering NSAIDS as a complement to opioid analgesic medications, it is important to consider the possible side effects as potentially harmful in the ICU patient population. Prostaglandins help protect the gastric mucosa, are active in the aggregation of platelets, and are involved in the autoregulatory response of the vasculature in the kidneys. The inhibition of prostaglandins by NSAIDS can lead to impaired platelet aggregation, renal dysfunction, and gastric irritation resulting in increased bleeding risk, sodium and water retention, increased creatinine, and gastric ulceration. NSAIDS can also put a patient at higher risks for bronchospasms. Due to these potential risks, NSAIDS should be used with extreme caution in any critically ill patient with renal dysfunction, cardiac failure, coagulation problems, or respiratory failure.[27] Second-generation NSAIDS, such as celecoxib (Celebrex) and rofecoxib (Vioxx), are more selective in their site of action and therefore do not cause these harmful adverse effects, but their slow onset of action may decrease their utility in critically ill patients. Long-term use of these agents can increase the risk of developing cardiovascular disease.[22]

Acetaminophen is commonly used in critical care. When it is combined with opioids, it produces a greater effect than opioids alone. In addition to mild analgesia, acetaminophen is an effective antipyretic; however, it does have the potential to cause hepatic damage. Dosages should be limited to a maximum of 2,400 mg/d if patients have a history of, or high potential for, liver impairment. Nonopioid analgesics that are commonly used in critical care and their recommended doses are listed in Table 5-6.

Opioid Analgesics

Opioids are the pharmacological cornerstone of postoperative pain management. They provide pain relief by binding to various receptor sites in the central and peripheral nervous systems, thus changing the perception of pain. Opioids are selected based on individual patient needs and the potential for adverse effects. Commonly used opioids are compared in Table 5-7. According to the SCCM, morphine sulfate, fentanyl (Duragesic), and hydromorphone (Dilaudid) are the preferred agents when IV opioids are needed.[22] Other opioids used in critical care include codeine, oxycodone (OxyContin), and methadone.

Meperidine (Demerol) is the least potent opioid and is administered in the largest doses. For example, to produce a level of analgesia comparable to 10 mg of morphine every 4 hours, 100 to 150 mg of meperidine every 3 hours would be needed. Meperidine is commonly underdosed and given at intervals too infrequent to be effective. Meperidine also produces a toxic metabolite that can cause significant negative physiological effects when used long term. Therefore, even though meperidine continues to be widely used in some settings, national experts and national practice guidelines do not recommend it for most patients.[22,28] Rationale for avoiding this drug is summarized in Box 5-4.[28]

DOSING GUIDELINES. Equianalgesia means approximately equal analgesia. This term is used when changing a patients' regimen from one analgesic to another. Morphine, 10 mg IM, is generally considered the gold standard dose for comparison. Dosing guidelines for opioid analgesics are presented in Table 5-7. Opioid dosage varies depending on the individual patient, the method of administration, and

Table 5-6 Nonopioid Analgesic Drugs

Drug	Adult Dose	Usual Pediatric Dose	Comments
Acetaminophen	325–650 mg every 4–6 h	10–15 mg/kg every 4–6 h	Available in liquid form, lacks anti-inflammatory action. Doses exceeding 4,000 mg/d increase the risk of hepatic toxicity.
Aspirin	325–650 mg every 4–6 h	10–15 mg/kg every 4–6 h	Can cause gastrointestinal or postoperative bleeding
Celecoxib (Celebrex)	100–400 mg twice a day		Less adverse effects than other nonsteroidal anti-inflammatory drugs, considerably more expensive
Ibuprofen (Motrin)	200–400 mg every 4–6 h	4–10 mg/kg every 6–8 h	Available in liquid form
Indomethacin (Indocin)	25–50 mg every 8–12 h		Available in rectal and IV forms, but high incidence of side effects
Ketorolac (Toradol)	30–60 mg IM initially, then: 30 mg IV every 6 h or 30 mg IM every 6 h 10 mg PO every 4–6 h		Available in parenteral form, limit use to 5 d, contraindicated with renal insufficiency
Naproxen (Naprosyn)	500 mg initially then 250 mg every 6–8 h	5 mg/kg every 12 h	Available in liquid form

All doses are oral, unless noted otherwise.

the pharmacokinetics of the drug. Adequate pain relief will occur once a minimum serum level of the opioid has been achieved. Each patient's optimal serum level will be different, and this level can change as pain intensity changes. Therefore, the dosing and titration of opioids must be individualized, and the patient's response and any undesirable effects, such as respiratory depression or oversedation, must be closely assessed. If the patient has previously received an opioid (eg, before surgery), doses should be adjusted above the previous required dose to achieve an optimal effect. Factors such as age, individual pain tolerance, coexisting disease(s), type of surgical procedure, and the concomitant use of sedatives warrant consideration as well. Older patients are often more sensitive to the effects of opioids; therefore, decreasing the initial opioid dose and slow titration are recommended for older patients.

METHODS OF ADMINISTRATION.

Oral Administration. Oral administration is simple, noninvasive, and inexpensive and provides effective analgesia. Oral is the preferred route for patients with cancer and chronic nonmalignant pain. The oral route is used infrequently in the ICU setting, however, because many patients are unable to take anything by mouth. Serum drug levels obtained after oral administration of opioids are variable and difficult to titrate. In addition, the transformation of oral opioids by the liver causes a significant decrease in serum levels.

Rectal Administration. Morphine and hydromorphone are available in a rectal form. This provides an alternative for patients who cannot take anything by mouth. Unfortunately, this mechanism has many of the same disadvantages as oral administration, including variability in dosing requirements, delays to peak effect, and unstable serum drug levels.

Transdermal Administration. Fentanyl is available in a transdermal patch format. This form is used primarily to control chronic cancer pain because it takes 12 to 16 hours to see substantial therapeutic effects and up to 48 hours to achieve stable serum concentrations. If used for acute pain, such as postoperative pain, high serum concentrations may remain after the pain has subsided, putting the patient at risk for respiratory depression.[29] The nurse must exercise caution when administering fentanyl by a transdermal patch. Gloves should be worn when handling the patch to avoid accidental exposure to the drug. The site where the patch will be applied should be chosen carefully. The skin must be intact. Any open areas in the skin (abrasions, rashes, wounds, etc.) could cause the drug to be absorbed more rapidly than anticipated, leading to increased serum concentrations and a possible overdose. Avoid using lotion in the area where the patch will be applied. The lotion can act as a barrier and reduce or prevent the absorption of the drug, thus decreasing the serum concentration and the analgesic effect. When the fentanyl patch is changed, the old patch must be disposed of properly to avoid accidental exposure to the drug or illicit use of the discarded patch.

Intramuscular Injection. Intramuscular (IM) injections should not be used to provide acute pain relief for the critically ill patient for several reasons:

- IM injections are painful.
- IM drug absorption is extremely variable in critically ill patients because of alterations in cardiac output and tissue perfusion.
- Anticipated discomfort associated with the injection increases the patient's anxiety.
- Repeated IM injections can cause muscle and soft-tissue fibrosis.

Table 5-7 Opioid Analgesic Drugs

Drug	Equianalgesic Dose (mg)		Comments	Precautions
	Oral	IM/IV		
Morphine	10–30 every 4 h	5–10 IM or 1–4 IV every 4 h	Considered the gold standard of comparison for opioids. Oral sustained-release, once-a-day, and rectal forms available	Use caution with impaired ventilation. Not recommended with hemodynamic instability or renal insufficiency.
Fentanyl		0.25–0.5 IV every 1–2 h	Drug of choice for rapid onset of analgesia in acutely distressed patients. Rectal and transdermal forms available	With transdermal form—12–24 h delay to peak effect, and fever increases dose and absorption rate.
Hydromorphone (Dilaudid)	2–8 every 3–4 h	0.7–2 every 1–2 h	More potent and slightly shorter duration than morphine. Rectal form available	
Meperidine (Demerol)	50–150 every 2–4 h	50–75 IM every 3–4 h	Not recommended (See Box 5-4.) Slightly shorter acting than morphine	Toxic metabolite accumulates, causing central nervous system excitation Limit use to <48 h
Methadone (Dolophine)	2.5–10 every 8–12 h	2.5–10 IV/IM every 8–12 h	Good oral potency, long half-life (24–36 h)	Accumulates with repetitive dosing, causing excessive sedation
Oxycodone (OxyContin)	2.5–30 every 4–6 h		Used for moderate pain when combined with a nonopioid (eg, Percocet). As a single entity—useful for severe pain	Dosing must be individualized due to high variability in pharmacokinetics.

Intravenous Injection. Intravenous (IV) administration is usually the preferred route for opioid therapy, especially when the patient requires short-term acute pain relief, for example, during procedures such as chest tube removal, diagnostic tests, suctioning, or wound care. IV opioids have the most rapid onset and are easy to administer. With morphine, the time to peak effect is 15 to 30 minutes; for fentanyl, peak effect is achieved within 1 to 5 minutes. However, the duration of analgesia is shorter with intermittent IV injections, and this can cause serum drug levels to fluctuate.

Continuous IV administration has many benefits for critically ill patients, especially those who have difficulty communicating their pain because of an altered level of consciousness or an endotracheal tube. Continuous IV infusions are easily initiated and maintain consistent serum drug levels. For continuous IV opioid infusions, fentanyl and morphine are commonly used because of their short elimination half-life (compared with other available opioids). Before starting a continuous IV infusion, an initial IV loading dose(s) is given to achieve an optimal serum level. Appropriate dosing and titration must be individualized, and this can be difficult because many critically ill patients have hepatic or renal dysfunctions that result in decreased metabolism of the opioid. A disadvantage of continuous IV infusions is that pain occurring during painful procedures may not be managed unless additional IV bolus injections are given.

PCA is an effective method of pain relief for the critically ill patient who is conscious and able to participate in the pain management therapy. The PCA method of opioid administration produces good quality analgesia, stable drug concentrations, less sedation, less opioid consumption, and fewer adverse effects.[22] Effective use of PCA is based on the assumption that the patient is the best person to evaluate and manage his or her pain. PCA individualizes pain control therapy and offers the patient greater feelings of control and well-being.

BOX 5-4 PATIENT SAFETY

Precautions and Concerns Associated with Meperidine Use

Meperidine (Demerol) is a dangerous analgesic that continues to be used in some settings. For safety's sake, it is not recommended for the following reasons:

- Low potency—requires unusually high doses
- Produces toxic metabolite—normeperidine
- May cause central nervous system excitation, anxiety, tremors, seizures
- Intramuscular administration produces fibrosis
- Contraindicated in patients with compromised renal function
- Contraindicated in elderly patients
- Should not be used for more than 48 hours
- Dose should not exceed 600 mg/24 h
- Should not be used for chronic pain treatment
- Use in sickle cell disease creates high risk for seizures
- Coadministration with monoamine oxidase (MAO) inhibitors can be lethal

Raymo LL, Camejo M, Fundin J: Eradicating analgesic use of meperidine in a hospital. Am J Health Syst Pharm 64(11):1148–1153, 2007

With PCA, the patient self-administers small, frequent IV analgesic doses using a programmable infusion device. Most often, morphine sulfate or fentanyl is used. The PCA device limits the opioid dose within a specific time period; thus preventing oversedation and respiratory depression. If the patient is physically or cognitively unable to use "conventional" PCA, other adaptations can be made. For example, the PCA pump can be activated by a designated family member. This family member will need thorough education in terms of how to assess for the presence of pain, how to administer the medication, and how to assess for oversedation and respiratory depression. The PCA pump can be also be activated by the patient's nurse.

Subcutaneous Administration. In some situations, venous access may be limited or impossible to obtain. When this occurs, continuous subcutaneous infusion and subcutaneous PCA may be used.

Spinal Administration. Spinal opioids can provide superior pain management for many patients. Spinal opioids selectively block opioid receptors, while leaving sensation, motor, and sympathetic nervous system function intact. This results in fewer opioid-related side effects than oral, IM, or IV routes of administration. Analgesia from spinal opioids has a longer duration than other routes, and significantly less opioid is needed to achieve effective pain relief. Opioids, such as fentanyl or morphine, can be given as a single injection in the epidural or intrathecal space, as intermittent injections, or continuous infusions through an epidural catheter, or epidural PCA.

Epidural analgesia is noted for providing effective pain relief and improved postoperative pulmonary function. This method is especially beneficial for critically ill patients after thoracic, upper abdominal or peripheral vascular surgery, rib fractures, orthopedic trauma, or postoperative patients with a history of obesity or pulmonary disease. With epidural analgesia, opioids are administered through a catheter inserted in the spinal canal between the dura mater and vertebral arch. Opioids diffuse across the dura and subarachnoid space and bind with opioid receptor sites.

Intermittent injections may be given before, during, or after surgical procedures. For more sustained pain relief, continuous epidural infusions are recommended. For patient-controlled epidural analgesia, the same parameters are used as with IV PCA, except that smaller opioid doses are used. Contraindications to epidural analgesia include systemic infection/sepsis, bleeding disorders, and increased intracranial pressure.

Preservative-free morphine and fentanyl are most commonly used for epidural analgesia because preservatives can be neurotoxic and may cause severe spinal cord injury. Morphine is more water soluble than fentanyl and thus is more likely to accumulate in the cerebrospinal fluid and systemic circulation. With increased accumulation, side effects are more likely. Fentanyl diffuses more quickly to the opioid receptors and causes fewer opioid-related side effects. The most serious adverse effect of epidural analgesia is respiratory depression. Although the incidence of serious respiratory depression is extremely low with epidural analgesia, respiratory assessments should be performed hourly during the first 24 hours of therapy and every 4 hours thereafter.

Because epidural analgesia is more invasive than the other drug delivery methods discussed, the patient must be closely monitored for signs of local or systemic infections. The insertion site is covered with a sterile dressing, and the catheter is taped securely. To avoid accidental injection of preservative-containing medications, the epidural catheter, infusion tubing, and pump should be clearly marked.

With intrathecal analgesia, the opioid is injected into the subarachnoid space, located between the spinal cord and dura mater. Intrathecal opioids are significantly more potent than those given epidurally; therefore, less medication is needed to provide effective analgesia. The intrathecal method is usually used to deliver a one-time dose of analgesic, such as before surgery, and is infrequently used as a continuous infusion due to the risk of central nervous system infection.

With epidural or intrathecal analgesia, a local anesthetic, such as bupivacaine (Marcaine), can be added to the continuous opioid infusion. Local anesthetics block pain by preventing nerve cell depolarization. They act synergistically with intraspinal opioid and have a dose-sparing effect. Less opioid is needed to provide effective analgesia, and the incidence of opioid-related side effects is decreased. This combination is more commonly administered via the epidural route.

OPIOID EFFECTS. Opioids cause undesirable side effects, such as constipation, urinary retention, sedation, respiratory depression, and nausea. These side effects represent a major drawback to their use. Opioid-related side effects are best managed in the following ways:

- *Decreasing the opioid dose:* This is the most effective strategy because it is directed at the *cause* of the side effect. Side effects are usually seen with excessively high serum levels of the drug. Decreasing the opioid dose can alleviate the side effect while still providing effective pain relief.
- *Avoiding PRN dosing:* When opioids are administered on a PRN basis, fluctuating serum drug levels occur, causing a greater tendency toward sedation and respiratory depression. Around-the-clock administration of analgesics, including opioids, is recommended.
- *Adding an NSAID to the pain management plan*: Using an NSAID in addition to an opioid can decrease the amount of opioid needed, still provide effective pain relief, and decrease opioid-related side effects.

Medications can be given to minimize or alleviate some side effects (eg, stool softeners for constipation, antihistamines for pruritus, and antiemetics for nausea). However, medications commonly prescribed to treat the opioid-related adverse effects can actually cause other adverse effects. For example, promethazine, a commonly prescribed antiemetic, can cause hypotension, restlessness, tremors, and extrapyramidal effects in the older patient.

Respiratory depression, a life-threatening complication of opioid administration, is often a concern for nurses and physicians. The incidence of true opioid-induced respiratory depression is low in most patients. In some cases, a respiratory rate as low as 10 may not be significant if the patient is still breathing deeply. Patients most at risk for respiratory

depression are infants, elderly people who have not recently used opioids, and patients with coexisting pulmonary, renal, or hepatic disease.

Opioid Antagonists

If serious respiratory depression does occur, naloxone (Narcan), a pure opioid antagonist that reverses the effects of opioids, can be administered. The dose of naloxone is titrated to effect—which means reversing the oversedation and respiratory depression, not reversing analgesia. This usually occurs within 1 to 2 minutes. After giving naloxone, the nurse needs to continue observing the patient closely for oversedation and respiratory depression because the half-life of naloxone (1.5 to 2 hours) is shorter than most opioids.

Naloxone should be diluted (0.4 mg in 10 mL of saline solution) and given IV, very slowly. Giving the drug too quickly or giving too much can precipitate severe pain, withdrawal symptoms, tachycardia, dysrhythmias, and cardiac arrest. Patients who have been receiving opioids for more than a week are particularly at risk.

Sedatives and Anxiolytics

Acute pain is frequently accompanied by anxiety, and anxiety can increase the patient's perception of pain. When treating acute pain, anxiolytics can be used to complement analgesia and improve the patient's overall comfort. This is an important consideration, especially prior to and during painful procedures.

Benzodiazepines

Benzodiazepines, such as midazolam (Versed), diazepam (Valium), and lorazepam (Ativan), can control anxiety and muscle spasms, and produce amnesia for uncomfortable procedures. In the ICU, benzodiazepines may be given IV as an intermittent bolus or by continuous infusion and titrated according to the patient's response. Because these medications have no analgesic effect (except for controlling pain caused by muscle spasm), an analgesic must be administered concomitantly to relieve pain. If an opioid and benzodiazepine are used together, the doses of both medications should usually be reduced because of their synergistic effects. The patient should also be closely monitored for oversedation and respiratory depression.

Midazolam is recommended for conscious sedation and short-term relief of anxiety because of its rapid onset (1 to 5 minutes with IV administration) and its short half-life (1 to 12 hours). Another advantage is its retrograde amnesia effect, which is particularly beneficial during procedures. The duration of effect of midazolam can be longer in older or obese patients and those with liver disease.[22]

A major advantage of benzodiazepines is that they are reversible agents. If respiratory depression occurs from benzodiazepine administration, flumazenil (Romazicon) can be administered IV. Flumazenil is a benzodiazepine-specific reversal agent that reverses the sedative and respiratory depressant effects without reversing opioid analgesics. The dosing of flumazenil should be individualized and titrated so that only the smallest effective amount is used. After prolonged benzodiazepine therapy, flumazenil should be used with caution due to the potential for stimulating withdrawal symptoms, increasing intracranial pressure, causing hypertension, and lowering the seizure threshold.[29]

Critically ill patients who are receiving repeated doses or continuous infusions of sedatives are given a break from sedation at least once a day. Administration should be interrupted until the patient is fully awake. This helps to prevent oversedation, which can inhibit weaning from mechanical ventilation.

Propofol

Propofol (Diprivan) is a rapid-acting sedative/hypnotic agent that has no analgesic properties and minimal amnesic effects. With appropriate airway and ventilatory management, propofol can be an ideal agent for patients requiring sedation during painful procedures. Because of its ultrashort half-life and high rate of elimination, it is reversible simply by discontinuing the infusion and patients will awaken within a few minutes. Propofol can also be used as a continuous infusion for mechanically ventilated patients who require deep, prolonged sedation.

Because propofol is only slightly water soluble, it is formulated in a white, oil-based emulsion containing soybean oil, egg lecithin, and glycerol. It is contraindicated, therefore, in patients allergic to eggs or soy products. Propofol contains no preservatives. Each ampule or vial must be used as a "single-dose" product vial and should be discarded within 6 to 12 hours after breaking the sterile seal to minimize the risk of systemic infections. Adverse effects commonly associated with propofol include respiratory depression, hypotension, elevated triglycerides, and pain and stinging at the injection site. Table 5-8 provides a comparison of sedatives commonly used in critical care.[35]

Nonpharmacological Comfort Measures

The combination of nonpharmacological and pharmacological interventions provide better pain control, with less use of opioid analgesics, decreased incidence of anxiety, and increased patient satisfaction.[27,28] These nonpharmacological approaches, which include interventions, such as distraction, relaxation, music, therapeutic touch, and massage, can be challenging to provide in the critical setting.

Environmental Modification

In critical care, the most basic and logical nonpharmacological intervention is environmental modification. The excessive noise and light in ICUs can disrupt sleep and increase anxiety and agitation, in turn contributing to pain and discomfort. Sources of noise may include alarms, equipment, telephones, ventilators, and staff conversations. The severity of illness has also been shown to be associated with sleep disturbances. Care should be preplanned to minimize noise and disruptions during normal sleeping hours and to create a pattern of light that mimics normal day–night patterns. Earphones, with music of the patient's choice, and earplugs have also been recommended for use in the ICU.[30,31]

Table 5-8 Comparison of Sedatives Commonly Used in Critical Care

Drug	Recommended Use	Onset (IV)	Unique Adverse Effects
Diazepam (Valium)	For rapid sedation of acutely agitated patients.	2–5 min	Phlebitis
Lorazepam (Ativan)	For long-term sedation of most patients via intermittent or continuous infusion	5–20 min	Acidosis/renal failure with high doses
Midazolam (Versed)	For conscious sedation and rapid sedation of acutely agitated patients. For short-term use only.	2–5 min	Prolonged wakening and delayed weaning from ventilator, if used long term
Propofol	Preferred sedative when rapid awakening is important.	1–2 min	Pain on injection and elevated triglycerides

Honiden S, Siegel MD: Analytic reviews: Managing the agitated patient in the ICU: Sedation, analgesia and neuromuscular blockade. J Intensive Care Med 25(4):187–204, 2010

Distraction

Distraction helps patients direct their attention away from the source of pain or discomfort toward something more pleasant. Patients, families, and nurses often use distraction routinely without giving it much consideration. Initiating a conversation with the patient during an uncomfortable procedure, watching television, and visiting with family are all excellent sources of distraction.

Relaxation Techniques

Relaxation can be described as a state of calmness or peacefulness. It is a state that is free from anxiety and skeletal muscle tension.[9] Relaxation exercises involve repetitive focus on a word, phrase, prayer, or muscular activity and a conscious effort to reject other intruding thoughts. Relaxation can give the patient a sense of control over a particular body part. Most relaxation methods require a quiet environment, a comfortable position, a passive attitude, and concentration. Each of these can be challenging to achieve in an ICU.

Breathing exercises have been used with much success in childbirth. They can also be used successfully in the critically ill patient. The quieting reflex is a breathing and relaxation technique that reduces stress and can easily be taught to the conscious and coherent patient. Instructions regarding the quieting reflex are given in Box 5-5. The nurse encourages the patient to perform the quieting reflex frequently during the day. This relaxation technique can be done in only 6 seconds, and it calms the sympathetic nervous system and gives the patient a sense of control over stress and anxiety.

BOX 5–5 TEACHING GUIDE *Instructions for the Quieting Reflex*

1. Inhale using an easy, natural breath.
2. Think "alert mind, calm body."
3. Smile inwardly (with your internal facial muscles).
4. As you exhale, allow your jaw, tongue, and shoulders to go loose.
5. Allow a feeling of warmth and looseness to go down through your body and out through your toes.

Touch

Historically, one of the greatest contributions nurses have made is the comfort and caring of presence and touch. These contributions still have an important place in today's highly technical ICUs. Nurses may feel that touching is too simple to be effective. However, few medical advances can replace the benefits of warm and caring touch. The need for touch is thought to intensify during times of high stress and cannot be totally met by other forms of communication. Nurses, when using touch, are usually trying to convey understanding, support, warmth, concern, and closeness to the patient. Touching not only contributes to the patient's sense of well-being but also promotes physical recovery from disease. It has a positive effect on perceptual and cognitive abilities and can influence physiological parameters, such as respiration and blood flow. Touch represents a positive therapeutic element of human interaction.

The effects of touch in the clinical environment are far-reaching. Touch has played a major part in promoting and maintaining reality orientation in patients prone to confusion about time, place, and personal identification. Nursing touch may be most helpful in situations in which people are experiencing fear, anxiety, depression, or isolation. It may also be beneficial for patients who have a need for encouragement or nurturing, who have difficulty verbalizing needs, or who are disoriented, unresponsive, or terminally ill. Patients often feel that the desire for touch increases with the seriousness of the illness.

Massage

Superficial massage initiates the relaxation response and has been shown to increase the amount of sleep in ICU patients, decrease fatigue, improve the immune system, and help relieve pain, anxiety, and nausea.[32] While the back is the most common location used for massage, backs are often difficult to access in ICU patients. Hands, feet, and shoulders are also good sites for massage. Massage is an excellent intervention for family members to use in their attempts to provide comfort to the critically ill.

Patient Education

To educate the patient about pain and pain relief, the critical care nurse must be familiar with the patient's pain

management plan and therapy being used. Communication between the nurse and patient is essential. Any information given should be reinforced periodically during the course of therapy, and the patient should be encouraged to verbalize any questions or concerns. Family members should be included whenever possible. Plans for pain management should be discussed with patients when they are most able to understand, for example, prior to surgery rather than during the recovery period. Emphasis is on prevention of pain because it is easier to prevent pain than to treat it once it becomes severe.

Patients need to know that most pain can be relieved and that unrelieved pain may have serious consequences on physical and psychological well-being and may interfere with recovery. The nurse helps patients and families understand that pain management is an important part of their care and that the health care team will respond quickly to reports of pain. Patients should also be given instructions about nonpharmacological interventions and traditional methods to minimize pain. Splinting the incisional area with a pillow while coughing or ambulating is a traditional pain-relief measure.

The potential for drug addiction or overdosage is often a major concern for the patient and family. These issues should be addressed and clarified because they create a barrier to effective pain relief. The patient also needs a clear understanding of any specialized pain management technology, such as PCA to alleviate the fear of overdosage.

BOX 5-6 CONSIDERATIONS FOR THE OLDER PATIENT

In Pain

- Painful chronic diseases often compound the acute pain of critical illness in older patients.
- Arthritis, the most common cause of chronic pain in older patients, often affects the back, hips, knees, and shoulders, increasing the patient's pain when being turned, particularly in the ICU.
- Some older patients can experience acutely painful conditions, such as myocardial infarction or appendicitis, without feeling pain.
- Older patients often use words such as "aches" or "tenderness" rather than "pain."
- Family care givers can help assess pain in older patients who have cognitive or language impairments.
- Older patients are particularly sensitive to opioids; higher peak concentrations and longer duration of actions are achieved in older adults.
- Meperidine (Demerol), pentazocine (Talwin), propoxyphene (Darvon), and methadone should not be used to treat pain in older adults.[29]
- Some older patients often have an increased need for meaningful touch during episodes of crisis.

▲ Pain Management in Specific Populations

Some critically ill populations create unique pain management challenges. Special considerations for the elderly patient are noted in Box 5-6.[29] Another population that is particularly challenging is patients who are known to be dying. Pain is a primary concern for patients and their families at the end of life. Initiatives to promote high-quality palliative and end-of-life care have contributed to the diligence of health care providers in their efforts to understand and control pain in dying patients.[33,34]

Progression toward death is often marked by decreased cardiac output, decreased perfusion, and failure of major organ systems. This can create problems with excessive accumulation of analgesics and their metabolites resulting from limited hepatic and renal function. In such cases, hydromorphone, oxycodone, and fentanyl are preferred agents because of their short half-lives. If pain or dyspnea becomes uncontrollable despite aggressive analgesic administration, high doses of sedatives may also be employed. In such cases, the goal of sedation is comfort and relief of suffering, and the common by-product is end-stage unconsciousness.[35]

Critical illness is painful. The subjective nature of pain, coupled with communication impairments and the interactions of acute and chronic disease processes, makes pain assessment and treatment particularly challenging in the ICU. Misconceptions and knowledge deficits on the part of health care providers, patients, and family members must be recognized and corrected. When providing pain management, all available modes of therapy—opioids, adjuvants, and nonpharmacological therapies—should be integrated into the plan of care. Critical care nurses must stay abreast of current pain management research and resources to provide the highest quality of pain and comfort management.

▲ Clinical Applicability Challenges

CASE STUDY

Mr. B., a 31-year-old construction worker who fell three stories onto concrete, was just admitted to the trauma intensive care unit from the Emergency Department's trauma resuscitation room. Mr. B. is alert, oriented, and reporting that he is in severe pain despite receiving several IV morphine injections. His injuries include fractures of the seventh and eighth ribs on the right with a hemopneumothorax, a fracture of the right radius and ulna, and a compound fracture of the right tibia and fibula. Blood in his urine and bruising in the right flank area also indicate a probable injury to the right kidney. While Mr. B. was being evaluated in the trauma room, a

(continued on page 65)

CASE STUDY *(Continued)*

chest tube was inserted to correct the hemopneumothorax; the fractures of his radius and ulna were reduced and a cast was applied; a Foley catheter was inserted to monitor his urinary output; and the compound fractures of his tibia and fibula were reduced and a splint was applied. Mr. B. will be taken to the operating room later in the day for débridement of the wound on his lower leg and external fixation of his fractures.

1. Often an NSAID is prescribed in conjunction with opioid drugs for optimal pain control. This patient is in extreme pain. Should he be receiving an NSAID in addition to his morphine and why?

2. Mr. B. is prescribed morphine 4 mg/IV every 2 hours for pain, yet he is still rating his pain at 10/10. The chief surgical resident has been contacted to write new orders for a higher or more frequent morphine dose; however, he is hesitant to increase the morphine due to the impending surgery to repair Mr. B.'s leg fractures. How could uncontrolled pain negatively affect Mr. B.'s condition?

3. Mr. B.'s pain remains 10/10, and the nurse contacts the attending trauma surgeon, explaining the situation and the reluctance of the surgical resident to increase the morphine dose. The attending surgeon decides to take an alternate approach that will increase the effectiveness of the morphine without increasing the dose. What alternative method of pain management would achieve these goals?

References

1. Puntillo K, Pasero C, Li D, et al: Evaluation of pain in ICU patients. Chest 135(4):1069–1074, 2009
2. Sessler CN, Varney K: Patient-focused sedation and analgesia in the ICU. Chest 133(2):552–565, 2008
3. Li D, Puntillo K: A pilot study on coexisting symptoms in intensive care patients. Appl Nurs Res 19(4):216, 2006
4. Siffleet J, Young J, Nikoletti S, et al: Patients self-report of procedural pain in the intensive care unit. J Clin Nurs 16(11):2142–2148, 2007
5. Gelinas C, Fillion L, Puntillo KA, et al: Validation of the critical-care pain observation tool in adult patients. Am J Crit Care 15(4):420–427, 2006
6. International Association for the Study of Pain: Task force on taxonomy. Part III: Pain terms, a current list with definitions and notes on usage (pp 209–214). In Merskey H, Bogduk N (eds): Classification of Chronic Pain: Descriptions of Chronic Pain Syndromes and Definitions of Pain Terms, 2nd ed. Seattle, WA: IASP Press, 1994
7. McCaffery M: Nursing Practice Theories Related to Cognition, Bodily Pain and Man-Environment Interaction. Los Angeles, CA: University of California at Los Angeles, 1968
8. American Chronic Pain Association: ACPA Consumer Guide to Pain Medication & Treatment (PDF). Retrieved April 1, 2010, from http://www.theacpa.org/people/medication.asp
9. Erstad B, Puntillo K, Gilbert H, et al: Pain management principles in the critically ill. Chest 135:1075–1086, 2009
10. Wiencek C, Winkelman C: Chronic critical illness: Prevalence, profile, and pathophysiology. AACN Adv Crit Care 21(1):44–61, 2010
11. Puntillo KA, Thompson CL, Stanik-Hutt J, et al: Pain behaviors observed during six common procedures: Results from Thunder Project II. Crit Care Med 32(2):421–427, 2004
12. Puntillo K, White C, Morris A, et al: Patients' perceptions and responses to procedural pain: Results from the Thunder Project II. Am J Crit Care 10:238–251, 2001
13. Stanik-Hutt JA, Soeken KL, Blcher AE, et al: Pain experiences of traumatically injured patients in a critical care setting. Am J Crit Care 10:252–259, 2001
14. Rawe C, Trame CD, Moddeman G, et al: Management of procedural pain: Empowering nurses to care for patients through clinical nurse specialist consultation and intervention. Clin Nurse Spec 23(3):131–137, 2009
15. Ead H: Improving pain management for critically ill and injured patients. Dynamics 16(3):26–31, 2005
16. Yeager MP, Glass DD, Neff RK, et al: Epidural anesthesia and analgesia in high risk surgical patients. Anesthesiology 66(6):729–736, 1987
17. Pasero C, Puntillo K, Li D, et al: Structured approaches to pain management in the ICU. CHEST 135(6):1665–1672, 2009
18. American Pain Society: Definitions related to the use of opioids for the treatment of pain. Retrieved April 1, 2010, from http://www.ampainsoc.org/advocacy/opioids2.htm
19. Richebe P, Beaulieu P: Perioperative pain management in the patient treated with opioids: Continuing professional development. Can J Anaesth 56(12):969–981, 2009
20. Chisholm-Burns M, Wells B, Schwinghammer T, et al: Pharmacotherapy Principles & Practice. New York: McGraw Hill, 2008
21. Agency for Healthcare Research and Quality: National Guideline Clearinghouse. Retrieved February 14, 2012, from http://www.guideline.gov/search/search.aspx?term=pain
22. Vadivelu N, Mitra S, Narayan D: Recent advances in post operative pain management. Yale J Biol Med 83(1):11–25, 2010
23. Karp JF, Shega JW, Morone NE, et al: Advances in understanding the mechanisms and management of persistent pain in older adults. Br J Anesth 101(1):111–120, 2008
24. AGS Panel on the Pharmacological Management of Persistent Pain in Older Adults: Pharmacological management of persistent pain in older persons. J Am Geriatr Soc 57(8):1331–1346, 2009
25. National Cancer Institute: Pain (PDQ). Retrieved April 1, 2010, from http://www.cancer.gov/cancertopics/pdq/supportivecare/pain/Patient/page1
26. Gelinas C, Johnston C: Pain assessment in the critically ill ventilated adult: Validation of the critical-care pain observation tool and physiologic indicators. Clin J Pain 23(6):497–505, 2007
27. Ashley E, Given J: Pain management in the critically ill. J Perioper Pract 18(11):504–509, 2008
28. Raymo LL, Camejo M, Fundin J: Eradicating analgesic use of meperidine in a hospital. Am J Health Syst Pharm 64(11):1148–1153, 2007
29. Mirski MA, Lewin JJ: Sedation and pain management in acute neurological disease. Semin Neurol 28(5):611–630, 2008
30. Scotto CJ, McClusky C, Spillan S, et al: Earplugs improve patients' subjective experience of sleep in critical care. Nurs Crit Care 14(4):180–184, 2009
31. Bijwadia JS, Ejaz MS: Sleep and critical care. Curr Opin Crit Care 15(1):25–29, 2009

32. Russell NC, Sumler SS, Beinhorn CM, et al: Role of massage therapy in cancer care. J Altern Complement Med 14(2):209–214, 2008
33. Truog RD, Meyer EC, Burns JP: Toward interventions to improve end-of-life care in the pediatric intensive care unit. Crit Care Med 34(11 Suppl):S373–S379, 2006
34. Fineberg IC, Wenger NS, Brown-Saltzman K: Unrestricted opiate administration for pain and suffering at the end of life: Knowledge and attitudes as barriers to care. J Palliat Med 9(4):873–883, 2006
35. Mularski RA, Puntillo K, Varkey B, et al: Pain management within the palliative and end-of-life care experience in the ICU. Chest 136(2):1360–1369, 2009

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

6 Palliative Care and End-of-Life Issues in Critical Care

Garrett K. Chan

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. List at least three end-of-life issues related to critical care nursing.
2. List at least three components of palliative care.
3. Describe how palliative care can be integrated into curative or disease-modifying care.
4. Identify at least three symptoms commonly experienced at the end of life.
5. Recognize the importance of flexible visiting hours for a patient at the end of life.
6. Describe activities by the nurse in preparing for and coordinating a family conference.
7. Identify strategies for self-care of the nurse.

About 2 million people die in the United States every year. Although some people die in peace and comfort, others die in severe distress and suffering. Over the past decade, nurses in the acute care setting have been increasingly concerned about how people die. Thoughts about critical care have slowly shifted; clinicians now recognize that death may be inevitable and that the use of technology to prevent death is limited. Critical care nurses are well positioned to help patients and families during this difficult transitional period. "Being with" patients and families in addition to "doing things to" them enables critical care nurses to provide the holistic care that is central to nursing.[1]

▲ Need for Quality End-of-Life Care in the Critical Care Setting

In the early 20th century, the average life expectancy was 50 years. Common causes of death included infection, accidents, and childhood mortality.[2] Few life-extending measures were available, and death occurred after hardly any interventions. The focus was on caring for the dying person by family members, who witnessed the death.

However, in the mid to late 20th century, medical interventions such as antibiotics, cardiopulmonary resuscitation (CPR), mechanical ventilation, dialysis, intra-aortic balloon pumps, and pulmonary artery catheters were discovered and routinely used to combat morbidity and mortality. These technologies, combined with other public health initiatives such as improved sanitation, carried the promise of treating the causes of death and therefore extending life. By the year 2000, the average life expectancy had been extended to 77 years.

Critical care nurses became focused on these life-extending procedures, and critical care units were developed to house seriously ill patients in one area of a hospital and closely monitor their response to curative, lifesaving, and aggressive treatments.[3] Increasingly, people died in the hospital setting surrounded by health care practitioners rather than their families. Over the course of the years of technological advancement, nurses increasingly viewed patients in terms of disease processes or technologies. The concept that the patient lying in the bed was a person who experienced physical, emotional, psychological, social, and spiritual suffering was lost.[4]

Over the past decade, evidence shows that skillful communication, family centered care, and shared decision making among the patient, family, and the interdisciplinary team can lead to healthy work environments, decreased moral distress, and mitigation of traumatic stress experienced by all persons involved in the care of the patient.[5–7] It is important to recognize that people approach death in a variety of ways. Therefore, instituting these caring practices from the moment the patient reaches the critical care environment is vital to providing good care. While palliative and end-of-life care education is important, there has been a paucity of prelicensure and continuing education programs addressing these topics.[8,9] The End-of-Life Nursing Education Consortium-Critical Care (ELNEC-CC) project was designed specifically to educate nurses about palliative and end-of-life care in critical care settings.[10]

Understanding Human Death

Over the past decade, there has been an increase in understanding the human experience of dying in the acute care setting. In 1995, a major study titled the *Study to Understand Prognoses and Preferences for Outcomes and Risks of Treatment* (SUPPORT) was published.[11] This study, conducted in five major academic medical centers across the United States, involved more than 9,000 seriously ill patients. The goal was to improve end-of-life decision making and reduce the frequency of mechanically supported, painful, and prolonged death. Despite use of an intervention designed to communicate preferences among providers, patients, and families, such wishes were often unknown, and aggressive treatment was common. Physicians were not aware that their patients preferred to avoid CPR. In addition, nearly 40% of patients who died spent at least 10 days in an intensive care unit (ICU), and 50% of family members of conscious patients reported that the patients were in moderate to severe pain at least half the time.

After publication of the SUPPORT study, the Institute of Medicine (IOM) released a report titled *Approaching Death: Improving Care at the End of Life*.[12] The IOM group of experts listed seven recommendations to improve end-of-life care (Box 6-1). These recommendations are important for critical care nurses because it is estimated that about 20% of deaths in the United States occur while patients are using ICU services.[13] Critical care nurses play an important role in recognizing opportunities for interventions that support patients, families, and other staff members during this difficult transition in life. Although technology, urgency, uncertainty, and conflict are common in critical care practice, these characteristics may inhibit or fragment a coordinated effort that aims to provide good end-of-life care.[1]

BOX 6-1 Recommendations to Improve End-of-Life Care

1. People with advanced, potentially fatal illnesses and those close to them should be able to expect and receive reliable, skillful, and supportive care.
2. Physicians, nurses, social workers, and other health professionals must commit themselves to improving care for dying patients and to using existing knowledge effectively to prevent and relieve pain and other symptoms.
3. Because many deficiencies in care reflect system problems, policy makers, consumer groups, and purchasers of health care should work with health care providers and researchers to:
   a. Strengthen methods for measuring the quality of life and other outcomes of care for dying patients and those close to them
   b. Develop better tools and strategies for improving the quality of care and holding health care organizations accountable for care at the end of life
   c. Revise mechanisms for financing care so that they encourage rather than impede good end-of-life care and sustain, rather than frustrate, coordinated systems of excellent care
   d. Reform drug prescription laws, burdensome regulations, and state medical board policies and practices that impede effective use of opioids to relieve pain and suffering
4. Educators and other health professionals should initiate changes in undergraduate, graduate, and continuing education to ensure that practitioners have the relevant attitudes, knowledge, and skills to care well for dying patients.
5. Palliative care should become, if not a medical specialty, at least a defined area of expertise, education, and research.
6. The nation's research establishment should define and implement priorities for strengthening the knowledge base for end-of-life care.
7. A continuing public discussion is essential to develop a better understanding of the modern experience of dying, the options available to dying patients and families, and the obligations of communities to those approaching death.

Adapted from Field MJ, Cassel CK: Approaching Death: Improving Care at the End of Life. Washington, DC: Institute of Medicine, 1997

Palliative Care

The introduction of palliative care principles into critical care can provide a framework to address these end-of-life issues. Palliative care originated from hospice care, which was designed to improve the quality of death and dying for patients and their families by addressing aspects of care that are unrelated to disease-specific treatments, cure, or rehabilitation.[14] According to the World Health Organization[15] and the IOM,[12] palliative care from an interdisciplinary perspective includes the following core principles: symptom management; advanced care planning; family centered care; emotional, psychological, social, and spiritual care; facilitating communication; awareness of ethical issues, and caring for the care giver. These principles should be addressed and incorporated into the total care of the patient, even when disease-modifying or curative therapies are used. In critical care nursing, it is vital that these core palliative care principles are incorporated in the daily plan of care of patients using an interdisciplinary approach.[16–18] Figure 6-1 illustrates how palliative care can be incorporated throughout the patient's illness.

Palliative care services in critical care have demonstrated an improvement in symptom management, family support, reduction in length of hospital stay, increased discharges to home with hospice referrals, and reduced costs.[16,19,20] The American Association of Critical-Care Nurses (AACN) has developed protocols for critical care practice in palliative and end-of-life care.[21] These protocols provide a good overview of core issues and clinical recommendations for critical care nurses. Resources to assist nurses in addressing issues surrounding the end-of-life phase are given in Table 6-1.

A particular therapeutic intervention may be either curative or palliative depending on its intent. For example, a packed red blood cell transfusion may be curative in a patient with an acute hemorrhage or palliative in a patient with chronic anemia and severe fatigue following chemotherapy. Whether an intervention attempts to cure or to palliate determines whether it is curative or palliative. Additional examples of treatments that can be either curative or palliative are surgeries that resect the bowel to remove a tumor that is causing an intestinal obstruction or administering furosemide in a patient who has severe pulmonary edema. If the treatment relieves the patient's suffering, then it is considered palliative.

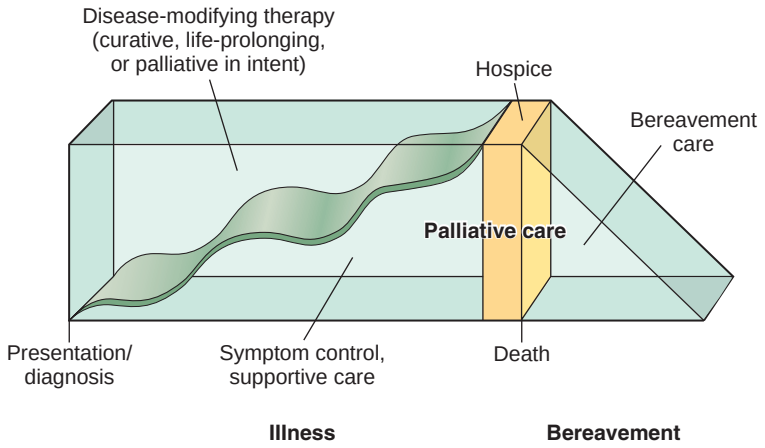


FIGURE 6-1 ▲ The continuum of care. (Adapted from Emanuel L, von Gunten C, Ferris F, et al (eds): The Education in Palliative and End-of-Life Care [EPEC] Curriculum: The EPEC Project. Chicago, IL: Author, 2003.)

▲ Symptom Management

In critical care, patients experience a wide range of symptoms from their diseases as well as from the therapies that are used to treat those diseases. Common symptoms at the end of life include pain, dyspnea, anxiety and agitation, depression, delirium, and nausea and vomiting. The nurse assesses for the presence and severity of each of these symptoms. Interventions appropriate for the symptoms and an evaluation of those interventions are crucial in providing good end-of-life care.

Pain

Pain is the most prevalent symptom in critical care units and is distressing to patients and families.[11] Diseases, procedures, and interventions such as turning, suctioning, and wound care can be sources of painful stimuli.[22,23] Assessing for the presence of pain and intervening to prevent or treat it using pharmacological and nonpharmacological interventions should be incorporated into every patient's care plan. Dose escalation to treat severe pain is an appropriate treatment modality provided there are accurate pain assessments and in the hands of skilled clinicians.[22] Including a bowel regimen to prevent constipation is crucial in the management of pain. Chapter 5 describes, in detail, the assessment of pain and nursing interventions that can be used to treat it.

Dyspnea

It is estimated that dyspnea is present in 21% to 90% of all patients with a life-threatening illness.[24] Causes of dyspnea can include the underlying disease pathology (eg, chronic obstructive pulmonary disease, pulmonary embolism, pleural effusion); anxiety; or family, spiritual, or social issues. Investigation for the source of the dyspnea directs the nurse to the appropriate intervention. Accurate and frequent assessment of dyspnea will also alert the nurse to the presence and severity of this distressing symptom.[25] Instruments that can accurately assess dyspnea include the Modified Borg Scale[26] for patients who can verbally respond and the Respiratory Distress Observation Scale (RDOS)[27] for patients who cannot verbally respond. Common interventions used for dyspnea include oxygen, opioids, and anxiolytics.[25] Nonpharmacological interventions such as pursed-lip breathing, relaxation strategies, reducing the room temperature (but not chilling the patient), reducing the number of people in the room at one time, keeping an unobstructed line of sight between the patient and the outside environment, and using a fan to blow gently across the face (not

Table 6-1 End-of-Life Care Resources

Organization	Website
End-of-Life Nursing Education Consortium (ELNEC)	http://www.aacn.nche.edu/elnec/
Education in Palliative and End-of-Life Care (EPEC)	http://www.epec.net
End-of-Life/Palliative Education Resource Center (EPERC)	http://www.eperc.mcw.edu
City of Hope Pain & Palliative Care Resource Center	http://www.cityofhope.org/prc/
National Hospice and Palliative Care Organization	http://www.nhpco.org
Nursing Leadership Academy on End-of-Life Care	http://www.palliativecarenursing.net
Center to Advance Palliative Care (CAPC)	http://www.capc.org
National Consensus Project for Quality Palliative Care (NCP)	http://www.nationalconsensusproject.org
Hospice and Palliative Nurses Association (HPNA)	http://www.hpna.org
American Association of Critical-Care Nurses (AACN)	http://www.aacn.org
Emergency Nurses Association (ENA)	http://www.ena.org
Association of Organ Procurement Organizations	http://www.aopo.org

directly into mucous membranes) have all been found to be effective in decreasing dyspnea.[25,28]

Anxiety and Agitation

Patients and families who face life-threatening illnesses commonly experience anxiety.[28] Anxiety can be related to any number of physical, emotional, psychological, social, practical, and spiritual issues. Assessment of anxiety can be complex, and an interdisciplinary approach, including nursing, social services, psychology, and chaplaincy, may be needed to evaluate the patient accurately and treat the anxiety properly. Nonpharmacological interventions may include counseling, taking care of practical matters (eg, arranging for the care of a pet), and arranging for spiritual concerns to be addressed (eg, arranging for a visit from a clergy member). If medication is needed, short- or long-acting benzodiazepines and atypical antidepressants may be helpful. Additional interventions for anxiety are discussed in Chapter 2.

Depression

When confronted with a serious illness, many patients experience intense sadness and anxiety accompanied by depressive symptoms such as anhedonia (loss of pleasure); loss of self-esteem; pervasive despair; thoughts of suicide; or feelings of helplessness, hopelessness, or worthlessness.[28] These are natural feelings and are usually present for only a short time. It is a myth that depression is "normal" at the end of life. If these feelings of depression persist, appropriate treatment needs to be initiated using a multidimensional approach, such as supportive psychotherapy, cognitive–behavioral therapy, and antidepressants.

Delirium

Delirium is an acute change in awareness or cognitive status that may manifest as agitation, withdrawal, or confusion. "Confusion" is an all-inclusive term that refers to inappropriate behavior, disorientation, or hallucinations. Terminal delirium is common in patients near death and may manifest as day–night reversal.[28] Management of delirium during the end-of-life phase is focused more on symptom control and relief of the distress of the patient and family than on the diagnosis and treatment of the underlying cause of the delirium. Benzodiazepines or neuroleptics (eg, haloperidol) are helpful in managing this symptom.

Nausea and Vomiting

Nausea is very common in patients with advanced disease. Nausea can be acute, delayed, or anticipatory. It can be exhausting, debilitating, and frustrating for the patient and the family. The pathophysiology of nausea and vomiting is complex and can vary based on the underlying etiology. Causes of nausea and vomiting may include physiological factors such as gastrointestinal causes (eg, intestinal obstruction, constipation, pancreatitis); metabolic causes (eg, hypercalcemia, uremia); central nervous system causes (eg, increased intracranial pressure); emotional factors; treatment-related factors (eg, chemotherapy); and vestibular disturbances.

A careful assessment and investigation of the source of nausea and vomiting is important in determining the appropriate treatment course. Drug classes that are commonly used to treat nausea and vomiting are serotonin (5-HT) receptor agonists (eg, ondansetron), anticholinergics (eg, hyoscine hydrobromide), antihistamines (eg, dimenhydrinate), phenothiazines (eg, prochlorperazine), steroids (eg, dexamethasone), prokinetic agents (eg, metoclopramide), butyrophenones (eg, haloperidol), and benzodiazepines (eg, lorazepam). A nasogastric tube may be used, but it may cause discomfort. To relieve persistent nausea and vomiting, surgery to resect an intestinal obstruction may be appropriate. If a patient has an unresectable intestinal obstruction, a draining percutaneous endoscopic gastrostomy tube may also be placed. Lastly, patients should be positioned to avoid any aspiration of emesis.

Palliative or End-of-Life Sedation

Palliative sedation, also known as end-of-life or terminal sedation, may be considered when all interventions have failed to control the symptoms. Palliative sedation is used when the patient (1) is experiencing unbearable and unmanageable pain or other symptoms, and (2) is approaching the last hours or days of his or her life.[28] The goal of end-of-life sedation is to produce a level of obtundation sufficient to relieve suffering without hastening death.[28] Before palliative sedation is considered, specialists in pain or palliative care are consulted, and it is verified that all therapies have been attempted without success. In addition, other disciplines such as social services, chaplaincy services, and psychology, should be consulted to investigate other potential causes of suffering before resorting to palliative sedation.

▲ Advanced Care Planning

Advanced care planning involves deciding how a patient would like to be treated in the event that he or she becomes unable to make decisions or communicate his or her wishes regarding care.[6] Advanced care planning involves more than just advance directives—it also involves issues such as determining health care proxies as well as trying to discover from the patient or the health care proxy the preferences for the goals of care during the end-of-life phase.

The critical care nurse communicates with the patient's primary care provider, who may have a long-standing relationship with the patient and know the patient's preferences regarding end-of-life treatment. The primary care provider may have had conversations with the patient on this subject. It is important to note that some patients want aggressive treatment despite a poor prognosis, whereas other patients want to forego any aggressive treatment despite the treatment's probable success. Patients are allowed, by federal law, to refuse treatment.

Advance Directives

Advance directives are written or oral instructions about future medical care that are to be followed in the event that the person loses the capacity to make decisions.[29] Types of advance directives include living wills and health care proxies (durable powers of attorney for health care). Each state regulates the use of advance directives differently. Advance directives are not "set in stone." They can be revised, orally or in writing, at any time.

A health care proxy is a person who has been designated to make decisions in the event that the patient cannot make decisions for himself or herself. The designation of a person as a health care proxy must be in written form and should always be up to date. The proxy should know the preferences of the patient and be able to communicate and adhere to those preferences. He or she should not confuse his or her own wishes and desires with those of the patient. Health care proxies are also known as "surrogate decision makers" or "health care agents."

Do Not Resuscitate and Do Not Attempt Resuscitation Orders

The standard of care for patients who suffer a cardiac or respiratory arrest is to initiate CPR. Do not resuscitate (DNR) or do not attempt resuscitation (DNAR) orders are orders placed by a physician, most often with the consent of the patient or the health care proxy, to alert other care givers that if the patient has a cardiac or pulmonary arrest, no attempts to restore cardiac or pulmonary function should occur.[12,30]

Although resuscitation efforts should not be initiated for a patient with a DNR or DNAR order, the patient should continue to receive appropriate care. In one study of critically ill cancer patients in a surgical ICU, researchers noted that the patients with a DNR or DNAR order received less medical care than other patients.[31] However, supportive nursing care remained unchanged. It is important to recognize that DNR and DNAR are not intended to give the impression that nurses should give inappropriate care.[32]

▲ Family Centered Care

Family centered care is a cornerstone of critical and palliative care. In palliative care, the patient is recognized as being part of a larger social network. Serious illness and death affect not only the patient but also the family. The Society of Critical Care Medicine published clinical practice guidelines[7] delineating recommendations to support families during critical illness. These recommendations include endorsement of a shared decision-making model, early and repeated care conferencing to reduce family stress and improve consistency in communication, honoring culturally appropriate requests for truth-telling and informed refusal, spiritual support, staff education and debriefing to minimize the impact of family interactions on staff health, family presence at both rounds and resuscitation, open flexible visitation, way-finding and family friendly signage, and family support before, during, and after a death.

When the patient can communicate, according to Stannard,[33] the ideal definition of family is whomever the patient defines as his or her family. When a patient is unable to communicate, the practical definition of family is anyone who shares a history and a future with the patient. The legal definition of family is based on blood relations and is purposefully narrow and limiting to clearly distinguish who may have authority over the patient should the patient lose decision-making capacity.

Family Presence During Resuscitation

In a critical review of the literature, Halm[34] noted that research has found that families have a right to be present during resuscitation; in addition, families report that being present during resuscitation was helpful during the bereavement process. Family members who have been present during resuscitations have not experienced more anxiety, depression, grief, intrusive imagery, or avoidance behavior compared with family members who did not witness resuscitations. In addition, there is no evidence to substantiate that the presence of family members invites litigation.

However, research studies do report that many health care providers are uncomfortable with family presence. Nurses who have less experience with resuscitation report more discomfort with family presence than nurses who have more such experience. In addition, the staff surveyed expressed concern that family members may take time and attention away from the patient. The AACN recommends that hospitals should have policies and procedures about how family presence during resuscitation is to be handled in their institutions.[35] It has been suggested that a successful family presence program depends on having a dedicated staff member attend to the family witnessing the resuscitative efforts. See Evidence-Based Practice Highlight 6-1.

Visitation

To the greatest extent possible, families should be free to visit a patient who is near death, to allow for coping during this period. Family members may communicate with and touch the patient, which may reassure both the patient and family. During this period of closure, cultural or spiritual ceremonies may also take place. Staff who have developed a relationship with the family should continue to work with the patient and family to the greatest extent possible. The extended visiting hours provide a continuity of care that is invaluable to families and helps cultivate a trusting relationship to reassure families that the nurses are working for the benefit of the patient.

It is important to be aware of the dynamics of each individual family. For example, if there is tension among certain family members, a visiting schedule may need to be established to allow family members to see the patient without crossing paths. In addition, the nurse should be alert to any signs from the patient that a particular family member is unwelcome. The patient may exhibit signs of agitation when that person is in the room. The nurse acts as an advocate to uphold the patient's wishes. Visitation advocacy as it relates to families and the critical care environment is discussed in more detail in Chapter 3.

EVIDENCE-BASED PRACTICE HIGHLIGHT 6-1

Family Presence During Resuscitation and Invasive Procedures

△ Expected Practice

- Family members* of all patients undergoing resuscitation and invasive procedures should be given the option of presence at the bedside. (Level B)
- All patient care units should have an approved written practice document (ie, policy, procedure, or standard of care) for presenting the option of family presence during resuscitation and bedside invasive procedures. (Level D)

△ Supporting Evidence

- Research [1–11] and public opinion polls [12–14] have found that 50% to 96% of consumers believe family members should be offered the opportunity to be present during emergency procedures and at the time of their loved one's death.
- Despite support by professional organizations and critical care experts, [15–24] only 5% of critical care units in the United States have written policies allowing family presence.[25] Surveys of nurses' practice find that most critical care nurses have been requested by family members to be present during resuscitation and invasive procedures and have brought families to the bedside, despite the lack of formal hospital policies.[25–27]
- Studies find the following benefits of family presence:
  - For patients: Almost all children want their parents present during medical procedures;[28–30] and adult patients report that having family members at the bedside comforted and helped them.[3,31–32]
  - For family members: Their presence at the bedside helped in removing doubt about the patient's condition by witnessing that everything possible was being done.[8,9,32–35] It decreased their anxiety and fear about what was happening to their loved one.[7,10,29,32,36–37] It facilitated their need to be together[8,10] and the need to help and support their loved one. [8–11, 33,34,36] They experienced a sense of closure,[3,8,11,34] and their presence facilitated the grief process should death occur.[3,5,11,32–36]
- Studies show that 94% to 100% of families involved in family presence events would do so again.[3,7–9,33,36]
- Studies also find that there are no patient care disruptions, no negative outcomes during family presence events,[8,9,29,32–34,38–39] and no adverse psychological effects among family members who participated at the bedside.[8–10,32,40]

AACN Evidence Leveling System

Level A Meta-analysis of quantitative studies or metasynthesis of qualitative studies with results that consistently support a specific action, intervention, or treatment.
Level B Well-designed, controlled studies with results that consistently support a specific action, intervention, or treatment.
Level C Qualitative studies, descriptive or correlational studies, integrative review, systematic reviews, or randomized controlled trials with inconsistent results.
Level D Peer-reviewed professional organizational standards with clinical studies to support recommendations.
Level E Multiple case reports, theory-based evidence from expert opinions, or peer-reviewed professional organizational standards without clinical studies to support recommendations.
Level M Manufacturer's recommendations only.

Excerpted from American Association of Critical-Care Nurses Practice Alert. Available online at http://aacn.org. All references cited in this alert are available with the associated resources related to this chapter. Visit: http://thepoint.lww.com

*Family members are those individuals who are relatives or significant others with whom the patient shares an established relationship.

Family Conferences

The family conference is a mechanism for sharing information in an organized way among clinicians and family members. During the family conference, the health care team (1) provides information about the condition of the patient and the patient's prognosis and (2) reviews recommendations from the primary and consult services. Family conferences also serve as a forum for exploring future care preferences with the family—how family members may wish to participate in determining the goals of care for the patient.[36] Cultural or religious beliefs may influence how these conversations develop and how the family reacts to the information.

Careful planning should occur before the family conference as family conferences can be very stressful to all participants. Curtis et al[36] describe the nurse's role before and after the family conference (Box 6-2). Box 6-3 describes how to facilitate a family conference. Encouraging the family to be active participants during the family conference increases their level of satisfaction and improves the quality of communication among providers and families.[37] Early and proactive multidisciplinary meetings help reduce confusing or conflicting messages and improve emotional and spiritual support to the family.[38]

Bereavement Care

The death of a patient can affect the family members and the staff in different ways. Previous coping skills, cultural and spiritual beliefs, and the circumstances surrounding the death influence the grief experience. A multidisciplinary team consisting of other nurses, social workers, chaplains, physicians, and volunteers can assist family members and staff with managing their grief. Critical care nurses should be familiar with the bereavement information and support services available within their institutions for both family members and themselves. Bereavement support includes providing family members with information regarding what to do after the death and who can be contacted at the hospital if questions arise.

Critical care staff should do everything possible to allow the family sufficient time to go through their leave-taking rituals. Bed shortages can make this difficult. However, not allowing family members the chance to say goodbye can complicate the grieving process. Survivors have reported

BOX 6-2 The Nurse's Role Before and After the Family Conference

Before the Conference

- Explain to the family about the patient's medical equipment and therapies.
- Tell the family what to expect during their conference with the health care team members.
- Talk with the family about their spiritual or religious needs, and take actions to address the unmet spiritual or religious needs.
- Talk with the family about specific cultural needs and take actions to address unmet cultural needs.
- Talk with the family about what the patient valued in life.
- Talk with the family about the patient's illness and treatment.
- Talk with the family about their feelings.
- Reminisce with the family about the patient.
- Tell the family it is all right to talk to and touch their loved one.
- Discuss with the family what the patient might have wanted if he/she were able to participate in the treatment decision-making process.
- Locate a private place or room for the family to talk among themselves.

After the Conference

- Talk with the family about how the conference went.
- Talk with any other health care team members who were present at the conference about how the conference went.
- Ask the family if they had any questions following the conference.
- Talk with the family about their feelings.
- Talk with the family about any disagreement among the family concerning the plan of care.
- Talk with the family about changes in the patient's plan of care as a result of the conference.
- Support the decisions the family made during the conference.
- Assure the family that the patient will be kept comfortable.
- Tell the family it is all right to talk to and touch their loved one.
- Locate a private place or room for the family to talk among themselves.

From Curtis JR, Patrick DL, Shannon SE, et al: The family conference as a focus to improve communication about end-of-life care in the intensive care unit: Opportunities for improvement. Crit Care Med 29(2 Suppl): N26–N33, 2001

that they remember unsatisfactory interactions with staff for a very long time. Sensitivity must be exercised during this potentially traumatic period.

▲ Emotional, Psychological, Social, and Spiritual Care

Patients nearing the end of their lives may experience emotional, psychological, social, and spiritual crises. Critical care nurses play a vital role in helping patients identify these concerns. An interdisciplinary team can attend to these potential feelings of loss, isolation, fear, and existential distress. At times, these crises can manifest as physical symptoms, such as pain, dyspnea, and fatigue. To assist patients at the end of life, assessment and interventions by social services, chaplaincy, psychologists, and volunteers are encouraged. One way to conduct a spiritual assessment is by using the FICA tool (see Box 6-4).[39]

BOX 6-3 Facilitating a Family Conference

Preparing for an ICU Family Conference About End-of-Life Care

- Review previous knowledge of the patient and/or family.
- Review previous knowledge of the family's attitudes and reactions.
- Review your knowledge of the disease—prognosis, treatment options.
- Examine your own personal feelings, attitudes, biases, and grieving.
- Plan the specifics of location and setting: a quiet, private place.
- Discuss with the family in advance about who will be present.

Holding an ICU Family Conference About End-of-Life Care

- Introduce everyone present.
- If appropriate, set the tone in a nonthreatening way: "This is a conversation we have with all families…."
- Discuss the goals of the specific conference.
- Find out what the family understands.
- Review what has happened and what is happening to the patient.
- Discuss prognosis frankly in a way that is meaningful to the family.
- Acknowledge uncertainty in the prognosis.
- Review the principle of substituted judgment: "What would the patient want?"
- Support the family's decision.
- Do not discourage all hope; consider redirecting hope toward a comfortable death with dignity if appropriate.
- Avoid temptation to give too much medical detail.
- Make it clear that withholding life-sustaining treatment is not withholding caring.
- Make explicit what care will be provided including symptom management, where the care will be delivered, and the family's access to the patient.
- If life-sustaining treatments will be withheld or withdrawn, discuss what the patient's death might be like.
- Use repetition to show that you understand what the patient or family is saying.
- Acknowledge strong emotions and use reflection to encourage patients or families to talk about these emotions.
- Tolerate silence.

Finishing an ICU Family Conference About End-of-Life Care

- Achieve common understanding of the disease and treatment issues
- Make a recommendation about treatment.
- Ask if there are any questions.
- Ensure basic follow-up plan, and make sure the family knows how to reach you for questions.

From Curtis JR, Patrick DL, Shannon SE, et al: The family conference as a focus to improve communication about end-of-life care in the intensive care unit: Opportunities for improvement. Crit Care Med 29(2 Suppl): N26–N33, 2001

▲ Facilitating Communication

Communication among the health care team, the patient, and family is the most important aspect of care giving in critical care, especially at the end of life. Through good communication, all people involved in the patient's care have a better understanding of how to care for the patient and family through this hospitalization. In addition, good

BOX 6-4 Spiritual Assessment

F	Faith, Belief, Meaning "Do you consider yourself spiritual or religious?" or "Do you have spiritual beliefs that help you cope with stress?" If the patient responds "no," the nurse might ask "What gives your life meaning?"
I	Importance and Influence "What importance does your faith or belief have in your life? Have your beliefs influenced you in how you handle stress? Do you have specific beliefs that might influence your health care decisions?"
C	Community "Are you a part of a spiritual or religious community? Is this of support to you and how? Is there a core group of people you really love or who are important to you?" Communities such as churches, temples, and mosques can serve as strong support systems for some patients.
A	Address/Action in Care "How should the health care provider address these issues in your health care?" Referral to chaplains, clergy, and other spiritual care providers.

Puchalski CM. Spirituality and the care of patients at the end-of-life: An essential component of care. Omega (Westport) 56(1):33–46, 2007

communication facilitates a healing environment that supports the physical and psychosocial needs of the patient, family, and providers. Three significant communication issues that surface frequently in end-of-life care include establishing treatment goals and priorities, ensuring interdisciplinary communication, and delivering bad news.

Establishing Treatment Goals and Priorities

Establishing goals and treatment priorities is essential to facilitating decision making with regard to care. The way in which options are presented can influence the decisions the patient and family make. For example, if a nurse asks the family, "Do you want the health care team to do everything for your loved one," it sets the family up for a "yes" answer. In the family's mind, the opposite of "everything" is "nothing." So, if the family answers "no" to the question, they may feel as if they are abandoning their loved one. In addition, it is important that nurses avoid ambiguous language and clearly define terms to ensure a shared knowledge. For example, the critical care nurse's understanding of "everything" commonly means intubation, CPR, defibrillation, and other aggressive procedures, whereas the family's understanding of "everything" may include only those interventions that might be helpful and calling a spiritual leader.

Emanuel et al[28] suggest a seven-step approach to help negotiate goals for care for patients.

1. Create the proper setting. Sit down, ensure privacy, and allow adequate time.
2. Determine what the patient and family know. Clarify the current situation and the context in which decisions about goals of care should be made. For example, if the family thinks that the renal failure is transient, yet the nurses believe the kidneys are beyond recovery, the determination of goals of care must be delayed until everyone has agreed about the clinical situation.
3. Explore what the patient and family are expecting or hoping for, such as asking the family what they hope will happen during this last hospitalization, or asking the family what outcomes they think will be attained while in the ICU. Understanding these hopes and expectations will assist the nurse in tailoring communication and reorienting families to what is or might be possible. Focus on what you will do to achieve those expectations and hopes. As appropriate, identify those things that you cannot do, perhaps because they will not help achieve the goals or because they are not possible.
4. Suggest realistic goals. To assist with decision making, share your knowledge about the patient's illness, its natural course, the experience of patients in similar circumstances, and the effects that contemporary health care may have. After sharing this information, suggest realistic goals (eg, comfort, peace, closure, loving care, withdrawal of interventions) and how they can be achieved. Work through unreasonable or unrealistic expectations.
5. Respond empathically to the emotions that may arise.
6. Make a plan and follow through with it.
7. Review and revise the goals and treatments as appropriate.

Ensuring Interdisciplinary Communication

A clear and a unified communication process is important to minimize confusion and distress among patients, families, and the health care team.[40] Critical care nurses should explore their understandings and beliefs about prognosis, goals, and plans of care and share these understandings with other health care providers to develop a unified message before discussing these issues with the families. An interdisciplinary approach in which all nurses are giving the same information consistently is ideal. Consensus among providers is an important step in deciding how treatment choices are presented.[40] Providing conflicting information creates confusion for everyone involved and may lead families to request nonbeneficial interventions. Being asked to provide care that is not helpful for the patient can create moral distress in nurses. Other disciplines such as social services, chaplaincy, and the bioethics committee can assist in clarifying issues and values among patients, families, and providers.

Delivering Bad or Serious News

Despite the best efforts of the health care team, patients may not respond positively to interventions. Keeping an honest and open line of communication is essential to preserve the trust of the patient and family. For this reason, it is important that critical care nurses practice strategies for delivering bad news. Such news can range from reporting that an antibiotic is not reducing an infection or a vasopressor medication is not maintaining an acceptable blood pressure to telling a family member that a patient has died. Because nurses are at the bedside 24 hours a day, communicating with families early that a patient is not doing well may help avoid a "surprise" announcement that the patient has died. Critical care nurses must remember

that family members are not health care professionals. The health care system requires that patients and their proxy decision makers be active in making decisions about health care treatment. However, at times, the health care team tries to place the responsibility of making a crucial decision, such as withdrawing mechanical ventilation, on the family because clinicians may fear a threat of legal retaliation and may try to abdicate responsibility for the decision. A better approach would be to help the family understand the benefits and drawbacks of continuing mechanical ventilation and making the decision jointly. Even if family members are health care professionals, they are family members first and health care professionals second, and they may make decisions based more on their relationship with their loved one than on sound medical or nursing decisions.

Simple strategies for communicating bad news may include phrases such as the following:

- "The blood pressure is worrisome given the amount of medication that we are giving your sister. We have reached the limit of how much we can safely give, and her blood pressure is not responding."
- "The ventilator alarm keeps ringing. It is letting me know that your father's lungs are becoming more resistant to mechanical ventilation. This is not a good sign."
- "I have noticed that your mother's kidneys have not been working well for the past couple of days. We have been trying to reverse her disease. However, now it seems that her heart and lungs are having difficulty as well."

Phrasing bad news in this way clearly indicates that the patient is not doing well but that the health care team is doing its best to help the patient. If discussions regarding withholding or withdrawing life-sustaining measures become necessary, the family may be more receptive because they see what the nurses are seeing.

Notifying the family members that the patient has died is a special case of delivering bad news. The manner that the nurse uses to deliver the bad news has a significant impact on how the family members remember the last moments of the patient's life. An excellent resource to help nurses learn more about how to communicate bad news to families is the book by Dr. Kenneth Iserson, *Grave Words: Notifying Survivors About Sudden, Unexpected Deaths*.[41] This book recommends that nurses divide death notification into four stages: prepare, inform, support, and afterwards.

1. In the preparation stage, the nurse gathers all the facts surrounding the death of the patient in order to answer any questions that might come up. Family members try and make sense of the death and request information.
2. In the inform stage, the nurse uses the person's name instead of "the patient" or "the deceased."
3. In the support stage, the nurse is available to the family members to answer any questions.
4. In the afterwards stage, the nurse provides information for the family, such as the names of funeral homes, medical examiner/coroner's office information, and who to contact at the hospital if the family has any questions.

Many more interventions and how to discuss these issues with the family are found in Dr. Iserson's book. Using clear, unambiguous language is important when delivering the bad news. Supporting the family members after the notification is essential. Becoming comfortable with the wording of the message (eg, by practicing phrases before they are needed) allows the nurse to focus on the family and their reaction to the message, instead of the message itself and how that message is delivered.

▲ Ethical Issues

Ethical issues affect how nurses work and provide care in the critical care environment. Ethical and legal issues are discussed in general terms in this chapter and in Chapter 8. Four ethical issues with special significance when talking about end-of-life care are the principle of double effect, moral distress, the withdrawal of life-sustaining technology, and organ and tissue donation.

Principle of Double Effect

The principle of double effect is an ethical principle that distinguishes between consequences a person intends and consequences that are unintended but foreseen and may be applicable in various situations where an action has two effects, one good and one bad.[42] The principle of double effect is most commonly applied to the administration of pain medications to patients who are dying. Opioids are used to relieve pain and other symptoms of suffering (ie, the good effect). However, opioids also may cause respiratory and cardiovascular depression that may, if left untreated, lead to death (ie, the bad effect). If the primary intention is to relieve pain and suffering with the recognition that the patient may die, it is morally and legally permissible to administer the opioid. If the primary intention is to cause death, it is not morally or legally permissible to administer the opioid.

The End-of-Life/Palliative Education Resource Center (EPERC) has created Fast Facts, which are quick step-by-step instructions about how to deal with a variety of end-of-life issues (see Table 6-1, p. 69).

Moral Distress

Moral distress occurs when nurses cannot turn moral choices into moral action.[43,44] This distress occurs when the nurse knows the proper course of action to take, but institutional or interpersonal constraints make it nearly impossible to pursue it.[43] For example, nurses tend to recognize when therapies are no longer beneficial or helpful to a patient sooner than family members. For families, it is difficult to realize that therapies may no longer be helpful. Moral distress can arise when the family's understanding about the utility of therapy differs from that of the nurse.

The AACN has identified moral distress as a key issue affecting the work environment. To produce a more healthy occupational environment, the AACN has developed a resource for nurses to use to address this issue.[45] This resource, *The Four A's to Moral Distress*, provides a framework for nurses to address their moral distress and find avenues for its

resolution. The four A's are ask, affirm, assess, and act, which facilitates change, thus creating a healthier nursing environment. Copies of this resource are available to AACN members or by contacting the AACN office (see Table 6-1, p. 69). In addition, hospital bioethics or ethics committees are available to staff to help work through situations in which moral distress is a factor.

Withholding or Withdrawing Life-Sustaining Measures

When it becomes clear to both the nurse and family that additional treatment will not be beneficial, the decision may be made to withdraw life-support methods. Mechanical ventilation is one intervention that is often withdrawn in such circumstances. Other life-sustaining measures that may be stopped include implantable cardiac defibrillators or pacemakers and hemodialysis.

When the decision is made to withdraw a therapy, special considerations are taken to reduce the suffering of the patient and to minimize the exhibition of distress to the family members. In the case of withdrawing mechanical ventilation, the decision is first made jointly with the family. In the case of extubation, opioids and sedatives are administered to the patient to reduce the pain and discomfort. In addition, the alarms to both the ventilator and the cardiac monitor are silenced to focus the family on the patient rather than the technology. Campbell[46] has published recommendations to successfully withdraw a mechanical ventilator. Additionally, many of the Fast Facts on the EPERC website relate to withdrawal of therapies, including mechanical ventilation and tube feeding (see Table 6-1, p. 69).

Organ and Tissue Donation

Organs and tissues can be procured after cardiac death or brain death. Federal law (Public Law 99-5-9; section 9318), Medicare, and the Joint Commission (formerly the Joint Commission on Accreditation of Healthcare Organizations) all require that (1) hospitals have written protocols regarding organ and tissue donation and (2) these institutions give the surviving family members the chance to authorize donation of their family member's tissues and organs.[47] When organ or tissue procurement is an issue, it is important that all family members are given the information they need to make a decision with which they are comfortable and that their grief is respected. In some cases, family members have initiated the conversation with their health care providers independently.

The local organ procurement organization (OPO) can provide additional resources. To find the OPO in your area, see Table 6-1 on page 69.

▲ Caring for the Nurse

Some deaths affect some people significantly. The death of a child, the death of a friend or colleague, mass casualties, or a particularly horrific, traumatic death can have a profound effect on the nurse. Colleagues must be supportive, exploring ways to support each other rather than dismissing the impact the death has on a colleague. According to Badger,[48] some self-care strategies to cope with the event include asking to be relieved from care responsibilities and to take a break; discussing the experience with a colleague, friend, or a nurse leader; taking a moment to reflect on one's feelings after the event; focusing on what was done right; and following basic health principles such as physical exercise, meditation, humor, music, eating properly, and obtaining adequate rest.

Working in a critical care unit is demanding physically, intellectually, and emotionally. Dealing with death on a consistent basis can take its toll on the nurse's well-being.[48] In the critical care setting, nurses caring for the patient may delay attending to their own grief because the demands of the unit and the needs of the family members may take precedence. It is important to be vigilant in recognizing signs and symptoms of unexpressed grief, burnout, and posttraumatic stress. Symptoms may include an increase in the number of sick days; indecision; difficulty with problem solving; isolation or withdrawal; behavioral outbursts; denial and shock; fixation on a single detail; immobilization; a feeling of extreme serenity; emotionally numbing responses, such as withdrawal, pessimism, or a diminished capacity for experiencing pleasure; and intrusive responses, such as unwanted or unpleasant recollections or flashbacks.[48] To maintain emotional health, it is important to seek assistance in dealing with these issues. Nurse leaders and human resources representatives can provide resources to assist with the stresses of working in critical care.

▲ Clinical Applicability Challenges

CASE STUDY

You are caring for Mr. J., a 42-year-old man who suffered a massive myocardial infarction. From the emergency department, he was admitted to the cardiac catheterization laboratory (CCL). He had occlusions in the left anterior descending artery and the left circumflex coronary artery that were opened via angioplasty and stent. However, in the CCL, the patient had persistent hypotension of systolic blood pressures in the 70s, cardiac index of 1.2, and pulmonary edema suggestive of cardiogenic shock. Dobutamine and an intra-aortic balloon pump were initiated in the CCL, and the patient was transferred to the ICU.

1. When the cardiologist arrives to discuss the events and prognosis with Mr. J.'s family, the cardiologist asks, "Do you want us to do everything if his heart stops beating?" The family looks puzzled at the question. As the nurse, what should you do?
2. On hospital day 7, the patient's blood pressure, cardiac index, and mentation have not improved, and the interdisciplinary team has scheduled a family conference. As the nurse, how will you prepare the family for the conference and how will you debrief with them after the conference?
3. The patient continues to decline, and on hospital day 10 the family decides to withdraw the intra-aortic balloon pump and dobutamine. It is 6:30 PM, and Mr. J.'s wife wants to go home to take a shower and eat dinner and come back at 9:00 PM, which is past the posted visiting hours. In addition, the change of shift will occur at 7:00 PM, and you will transfer care to the oncoming nurse. As the nurse, how will you handle her request?

References

1. Rushton CH, Williams MA, Sabatier KH: The integration of palliative care and critical care: One vision, one voice. Crit Care Nurs Clin North Am 14:133–140, 2002
2. Ariès P: The hour of our death. New York, NY: Knopf, 1981
3. Luce JM, Prendergast TJ: The changing nature of death in the ICU. In: Curtis JR, Rubenfeld GD (eds): Managing Death in the Intensive Care Unit. The Transition from Cure to Comfort. New York, NY: Oxford University Press, 2001, p 388
4. Benner P, Kerchner S, Corless IB, et al: Attending death as a human passage: Core nursing principles for end-of-life care. Am J Crit Care 12(6):558–561, 2003
5. American Association of Critical-Care Nurses: AACN standards for establishing and sustaining healthy work environments: A journey to excellence. Am J Crit Care 14(3):187–197, 2005
6. Curtis JR, Treece PD, Nielsen EL, et al: Integrating palliative and critical care: Evaluation of a quality-improvement intervention. Am J Respir Crit Care Med 178(3):269–275, 2008
7. Davidson JE, Powers K, Hedayat KM, et al: Clinical practice guidelines for support of the family in the patient-centered intensive care unit: American College of Critical Care Medicine Task Force 2004–2005. Crit Care Med 35(2):605–622, 2007
8. Ferrell BR, Virani R, Grant M, et al: Evaluation of the End-of-Life Nursing Education Consortium undergraduate faculty training program. J Palliat Med 8(1):107–114, 2005
9. Malloy P, Ferrell BR, Virani R, et al: Evaluation of end-of-life nursing education for continuing education and clinical staff development educators. J Nurses Staff Dev 22(1):31–36, 2006
10. Ferrell BR, Dahlin C, Campbell ML, et al: End-of-life nursing education consortium (ELNEC) training program: Improving palliative care in critical care. Crit Care Nurs Q 30(3):206–212, 2007
11. SUPPORT Investigators: A controlled trial to improve care for seriously ill hospitalized patients. The study to understand prognoses and preferences for outcomes and risks of treatments (SUPPORT). The SUPPORT Principal Investigators [see comments] [published erratum appears in JAMA 275(16):1232, 1996]. JAMA 274(20):1591–1598, 1995
12. Field MJ, Cassel CK: Approaching death. Improving care at the end of life. Washington, DC: Institute of Medicine, 1997, 0-309-06372-8
13. Angus DC, Barnato AE, Linde-Zwirble WT, et al: Use of intensive care at the end of life in the United States: An epidemiologic study. Crit Care Med 32(3):638–643, 2004
14. Egan KA, Labyak MJ: Hospice care: A model for quality end-of-life care. In: Ferrell BA, Coyle N (eds): Palliative Nursing. New York, NY: Oxford University Press, 2001, pp 7–26
15. World Health Organization: Cancer Pain Relief and Palliative Care. Geneva, Switzerland: Author, 1990
16. Curtis JR: Caring for patients with critical illness and their families: The value of the integrated clinical team. Respir Care 53(4):480–487, 2008
17. Hanson LC, Dobbs D, Usher BM, et al: Providers and types of spiritual care during serious illness. J Palliat Med 11(6):907–914, 2008
18. McCormick AJ, Curtis JR, Stowell-Weiss P, et al: Improving social work in intensive care unit palliative care: Results of a quality improvement intervention. J Palliat Med 13(3):297–304, 2010
19. Campbell ML: Palliative care consultation in the intensive care unit. Crit Care Med 34(11 Suppl):S355–S358, 2006
20. Crighton MH, Coyne BM, Tate J, et al: Transitioning to end-of-life care in the intensive care unit: A case of unifying divergent desires. Cancer Nurs 31(6):478–484, 2008
21. Medina J, Puntillo KA: AACN Protocols for Practice: Palliative Care and End-of-Life Issues in Critical Care. Sudbury, MA: Jones & Bartlett, 2006
22. Mularski RA, Puntillo K, Varkey B, et al: Pain management within the palliative and end-of-life care experience in the ICU. Chest 135(5):1360–1369, 2009

23. Puntillo KA, White C, Morris AB, et al: Patients' perceptions and responses to procedural pain: Results from Thunder Project II. Am J Crit Care 10(4):238–251, 2001.
24. Zeppetella G: The palliation of dyspnea in terminal disease. Am J Hosp Palliat Care 15(6):322–330, 1998.
25. Mahler DA, Selecky PA, Harrod CG, et al: American College of Chest Physicians consensus statement on the management of dyspnea in patients with advanced lung or heart disease. Chest 137(3):674–691, 2010
26. Burdon JG, Juniper EF, Killian KJ, et al: The perception of breathlessness in asthma. Am Rev Respir Dis 126(5):825–828, 1982
27. Campbell ML, Templin T, Walch J: A respiratory distress observation scale for patients unable to self-report dyspnea. J Palliat Med 13(3): 285–290, 2010
28. Emanuel LL, von Gunten CF, Ferris FD, et al: The Education in Palliative and End-of-Life Care (EPEC) Curriculum: The EPEC Project. Chicago, IL: Author, 2003
29. National Institue on Aging. Long Distance Caregiving: Chapter 19: What is the difference between an advance directive and a living will? 2006; http://www.nia.nih.gov/HealthInformation/Publications/LongDistanceCaregiving/chapter19.htm. Accessed October 20, 2006
30. Burns JP, Edwards J, Johnson J, et al: Do-not-resuscitate order after 25 years. Crit Care Med 31(5):1543–1550, 2003
31. Keenan CH, Kish SK: The influence of do-not-resuscitate orders on care provided for patients in the surgical intensive care unit of a cancer center. Crit Care Nurs Clin North Am 12(3):385–390, 2000
32. Fields L: DNR does not mean no care. J Neurosci Nurs 39(5):294–296, 2007
33. Stannard D: Family care. In: Schell HM, Puntillo KA (eds): Critical Care Nursing Secrets. St. Louis, MO: Mosby Elsevier, 2006, pp 767–772
34. Halm MA: Family presence during resuscitation: A critical review of the literature. Am J Crit Care 14(6):494–511, 2005
35. Family presence during CPR and invasive procedures. Practice Alert. http://www.aacn.org/AACN/practiceAlert.nsf/Files/FP/$file/Family%20Presence%20During%20CPR%2011–2004.pdf. Accessed October 20, 2006
36. Curtis JR, Patrick DL, Shannon SE, et al: The family conference as a focus to improve communication about end-of-life care in the intensive care unit: Opportunities for improvement. Crit Care Med 29 (2 Suppl):N26–N33, 2001
37. McDonagh JR, Elliott TB, Engelberg RA, et al: Family satisfaction with family conferences about end-of-life care in the intensive care unit: Increased proportion of family speech is associated with increased satisfaction. Crit Care Med 32(7):1484–1488, 2004
38. Machare Delgado E, Callahan A, Paganelli G, et al: Multidisciplinary family meetings in the ICU facilitate end-of-life decision making. Am J Hosp Palliat Care 26(4):295–302, 2009
39. Puchalski CM: Spirituality and the care of patients at the end-of-life: An essential component of care. Omega (Westport) 56(1):33–46, 2007
40. Curtis JR, Patrick DL: How to discuss dying and death in the ICU. In: Curtis JR, Rubenfeld GD (eds): Managing Death in the Intensive Care Unit. New York, NY: Oxford University Press, 2001, pp 85–102
41. Iserson KV: Grave Words: Notifying Survivors About Sudden, Unexpected Deaths. Tuscon, AZ: Galen Press, 1999
42. Williams G: The principle of double effect and terminal sedation. Med Law Rev 9(1):41–53, 2001
43. Jameton A: Nursing Practice: The Ethical Issues. Englewood Cliffs, NJ: Prentice Hall, 1984
44. Rushton CH: Defining and addressing moral distress: tools for critical care nursing leaders. AACN Adv Crit Care 17(2):161–168, 2006
45. The four A's to rise above moral distress. http://www.aacn.org/AACN/practice.nsf/Files/4as/$file/4A's%20to%20Rise%20Above%20Moral%20Distress.pdf. Accessed October 20, 2006
46. Campbell ML: How to withdraw mechanical ventilation: A systematic review of the literature. AACN Adv Crit Care 18(4):397–403; quiz 344–395, 2007
47. Campbell ML, Zalenski R: The emergency department. In: Ferrell BA, Coyle N (eds): Textbook of Palliative Care, 2nd ed. New York, NY: Oxford University Press, 2006 pp 861–869
48. Badger JM: Understanding secondary traumatic stress. Am J Nurs 101(7):26–32, 2001

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

PART 2

Professional Practice Issues in Critical Care

7

Ethical Issues in Critical Care Nursing

Connie M. Ulrich and Christine Grady

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Explain the way ethics assists nurses and other clinicians in resolving moral problems.
2. Name and describe ethical principles applicable to clinical ethics.
3. Describe steps in the process of ethical decision making.
4. Identify resources available to nurses to help resolve ethical dilemmas.
5. Discuss an example of an ethical issue confronted by critical care nurses in practice and how applying ethical principles may help to resolve it.

Critical care nurses face some of the most daunting ethical challenges in clinical practice. Nurses who work in intensive care and step-down units, operating rooms, emergency rooms, and other fast-paced, highly specialized settings must be able to recognize ethical issues they face in the daily care of patients and be prepared to address them in collaboration with patients, families, colleagues, administrators, and relevant others. Because of the precarious condition of critically ill patients, difficult ethical issues often consist of conflicts among members of the health care team surrounding the benefits and burdens of treatment. Some issues may include the following: How to alleviate suffering when the patient and/or family want to continue aggressive measures? How do nurses and other members of the health care team openly discuss end-of-life issues when often these discussions are perceived as giving up hope? How do nurses provide beneficent care in the context of finite resources? And, how can nurses feel good about the choices that are made in critical practice environments, reflecting their professional goals for optimal care? This chapter provides a foundational overview of nursing ethics, the application of ethical principles, and ethical reasoning skills in critical care to enable nurses to confidently advocate for the best interests of their patients.

▲ Differentiating Between Ethics and Morals

Ethics is the study of morality or standards of conduct[1] and a critical reflection or evaluation of moral choices that are made.[1,2] *Morals*, on the other hand, are commonly shared beliefs by members within a society about the "rightness" or "wrongness" of actions and behaviors that include the "shoulds," "should nots," "oughts," and "ought nots" and are usually learned through family systems as well as religious and cultural traditions.[1,2]

Nurses often struggle with ethical issues because they are strong patient advocates and develop intimate human relationships when caring for critically ill patients. These are especially challenging when there is limited consensus about what the *ethically right or wrong* approach may be in any given situation. For example, what "ought" the nurse do if a family member asks that the patient with a poor prognosis not be told about a cancer diagnosis? Should the truth always be told in these kinds of situations? Ulrich et al[3] noted that an *ethical problem* can occur in any clinical or research situation when there are salient questions about the *rightness* or *wrongness*

of particular aspects of the care and treatment of patients. Applying ethical principles and considerations helps nurses to articulate sound reasons for ethical positions, clarify the ethical principles that might be in conflict, and address the issues they encounter.

▲ Ethical Principles

Ethical principles serve as general guidelines in health care decision making. Principles, such as autonomy, beneficence, nonmaleficence, veracity, fidelity, and justice, can guide conduct and provide a basis for ethical reasoning. These principles are not, however, absolute and can conflict with one another. Because critical care nurses address ethically laden patient care problems daily, understanding and applying these principles can assist them in determining an appropriate course of care for their patients.

Autonomy

Autonomy is the right to self-determination or the right to make independent decisions about one's own body or actions free from interference or coercion from others.[1] It is a central value in the Western world and reflects personal values, goals, and convictions for self-governance.

Critical care nurses are often recognized for their advocacy roles and their willingness to speak up for their patients. In fact, the patient care population that they care for seems to demand it. These patients range from those who are ventilator dependent for long periods of time to patients whose families must make immediate decisions pertaining to end-of-life care. It is not unusual for nurses to be "caught in the middle"[4] as they struggle to promote autonomous decision making of their patients, including ensuring that patients have given their informed consent and have opportunities to plan advance directives. In a study investigating the everyday ethical issues that nurses face, the authors found that nurses (including those in acute care settings) reported frequently encountering issues associated with protecting patients' rights and informed consent to treatment procedures.[3]

Informed consent respects autonomy by providing patients and families with the pertinent information they need to make an ethically sound decision. In essence, "respect for autonomy obligates professionals in health care and research involving human subjects to disclose information, to probe for and ensure understanding and voluntariness, and to foster adequate decision making."[1] Conflict can occur when health care professionals have views about care in the patient's best interests that do not match the patient's autonomous wishes. Critical care nurses can use the "Nursing Interventions Classification" (NIC)[5] system for guidance when developing activities to facilitate autonomous decision making in the critical care setting (Box 7-1).

BOX 7-1 NURSING INTERVENTIONS CLASSIFICATION

Supporting Autonomous Decision Making

Definition

Providing information and support for a patient who is making a decision regarding health care.

Activities

- Determine whether there are differences between the patient's view of his or her own condition and the view of health care providers.
- Assist patient to clarify values and expectations that may assist in making critical life choices.
- Inform patient of alternative views or solutions in a clear and supportive manner.
- Help patient identify the advantages and disadvantages of each alternative.
- Establish communication with patient early in admission.
- Facilitate patient's articulation of goals for care.
- Obtain informed consent, when appropriate.
- Facilitate collaborative decision making.
- Be familiar with institution's policies and procedures.
- Respect patient's right to receive or not to receive information.
- Provide information requested by patient.
- Help patient explain decision to others, as needed.
- Serve as a liaison between patient and family.
- Serve as a liaison between patient and other health care providers.
- Use interactive computer software or Web-based decision aides as an adjunct to professional support.
- Refer to legal aid, as appropriate.
- Refer to support groups, as appropriate.

From Bulechek GM, et al: Nursing Interventions Classification (NIC), 5th ed. St. Louis, MO: Mosby, 2008, p 247, with permission.

Beneficence

Nurses are ethically, legally, and professionally obligated to avoid harm and promote benefit for their patients; the American Nurses Association (ANA)[6] notes that nurses' primary commitment is to the "health, welfare, and safety of the client." This includes acts of caring and compassion as well as protection from harm. The ethical principle of beneficence is the *sine qua non* for the nursing profession, guiding all nurses in their daily interactions and activities with patients. The principle of beneficence presupposes that any harm patients are exposed to is justified by the potential benefits. Beauchamp and Childress outline three obligations of beneficence, including preventing harm to others, promoting and acting for the good of others, and eliminating untoward or harmful circumstances.[1]

In the critical care setting, the ethical principle of beneficence often conflicts with other ethical principles, including that of autonomy. Patients and their families in some cases may be more comfortable deferring treatment decisions to their health care provider, especially since the crisis and chaotic nature of intensive care may make it difficult to make decisions. The critical care nurse and physician often speak and act on behalf of a patient who, for example, is neurologically or cognitively impaired. Without knowledge of the patient's autonomous wishes, the nurse acts to promote the patient's best medical interests. Intentionally acting on behalf of another without the other's explicit consent may be considered paternalistic. "Paternalism is commonly framed in terms of the conflict between the primary obligation of physicians, nurses, and other provider–practitioners to abide by the principle of beneficence in their practice and the

assertion of the rights of persons who are receiving services to make autonomous decisions about their lives."[7] Paternalism can be acceptable for patients in the critical care setting when there are benefits to the patient that clearly outweigh the paternalistic action. A common example is helping the patient turn, breathe deeply, cough, and ambulate following an extensive surgical procedure when the patient is in pain, is sleep deprived, or wants to be left alone.

Nonmaleficence

The ethical principle of nonmaleficence obligates us to avoid causing undue pain and suffering or harm to another. This principle is often discussed along with the principle of beneficence because the two are intricately linked. Nonmaleficence is a *prima facie* duty for the nurse, meaning that it is morally binding unless there are other conflicting moral obligations that outweigh its primacy.[1,2,8] Nonmaleficence obligates nurses to avoid harm of not only a physical or bodily nature but also psychological or emotional distress; in some cases, harm can be caused by a breach of professional standards of care. Nurses are constantly balancing the benefits and harms of decisions, treatments, and procedures associated with critically ill patients. Indeed, administration of medications meant to improve a patient's condition, including steroids, morphine, amphotericin B, heparin, and other pharmacological agents, may also have serious adverse effects. Nurses must constantly apply the principle of nonmaleficence in critical care where the quality of life of patients is an important consideration.

Veracity

Nurses remain one of the most trusted professional groups in the United States,[9] and therefore, veracity or truth-telling is foundational to the nurse–patient relationship. The American Nurses Association Code for Nurses states: "Truth-telling and the process of reaching informed choice underlie the exercise of self-determination, which is basic to respect for persons…. Clients have the moral right to determine what will be done with their own person; to be given accurate information, and all the information necessary for making informed judgments."[6]

Much current research suggests that patients want to be told the truth about their diagnosis and need full disclosure of the potential risks and benefits of specific treatments and alternatives to make an informed decision related to their care.[10–12] Sometimes, information can be presented in ways that are misleading or biased even though accurate; this can create conflict and uneasiness. Truth-telling in the clinical setting preserves autonomy and trust and maintains sound communication patterns with patients.

Fidelity

Fidelity is the duty to be faithful to one's patients by keeping promises and fulfilling contracts and commitments. It is the moral covenant between individuals in a relationship.[1] Nurses are involved in many aspects of a patient's immediate care: physical, emotional, spiritual, and psychological. For this reason, they often develop close relationships with patients and their families and are called upon to be forthright and to keep their promises to inform, update, and communicate unforeseen circumstances that might occur in the nurse–patient–family relationship. However, there are times when keeping promises can be problematic, for example, in the following case of a husband asking the nurse to keep a secret from his ill wife. The husband of a 50-year-old woman who is suffering from sudden onset bacterial pneumonia with severe hypoxia, respiratory distress, and receiving mechanical ventilation in the intensive care unit (ICU) tells the primary nurse that he is HIV positive and has known his status for the past year. He has been afraid to tell his wife and now wonders if she also has the disease. Should the nurse agree to keep his secret and withhold the information from the patient until her husband can work through his emotions and the best way to discuss the issue as he is terrified that it will destroy their relationship? Does the nurse have obligations based on an implicit promise to the patient to make sure that her current illness is not HIV related? How should the nurse handle it after the patient is weaned from the ventilator and asks what is wrong with her and why she went into respiratory distress?

Confidentiality is an aspect of fidelity that is an essential component of trusting relationships. Patients have the right to know who has access to their personal health care information and that it will be kept confidential and that there are safeguards in place for privacy, security, and authorized access.[13] A commitment to maintain confidentiality in patient–provider relationships is part of every professional health care code of ethics, but it is not absolute. In certain circumstances, confidentiality can be broken. This can occur if there is imminent danger to the patient or other third party. Additionally, disclosure is required for public health reporting purposes related to certain infectious diseases or abusive situations.[14]

Justice

Justice is an important ethical principle in critical care settings and is usually defined in terms of fairness or that which is due to others. In health care, it is often discussed in terms of distributive justice or how one allocates scarce or finite resources. The most recent national health care debate has brought to light many ethical concerns associated with the escalating costs of U.S. health care as well as the quality of care that is provided to the public and the cost-effectiveness and cost-benefit analysis of current standard therapies. Questions of distributive justice often arise in intensive and emergency settings because of the need to prioritize care; this happens on a daily basis. ICU care is expensive and unfortunately, about one of five Americans die in ICUs each year with the elderly accounting for up to half of all ICU admissions.[15] Not only does distributive justice concern the appropriate allocation of technology and aggressive measures but also the unit and nursing-based resources needed to provide that care.

Hospital understaffing has emerged as a major factor in the care of critically ill patients. Aiken et al[16] at the University of Pennsylvania reported an association between nurse staffing levels and patient mortality and failure to rescue (mortality following complications) in adult surgical patients cared for in Pennsylvania hospitals. In this research, nurses were found

to be important surveillance and safety measures within hospitals because higher patient-to-nurse ratios negatively impacted outcomes of care. Staffing is an ethical concern and an indicator of distributive justice because it directly affects the beneficent care of patients. Staffing inadequacies have been found to be a major source of ethics-related stress that ultimately impacts job satisfaction and retention of nurses.[3] Critical care nurse managers and staff nurses are constantly allocating resources as they weigh the patient care needs required on their units, the number of admissions and available beds, and the ability of staff to meet those needs. Staffing shortages sometimes require utilizing personnel who are not readily familiar with a particular unit or hospital, and this could include novice nurses or those designated as float staff. Critical care unit-based orientation and other special training sessions are imperative to address standards of practice, policies, procedures, and additional technological aspects of caring for critically injured patients.

Withholding and Withdrawing Treatment

Determining when to withhold or withdraw treatment for critically ill or injured patients is stressful for all of those involved. This is true not only for the families who surrender their hope for continued survival but also for those health care providers who have been caring for a patient for an extended period of time. Withholding care may be at the request of the patient or family and is sometimes a consideration for those patients deemed critically or irreversibly ill and for whom there is no foreseeable recovery regardless of the treatment offered. Withdrawing life supportive treatments (eg, ventilator and respiratory support, nutrition, and hydration) once they have been started is also sometimes ethically appropriate, but generally creates more controversy and distress for physicians and nurses. Ethical and legal advice is often sought, and professional guidelines (eg, Society of Critical Care Medicine) and in-house hospital and ethics committees' policies and procedures can assist both physicians and nurses when making these determinations. Brock notes that "any set of circumstances that would morally justify not starting life-sustaining treatment would justify stopping it as well."[17]

Open communication is imperative in critical care as "the majority of deaths in the intensive care setting involve withholding or withdrawing multiple life-sustaining therapies."[18] Families are often confronted with the difficult task of deciphering medical information about the prognosis of their loved one. Some patients and their families prefer a shared decision-making model or one in which decisions are made with clinicians; others prefer recommendations by the physician.[19] Critical care nurses can help prepare families for difficult discussions about withdrawing or withholding life supportive treatments by facilitating care conferences to address cultural, spiritual, and value-based needs of the patient. When a patient cannot speak for himself, it is important to talk with families about the wishes of their loved one and to present information about the patient's plan of care in a timely fashion with up-to-date, clear, and honest expectations about the situation at hand.

Medical Futility

Medical futility has been defined in various ways and is frequently discussed in critical care situations. When the health care team believes that an intervention has no prospect of helping the patient, they may describe the intervention as futile. This can create much conflict and turmoil if family members hold out hope of recovery and seek to pursue all aggressive measures. Medical futility includes both a qualitative (ie, value judgment) and a quantitative aspect.[20] Schneiderman et al[20] note that "when physicians conclude (either through personal experience, experiences shared with colleagues, or consideration of reported empirical data) that in the last 100 cases, a medical treatment has been useless, they should regard that treatment as futile. Furthermore, the Hastings Center notes that physicians have no obligation to comply with a patient or surrogate's request for treatment if that treatment is deemed to provide no physiological benefit.[21]

▲ Ethics as a Foundation for Nursing Practice

Nursing is guided by historical precepts and ethical norms that form the basis of nursing's contract with society. This fiduciary relationship is based on trust. In fact, in public polls about health care professionals, nurses consistently rank highly on issues of trust and ethical standards. Nursing's primary commitment is to the patient and to ensure the delivery of safe, quality care; however, there is also a collective moral responsibility to serve the good of society. All professional groups define themselves by their body of scholarly knowledge, degree of self-regulation, and the professional code of ethics that underlie their beliefs, practices, and norms.[22,23]

Ethics is important in understanding how the philosophy of the profession of nursing as well as its values, duties, and obligations to patient care remain constant within a society with a changing demography (ie, chronically ill and aging society) and rising health care costs. Ongoing evaluations of the professional standards of nursing and specifically, critical care nursing, are necessary as the profession continues to evolve and develop new knowledge that will address advancing technology and its use in the care of the critically ill.

▲ Nursing Code of Ethics

The American Nurses Association (ANA) *Code of Ethics for Nurses* was first adopted in 1950 but has gone through several revisions to reflect the ethical, professional, and societal concerns of the day.[24] The most recent *Code of Ethics for Nurses with Interpretive Statements*[6] (2001) is a document that embodies the moral values of the profession. It represents not only the expectations for ethical conduct in clinical practice but also the duty of the nurse to protect his or her own integrity within the health care environment. The Code symbolizes a collective agreement among its members for societal good and serves as a guideline for nurses to follow in ethical decision making related to patient, family, and community care. There may be times when the law and ethics conflict; however, every discipline, including nursing, is bound to a code of ethics that represents the moral basis for its professional standards of practice and helps to ensure the public's trust with their care. The nine provisions of the code are outlined in Box 7-2.

BOX 7-2 ANA Code of Ethics for Nurses

1. The nurse, in all professional relationships, practices with compassion and respect for the inherent dignity, worth, and uniqueness of every individual, unrestricted by considerations of social or economic status, personal attributes, or the nature of health problems.
2. The nurse's primary commitment is to the patient, whether an individual, family, group, or community.
3. The nurse promotes, advocates for, and strives to protect the health safety, and rights of the patient.
4. The nurse is responsible and accountable for individual nursing practice and determines the appropriate delegation of tasks consistent with the nurse's obligation to provide optimum patient care.
5. The nurse owes the same duties to self as to others, including the responsibility to preserve integrity and safety, to maintain competence, and to continue personal and professional growth.
6. The nurse participates in establishing, maintaining, and improving health care environments and conditions of employment conducive to the provision of quality health care and consistent with the values of the profession through individual and collective action.
7. The nurse participates in the advancement of the profession through contributions to practice, education, administration, and knowledge development.
8. The nurse collaborates with other health professionals and the public in promoting community, national, and international efforts to meet health needs.
9. The profession of nursing, as represented by associations and other members, is responsible for articulating nursing values, for maintaining the integrity of the profession and its practice, and for shaping social policy.

From American Nurses Association Code of Ethics for Nurses. Washington, DC: ANA, 2001.

▲ Ethical Issues

An ethical issue or problem can occur in any clinical situation where there are concerns about what might be "morally right or wrong" in the care of the critically ill patient and his or her family. There are many ethical issues in critical care. These can include but are not limited to end-of-life treatment decisions, truth-telling, transplantation, do not resuscitate (DNR), informed consent, clinical research with critically ill patients, assisted suicide, the use of aggressive measures, conflict among colleagues, and resource allocation. Ulrich et al[3] reported that these everyday ethical issues occur frequently and often reflect providers' concerns about protecting the rights of their patients and informed consent to treatment procedures as well as issues of advance care planning and surrogate decision-making.

Although nurses interact daily with patients and their families, some evidence suggests that they are hesitant to speak about the ethical issues they encounter for fear of retaliation.[25] Sulmasy et al[26] stress that nurses can be included in sensitive conversations (eg, DNR) because they are both confident and capable. They are in a key position to listen, explain, and comfort patients and address unmet needs. Health care organizations should develop policies and procedures that provide support to nurses to seek consultation for ethical concerns. Ethics education, in professional training and continuing education, is also essential. Interdisciplinary team work is also needed as the complex needs of intensive care patients require all members of the health care team, including nurses to be part of the discussion.

Moral Distress

Moral distress has become a frequent topic of discussion in the literature and remains a common concern among health care providers, including nurses and physicians. This phenomenon was originally defined by Andrew Jameton[27] more than 20 years ago as the distress that one feels when one knows the ethically correct action to take but is precluded from doing so. Constraints can include those at the individual, societal, or organizational level and consist of patient demands, limited resources, or circumstances within the workplace where one feels powerless because of authoritative structures. Bell and Breslin[28] further add that acting against one's personal and professional values creates moral distress. Physical and emotional symptoms, such as anger, anxiety, frustration, powerlessness and fatigue, are common outcomes associated with moral distress. Additionally, studies have shown that moral distress is linked to nurses' intent to leave their positions.[29,30]

Strategies to minimize moral distress and to create a healthy workplace have been outlined by the American Association of Critical Care Nurses (AACN) and include the four As: Ask, Affirm, Assess, and Act[31] (see Fig. 7-1). Ulrich and Hamric[32] also urge nurses to foster an open communication plan with other health care team members, seek ethics consultation services as needed, uphold a zero tolerance policy for retaliation if members speak out, and continually assess and dialogue about the needs of complex patients.

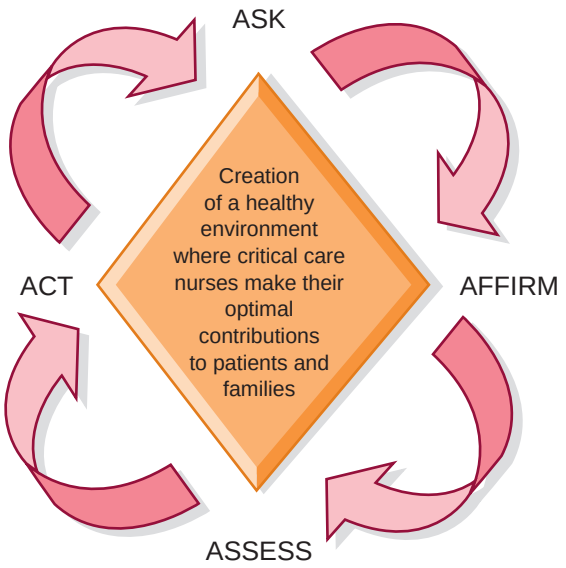


FIGURE 7-1 ▲ Framework to address moral distress. (From American Association of Critical-Care Nurses: Position statement: Moral distress. Aliso Viejo, California, July 8, 2004, AACN).

▲ Ethical Decision Making

Several models exist to assist clinicians in the ethical decision-making process. A pragmatic case method approach is useful because it allows all members of the health care team to focus on a specific clinical case, address the ethical issues that may be morally problematic, and develop a plan of action.

The Case Method Approach

Steps in the case method of ethical decision making are listed in Box 7-3. These steps are similar to the nursing process.

Step One

Assessment is an initial task of any medical or nursing clinician and applies to the ethical decision-making process as well. Determining or identifying the ethical problem is the most critical part of the assessment. One needs to understand the patient's medical condition/problem, as well as the prognosis, potential sequelae, and treatment goals, and any additional contextual factors that may influence the ethical decision making process, including family and organizational systems. It is always important to gather sociodemographic information, such as age, gender, education, setting of care, and other factors (eg, religion, culture, language), that might influence decisions or communication. For example, assessment questions that might be helpful include the following: Is the patient legally competent and capable of making a decision associated with his or her care? What are the preferences of the patient and family? Are there advance directives that outline the patient's wishes for treatment? Are there competing interests that need further discussion such as conflict amongst family members, and/or conflict between the patient, family members, and clinicians? Finally, are institutional factors contributing to the ethical problems presented by the clinical case? The usual goals in the critical care setting, for example, trend toward aggressive treatment rather than palliation or support.

Step Two

Explicating and ranking the relevant ethical issues or moral considerations associated with a particular case of interest is the second step in the ethical decision-making process. Clinicians can use relevant literature, institutional policies, professional groups, commissions, their past clinical experiences, or other resources to seek out information on cases that pose similar ethical challenges. This can help illuminate how to resolve the specific problem or if there are different morally acceptable options that are available to the clinician.

BOX 7-3 Steps in Ethical Decision Making

1. Assessment of the problem
2. Define the ethical issues
3. Delineate the goals; decision making and implement plan of action
4. Evaluation of the process and modify action as needed.

Adapted from Spencer E: A case method for consideration of moral problems. In: Fletcher JC, Spencer EM, Lombardo PA (eds): Fletcher's Introduction to Clinical Ethics, 3rd ed. Hagerstown, MD: University Publishing Group, 2005.

Step Three

Step three focuses on goal setting, decision making, and implementation. During this step, all personnel involved in the decision-making process are encouraged to clarify their own values and moral convictions so that the needs of the patient can be discussed forthrightly. Spencer notes that "moral problem-solving and planning for the care of the patient go hand in hand."[33] Weighing the benefits and burdens of each option as well as examining the ethical principles and underlying theories in regard to the case at hand can assist clinicians in making a decision. For instance, in the case study presented in the Clinical Applicability Challenges at the end of this chapter about Ms. X., is the autonomy of the patient being promoted by allowing her to die? How do we reconcile the moral distress it creates for the staff and the perception that it violates the norms of professional behavior by the resident in the case? Sometimes, ethics consultation, referral to an ethics committee, or other types of assistance are needed to provide an objective outside voice and to facilitate open dialogue among clinicians or family members when discussions appear to be at an impasse.

Step Four

Evaluation of an ethical decision allows all parties to reflect on the decision at hand and address any missed opportunities. Some questions to consider during this step include: Was the ethical problem adequately addressed? Were the goals of treatment met? If not, why? Were all parties satisfied with the outcome? Is there an alternative option that could be used if there is a need to rethink the plan of care? What factors contributed to a positive resolution of the case? What factors led to a less than desirable outcome? What have I/we learned from this process? Finally, what, if any, educational changes can be made in the clinical environment to resolve similar ethical challenges in the future?

▲ Strategies for Promoting Ethical Decision Making

The highly charged environment in which critical care nurses work calls for strategies to assist them with the increasing demands they encounter. Two strategies for developing an environment that is supportive of ethical concerns are briefly discussed here: institutional ethics committees and ethics rounds and conferences.

Institutional Ethics Committees

Many health care facilities have institutional ethics committees (IEC) to help resolve ethical conflicts in patient care as well as address ethics-related questions that might arise in clinical practice.[34–36] Ethics committees usually involve a diverse group of individuals, including physicians, nurses, social workers, pastoral care counselors, legal counsel, administrators, and community members. IECs provide ethics consultation and can assist practitioners by providing an external voice on contentious ethical issues that may seem irresolvable and facilitate education for all members of the health care team. Recommendations by the IEC can be either binding

or nonbinding, depending on the individual committee. However, an IEC can offer support to practitioners, patients, and families and improve their satisfaction with the delivery of care. Some institutions provide an ethics consultation service by a subset of the IEC or an independent consultant.

Ethics Rounds and Conferences

Ethics rounds may be an important and useful aspect of patient care, as they provide an opportunity to discuss a particular patient's health care needs in-depth and identify clinical, social, or ethical problems early in the course of care. Importantly, ethics rounds allow providers to express their views on issues of concern as well as clarify the most salient values and preferences at stake. This can include questions on DNR status, palliative care versus curative care, advance directives, conflicts associated with family systems, disagreements among health care providers about future goals of care, organ donation, cultural and religious differences, and many others. Nurses can make ethics rounds a regular part of their unit's activities by establishing a set time to focus on a specific case that raised ethical concerns or was particularly distressing for the staff. Interdisciplinary ethics rounds also provides an opportunity to build trusting work relationships with multidisciplinary care providers and develop a team approach to addressing value conflicts that arise in the unit. An individual patient ethics conference can be useful in resolving complex cases and opening a dialogue with patients, families, and the nursing staff or a multidisciplinary group.

▲ Clinical Applicability Challenges

CASE STUDY

Ms. X. is a 72-year-old female who has been on dialysis for the past 4 years and has a history of hyperlipidemia and chronic obstructive pulmonary disease. Ms. X. presents with an aortic aneurysm and seeks surgical repair for her condition. The patient survives the surgery and receives standard postoperative nursing care interventions for an aortic aneurysm repair. This includes neurological checks and a 10-minute neurological assessment every hour, mechanical ventilator support measures, hypovolemic shock and hemodynamic prevention activities, and monitoring mean arterial pressure. She also remains on continuous dialysis, and her postoperative course is relatively stable.

On postoperative day 5, Ms. X. remains in serious condition but is doing remarkably well considering her diagnosis and surgical repair. However, she writes a note to the nurse that reads: "I want to die." The nurse reassures the patient that she is doing very well for her postoperative course, and no further action was taken at that point regarding the patient's note. On postoperative day 6, the patient is extubated and on oxygen via nasal cannula as well as some vasoactive medications. Within an hour, Ms. X. begins to belly breathe and is having some respiratory distress. She is given 40% oxygen via face mask. Oxygen saturation levels remain at 94% and greater and she stabilizes with oxygen by nasal cannula at 6 L/min. Regarding long-standing back pain, the patient indicates, however, that "the pain is unbearable," and again states that "I want to go." She was offered an epidural at the bedside for the back pain, but refused the epidural and simply stated that she was "done and does not want to fight anymore." Her family agreed with her request that she should be allowed to die and to stop all interventions/treatments. The attending physician does not agree and wants postoperative care to continue. An ethics, legal, and psychological consult is called by the nurse. Ms. X., however, refuses to see the psychologist and asks that he not come into her room as she is completely lucid and "wants to die." The resident argues, "This is homicide" and wants no part of Ms. X.'s care. The nurse thinks that the patient's wishes should be taken seriously, consistent with respect for her autonomy, but that careful steps should be taken to assess the basis for her words and what she understands and means by them.

1. How should we evaluate a patient's request that reverses her previous preferences for treatment?
2. What factors might have a bearing on the patient's plan of care?
3. What possible learning needs would you anticipate for all those involved in this case?
4. What interventions should be initiated to address the concerns of the patient?
5. How would you determine the best course of action for this patient's plan of care?

Acknowledgment

Jill Gehman, RN, BSN for her assistance on clinical applicability challenges with critically ill patients.

References

1. Beauchamp TL, Childress JF: Principles of Biomedical Ethics, 6th ed. New York, NY: Oxford University Press, 2009
2. Veatch RM: Medical ethics: An introduction. Boston, MA: Jones & Bartlett Publishers, 1989
3. Ulrich C, Taylor C, Soeken K, et al: Everyday ethics: Ethical issues and stress in nursing practice. J Adv Nurs 66(11):2510–2519, 2010. doi: 10.1111/j.1365-2648.2010.05425.x
4. Hamric A: Reflections on being in the middle. Nurs Outlook 49: 254–257, 2001
5. Bulechek GM, Butcher HK, Dochterman JM: Nursing Interventions Classification (NIC), 5th ed. St. Louis, MO: Mosby, 2008
6. American Nurses Association (ANA): Code of Ethics for Nurses. Washington, DC: ANA, 2001
7. Cody WK: Paternalism in nursing and healthcare: Central issues and their relation to theory. Nurs Sci Q 16:288–296, 2003
8. Garrett TM, Baillie HW, Garrett RM: Health Care Ethics: Principles and Problems, 3rd ed. Upper Saddle River, NJ: Prentice Hall, 1998
9. Jones J: Nurses Top Honesty and Ethics List for 11th Year. 2010. Retrieved from http://www.gallup.com/poll/145043/Nurses-Top-Honesty-Ethics-List-11-Year.aspx, December 3.
10. Washington G: Trust: A critical element in critical care nursing. Focus Crit Care 17(5):418–421, 1990
11. Gallagher TH, Waterman AD, Ebers AG, et al: Patient's and physicians' attitudes regarding the disclosure of medical errors. JAMA 289(8): 1001–1007, 2003
12. Thomasma DC: Telling the truth to patients: A clinical ethics exploration. Cambridge Quarterly of Healthcare Ethics 3:375–382, 1994
13. U.S. Department of Health and Human Services: Understanding Health Information Privacy. Washington, DC: U.S. Department of Health and Human Services, 2010. Retrieved from: http://www.hhs.gov/ocr/privacy/hipaa/understanding/index.html
14. Pettrey L: Patient confidentiality: Is it ever OK to tell? AACN News 17(4):5, 2000
15. Angus DC, Barnato AE, Linde-Swirble WT, et al; Robert Wood Johnson Foundation ICU End-of-Life Peer Group: Use of intensive care at the end of life in the United States: An epidemiologic study. Crit Care Med 32(3):638–643, 2004
16. Aiken LH, Clarke SP, Sloane DM, et al: Hospital nurse staffing and patient mortality, nurse burnout, and job dissatisfaction. JAMA 288(16):1987–1993, 2002
17. Brock DW: Death and dying. In Veatch RM (ed): Medical Ethics: An Introduction. Boston, MA: Jones & Bartlett Publishers, 1989, pp 329–356
18. Gerstel E, Engelberg RA, Koepsell T, et al: Duration of withdrawal of life support in the intensive care unit and association with family satisfaction. Am J Respir Crit Care Med 178:798–804, 2008
19. McKinstry B: Do patients wish to be involved in decision-making in the consultation? A cross-sectional survey with video vignettes. Br Med J 321:867–871, 2000.
20. Schneiderman LJ, Jecker NS, Jonsen AR: Medical futility: Response to critiques. Ann Intern Med 125:669–674, 1996
21. Hastings Center Task Force: Guidelines on the termination of life-sustaining treatment and the care of the dying: A report of the Hastings Center. New York, NY: Briarcliff Manor, 1987
22. Greenwood E: Attributes of a profession. In Allhoff F, Vaidya AJ (eds): Professions in Ethical Focus: An Anthology. Toronto, ON: Broadview Press, 2008, pp 13–23
23. Sullivan W: Challenges to professionalism: Work integrity and the call to renew and strengthen the social contract of the professions. Am J Crit Care 14(1):78–84, 2005
24. Davis AJ, Fowler MD, Aroskar MA: Ethical Dilemmas & Nursing Practice, 5th ed. Upper Saddle River, NJ: Pearson, 2010
25. Danis M, Farrar A, Grady C, et al: Does fear of retaliation deter requests for ethics consultation? Med Health Care Philos 11(1): 27–34, 2007
26. Sulmasy DP, He MK, McAuley R, et al: Beliefs and attitudes of nurses and physicians about do not resuscitate orders and who should speak to patients and families about them. Crit Care Med 36(6): 1817–1822, 2008
27. Jameton A: Nursing Practice: The Ethical Issues. Englewood Cliffs, NJ: Prentice Hall, 1984.
28. Bell J, Breslin JM: Healthcare provider moral distress as a leadership challenge. JONAS Healthc Law Ethics Regul 10(4):94–97, 2008
29. Corley MC: Nurse moral distress: A proposed theory and research agenda. Nurs Ethics 9:636–650, 2002
30. Ulrich CM, O'Donnell P, Taylor C, et al: Ethical climate, ethics stress, and the job satisfaction of nurses and social workers in the United States. Soc Sci Med 65(8):1708–1719, 2007
31. American Association of Critical Care Nurses (AACN): Position statement: moral distress, Aliso Viejo, California, July 8, 2004, AACN.
32. Ulrich CM, Hamric AB: What is so distressing about moral distress in advanced practice nursing. Clinical Scholars Review. J Doc Nurs Pract 1(1):5–6, 2008
33. Spencer E: A case method for consideration of moral problems. In: Fletcher JC, Spencer EM, Lombardo PA (eds): Fletcher's Introduction to Clinical Ethics, 3rd ed. Hagerstown, MD: University Publishing Group, 2005
34. Bushy A, Rauh JR: Implementing an ethics committee in rural institutions. J Nurs Adm 21(12):18–25, 1991
35. Bartels D, Younger S, Levine J: Ethical committees: Living up to your potential. AACN Clin Issues Crit Care Nurs 5(3):313–323, 1994
36. Buchanan S, Cook L: Nursing ethics committees: The time is now. Nurs Manag 23(8):40–41, 1992

8 Legal Issues in Critical Care Nursing

Nayna C. Philipsen and Patricia C. McMullen

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Describe major areas of the law that affect critical care nursing practice.
2. Define the four elements of malpractice (professional negligence).
3. Delineate allegations commonly made against critical care nurses.
4. Explain types of vicarious liability.
5. Apply knowledge of informed consent and advance directives to critical care patient situations.

Because society in the United States seems to be more litigious than ever, legal issues involving critical care are of increasing concern. The number of malpractice suits that name or involve nurses is increasing. Issues from refusal and termination of treatment to the spiraling costs of the malpractice system are widely discussed. Legislators have become involved by introducing and/or enacting living will statutes and bills designed to address the malpractice crisis in their jurisdictions.

This chapter begins with an overview of the major areas of law of governmental organizations and major areas of law that impact the practice of nursing. Next, the legal principles of negligence, vicarious liability, and patient autonomy are reviewed, with pertinent critical care case examples. The chapter then proceeds to identify and address selected current legal issues that are most applicable to the practice of critical care nursing.

▲ An Overview of Governmental Organization and Major Areas of the Law

The United States Constitution provides the basic principles and guiding policy for all law in the land. Three branches of government were established to create a "balance of powers" to minimize the risk of abuse of the law by those in power. The structure of each state government is similar to the federal structure. The *Legislative Branch*, composed of representatives of the people, creates and modifies *statutes*. The *Executive Branch* is headed by the president or a state governor and consists of federal and state agencies that write and enforce *regulations* that give the people *notice* of what they need to do to comply with the statutes. The *Judicial Branch* consists of the federal and state court systems, which interpret statutes and regulations and examine constitutionality when disputes arise. The courts generate *case law*, which becomes *precedent or common law*, providing guidance for future interpretation of the law.

A legal dispute is first heard by a *trial court*, which examines the evidence and makes a decision based on the facts of the case. Some disputes then go on up to higher courts, called *appeals courts*. These courts make decisions about alleged legal and procedural mistakes by the judges or lawyers in the trial courts. They do not retry the initial dispute.

Three types of law affect the practice of critical care nurses. These are administrative law, criminal law, and civil law.

Administrative Law

Administrative law includes state and federal statutes, their accompanying regulations, and the regulatory agencies that enforce those laws. Nurses deal with state agencies and state laws and regulations when they apply for a nursing license to enter the profession. Every state legislature has enacted a Nurse Practice Act (NPA). The NPA defines the practice of nursing, requires nursing licensure, establishes standards for nursing schools, and delegates enforcement powers to a state agency, usually the State Board of Nursing. This agency develops regulations that give notice to the public and to nurses of how the NPA will be interpreted and implemented in that state. The Boards of Nursing work together to develop common standards and address common challenges through the National Council of State Boards of Nursing, Inc. (NCSBN), where their representatives conduct research, pool resources, and publish model practice acts and position statements. The NCSBN is also responsible for the development and integrity of the national licensure examination known as the NCLEX.

Practicing nurses are expected to know the provisions of the NPA and any regulations dealing with the practice of nursing in every state where they practice. If a nurse is unfamiliar with the NPA, it is important that he or she contact

the State Board of Nursing to obtain access to and review this act. State boards of nursing increasingly place their NPA and regulations on a Web site. The NCSBN Web site provides links to the Web sites of all of the boards of nursing, as well as other useful information related to legal aspects of nursing licensure and practice.[1]

Why do state governments regulate nursing practice? The United States Constitution charges the states with protecting the health, safety, and well-being of their citizens. For nurses, this begins with nursing licensure, a process in which the new nurse applicant must show that he or she can enter practice as a safe practitioner by completing an approved nursing education program, passing a national examination (the NCLEX), and demonstrating good moral character. While public protection during initial licensure is proactive, assurance of continuing competency after licensure is often reactive. As long as they continue to pay their renewal fees, nurses may be required to practice a certain number of hours per year or attend approved continuing education seminars, but they typically do not have to return to nursing school or face reexamination to renew a license. Instead, the boards identify loss of competency by responding to complaints.

Complaints, whether to the Board of Nursing or to the courts in the form of a lawsuit, are a special concern of critical care nurses. Because their patients are not as stable as patients in most other areas of health care, they are at high risk for an undesired outcome. These complaints can come from many sources, such as patients, their families, other health care workers, or the nurse's employer. Even a revengeful ex-spouse of a nurse can allege any of the violations listed in the NPA.

For example, if a patient feels that he or she has received incompetent or unethical nursing care, he or she may contact the State Board of Nursing and file a complaint against the nurse or nurses involved in the care. The state board then determines whether the complaint, if true, would violate the NPA and possibly result in limits on or loss of the nurse's license to practice. If so, the board conducts an investigation to determine whether the claim has merit.

Under the Fifth Amendment of the U.S. Constitution, all citizens are afforded the right to *due process* before the state or federal government can take any property. Case law indicates that a nurse's license is a form of property because it helps a person earn a living. Therefore, due process rights are attached to a nursing license. This means that the State Board of Nursing must meet certain procedural requirements before it can discipline by taking away or placing conditions or limitations on a nursing license. First, the nurse is entitled to *notice* that someone has filed a claim against his or her license. The nurse is also entitled to a hearing that follows the procedures outlined in the NPA, in order to answer and defend alleged violations.

In most states, the final defense against charges on a nurse's license takes the form of a hearing before the State Board of Nursing, whose members typically include nurses. To be sure that the hearing is fair, the nurse should attend the hearing and should arrange to have representation at the hearing by an attorney whose practice includes nursing licensure. The American Association of Nurse Attorneys (TAANA) is an excellent source of attorneys with this special expertise. At a hearing, the board members listen to the case against the nurse's license, which is presented by an attorney or other representative for the state. Witnesses who have knowledge of facts surrounding the complaint will testify and introduce evidence. These witnesses may include board staff members who investigated the complaint or who acquired the related medical records and other documents, the person who filed the complaint, any witnesses to the incident, and people who supervised the nurse. Then the nurse, either directly or through legal counsel, can question the board's witnesses and can introduce evidence or testimony to refute the allegations in the complaint. Based on the presentations by the state and by the nurse, the State Board of Nursing makes a determination as to whether the nurse violated the NPA and what, if any, discipline is warranted to protect the public. Typically, the decision of the State Board of Nursing is final, and unless there were violations of the nurse's due process rights, a court upholds the state board's findings if either side appeals.

Although the nurse's right of due process cannot be abridged, state boards of nursing have the right to *immediately* suspend a nurse's license for acts that the Board deems so dangerous to the public welfare that the complaint, if true, would constitute an emergency. When the state immediately suspends a license, the nurse must stop any nursing practice immediately. The NPA provides the nurse with the right to an accelerated hearing, within a short period of time from the date of the suspension.

HIPAA

A federal agency that has a major impact on nursing practice is the U.S. Department of Health and Human Services (HHS), which generates regulations and enforces federal statutes such as the Health Insurance Portability and Accountability Act (HIPAA), Medicare, and Medicaid, including the rules for reimbursement to providers and health care institutions. Critical care nurses are familiar with HIPAA, a statute enacted by Congress in response to concerns about the security of electronic health records. The Title II HIPAA Privacy Rule in particular has raised the awareness of critical care nurses about their need to verify who can have access to patient information before sharing it. HIPAA made it clear that patients have a great deal of control over their health information, and in many circumstances the right to decide with whom the nurse can share that information. HIPAA also provides guidelines for communicating this right to the patient. The exceptions include sharing information in the record needed for care with other care givers, information needed for billing with payers, information that is de-identified (personal and all possibly identifying information are removed), and information needed for the general welfare, such as public health data and the regulation of health care facilities and professionals, including HIPAA enforcement. HIPAA's security rules also impact nursing practice. As the use of electronic health records increases, nurses must comply with regulations designed to protect electronic information, including the use of passwords and biometric identification and the location of computer screens to protect them from unauthorized eyes. Violation of relevant federal regulations and standards can have serious consequences, including the loss of federal reimbursement for care.

▲ Employment Law

Workplace statutes and their supporting regulations form another area of administrative law that clearly impacts nursing practice. A full discussion of these is beyond the scope of this chapter, but critical care nurses should be aware that a number of statutes affect their practice, their conditions of employment, their right to a safe workplace, and the responsibilities of health care supervisors and employers. Each statute is enforced by the federal or state government agency that is also responsible for drafting and revising such regulations. For example, the Fair Labor Standards Act, the Occupational Health and Safety Act, and the Family Medical Leave Act are enforced by the U.S. Department of Labor. The U.S. Equal Employment Opportunity Commission enforces Title VII of the Civil Rights Act of 1964 with Amendments, including the Lilly Ledbetter Fair Pay Act of 2009. The National Labor Relations Board is a federal agency formed to enforce the National Labor Relations Act. A number of federal agencies share responsibility for enforcing the Americans with Disabilities Act, including the Office of Civil Rights of HHS and the U.S. Department of Justice.

Criminal Law

Criminal law is public law. It encompasses cases in which the local, state, or federal government has filed a lawsuit against an individual alleging that he or she has committed a wrongful and illegal act against society. In criminal cases, the victim has the role of witness for the state. If the nurse is the victim of the charged crime, he or she is a witness. If the state files charges against the nurse for committing a crime, the nurse is the defendant. If the defendant nurse is convicted of a crime, the penalty can include loss of liberty. Criminal cases in nursing have included intentional assault and battery, fraud, theft, negligent homicide, and murder. If a nurse intended harm to a patient through substandard treatment, the nurse may be charged by the state under criminal law, in addition to any civil case for malpractice damages filed by the patient. There is a growing body of law around workplace violence involving nurses. In general, however, criminal cases are unusual in nursing situations. Nurses caring for trauma patients whose injuries may have resulted from criminal acts sometimes find themselves involved in a criminal case as a witness to the injury. In some states, they may be required to report certain injuries to criminal law enforcement authorities. An example of a criminal case is presented with the case studies later in this chapter.

Civil Law

Civil law is private law, and deals with conflicts among individuals. Civil law includes tort law, contract law, and the concepts of vicarious liability and product liability. Torts include trespass, assault, battery, and negligence.

▲ Nursing Negligence in Critical Care

The fundamental legal responsibility of the registered nurse in critical care settings does not differ from that of the registered nurse in any work setting. The registered nurse adheres to five principles for the protection of the patient and the practitioner (Box 8-1). The most common lawsuits against nurses and their employers are based on *negligence by a professional*, which is called *malpractice*.

BOX 8-1 Five Legal Responsibilities of the Registered Nurse

- Performs only those functions for which he or she has been prepared by education and experience
- Performs those functions competently
- Delegates responsibility only to personnel whose competence has been evaluated and found acceptable
- Takes appropriate measures as indicated by observations of the patient
- Is familiar with policies of the employing agency

A nursing malpractice case can begin when a patient experiences bad bedside manner or an undesired outcome related to nursing care. If a significant financial loss can be attributed to that outcome, the distressed patient might file a lawsuit against the nurse in a civil court, alleging nursing malpractice. The nurse then becomes the defendant in a civil malpractice case. This experience is a great burden for any nurse, even in the best of times. First, the nurse should contact his or her malpractice insurance carrier and obtain legal counsel who has expertise in nursing malpractice. The nurse may soon be involved with the next step of the civil lawsuit, called *discovery*. This can involve requests for documents, preparing written answers to written questions called "interrogatories," and, most stressful of all, a deposition, which is oral testimony given under oath and recorded by a court reporter, but out of court. In the best case, the plaintiff patient's attorney will advise dropping the case after hearing the nurse's testimony, but in the worst case this testimony will be used later to challenge the nurse's credibility during the next major phase of the lawsuit, the *trial*. During trial, witnesses will testify for both sides and introduce evidence for cross-examination in the courtroom. As in all cases, the person filing the lawsuit (complainant) must prove his or her case before a defense is necessary. To prove a malpractice case, the complaining patient must prove all of the required "elements" of malpractice: duty, breach of duty, damages, and causation.

Duty

A duty is a legal relationship between two or more parties. In most nursing cases, duty arises out of a contractual relationship between the patient and the health care facility. That is, when a patient receives health care, an implied contract arises. The patient, the insurer, or both agree to pay for any health care services the patient receives. In return, the health care facility agrees to supply "reasonable care." One way the duty of an individual nurse to the patient may be confirmed is by the appearance of the nurse's name in the patient's record. A nurse has a legal duty to provide reasonable care to all of his or her patients. This means that the nurse must provide care that complies with the established *standard of care* for a reasonable nurse under the circumstances present at the time of the incident.

Arguing the standard of care is the core of many malpractice cases. In a typical malpractice lawsuit, this is done by both sides hiring expert witnesses, sometimes called "dueling experts," to introduce evidence arguing for their different versions of the standard of care. The following factors can be used to determine the standard of care for a critical care nurse:

- Testimony from experts in critical care, which may include a critical care nurse who is working as an expert witness
- The employing institution's procedure and protocol manuals
- Nursing job descriptions
- Nursing research, textbooks, professional journals, medication books
- Professional organization standards and guidelines (eg, Advanced Cardiac Life Support [ACLS] and Certified Critical Care Registered Nurse [CCRN] standards)
- Laws and regulations governing professionals and institutions
- Standards of private accrediting bodies
- Equipment manufacturers' instructions

Breach of Duty

What was the standard of care? What would a reasonable critical care nurse have done under the circumstances? After the plaintiff patient establishes duty, the patient must show that the nurse breached (violated) that duty, that is, the nurse was negligent. Professional negligence is determined by comparing the nurse's conduct with the standard of care. The critical care nurse who fails to meet the standard of care has breached (violated) his or her duty to the patient.

Negligence may be either "ordinary" or "gross." Ordinary negligence implies professional carelessness. Gross negligence suggests that the nurse willfully and consciously ignored a known risk for harm to the patient. Most cases involve ordinary negligence, but gross negligence can be found if, for example, the nurse ignored sound nursing advice or harmed a patient while under the influence of drugs or alcohol.

Causation

Malpractice law requires a causal relationship between the critical care nurse's breach of the standard of care and the injury to the patient. To prove malpractice, the plaintiff patient has to show that injury or harm occurred as a result of the nurse's action or inaction; that is, the injury would not have occurred without the conduct in question. The plaintiff must also show that the injury was reasonably anticipated. For example, if a critical care nurse administered digoxin to a cardiac patient who had a pulse of 30 beats/min and the patient suffered a cardiac arrest, the court would likely find that the critical care nurse caused the patient's arrest; that is, the wrongful administration of the digoxin was the "proximate cause" of the arrest. However, if the patient had a pulse of 70 beats/min when the digoxin was administered, and the patient suffered a totally unanticipated seizure, it is probable that the nurse's action would not be found to be careless such as to be the cause of the seizure. In this case, the nurse would normally be exonerated, because seizures are not an expected complication of digoxin administration.

Damages

The intent of malpractice negligence law is to make the injured party "whole." The law attempts to return the plaintiff as close as possible to a position that he or she would have been in had the nurse's conduct not caused this injury. Unfortunately, injuries sustained usually cannot be undone. Therefore, most court awards attempt to give the injured plaintiff an award of monetary "damages" that will compensate the plaintiff for his or her injuries. Monetary damages are grouped under the broad headings of economic and noneconomic damages.

Economic damages relate to those damages that can be calculated within a degree of certainty. Medical costs and lost wages are the two major types of economic damages. Noneconomic damages are more difficult to calculate. These damages include pain and suffering and loss of consortium (relations) that occurred as a result of the malpractice. Many state and federal governments place monetary limits on the amount a patient can recover for pain and suffering, regardless of the amount that may be awarded by a jury. "Loss of consortium" damages include the patient's inability to perform household tasks and the loss of marital relations.

The spouse and minor children of a patient may also be able to recover economic and noneconomic damages that they suffer as a consequence of injuries to the patient. When a minor child is the plaintiff, it is not unusual for the parents to file for noneconomic losses due to the loss of society and affection of their child. On the economic side, a parent can sue for his or her own lost wages due to a need to care for the child.

Many types of malpractice complaints are lodged against critical care nurses. The following cases illustrate reasons nurses are often named in malpractice suits.

CASE STUDY

Failure to Comply With Reasonable Standards of Care

"T" was 7 years old when his parents took him to the emergency department complaining of nausea, vomiting, and a fever. The child was examined and a complete blood count (CBC) ordered. The physician diagnosed a viral upper respiratory infection and discharged the boy. The next morning his parents brought T back to the hospital. His complaints now included abdominal and chest pains. T was classified as Emergent Level 2 and placed in an examination room. An hour later he was examined, and lab tests and chest x-ray were ordered. As a result, T was diagnosed with pneumonia a little over 2 hours after arrival. The doctor ordered transfer to a pediatric intensive care unit (PICU) at another hospital, as well as antibiotics and fluids, which were administered. The receiving hospital informed the doctor that they had to prepare a bed for T in the PICU, and wanted him transferred with a pediatric transfer team, after it completed its current run to another city. T's condition worsened. He was intubated and developed malignant hyperthermia. Nine hours after his arrival, doctors arranged for his transfer by helicopter. T was in the second hospital for several weeks, diagnosed with septic shock, which caused

(*continued on page 91*)

organ damage. His parents sued the first hospital and doctor on T's behalf, for negligence and for violation of the Emergency Medical Treatment and Active Labor Act. The evidence showed that two nurses and a doctor examined T and physically assessed him. The court found that this was reasonable care, ruled against T and his parents, and dismissed the case.[2]

In 1999 the Institute of Medicine (IOM) released a study, *To Err is Human: Building a Safer Health System*,[3] which raised awareness of medical errors, including serious medication errors, which occur in 5% to 10% of patients admitted to hospitals. Experts agreed that the most common cause of error is the system itself, not the individual practitioners in the system. This report triggered the formation of the National Patient Safety Foundation by the American Medical Association, and the creation of a nonpunitive sentinel events reporting system by the Joint Commission for the Accreditation of Healthcare Organizations (JCAHO), now known as the Joint Commission. Remaining controversies include what type of errors should be reported, and whether individual practitioners should be disciplined for this type of error. In the case of gross negligence, the nurse is likely to be found liable for a medication error, regardless of the answers to these questions.

CASE STUDY

Improper Medication Administration

Baby A was admitted to the pediatric intensive care unit (PICU) of a local hospital to determine why she developed respiratory dysfunction. A pulmonologist, a critical care specialist, neurologists, and geneticists examined her to rule out various disorders. During a right femoral central line placement, a doctor inadvertently cannulated the artery and immediately removed the line. Thereafter, inadequate perfusion of blood through her leg was noted. A vascular surgeon performed an emergent thrombectomy and administered 162 mg of papaverine. Shortly after that, Baby A went into cardiac arrest and died. Baby A's parents sued the doctor for administering an incorrect dosage of papaverine. The patient's expert witness stated that administering papaverine to the infant caused her death, and that the doctor's failure to ascertain the correct dose was a substantial factor in her death. The doctor argued that it was the responsibility of the operating room staff, specifically the circulating nurse, to prepare medications for administration, and the standard of care permitted the doctor to rely on that. In this case, an effort to transfer the responsibility for a medication error from the doctor to the nurse was unsuccessful. A trial was ordered on the malpractice claim against the doctor.[4]

CASE STUDY

Criminal Liability in Critical Care

Mr. D., an 86-year-old man, was admitted to the hospital with abdominal pain. He was diagnosed with a perforation in the proximal duodenum, causing diffuse peritonitis. The day after his surgery, he was found to have a serum potassium level of 3.2 mEq/L, below the normal level of 3.3 to 5.5 mEq/L. The intensive care unit (ICU) nurse administered an ordered dose of potassium chloride elixir through his nasogastric tube. However, subsequent laboratory tests showed this had not been well absorbed.

Mr. D.'s physician ordered the ICU nurse to administer 40 mEq of potassium chloride in 100 mL of saline solution in a bag of intravenous fluid. After the nurse informed the physician that the potassium chloride would need to be infused over the course of 1 hour, the physician ordered the nurse to draw up a syringe of 40 mEq of potassium chloride in 30 to 50 mL of saline solution. The nurse prepared the syringe but refused to administer it, knowing that this was dangerous. Another ICU nurse was also present and informed the physician that it was hospital policy to administer a maximum dosage of 40 mEq of potassium chloride over 1 hour. The physician then took the syringe from the nurse and administered the potassium chloride directly. During the injection, Mr. D. stopped breathing, and efforts at cardiopulmonary resuscitation were unsuccessful.

The physician's failure to use reasonable standards of medical care and total disregard of the cautions given by the ICU nursing staff resulted in criminal liability. A court convicted the physician of involuntary manslaughter, "the unlawful killing of a human being without malice in the commission, without due caution and circumspection, of a lawful act which might produce death." The sentence was 5 months' imprisonment, 36 months' supervised parole, a $100 assessment, and a $25,000 fine. The U.S. Court of Appeals for the 10th Circuit upheld the criminal conviction.[5]

Vicarious Liability

Vicarious liability means to hold someone (a person or institution) responsible for the actions of another. Critical care nurses sometimes find themselves in situations where various types of vicarious liability apply, including respondeat superior, corporate liability, negligent supervision, and the rule of personal liability.

Respondeat Superior

The doctrine of respondeat superior is translated as "let the master answer for the sins of the servant." Under this legal theory, hospitals are held liable for the negligence of their employees. Respondeat superior is a legal doctrine based on public policy that notes that because a hospital profits from the patients seeking care, the hospital should pay for some of the damages caused by hospital personnel if negligence occurs. This doctrine applies only when hospital employees act within the scope of their employment.

Respondeat superior does not apply in situations involving temporary agency personnel because they are usually employees of the agency, not of the hospital. Nor does it typically apply to physicians, because they usually are not employed by the hospital. This doctrine would not apply to nurses who are employed by a hospital but who are accused of malpractice for work outside of their hospital employment.

Because hospitals may be held liable for nursing activities conducted by their employees, they carry professional liability insurance for the activities of their employees, intended to cover the cost of defending individuals named in a malpractice case. However, many nurses also carry their own malpractice insurance, to cover off-the-job nursing activities and to cover the cost of choosing their own independent counsel to protect their rights in a case where multiple parties from their institution are sued, because they are likely to have conflicting interests.

Corporate Liability

Corporate liability is vicarious liability that occurs when a hospital is found liable for its own unreasonable conduct. For example, if it is found that a unit is chronically understaffed and a patient suffers an injury as a result of short staffing, the hospital can be held accountable. It is reasonable to expect any hospital that has an ICU or an emergency department to take precautionary measures to ensure that it is adequately staffed or that beds or admissions are reduced. Failure to ensure adequate staffing can lead to payment of monetary damages under the theory of corporate liability.

Corporate liability may also occur within "floating" situations. A nurse working in a critical care setting must be competent to make immediate nursing judgments and to act on those decisions. If the nurse does not possess the knowledge and skills required of a critical care nurse, he or she should not be attempting to render critical care. A nurse who is not well versed in critical care should notify the charge nurse or nursing supervisor of this fact. The nurse needs to clearly state which nursing care activities he or she can and cannot implement. The supervisor and charge nurse must then delegate the remaining nursing duties to staff members with adequate education, training, and experience. Box 8-2 addresses issues of concern to the floating nurse.

BOX 8-2 Commonly Asked Questions When Rotating to an Unfamiliar Unit

1. *If I am asked to go to another unit, must I go?*
   Usually, you will be required to go to the other unit. If you refuse, you can be disciplined under the theory that you are breaching your employment contract or that you are failing to abide by the policies and procedures of the hospital. Some nursing units negotiate with hospitals to ensure that only specially trained nurses rotate to specialty units.
2. *If I rotate to an unfamiliar unit, what types of nursing responsibilities must I assume?*
   You will be expected to carry out only those nursing activities that you are competent to perform. In some instances, this will be the performance of basic nursing care activities, such as blood pressures, and uncomplicated treatments. If you are unfamiliar with the types of medications used on the unit, you should not be administering them until you are thoroughly familiar with them. Consider the medication cards the student completes in nursing school. They were assigned because a reasonable, prudent nurse does not give medications without knowledge of their pharmacology, dosage, method of administration, side effects, and interactions with other medications. The same reasoning applies for any other type of critical care monitoring.
3. *What should I do if I feel unprepared when I get to the unit?*
   Suggest that you assist the unit with basic nursing care requirements and that specialized activities (eg, invasive monitoring, cardiac monitoring, or the administration of unfamiliar drugs) be performed by staff members who are adequately prepared. Do not feel incompetent because you are not familiar with all aspects of nursing care. After all, when is the last time you saw the neurologist go to labor and delivery and perform a cesarean birth?
4. *What if the charge nurse orders me to do something I am not able to do safely*?
   You are obligated to say you are unqualified and request that another nurse carry out the task. The charge nurse also needs to remember that she could be held liable for negligent supervision if she orders you to do an unsafe activity and a patient injury results.

Negligent Supervision

Negligent supervision is vicarious liability claimed when a supervisor fails to reasonably supervise people under his or her direction. For example, if a nurse rotates to an unfamiliar unit and informs the charge nurse that he or she has never worked in critical care, it would be unreasonable for the charge nurse to ask the floating nurse to perform invasive monitoring. If the charge nurse did assign such responsibilities to the floater and a patient injury resulted, the charge nurse could be held accountable to the patient for negligent supervision.

Captain of the Ship Doctrine

At one time, the physician was viewed as the "captain of the ship" in relation to the nurse. Therefore, the nurse was expected to follow any order from the physician. This doctrine has largely been replaced by a legal concept known as the *rule of personal liability*. As a result, nurses are responsible for making sound decisions by virtue of their own specialized education, training, and experience. A critical care nurse today who is unsure about whether a physician's order is safe or appropriate for a patient should not follow it as a matter of course, but should seek clarification from the physician or, if needed, from the nursing supervisor.

CASE STUDY

Rule of Personal Liability and Independent Nursing Judgment

Mr. S., a 46-year-old man with a history of ventricular tachycardia, was prescribed flecainide acetate (Tambocor) for his heart condition. One evening he reported his heart "started feeling funny" and had a friend take him to the emergency department. An electrocardiogram on admission to the emergency department revealed he was experiencing ventricular tachycardia.

Mr. S. told a nurse and a physician in the emergency department that he did not want cardioversion. The emergency department physician, in telephone consultation with a cardiologist, ordered 5 mg of verapamil. Within 2 minutes, Mr. S.'s blood pressure crashed, he had a seizure, and he went into cardiac arrest. As a result, Mr. S. suffered brain damage and was forced to reside in a nursing home because he lacked independent motor function and was unable to speak.

The emergency department nurse, physician, and hospital were sued for several reasons, including malpractice. During deposition, the nurse testified that she was ACLS certified and admitted to knowing that verapamil was contraindicated in patients with ventricular tachycardia. She related that she had serious questions about administering the verapamil but acceded to the physician's orders.

The court found that the standard of reasonable nursing care required the nurse to intervene to prevent complications and that failure to intervene was a violation of the nursing standard of care. In addition, the court determined that the standard of nursing care requires a nurse to exercise independent nursing judgment if he or she believes that an order may have adverse consequences for the patient.[6]

The Questionable Medical Order

The above case describes a difficult situation for nurses, who frequently feel that their employment may be threatened if they do not follow the order of a physician. However, an order that is patently wrong can harm the patient if it is followed. A secondary consequence can be liability for the physician, the nurse, and the hospital (as the employer) if the patient suffers harm as a direct result of the order. Both ethics and professional preservation (where can a critical care nurse work without a license?) argue the wisdom of refusing to obey orders in some situations.

A policy statement should exist in procedures or by directive that indicates the manner of resolving the issue of the "questionable" medical order. This is important for all medical orders, but particularly for those given for critically ill patients where unusual doses of medication are frequently ordered. The nurse who questions an order should express his or her specific reasons for concern to the physician who wrote the order. This initial approach frequently results either in an explanation of the order and a medical justification for the order in the patient's medical record, or in an alteration of the order based on additional information from the nurse. If this approach is unsuccessful, many hospitals require that the attending physician or the nursing supervisor be notified. Others have a policy that the chief of the service must be consulted about questionable orders. If these options are unavailable or are unsuccessful, a critical care nurse or any other nurse can refuse to give a medication, and should if patient harm is the expected result.

Establishment of Protocols

If the critical care nurse is required to perform medical acts and is not under the direct and immediate supervision of the delegating physician, the activities must be based on established protocols. These protocols should be created by the medical and nursing departments and should be reviewed for compliance with the state's NPA. Protocols must be reviewed frequently so health care professionals can make sure that they reflect current medical and nursing regulations and standards of care. In the event of a malpractice suit, the critical care protocols and procedures can be introduced as evidence to help establish the applicable standard of care. Although it is important that protocols provide direction, excessive detail restricts the critical care nurse's flexibility when selecting a proper course of action and is more likely to be different from actual practice, increasing the liability risk of the nurses and the institution.

Liability for Defective Medical Equipment

A medical device, defined as virtually anything used in patient care that is not a drug, includes intricate pieces of equipment (eg, intra-aortic balloon pumps, endotracheal tubes, pacemakers, defibrillators), along with less complicated ones, such as bedpans, suture materials, patient restraints, and tampons. Before 1976, medical devices were unregulated; since 1976, the U.S. Food and Drug Administration (FDA) within HHS has regulated medical devices sold in the United States. Before November 1991, hospitals, their employees, and staffs were permitted, but not required, to report device malfunctions to the device manufacturer or to the FDA. The Safe Medical Devices Act of 1990 requires user facilities (which include hospitals and ambulatory surgery facilities, but not physician offices) to report to the manufacturer medical device malfunctions that result in serious illness, injury, or death to a patient. They are also required to report to the FDA those that result in a patient's death. A serious illness or injury includes not only a life-threatening injury or illness but also an injury that requires "immediate medical or surgical intervention to preclude permanent impairment of a body function or permanent damage to a body structure."[7] Therefore, the rupture of an intra-aortic balloon pump that requires that the balloon-dependent patient immediately be transported to the operating room for removal and replacement of the device is a reportable event. Nursing and other staff must now participate in reporting device malfunctions, including those associated with user error, to a designated hospital department. Personnel in that area are usually responsible for determining which malfunctions engender an obligation to report and to whom they should be reported.

In 2009 the Secretary of HHS directed the FDA commissioner to issue regulations for class III devices, those with the greatest risk to harm patients, to ensure that they are approved through the most stringent premarket review process.[8] There is a duty not to use equipment that is patently defective. If the equipment suddenly ceases to do what it was intended to do, makes unusual noises, or has a history of malfunction and has not been repaired, the hospital could be liable for damage caused by it. Likewise, the nurses could be liable if they know or should know of these problems and use the equipment anyway. The following cases involved liability for defective equipment.

CASE STUDY

Defective Equipment and Negligence

Mrs. V was admitted to a hospital in stable condition for an elective ambulatory cardiac catheterization. Doctors determined that she needed an angioplasty and stent procedure, performed the next day. Complications arose, and Mrs. V was transferred to ICU, where she suffered a cardiorespiratory arrest, entered into a comatose state, and died of abdominopelvic hemorrhage 2 days after her initial admission. Her children sued the hospital for the malpractice of its employee nurses under the vicarious liability doctrine, alleging that the nurses failed to notify the doctors immediately of the emergency. They also sued the hospital for negligence citing its failure to have a CT scan machine on its premises, characterizing it as "adequate and necessary" medical equipment. The court denied the hospital's motions to dismiss this case.[9]

Patient Decision-Making Autonomy

Laws protecting patient autonomy require that patients receive enough information to make an informed, intelligent decision to accept or reject a proposed treatment. This is called *informed consent*. It can be especially challenging

for nurses caring for patients who are critically ill. Obtaining informed consent from the patient, or from the family or designated proxy in the case of a patient who is not competent, is the responsibility of the care giver who is ordering the care, usually a physician. The nurse is frequently asked to witness the signing of the consent form. In these cases, the nurse is merely attesting that the signature on the consent form is that of the patient or the patient's surrogate decision maker. When the nurse actually witnesses a physician's explanation concerning the nature of the proposed treatment, the risks and benefits of the treatment, alternative treatments, and potential consequences if the patient decides to do nothing, the nurse may place a note in the nurse's notes or designated part of the patient's record stating, "consent procedure witnessed." This information may be vital in the rare case in which the patient or family alleges that the care giver failed to provide informed consent.

▲ Advance Directives: Living Wills and Powers of Attorney

Advance directives are legal documents that preserve the patient's right to determine his or her care by permitting a patient to make decisions about health care ahead of time, in case the patient becomes incompetent to make decisions later. Incompetence can occur due to illness, age, trauma, or court determination. If a patient is deemed incompetent, the nurse must identify the patient's surrogate decision maker and contact the surrogate to make health care decisions on the patient's behalf.

If the patient's surrogate is not designated in writing, state law identifies the appropriate family members to make decisions. The surrogate should refer to the patient's advance directives and any other known wishes for guidance. This situation can become complex, especially when the surrogate decision maker disagrees with the advice of the physician or other care giver, with the wishes stated by the patient in an advance directive or otherwise, or with another surrogate, which can occur in a situation where the patient's children, parents, or siblings share this responsibility. While the nurse should not implement procedures that he or she identifies as unethical, neither nurses nor physicians can substitute their judgment for the patient's right to make the decision about accepting or rejecting available procedures.

A *living will* is a written directive from a competent patient to family and health care team members concerning the patient's wishes in the event the patient is unable to express these wishes in the future. A living will only applies to the limited situations that it describes, which may not include the particular decision that must be made. A living will becomes effective only if the patient is both terminally ill and incompetent to communicate his wishes, or is permanently comatose. Consequently, when the patient is critically ill or temporarily unable to make health care decisions, the living will may not be operative.

To provide broader coverage, patients must prepare a *durable power of attorney for health care*. This is the legal document that allows the patient to appoint a surrogate decision maker while he or she is still competent. The surrogate, also known as a health care agent or proxy, has authority to make treatment and health care decisions in the event that the patient is not able to do so. This type of document allows a trusted friend or relative to "stand in the shoes of the patient" to make health care decisions when the patient is not able to make them.

Savvy patients will prepare both a living will and the durable power of attorney for health care. This ensures that the decision maker's decisions will be as close as possible to what the patient wants. Many advance directives give the health care agent specific instructions concerning health matters. For example, the advance directive may provide instructions concerning artificial nutrition and hydration, or it may outline specific treatments, such as a "no code" status under specified circumstances.

In response to a federal statute referred to as the Patient Self-Determination Act of 1991,[10] all 50 states have statutes that allow patients to execute living wills, durable powers of attorney for health care, and advance directives. However, each state may place unique requirements on the drafting of these documents. Some states require that the directive be notarized. Other states mandate that a state-appointed ombudsman who outlines the pros and cons associated with the advance directive counsel the patient. Witness requirements also vary from state to state. Consequently, it is important to know the laws concerning advance directives that apply in your state. An excellent starting point is the web page of the National Hospice and Palliative Care Organization (caringinfo.org), where you can download advance directives and instructions from each state. The Web site of the American Association of Retired Persons (aarp.org) provides lay people and health care providers with up-to-date information about advance planning for health care.

In most states, it is likely that a recent living will would be taken as evidence of what the patient would have wanted had he or she been competent when the decision was presented. Although there have not been any cases concerning a written living will, there have been several involving patients who had expressed wishes orally about life-sustaining measures.

▲ Issues That Involve Life-Support Measures

Several basic issues regarding refusal and termination of treatment can involve the critical care nurse. Do not resuscitate (DNR) orders, refusal of treatment for religious reasons, advance directives, and withdrawal of life support all fall into this category.

Do Not Resuscitate (DNR) Orders

Cardiopulmonsry resuscitation (CPR) success rates for those receiving in-hospital care are variable and are affected by patient environment and resuscitative factors.[11] However, CPR is not appropriate for all patients who experience a cardiac arrest, because it is highly invasive and may constitute a "positive violation of an individual's right to die with dignity." Furthermore, CPR may not be indicated when the

illness is terminal and irreversible and when the patient can gain no benefit.

Prestigious authorities (eg, the President's Commission for the Study of Ethical Problems in Medicine and Biomedical and Behavioral Research; hereafter "the President's Commission") have recommended that hospitals have an explicit policy on the practice of writing and implementing DNR orders.[12] Most hospitals and medical societies, and some states, have published DNR policies.[13]

Whether to resuscitate any patient is a decision that is made with the attending physician, the patient, and the family, although critical care nurses and other nurses often have substantial input into the decision. However, in general, the consent of a competent patient or the patient surrogate is required when a DNR decision is made and the order is written.

Once the DNR decision has been made, the order should be written, signed, and dated by the responsible physician. It should be reviewed periodically; hospital policies may require review every 24 to 72 hours. The more informal methods of designating patients with whom CPR is not to be undertaken can lead to uncertainty and inappropriate responses if an arrest occurs. If an arrest occurs in an emergency department or in another situation in which a formal DNR decision has not been made and written, the presumption of the medical and nursing staffs should be in favor of intervention, and a code should be called. A "slow code" (in which the nurse takes excessive time to call or the health care team takes its time responding) is never permissible. Either CPR is indicated, or it is not.

Courts sometimes become involved in DNR decisions and provide legal guidance through case law. In 2004, a California court ruled that a physician may lawfully write a DNR order for a minor patient for whom there is no lifesaving or life-prolonging treatment. This case involved a DNR order for Christian, an 11-year-old boy with cystic fibrosis who developed the flu and then pneumonia after his parents had rejected the flu shot for him. A nurse testified that she was present during the discussion between the doctor and the parents, and that both parents agreed to the DNR order "in view of (Christian's) chronic irreversible condition."[14]

CASE STUDY

Discontinuation of Ventilator Support

Dr. B. was a 79-year-old nuclear engineer in the end stages of Alzheimer's disease who was admitted to the hospital in a vegetative state with aspiration pneumonia. Mr. B. had no advance health care directive. A family dispute evolved among his sons over his care. After hearings, the court appointed one son as Dr. B's guardian and surrogate decision maker. In 2009, the court denied the request of another son for a hearing on the issue of whether his brother may authorize the removal of hydration, nutrition, and respiratory care.[15]

Unfortunately, it is estimated that as few as 4% to 24% of Americans have an advance directive.[16–18] An advance directive can help with troublesome decisions, such as those that were faced by both the family and the health care team in this case. It is also important for patients to speak with their families and their attending physician concerning end-of-life decisions.

Right to Refuse Treatment for Religious Reasons

CASE STUDY

Lifesaving Transfusion for a Jehovah's Witness

Petitioner was a 17-year-old Jehovah's Witness diagnosed with lymphoblastic leukemia when his doctors wrote a letter to the court requesting an order authorizing them to be able to transfuse him to prevent death or serious harm. A juvenile court issued an order authorizing the administration of transfusions using whole blood products despite his religious beliefs and the wishes of his parents. Petitioner filed an appeal, asking the court to establish a common-law "mature minor" exception to the statute that allowed a judge in the juvenile court to authorize emergency medical care. The exception would have allowed the mature minor to make these decisions. The court did not rule in this case, because Petitioner turned 18 and was now legally able to make his own health care decisions, including refusal of transfusions to treat his leukemia.[19]

Some courts are unwilling to rule against the religious-based decisions of a patient to refuse treatment, but they are most likely to do so if the welfare of a dependent child is at stake. For example, in one case, the Connecticut Supreme Court found that a hospital could not "thrust unwanted medical care on a patient who… competently and clearly denied that care." Critical care nurses need to consult the hospital's risk management department or legal counsel in such situations to ensure proper handling of these types of legal issues.[20] There are exceptions to the informed consent requirements. For example, an emergency situation in which time and circumstances create critical barriers does not require informed consent before initial procedures are implemented. A patient can also waive the right to informed consent by stating that he or she does not want information about a proposed treatment or procedure.

Landmark Legal Cases on Withdrawal of Treatment

What constitutes life support, when these measures must be used, and when they may be terminated are issues that have been raised in many court cases. However, the law in these areas is still developing and will continue to do so as each state creates its own guidelines and as technology continues to introduce new possibilities.

In the matter of *Schiavo v. Schiavo*,[21] the United States was drawn into a legal and emotional battle over the issue of the right to die and, barring advance directives, who may speak for an unconscious patient. Theresa (Terri) Schiavo was in a vegetative state for 13 years. In 2003, Ms. Schiavo's husband, Michael, petitioned to cease his wife's feeding and hydration over the objections of her parents and brother.

In response, Terri's parents, Robert and Mary Schindler, led a battle before the Florida State Courts, the U.S. Courts of Appeal, the Florida State Legislature and Executive Branch, the U.S. Congress, the White House, and, eventually, the U.S. Supreme Court.[22] Aside from the legal activities, the parties to the action also tried Terri's circumstances and future before the court of public opinion.

Although it is unusual for this type of case to be heard in the U.S. Supreme Court, lower court involvement in this type of situation is not unusual. When a disabled person is unable to understand and coherently speak on his or her own behalf, the law requires that health care providers first obtain the patient's personal advance directives. Courts have repeatedly struggled over who should make decisions about the disabled patient's care when there is no advance directive and relatives cannot agree on the appropriate plan of care for the patient, such as occurred in the Schiavo case.

Initially the Schiavo case was representative of a typical guardianship action. The petitioner (eg, husband Michael Schiavo) petitioned the court to act on behalf of the alleged disabled patient (eg, his wife, Terri). Notice of the proposed order to name Mr. Schiavo as Terri's guardian was given to all interested persons, including her parents Mr. and Mrs. Schindler.[21] In addition, courts may appoint a temporary *guardian ad litem*, charged with the duty to represent the interests of the alleged disabled. The temporary guardian ad litem, usually an attorney, is an independent, disinterested party whose responsibilities are to review the medical records, interview all medical providers, interview the petitioner (ie, Mr. Schiavo) and any interested persons, and most importantly, interview the alleged disabled as to his or her mental and physical capacity. The temporary guardian ad litem reports independently to the court on his or her findings of the status of the alleged disabled.

In the Schiavo case, a custodial hearing was held allowing the petitioner, witnesses, and any other interested persons to testify as to (1) the competency of the alleged disabled, (2) whether the alleged disabled would be best served with a guardianship, and (3) who would be best to act as *permanent* guardian ad litem. In deciding these issues, the courts place great weight on the findings of the temporary guardian ad litem.

Michael Schiavo prevailed in maintaining his position as permanent guardian ad litem of Terri,[22] and her tube feeding was discontinued. As a result of this and other cases, families throughout the nation became more aware that they might be faced with the awful possibility of being pitted emotionally, legally, and monetarily against each other when advance directives are not executed.

Nurses, particularly those working in critical care areas, are frequently confronted with patients who are incompetent to understand the nature of their care. Nurses are instrumental in educating patients, families, friends, and society about the importance of understanding and executing advance directives. From an attorney's perspective, the consensus is, "Pay me a little now to prepare a valid advance directive, or pay me dearly when we need to litigate guardianship and end-of-life issues."

Given the regularity with which life-support decisions must be made in health care facilities, it is remarkable that it was not until 1976 that the first case, *In re Quinlan*, focused national attention on the "right to die" controversy. The cases concern competent minors and adults who have a disease or condition that would eventually be terminal. States have not been consistent in their decisions, even when the situations are arguably similar. For example, the New Jersey court in the case of Karen Ann Quinlan, a 21-year-old woman in a persistent vegetative state, held that the decision about treatment is in the hands of the patient's guardian in consultation with the hospital ethics committee.[23] The President's Commission stated (p 6) that judicial review of these decisions should be reserved for occasions when "adjudication is clearly required by state law or when concerned parties have disagreements that they cannot resolve over matters of substantial import."[12]

CASE STUDY

Right to Restrict Food and Fluids

Nancy Cruzan, a young woman who suffered anoxic brain damage in an automobile accident, remained in a persistent vegetative state in Missouri and was fed by gastrostomy. After rehabilitation was unsuccessful, Ms. Cruzan's parents (as co-guardians) requested withdrawal of the feeding tube. After the employees of the residential rehabilitation center where Ms. Cruzan was receiving care refused to withdraw the feedings, her parents sought judicial review of their request. After testimony, the trial court approved the parents' request.

On appeal, the Missouri Supreme Court reversed the lower court. First, it held that Missouri law does not permit surrogate decision making in decisions of this importance. For a person to exercise the right to terminate artificial feeding in Missouri, that person must have previously expressed his or her wishes, either orally or in writing. Evidence of those wishes had to meet a relatively high evidentiary standard, a standard that the court held had been met in the lower court proceeding.

This case was appealed to the U.S. Supreme Court, and in 1990, it was affirmed on constitutional grounds.[24] After the decision was issued, the Cruzans returned to the Missouri lower court and presented further evidence (through additional witnesses) about what their daughter had expressed while competent. The lower court found that they had presented clear and convincing evidence and affirmed the rights of the co-guardians to authorize withdrawal of the feeding tube.

It is important to note that although the Cruzan case is still applicable law, the way in which this landmark case has been interpreted and implemented has been extremely variable at state court levels. This case received much publicity, but it did not change the law in any state except Missouri. Most states continue to permit surrogate decision making by relatives and require a lower evidentiary standard than that required in Missouri. Also note that, unlike the Schiavo case described previously, family members were all in agreement that Ms. Cruzan's artificial nutrition and hydration should be discontinued.

The right to consent to treatment is meaningless without the right to refuse treatment before or after it is initiated. In recent years, as health care providers have become more comfortable recommending rejection or termination of treatment in selected cases, they have met resistance from some families who wish to continue treatment no matter what the chance of success. Although no law or legal principle requires that extraordinary, but clearly futile, treatment be provided, it is probably also true that health care providers have little legal recourse against families who refuse to withdraw life support, that is, unless the patient has left written indications of his or her wishes before incompetence. This may change as society reexamines the allocation of limited resources in health care.

In most states, problems of terminating treatment need not be resolved in court. This is not an easy decision, but decisions regarding treatment or nontreatment that meet accepted medical standards and with which the patient concurs are made virtually every day in health care settings such as critical care. Hospital ethics committees typically play a crucial role in such circumstances.

A distinction should be made between termination of treatment and termination of care. Ending treatment is not the same as giving up. Patients who are not being "treated" for their terminal condition require competent and sensitive nursing and medical care. Palliative care provides pain relief and symptom management, and a better quality of life for those nearing the end of life and their families. The duty to provide good nursing care does not end with the decision to move from treatment for cure to providing comfort care and (ideally) timely referral to hospice.

Brain Death

In 1968, an ad hoc committee at Harvard Medical School established the Harvard criteria for determining brain death, or irreversible coma. In 1981, the President's Commission for the Study of Ethical Problems in Medicine and Biomedical and Behavioral Research published *Defining Death*. The Commission recommended a uniform statute defining death, to address "general physiological standards rather than medical criteria and tests, which will change with advances in biomedical knowledge and refinements in technique."[25] All states have laws addressing the definition of death in the state. Some states adopted the Harvard criteria by statute, whereas other states enacted legislation that defines brain death in broader, less restrictive terms. Some states use brain death as the sole criterion; other states rely on a number of factors, such as response to pain and cessation of cardiac function. It is important that the nurse know the legal definition of death in any state where he or she is practicing, although such a determination typically rests with the patient's attending physician and may require the concurrence of other consulting physicians.

A patient who is brain-dead is legally dead, and there is no legal duty to continue to treat him or her. It is not necessary to obtain court approval to discontinue life support on a patient who is brain-dead. Furthermore, although it can be desirable to obtain family permission to discontinue treatment of a brain-dead patient, it is not a legal requirement. Before terminating life support, physicians and nurses should determine whether the patient is an organ donor.

Organ Donation

Every state in the United States has a law based on the Uniform Anatomical Gift Act. The statutes establish the legality of organ donation by people and their families, and set procedures for making and accepting the gift of an organ. Every state also has some provision to enable people to consent to organ donation using a designated place on a driver's license. More recently, many states have enacted "required request" laws. These laws attempt to increase the supply of organs for transplantation by requiring hospital personnel to ask patients' families about an organ gift at the time of the patient's death.

▲ Clinical Applicability Challenges

SHORT ANSWER QUESTIONS

1. Nurse Jacqui has just learned that a judge has ruled to dismiss a professional malpractice claim that was filed against her, because the patient alleged no damages that could form the basis of an award. What is the status of this patient's complaint against Jacqui?

2. A patient has filed a complaint with Nurse Jacqui's Board of Nursing, alleging that Jacqui failed to meet the standard of care. Jacqui learned of this by receiving an order informing her of the complaint and the decision of the Board to suspend her license. What is Jacqui's status now?

3. Nurse Port was on duty when she received a new admission with a diagnosis of "rule out pneumonia." The patient had several orders for an IV and for STAT medications. Nurse Port picked up the IV bag from the medication room and hung it. The patient died, and staff later discovered that Nurse Port had picked up the wrong bag. How should this situation be handled?

4. Nurse Good has a patient in intensive care who is in critical condition with uncontrolled bleeding after apparently becoming the victim of a hit-and-run driver. The patient is conscious but appears weak and confused. Nurse Good and law enforcement officers are desperately trying to find information about this patient. A visitor has just appeared who informs Nurse Good that the victim appears to be his brother. He is very distraught and asks whether his brother has had a blood transfusion. How should Nurse Good respond?

References

1. National Council of State Boards of Nursing. Retrieved December 14, 2010 at https://www.ncsbn.org
2. Wendy Guzman: Individually and as Next Friend of T. Guzman v. Memorial Hermann Hospital System, d/b/a/ Memorial Hermann Southeast Hospital. Civil Action No. H-07-3973. 637 F. Supp. 2d 464; 2009 U.S. Dist. LEXIS 50574
3. Kohn LT, Corrigan JM, Donaldson MS, (eds): Committee on Quality of Health Care in America. To Err is Human: Building a Safer Health System. Institute of Medicine. Washington, DC, National Academy Press, 1999

4. Martin v. Ricotta et al: Supreme Court of New York, 2009 NY Slip Op 32976U, 2009 N.Y. Misc., 2009
5. U.S. v. Wood, 207 F.3d 1222; 2000 U.S. App. LEXIS 5475, 2000 Colo J. C.A.R. 1645, 2000
6. Las Colinas Medical et al v. Bush, 122 SW3d 835 (TX App 2nd Dist), 2003
7. U.S. Department of Health and Human Services, Food and Drug Administration: Medical devices: Medical device, user facility, distributor, and manufacturer reporting, certification and registration. Fed Regist 56:64004–64182, 1991
8. Government Accountability Office. 2009. Medical Devices: FDA Should Take Steps To Ensure That High-Risk Device Types Are Approved Through The Most Stringent Premarket Review Process. GAO-09-190. Retrieved from on December 14, 2010 from http://www.gao.gov/products/GAO-09-190
9. Aurea Esther Ramirez-Velez, et al V. Centro Cardiovascular, et al: CIV. NO. 05-1732(PG), 2007 U.S. Dist. LEXIS 81956, 2007
10. Omnibus Budget Reconciliation Act of 1990. Pub. L. No. 101-508 §§4206, 4751 (codified in scattered sections of 42 USC, particularly §§1395cc, 1396a) (West Supp), 1991.
11. Dumot JA, Burval DJ, Sprung J, et al: Outcome of adult cardiopulmonary resuscitations at a tertiary referral center including results of "limited" resuscitations. Arch Intern Med 161(14):1751–1758, 2001
12. President's Commission for the Study of Ethical Problems in Medicine and Biomedical and Behavioral Research: Deciding to forego life-sustaining treatment. Washington, DC: U.S. Government Printing Office, March 1983
13. Harris DM. Contemporary Issues in Healthcare Law and Ethics, 3rd ed. Chicago, IL: Health Administration Press, 2010
14. Margherita Underhill v. Long Beach Memorial Hospital Center et al: 2007 Cal. App. Unpub. LEXIS 3387
15. Scot Bernstein v. The Superior Court of Ventura County, 2009 Cal. App. Unpub. LEXIS 894
16. Ngo-Metzger Q, August KJ, Sinivsan M, et al: End of life care: Guidelines for patient-centered communication. Am Fam Physician 77(2): 167–174, 2008
17. Ackermann RJ. Withholding and withdrawing life-sustaining treatment. Retrieved December 14, 2010, from http://www.aafp.org/afp/20001001/1555.html
18. Stamford Hosp. v. Vega, 646 (CT), 1996
19. Huffman GB. Benefits of discussing advance directives with patients. Retrieved December 14, 2010, from http://www.aafp.org/afp/20001001/1555.html
20. Law v. Camp et al: 116 F. Supp. 2d 295 (CT), 2000
21. Michael Schiavo, as Guardian of the person of Theresa Marie Schiavo, v. Jeb Bush, Governor of the State of Florida, Charlie Crist, Attorney General of the State of Florida, Florida Circuit Court Civil Case No. 03-008212-CI-20, 2004
22. Schiavo v. Schiavo, DC CV-05-00530-T, U.S. 11th Circuit Court of Appeals, 2005. See also, Bush v. Schiavo, Case No. SC04-925, Supreme Court of Florida. The U.S. Supreme Court denied the Schindlers' petition for a stay of action without any further opinion, thus allowing the removal of Terri's tube feeding (S Ct Order 04A825), March 24, 2005.
23. In re Quinlan, 70 NJ 10, 355 A2d 647, New Jersey, 1976.
24. Cruzan v. Director, Missouri Department of Health et al, III L Ed2d 224, 110 S Ct 2841, 1990.
25. President's Commission for the Study of Ethical Problems in Medicine and Biomedical and Behavioral Research: Defining death: A Report on the medical, legal and ethical issues in the determination of death. Washington, DC: U.S. Government Printing Office, July 1981.

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

9 Building a Professional Practice Model for Excellence in Critical Care Nursing

Janie Heath

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Discuss nursing professionalism and nursing excellence.
2. Recognize characteristics of professional development.
3. Explore personal and professional attributes to build a professional practice model of critical care nursing excellence.

In today's fast-paced critical care environment, nurses respond to the needs of patients and families who have entered a chaotic and frightening world of illness, trauma, and pain. Often, finding the time for professional growth can be challenging. Building a professional practice of excellence requires a "passion" to profoundly affect the lives of patients and families. At the same time, it requires advancing the critical care nursing profession through evidence-based practice, best practice models of care, or both. This chapter discusses how a professional practice for excellence in critical care nursing can be built with the attributes of values, vision, mastery, passion, action, and balance as the framework.

▲ Defining the Critical Care Nurse

Like their patients and patients' families, critical care nurses are an exceptional and diverse group of people. Knowledgeable, highly skilled, and caring are a few of the professional attributes that can be applied to critical care nurses. However, the term *nursing professionalism* may bring to mind different images, especially to health care consumers. To some, nursing professionalism still means wearing a crisp, clean, white uniform, whereas to others, regardless of the uniform, it means demonstrating a high level of intellectual, interpersonal, ethical, and clinical skills. Kalisch and Kalisch[1] first reported on the image of nursing in the early 1980s. They found that "90% of the public thought nurses were nice ladies who help doctors."

Critical care nurses know all too well that responding to lethal dysrhythmias, administering blood products, and weaning patients from ventilators is more about having a specialized body of knowledge, competent skills, and clinical experience in holistic nursing than about just "helping doctors." For both the novice and the experienced critical care nurse, the journey of nursing professionalism and nursing excellence goes beyond the bedside skills required to take care of the sickest and most vulnerable patients and families.

Critical care nursing started being recognized as a specialty when the first intensive care units (ICUs) emerged in the 1950s, yet Buresh and Gordon[2] have found increasing evidence of a large communication gap between the profession and the greater public. If critical care nursing is to be recognized as a respected and valued profession, nurses must boldly speak up to define who they are and what they do.

A starting point to define who critical care nurses are can be found in the annual demographic membership survey conducted by the American Association of Critical-Care Nurses (AACN),[3] the world's largest specialty nursing organization. Since 1969, the AACN has been serving the needs of more than 500,000 nurses who care for critically ill patients and their families. With a steady membership of approximately 80,000 nurses, the majority of members (60%) are between the ages of 40 and 59 years of age and have a bachelor of science in nursing (Fig. 9-1A).[3] With an average income between $55,000 and $74,999 per year, the majority of members (25%) have been in critical care practice for more than 20 years (see Fig. 9-1B).[3] Although the profession continues to be predominantly female (90%), the number of men in critical care is increasing (11%), according to the AACN membership survey.[3] The largest ethnic background represented was Caucasian (78%), followed by Asian (12%), African American (4%), Hispanic (3%), and Native American (1%).[3] Of interest, the AACN membership data are consistent with the average findings of today's 3 million registered nurses (RNs) reported in the initial findings from the 2008 National Sample Survey of Registered Nurses.[4]

It is important to evaluate such data because this helps drive decision making and determine trends, issues, and policy and advocacy implications that affect critical care nursing practice, patients and families, and systems. Currently, the majority of AACN members (18%) work in ICU settings followed by 13% working in combined ICU and coronary care unit (CCU) settings, 12% working in progressive care settings, 10% in cardiovascular–surgical ICU settings, 6% working in CCU, 5% working in medical ICU, 4% working in medical–surgical ICU, and 4% working in pediatric ICU.

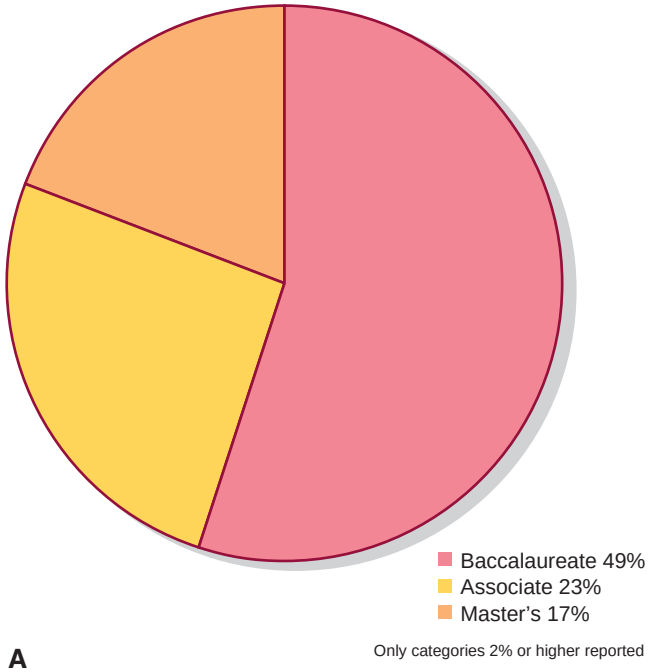


FIGURE 9-1 ▲ Mastery of the profession. **A**: The nursing degrees earned by critical care nurses. **B**: Years of experience in critical care nursing. (From American Association of Critical-Care Nurses: 2010 Demographics. Retrieved June 1, 2010, from http://www.aacn.org.)

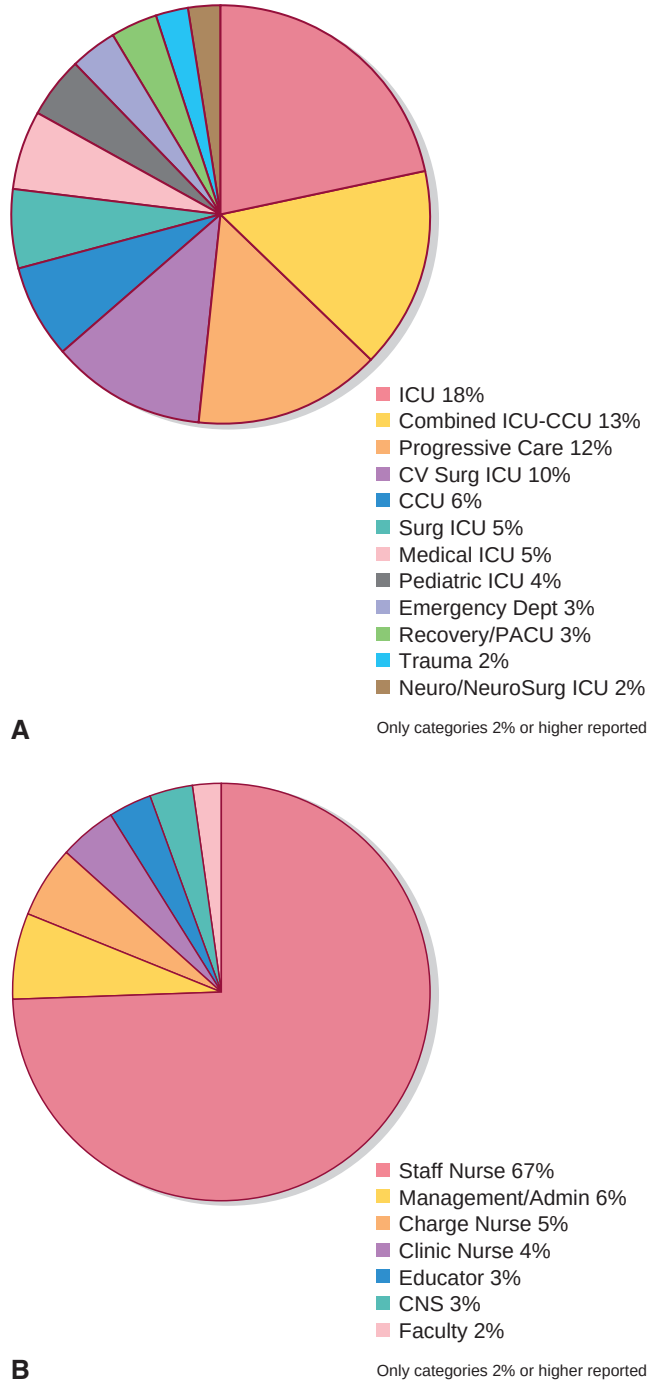


FIGURE 9-2 ▲ Who are critical care nurses? **A**: Practice settings for critical care nurses. **B**: The positions held by critical care nurses. (From American Association of Critical-Care Nurses: 2010 Demographics. Retrieved June 1, 2010, from http://www.aacn.org.)

The remaining top categories make up less than 4% of the total and include the emergency department, recovery room/postanesthesia care unit, trauma unit, and neuro/neurosurgical ICU (Fig. 9-2A).[3] The vast majority of the AACN members surveyed (67%) have positions providing direct care as staff nurses (see Fig. 9-2B). The AACN membership data are consistent with the average findings of Kirchhoff and Dahl's[5] national survey of facilities and units that provide critical care. Their study revealed that 74% of the facilities participating in the study were nongovernment, not-for-profit organizations with a mean of 217 operating beds and 13,000 admissions per year.[5]

▲ Defining Nursing Professionalism

The struggle to define nursing professionalism expands beyond critical care environments. There continues to be on going dialogue about whether nursing is a true profession. For more than two decades, Kelly emphasized that the status of nursing as a profession is important because it reflects the value society places on the work of nurses.[6] However,

some think that because entry into nursing practice does not require a baccalaureate degree, it is, at best, an emerging profession that requires new models of nursing education.[7] Such new models, the clinical nurse leader and doctorate of nursing practice, are believed to help ensure quality patient outcomes and patient safety.[7,8] However, others believe that the nursing profession has made adequate progress to meet full-fledged professional status with current models of nursing education.[9]

One of the first definitions of professionalism came from Abraham Flexner,[10] who wrote the classic Flexner Report in the early 1900s to reform medical education. Flexner defines professionalism as a process by which an occupation achieves professional status. Although other professions have developed their own criteria, Flexner's work remains the benchmark and foundation for many. In 1981, Kelly was the first to expand his work for the nursing profession by providing a theoretical framework from which professional nursing characteristics are defined today[6] (Box 9-1).

To a great extent, the criteria addressed by Flexner and Kelly are only as good as the person who takes personal responsibility and accountability for committing to a professional role. One legendary nurse leader, Margretta Styles, argued that professionalism of nursing can only be achieved through the "professionhood" of its members.[7] Like Kelly, Styles believes there must be a sense of social significance, commitment to the ultimacy of professional performance, and appreciation of collegiality and collectivity.[7] However, as Styles approached the end of her nursing career, she proposed a new term to phrase the work of nurses: *professionalist*.[7] In her words, "professionalists strive to build a solid foundation for their calling—an ethical, academic, political, and socioeconomic foundation to serve as the underpinning for a strong profession to evolve and serve." (p 89)[7]

Researchers have investigated how to describe professionalism among critical care nurses as well. In 1994, Holl[11] investigated such critical care nursing characteristics as professional beliefs, decision making, level of education, membership of professional nursing organization, and certification. Holl[11] found that nurses who continue their education and belong to professional organizations are more likely than others to be independent thinkers and to participate in creative problem solving. In a similar study, Heath et al[12] found that there was a high level of "passion about nursing and promoting the profession" and that self-motivation was the leading influential factor for fostering individual professional development among critical care nurses. Other professional development characteristics evaluated included participation in employing agency committees, community service, and recognition of peers.[12] Of note, several of the professional characteristics initially identified by Kelly and studied by others[13,14] are identified in this chapter as hallmarks of excellence for critical care nursing practice.

BOX 9-1 Kelly's Characteristics of a Profession

- The services provided are vital to humanity and the welfare of society.
- There is a special body of knowledge that is continually enlarged through research.
- The services provided involve intellectual activities where accountability is a strong feature.
- Practitioners are educated in institutions of higher learning.
- Practitioners are motivated by service, and work is an important component of their lives.
- There is a code of ethics to guide the decisions and conduct of practitioners.
- There is an association that encourages and supports standards of practice.

From Kelly L: Dimensions of Professional Nursing. New York, NY: Macmillan, 1981; and Joel L: Kelly's Dimensions of Professional Nursing, 9th ed. New York, NY: McGraw-Hill, 2003.

▲ Defining Nursing Excellence

The term *excellence* can be a misnomer similar to the expression *best practice*. No single definition captures the essence of excellence for everyone. For some, it is something that is seen, heard, or felt that describes excellence in nursing. For example, the way a critical care nurse sees a patient going "bad" before laboratory values or hemodynamic numbers are known may be evidence of excellence. Other descriptions of excellence may be the way a critical care nurse hears an S_3 or S_4 heart sound before a patient becomes symptomatic, or the way a critical care nurse "feels" the pain of a postoperative patient on neuromuscular blockade without analgesics.

Weston et al[15] define nursing excellence as a dynamic process that is continually redefined and reinforced. They further describe excellence as an ongoing comparison with a standard that one continuously tries to improve.[10] Six attributes for advanced-practice nursing excellence have been identified by Weston et al[15] as values, vision, mastery, passion, action, and balance. These attributes have been adopted and modified for this chapter to propose a professional practice model for critical care nursing (Fig. 9-3). The foundation for this model consists of strong values and a vision. The supporting structures of the model are composed of mastery, passion, action, and balance. The top of the model captures the essence of the structure: critical care nursing excellence. Each structure of the professional practice model has defining characteristics that are instrumental for ongoing self-reflection, which is necessary to develop and commit to excellence in critical care nursing.

Values

CASE STUDY

"Our lives begin to end the day we become silent about things that matter." (Martin Luther King Jr.)[16]

Reflection on "Values" for Critical Care Nursing Excellence

"A group of nurses, participating in a focus group for the AACN-VitalSmarts Silence Kills Study, describe a peer as careless and inattentive. Instead of confronting her, they double check her work—sometimes running into patient rooms to retake a blood pressure or redo a safety check. They have

(*continued on page 102*)

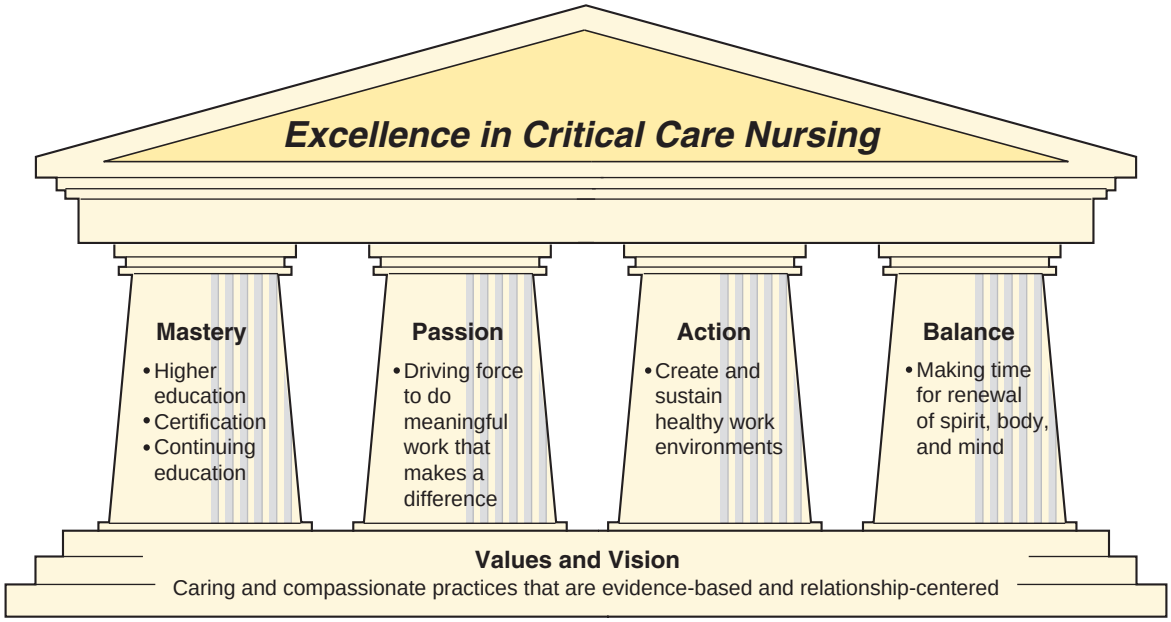


FIGURE 9-3 ▲ A professional practice model for critical care nursing.

'worked around' this nurse's weaknesses for over a year. The nurses resent her but never talk to her about their concerns, nor do any of the physicians who also avoid her and compensate for her." (p 2)[17] Further data reveal that out of 1,700 health care providers and administrators throughout U.S. hospitals, 84% of physicians and 62% of nurses and other clinical care providers have seen coworkers taking shortcuts that could be dangerous to patients.[17] McCauley summed the significance of the study well by stating, "This research (Silence Kills) validates what our 100,000 constituents have communicated to us as the number one barrier hindering optimal care for patients. Too often, improving workplace communications is seen as a 'soft' issue—the truth is, we must build environments that support and demand greater candor among staff if we are to make a demonstrable impact on patient safety."[18]

Do you value communication about competence and accountability? Do you value having a healthy work environment where skilled communication protects patients and fosters collaborative relationships? What are your values to foster care nursing excellence and make a difference in the lives of the acute and critically ill?

True excellence is seen when professionals reflect their core values. The values of one's profession, the values of one's employing organization, and one's own personal values are the behaviors that guide professional practice for excellence. The unique contributions that critical care nurses bring to the bedside are often a reflection of an inner core value of caring. It is this deep and personal connection to caring that brings many into the nursing profession. The word *nursing* is derived from the Latin word *nutrire*, which means "to nourish." The term *nurturing* describes an ability to care for, sustain, and provide for another. Critical care nurses are privileged to care for individuals who face life-threatening conditions during the most vulnerable and private times of their lives. It is through this value of altruism (the desire to help others) that critical care nurses have the ability to creatively bridge high-tech and high-touch with everyday practice.

The everyday busyness of critical care nursing is labor intensive, but taking the time to share joyful, painful, and tearful experiences with complex patients and families is the core of critical care nurses' existence. The art of nursing is probably what dominates the image of nursing for the public. For 8 years in a row, Gallup polls have reported that the public rates nursing as the most honest and ethical profession.[19] In their book *From Silence to Voice*, Buresh and Gordon[2] reported that the Gallup poll results about nurses reflect a paradox. As nonnurse authors, they discuss how the public holds nurses in the highest regard, even though the public has limited information about the science that nurses really practice.[2]

There is power in the nursing profession when nurses articulate not only the core values of caring but also evidence-based practice, advocacy, accountability, autonomy, and collaboration. It is now well established that nurses have been silent for too long and that the days of "It is just my job" or "I am just a nurse" need to come to an end.[2,17,20] Themes such as AACN's "Powered by Insight," "Reclaim our Priorities," "With Confidence," and "Act with Intention" help empower nurses to make their voice heard for their patients, families, and profession.[21–24] Strong personal and professional core values at AACN (Box 9-2) inspired a nine-person panel to develop the AACN Healthy Work Environment Standards to help address work environments that tolerate ineffective interpersonal relationships resulting in medical errors, ineffective delivery of care, and conflict and stress among health professionals.[20] Creating and sustaining healthy work environments ensure safe patient care and set a path toward the AACN core value to "commit to quality and excellence."[25]

BOX 9-2 Core Values: American Association of Critical-Care Nurses (AACN)

- **Be accountable** to uphold and consistently act in concert with ethical values and principles.
- **Advocate** for organizational decisions that are driven by the needs of patients and families.
- **Act with integrity** by communicating openly and honestly, keeping promises, honoring commitments, and promoting loyalty in all relationships.
- **Collaborate** with all essential stakeholders by creating synergistic relationships to promote common interest and shared values.
- **Provide leadership** to transform thinking, structures, and processes to address opportunities and challenges.
- **Demonstrate stewardship** through fair and responsible management of resources.
- **Embrace lifelong learning,** inquiry, and critical thinking to enable each to make optimal contributions.
- **Commit to quality and excellence** at all levels of the organization, meeting and exceeding standards and expectations.
- **Promote innovation** through creativity and calculated risk taking.
- **Generate commitment and passion** to the organization's causes and work.

From American Association of Critical Care Nurses: Core values. Retrieved June 1, 2010, from http://www.aacn.org/aboutaacn, with permission.

Vision

CASE STUDY

"There is no power greater than a community discovering what it cares about." (Margaret Wheatley)[16]

Reflection on "Vision" for Critical Care Nursing Excellence

What would a reality show film crew see if they followed you for a day? Someone focused and intentional in his/her actions? Or an endless string of hit-and-runs? "Intention is the why." It's the reason we do something. When our intentions are straightforward and confident, we carry them out because we know they will keep our patients safe and lead to best outcomes. Sometimes our intentions are clear-cut. Caring for someone with a gastrointestinal hemorrhage has three initial goals: Find the bleed. Stop the bleed. Replenish volume loss. Lack of intention has obvious negative consequences. Inability to tolerate an endoscopy delays finding and stopping the bleed. A clotted type and cross sample delays blood replacement. An overwhelmed family requires additional information and refocused energy.

Other times, intentions aren't so clear. Caring for someone with acute coronary syndrome makes you stop and ask questions like "Why do patients wait so long to come to the hospital" and "What stops nurses and docs from getting that first ECG, initial blood samples, and a focused history during the critical first 10 minutes?" When we step back and take in the bigger picture, our intentions become clear.

Do you remember Yoda, the character from the movie *Star Wars*? Diminutive, pointy ears, greenish hue, very wise. "Do or do not," he said, "There is no try." (p 22)[24]

Beth Hammer, RN, MSN, APRN-BC, Past President of AACN, Act with intention and speak with a bold voice.

Do you have a vision to act with intention and live that vision in all of your actions? Do you have a vision to have a bold voice for a culture in which people hold themselves and others accountable to professional standards and collaborative relationships? What is your vision to foster nursing excellence and make a difference in the lives of the acute and critically ill?

A clear vision, based on core values, is essential to building a professional practice model of critical care nursing excellence. It requires envisioning the future's possibilities and then taking the challenge of making the vision a reality. In 2001, the AACN made a strategic decision to promote the creation of healthy work environments that embrace a culture of excellence when taking care of acute and critically ill patients.[20] Based on the escalating evidence from the Institute of Medicine and the Joint Commission about unhealthy work environments and how they contribute to medical errors, the AACN had a vision to propose solutions to improve patient safety.[26–28] Through a partnership with VitalSmarts, a national study was conducted to evaluate communication and collaboration challenges among hospital providers of care. The findings of the study resulted in the need to develop standards addressing six essential areas: skilled communication, true collaboration, effective decision making, appropriate staffing, meaningful recognition, and authentic leadership.[20]

As risk takers and agents for change, today's critical care nurses are making history by embracing the AACN Healthy Work Environment Standards. Winston Churchill once said, "The pessimist sees difficulty in every opportunity and the optimist sees the opportunity in every difficulty."[16] It is challenging to create and sustain work environments of excellence, especially when there are concerns about nurse-to-patient ratios, mandatory overtime, unionization, nursing recruitment, and retention. It is also difficult to provide critical care nurses with the tools, resources, and support they need to meet patient and family needs effectively and at the same time enhance their own professional growth, learning, and satisfaction. Nursing has a long tradition of taking "bumpy" roads to make a vision reality. No matter how many times nurses fall down and get bruised on the way to reach a vision, they maintain their resilience by having the courage to listen, learn, and act for themselves, their patients, and their professional practice.

Mastery

CASE STUDY

"Education is our passport to the future, for tomorrow belongs to the person who prepares for today." (Malcolm X)[16]

Reflection on "Mastery" for Critical Care Nursing Excellence

"At 3:30 AM in a busy ICU, a nurse prepares to give insulin to a patient with an elevated blood glucose level. The sliding scale doses of insulin on the medication sheet are unclear, and the physician's order sheet is difficult to read. From past experience, the nurse knows how late night calls to this physician often result in verbal outbursts and demeaning slurs, no matter how valid the inquiry. Needing to act but not wanting another harassing encounter with the physician, she makes a judgment of the appropriate dose and administers the insulin. Two hours later, she finds the patient completely unresponsive. To treat the critically low blood glucose level, she administers concentrated injections of glucose and calls for additional emergency help. Despite all attempts to restore the patient's brain to consciousness, he never awakens and his brain never functions normally again." (p 10)[20] Barden summed this scenario well by stating, "Nurses must be as proficient at handling personal

(*continued on page 104*)

communication as they are in clinical skills. A culture of safety and excellence requires that individual nurses and healthcare organizations make it a priority to develop communication skills that are on par with expert clinical skills."[18]

What mastery of knowledge and skills have you achieved to stop verbally abusive behavior in the workplace? What mastery of knowledge and skills have you achieved to seek solutions that preserve a nurse's personal integrity and ensure a patient's safety? What are you mastering to foster nursing excellence and make a difference in the lives of the acute and critically ill?

Studer,[29] author of *Hardwiring Excellence*, believes that excellence in health care occurs when "employees feel valued, physicians feel their patients are getting great care, and patients feel the service and quality they receive are extraordinary." (p 45) Studer[29] further believes that creating and sustaining a culture of excellence requires the willingness to take individual ownership (be an owner, not a renter of an organization) of problems and opportunities. To value lifelong learning and to have a vision for personal mastery is essential to building a professional practice model for critical care nursing excellence (see Fig. 9-3). There are many pathways for personal mastery. Weston et al[15] believe that seeking feedback and peer review for self-improvement is one of the most effective pathways for building mastery. Other pathways for personal mastery include seeking a degree in higher education, making a commitment to ongoing continuing education, and demonstrating competence through certification. The rewards of mastery often go beyond these pathways. Mastery combines expert professional skills with leadership and interpersonal and organizational proficiency and often leads to the most coveted role of all, the role of mentoring.

The importance of demonstrating personal mastery of knowledge and skills can also be seen in the mounting evidence about how poor communication and lack of collaboration among health care professionals contributes to medical errors and staff turnover.[17,26,27] The Silence Kills Study revealed that 88% of physicians and 48% of nurses and other providers work with people who show poor clinical judgment, and unfortunately, fewer than 10% of them confront their colleagues about their concerns.[17] Avoiding crucial conversations about incompetent or inappropriate practices such as observing violation of infection standards or verbal abuse impairs patient safety and impedes quality care. It is essential that critical care nurses achieve the personal mastery of knowledge and skills that promote high-quality health care delivery and patient safety.

To emphasize the importance of validating clinical competency, on December 11, 2002, a compelling white paper on the benefits that specialty certification brings to the public, employers, and nurses was released.[28] The document, *Safeguarding the Patient and the Profession: The Value of Critical Care Nurse Certification*, raises awareness about the responsibility nurses have to honor and validate the public's trust for patient safety. The AACN believes that certification validates competency of knowledge, skills, and experience for quality patient care.[28] Consumers of nursing services must be able to recognize the contributions critical care nurses make to ensure high-quality and competent care to patients and families.

Barden (2003)[30] believes that there should be two types of critical care nurses at the bedside: (1) those who are certified, and (2) those who are in the process of becoming certified. Certification is a process of achieving the highest recognition of excellence. It is more than "another initial"[31]; it is a mark of excellence that can be referred to as "the Good Housekeeping Seal of Approval." Credentials on name badges, such as CCRN (critical care registered nurse), PCCN (progressive care certified nurse), or CCNS (clinical nurse specialist in acute and critical care), make personal mastery visible to the consumer and ensure public protection.

Since 1975, the AACN Certification Corporation has promoted and enhanced mastery of patient health and safety by certifying and recertifying nurses in the care of acute and critically ill patients.[32] Throughout the United States and Canada, nurses have received more than 410,000 certifications in 134 specialties. A total of 67 different certifying bodies granted these certifications and use at least 95 different credentials.[33] Currently, there are more than 40,000 certified critical care nurses with the credentials of CCRN, PCCN, CCNS, CMC (cardiac medicine certification), CSC (cardiac surgery certification), ACNPC (acute care nurse practitioner), or CNML (certified nurse manager and leader).[32]

Achieving a culture of excellence requires meaningful recognition of achievements such as mastery of certification.[20,28] Cary[33] reported four avenues through which certification status can be recognized: public acknowledgment, financial compensation, career advancement, and retention. In addition, findings revealed that there is a perception, especially among newly certified nurses, that certification gives autonomy, enhances collaboration with other health care providers, allows control over practice, and results in higher patient satisfaction ratings.[33]

A growing body of knowledge related to the value of specialty certification in critical care is being developed to determine the effect of certified nursing practice. Fitzpatrick et al[34] recently found AACN-certified nurses with higher empowerment scores were less likely to leave their current position, which demonstrates the importance of administrative support for certification. Kirchhoff and Dahl[5] found that 42% of CCUs provided public acknowledgment of certification and that 25% provided financial compensation with a certification bonus, whereas Ulrich and colleagues reported less support for an initial certification bonus (13.8%) and slightly higher support for certification recognition (45%) among critical care nurses.[35]

Passion

CASE STUDY

"Perpetual optimism is a force multiplier." (General Colin Powell, U.S. Army, retired, and former Secretary of State)[16]

Reflection on "Passion" for Critical Care Nursing Excellence

Excerpt from Dave Hanson RN, MSN, CCNS 2009 AACN President Speech: "Reclaiming our Priorities"

Throughout history it has been proven that one person, courageous and determined, can be the catalyst for great and lasting change. One person. Florence Nightingale. Rosa Parks. Martin Luther King, Jr. You know these names. You know the history they've made. You know their courageous

(*continued on page 105*)

acts and the priorities they reclaimed to change the course of the world we live in. Let me introduce names you may not know yet... Anne Marie McCarthy. One person. One nurse who has learned how to build good relationships with the family members of her patients, even the most difficult ones. When the staff nurse advisory team members were sharing their stories about how challenging some of their patients' families are to deal with, Anne Marie shared her approach: "I make it a point to meet my families at the door of the ICU. I introduce myself, being sure to shake their hands. Then I share with them the care plan for the day, what I think they can expect and how they can play a role in achieving the day's goals. I confidently assure them that I'm going to take really good care of their loved one. Since I started approaching families proactively like this, it seems as if they don't get in the way anymore. In fact, they often help me. Now, I actually ask for patients with the most difficult families." (p 6)[22]

Do you have a passion to reclaim your priorities and influence the care of patients and families by putting their care first regardless of the challenges? Do you have a passion to follow and/or lead evidence-based and relationship-centered initiatives at your organization? What are you passionate about to foster nursing excellence and make a difference in the lives of the acute and critically ill?

Just as a link is seen between values, vision, and mastery, passion is the essential thread to link together all the professional practice attributes for critical care nursing excellence (see Fig. 9-3). Passion involves enthusiastically striving for what is best for ourselves and those we serve. In *You Are the Leader You've Been Waiting for,* Klein described how to enjoy high performance and high fulfillment at work by being passionate about your calling or purpose.[36] He stated, "When your values, gifts, and calling operate in unison your work has a sense of inner congruence and outer effectiveness. You are clear about who you are and enjoy the ways in which you bring your gifts to life through your work." (p 119)[36] Similarly, others believe passion fuels results so that there is a "flywheel" effect, building in momentum with every step, action, decision, and turn.[36,37]

Weston et al[15] stated that "passion involves ardently striving for the best, even when repeated efforts seem tedious or appear exceedingly strenuous." (p 310) The truly passionate critical care nurse is not satisfied with providing less than the highest quality care possible to patients and families. Achieving this goal often requires going beyond an 8- or 12-hour shift. Acts of passion for critical care nursing excellence can be seen in bringing the latest research findings to the bedside, revising unit policy and procedure books with the most up-to-date procedures, and teaching coworkers the most effective therapies to produce the best outcomes for patients and families. Acts of passion for critical care nursing excellence can be seen when people take ownership to become engaged and transform work environments so that they are respectful, healing, and humane.

Passion can be felt in critical care nursing not only at the bedside but throughout the profession. Nursing leaders in critical care are partnering with interdisciplinary groups and talking to legislators to improve patient safety issues such as the workforce shortage, computerized physician and provider order entry, intensivist models of practice, evidence-based practice, and appropriate staffing. Being passionate about something requires time, energy, and commitment. The journey to excellence for healthy work environments started in 2001 for AACN because leadership was passionate about its mission of providing the highest-quality resources to maximize nurses' contribution to caring and improving the health care of critically ill patients and their families.[20]

In *Good to Great,* Collins describes why some organizations make the leap and sustain the leap from being a good organization to a great organization.[37] He challenges people and organizations to pick up their rocks and look at the "ugly squiggly things" underneath them, rather than putting the rocks back down and covering them up. Using resources from AACN, critical care nurses are picking up their rocks and addressing the "ugly squiggly things" in their workplace environments that impede quality patient care. Acts of passion for healthy work environments are taking place as the AACN standards for skilled communication, true collaboration, effective decision making, appropriate staffing, meaningful recognition, and authentic leadership are established.[20]

Action

CASE STUDY

"We are what we repeatedly do. Excellence then is not an act but a habit." (Aristotle)[16]

Reflection on "Action" for Critical Care Nursing Excellence

Excerpt from Mary Fran Tracey RN, PhD, CCNS, CCRN, FAAN 2007 AACN President Speech: "Empowered by Insight"

Our role is not simple. Our patients are really sick. We don't work on an assembly line doing exactly the same thing every day, knowing exactly what the result of our actions will be. Our patients count on us to maintain good judgment and the energy we need to be a part of solving our most complex problems. We can't fulfill this promise if we are allowing ourselves to work in constant crisis mode. It will take some doing to build in that much-needed slack time in our organizations. In the meantime, we must immediately begin employing a new strategy to maintain our good judgment every day. I call it stopping for a "thought pause."

A thought pause requires us to take a moment to *think* rather than *do*. To use our knowledge and the knowledge of our colleagues. To ask the right questions before we take action—especially high-risk actions like giving medications. To rise above the task for a moment.

We are not cogs in a machine. We are not a service that just comes with the bed. We as nurses are the creators and sharers of insights that ensure our patients' wishes are honored—that they are not in pain and that they are better for the care we provide. (pp 3,4)[23]

What action have you taken from a powerful insight to ensure safety becomes the norm and excellence the goal for managing the care of patients and their families? When was the last time you took time to think before taking action? What action will you take to foster nursing excellence and make a difference in the lives of the acute and critically ill?

Florence Nightingale once said, "One's feelings waste themselves in words, they ought all to be distilled into action which brings results." (p 44)[38] In other words, part of professionalism in critical care nursing is "to walk the talk" for excellence. As values, vision, mastery, and passion for critical care nursing excellence build, the attribute of action (see Fig. 9-3) becomes another essential pillar for the framework and one that resonates well for tangible results in critical care. Recognizing that so many vulnerable patients' lives are at risk and the invaluable contributions that nurses make, the AACN leadership decided it was time to act deliberately and definitively. In 2003, AACN launched the Beacon Award for Critical Care Excellence, an award specifically designed to recognize the leading critical care units in the United States.[39] Currently, more than 315 critical care units have been recognized for demonstrating high-quality standards, exceptional care of patients, families, and healthy work environments. Of those units, 41 have received the Beacon Award more than twice.[40]

When the Beacon Award was first launched, a *beacon* was defined as "a source of light, an inspiration, or signal of guidance" with the belief that "every critical care unit could be a Beacon unit."[40] Since that time, the 42-item Beacon application has been modified to a new application with 38 items, and any hospital unit that has high acuity and critically ill patients and meets evidence-based standards of excellence and patient safety is now eligible to apply. Although the new application includes three levels (bronze, silver, and gold) for units to chart their journey and receive a 3-year designation, the Beacon application continues to address innovation, excellence, or both in six categories: recruitment and retention; education, training, and mentoring; evidence-based practice and research; patient outcomes; creating and promoting healing environments; and leadership and organizational ethics. In addition, the Beacon Award provides a mechanism for individual and collective critical care units to measure progress against evidence-based initiatives and national criteria for performance, learn and refine their processes and systems, and be recognized for their achievements.[39] What bolder action or stronger message can be conveyed to the public and the patients whom critical care nurses serve than to validate excellence in practice?

Seeking opportunities and partnerships to further extend action to ensure optimal health outcomes for individuals experiencing an acute and life-threatening illness requires a relentless and fearless voice by critical care nurses. These nurses have been using their innate gift of inquiry for decades to tackle patient care issues. However, Nightingale is perhaps nursing's most famous leader who first used research to change practice.[38] Even though she lacked the theoretical bases that are known today, she had a core set of values, a vision, the mastery, and a passion to improve England's hospital care in the mid-19th century. Today, critical care nurses are seeing "sacred cows" that were once revered being thrown out of practice. For example, nursing research has well demonstrated that using dye in enteral feeding, restricting family presence during cardiopulmonary resuscitation, and restricting visitation hours in ICUs need to be eliminated from practice.

Even though new models of nursing education are evolving, it does not take a doctorate-prepared critical care nurse to raise questions and put a plan in place for collecting, analyzing, and reporting patient and family outcomes. Outcome-based practice is the responsibility of all nurses, whether one is actually doing the research, disseminating the research to the bedside, or reporting the research findings. In addition, it is important for all nurses to celebrate and showcase high-quality nursing outcomes, no matter what nursing specialty provided the body of knowledge. Focusing on quality indicators and performance improvement in acute and critical care settings as daily practice is a bold and powerful action for improving patient care.

This is the commitment and voice that needs to be heard about critical care nursing. Practice alerts from AACN are another example of efforts to prevent and minimize infection, reduce complications from critical illness, promote patient safety, and establish best practices. First launched in 2004, the AACN practice alerts are succinct dynamic directives that are well supported with current authoritative evidence to ensure best practice. There are more than a dozen practice alerts that (1) bridge the gap between practice and research, (2) provide guidance, (3) standardize care, and (4) identify and inform new trends and provide information about them.[41] A few recent practice alerts include oral care in the critically ill, noninvasive blood pressure monitoring, and severe sepsis.[41] Because critical care nurses have a great opportunity to promote the science of nursing through the AACN practice alerts, it is helpful to remember that "the public cannot protect a social resource that it does not know and understand; only nurses can give the public the knowledge and understanding necessary to protect human care giving." (p 42)[2]

Balance

CASE STUDY

"Balance isn't either/or, it's AND." (Steven Covey)[16]

Reflection on "Balance" for Critical Care Nursing Excellence

Excerpt from Caryl Goodyear-Bruch RN, PhD, CCRN 2009 AACN President Speech: "With Confidence"

With purpose come all kinds of possibilities for taking confident actions that matter to our patients. CCRN Marie Lasater recently wrote a short article for the *American Journal of Nursing* describing a difficult day on her step down unit. She vividly described the patients, the realities of our health care system and of her decision to prioritize being with a dying patient who was alone, without family. She wrote: "I have six patients this morning. One with a self-inflicted gunshot wound to the head who's expected to die on my shift. Two with recent strokes, one with a lumbar drain after back surgery, and one with a possible prion disease. Because of a sick call, we have only one patient care technician for 20 patients. I will be doing total patient care... As I'm paging the physician [regarding another patient]... I get a page that the oxygen saturation level of my dying patient is dropping. I race to don protective gear and help my stroke patient to the bedside commode and back to bed. I then check on my dying patient. I hold his hand for a moment. As I'm telling him he is in a safe place, that his family is safe, and that people care about him, I'm paged again. I'm getting another admission. I have been at work for an hour and 15 minutes, but I haven't even begun to give scheduled medications. We were recently told that we will be 'counseled' if we clock out late from our shifts. I tell the unit secretary I will be in my dying patient's room and ask the other nurses to handle my pages. I hold my patient's hand and keep repeating, 'I'm here'." (pp 3,4)[24]

How confident are you to put balance in your professional and personal life for renewal? When was the last time you had full presence with a dying patient in the midst of a chaotic workday? How will you start balancing your life to foster nursing excellence and make a difference in the lives of the acute and critically ill?

Balance is the final component of the professional practice model for critical care nursing excellence (see Fig. 9-3, p. 102). Balance can bring renewal to the spirit and allow for more moments of "full presence," which often breaks down in our busy professional and personal lives. Taking the time to care for one's self is essential to keeping the body and mind in balance. Otherwise, it can be difficult to keep perspectives clear. Today, nurses are increasingly blurring the lines between home and work and work and leisure. The lines of communication are continuously open because of the proliferation of pagers, cell phones, facsimile machines, text messages, e-mail, and voicemail. It is time to say "no" to being "supernurse" and "supermom" or "superdad" and find the time to take care of oneself. Nurses do not do their patients, their families, or themselves any good if they consistently place the needs of others above their own. Critical care nurses listen to patients and families 24 hours a day, 7 days a week. They must also have time to listen to their own minds and hearts and those who love them most.

In *You Are the Leader You've Been Waiting for,* Klein stresses the importance of letting go.[42] He believes letting go of the old can make space for something new. It is during a time of transformation that he believes one should not act but be still.[42] Nurses cannot stop the beeps, alarms, and phone calls at work, but when the shift is over, it is time to "let go." As difficult as it may be, it is time for nurses to let go of the complex, vulnerable, and unstable patients who were in their hands during their shift. It is a time to be "still;" nurses may turn the television off and read to the children, take a leisurely walk with a pet, or sit quietly listening to the sounds of life. The minds and hearts of nurses need time to renew and be recharged for the next day of taking care of critically ill patients and their families. When nurses are balanced, it is easier not to give in to the cynicism and frustration that abound in the workplace or home. To be truly engaged and energized requires nurturing oneself first and then empowering others to do the same. Look around your unit today and ask yourself some questions. Who are the critical care nurses who reach out to others the most? Who are the critical care nurses who smile the most? Who are the critical care nurses who say "thank you" and give compliments the most? You may find that your answer is with nurses who have discovered how to make balance a priority in their professional and personal lives.

BOX 9-3 Closing Thoughts on Critical Care Nursing Excellence

REMEMBER… Ready or not, someday it will all come to an end.
There will be no more sunrises, no shift work, no change of report.
All the things you valued, whether treasured or forgotten, will pass to someone else.
It will not matter what you owned or what you were owed.
Your challenges, frustrations, and disappointments will finally disappear.
So too your hopes, ambitions, and plans.
What will matter is not your success, but your significance.
What will matter is not what you learned, but what you taught.
What will matter is every act of integrity, compassion, courage, or sacrifice that enriched, empowered, or encouraged others to emulate your example of critical care nursing excellence.
Living a life that matters doesn't happen by accident.
It's not a matter of circumstance but of choice.
Live your contribution and choose to live a life that matters.
Become engaged and transform your practice for critical care nursing excellence

Modified from Josephson M: What will matter. Retrieved June 1, 2010, from http://www.charactercounts.org

▲ Conclusion

In today's fast-paced critical care environment, finding the time for professional growth can be challenging yet rewarding (Box 9-3). Building a professional practice of excellence requires a passion to profoundly affect the lives of those who trust critical care nurses most: complex, unstable, and vulnerable patients and their families. At the same time, it requires advancing the critical care nursing profession through a healthy work environment that is patient centered, collaborative, interdisciplinary, and evidence based. The desire and commitment for critical care nursing excellence requires self-reflection about the values, vision, mastery, passion, action, and balance in one's practice (see Fig. 9-3, p. 102). Critically ill patients and their families expect and deserve nothing but the best care.

Building a professional practice model of excellence can give critical care nurses the confidence to use their bold voice and presence to make significant contributions for improving the delivery of care to the patients and families who have entered a chaotic and frightening world of illness, trauma, and pain. Even in the fastest-paced critical care environments of today, finding the time for professional growth is challenging but, as evidence demonstrates, essential. For the profession of critical care nursing to advance, nurses must acquire the necessary clinical experience and competencies to provide best practice models of care for critically ill patients and their families. Whether it is participating on a patient safety hospital committee or recruiting youth to critical care nursing, endless opportunities exist for building a professional practice of excellence. The days of landmark studies reporting that nursing is a silent and unknown profession[43,44] will soon come to an end as more bold and committed voices are heard about excellence in critical care nursing practice.

▲ Clinical Applicability Challenges

SHORT ANSWER QUESTIONS

1. How can nurses speak up boldly about a model of practice for critical care nursing excellence that optimizes the contributions they make in the lives of acute and chronically ill populations, families, and systems?
2. How would patients and families identify an inner core value of clinical competence among critical care nurses at the bedside?
3. What would a potential new hire critical care nurse think about a health care organization that requires name badges to carry all nursing credentials including certification?

References

1. Kalisch P, Kalisch B: Working together for nursing. Focus Crit Care 10:12–14, 1983
2. Buresh B, Gordon S: From Silence to Voice: What Nurses Know and Must Communicate to the Public. Ottawa, Canada, Canadian Nurses Association, 2000
3. American Association of Critical-Care Nurses: 2010 Membership Demographics. Retrieved May 15, 2010, from http://www.aacn.org/WD/Memberships/Docs/membdemographics.pdf
4. Division of Nursing, Bureau of Health Professionals in the Health Resources and Services Administration, U.S. Department of Health and Human Services: Initial Findings from the 2008 National Sample Survey of Registered Nurses. Retrieved May 20, 2010, from http://bhpr.hrsa.gov/healthworkforce/rnsurvey/initialfindings2008.pdf
5. Kirchhoff K, Dahl N: American Association of Critical-Care Nurses' national survey of facilities and units providing critical care. Am J Crit Care 15(1):13–27, 2006
6. Joel L: Kelly's Dimensions of Professional Nursing. New York, NY: Macmillan, 2003
7. Styles M: Professionalists, all. J Contin Educ Nurs 31(2):88–89, 2000
8. Stanley JM, Gannon J, Gabuat J, et al: The clinical nurse leader: A catalyst for improving quality and patient safety. J Nurs Manag 16: 614–622, 2008
9. Dracup A, Cronenwett L, Meleis A, et al: Reflections on the doctorate of nursing practice. Nurs Outlook 53(6):177–182, 2005
10. Flexner A: A Medical Education in the United States and Canada: A Report to the Carnegie Foundation for the Advancement of Teaching. Bethesda, MD, Science & Health Publications, 1910
11. Holl R: Characteristics of the registered nurse and professional beliefs and decision making. Crit Care Nurs Q 17:60–66, 1994
12. Heath J, Andrews J, Graham-Garcia J: Assessment of professional development of critical care nurses: A descriptive study. Am J Crit Care 10(1):17–22, 2001
13. Manojlovich M: Predictors of professional nursing practice behaviors in hospital settings. Nurs Res 54(1):41–47, 2005
14. Wynd C: Current factors contributing to professionalism in nursing. J Prof Nurs 19(5):251–261, 2003
15. Weston M, Buchda V, Bergstrom D: Creating excellence in practice. In Stanley J (ed): Advanced Practice Nursing: Emphasizing Common Roles. Philadelphia, PA: FA Davis, 2005, pp 395–411
16. Famous Quotations Network. Retrieved May 1, 2010, from http://www.famous-quotations.com
17. Maxfield D, Grenny J, McMillan R, et al: Silence kills: The seven crucial conversations for healthcare. VitalSmarts L.C. 2005. Retrieved May 1, 2010, from http://www.silencekills.com
18. American Association of Critical-Care Nurses and VitalSmarts Press Release: New study finds U.S. hospitals must improve workplace communication to reduce medical errors, enhance quality of care. Washington, DC, January 26, 2005. Retrieved May 10, 2010, from http://www.silencekills.com/UPDL/PressRelease.pdf
19. Gallup Organization: Nurses remain at top of honesty and ethics poll. The Gallup Organization, November 22, 2009. Retrieved June 1, 2010, from http://www.gallup.com/poll/1654/Honesty-Ethics-Professions.aspx
20. American Association of Critical-Care Nurses: AACN Standards for Establishing and Sustaining Healthy Work Environments: A Journey to Excellence. Aliso Viejo, CA, AACN, 2005
21. Tracey MF: 2007 Presidential Speech: Empowered by Insight. Retrieved June 1, 2010, from http://www.aacn.org/DM/NTI2007/pages/images/PresidentSpeech-07.pdf
22. Hanson D: 2008 Presidential Speech: Reclaiming our Priorities. Retrieved June 1, 2010, from http://www.aacn.org/WD/NTI2008/Docs/presidentSpeech08.pdf
23. Goodyear-Bruch C: 2009 Presidential Speech: With confidence. Retrieved June 1, 2010, from http://www.aacn.org/wd/practice/content/president-theme09.pcms?menu=practice
24. Hammer B: Act with intention and speak with a bold voice. Bold Voices: Acute and Critical Care Nurses Making their Optimal Contribution. 1(11):22, 2009
25. American Association of Critical-Care Nurses: Core values. Retrieved May 12, 2010, from http://www.aacn.org/wd/memberships/content/mission_vision_values_ethics.pcms?menu=aboutus
26. Institute of Medicine: Crossing the quality chasm: A new health system for the 21st century. Washington, DC: National Academy Press, 2001
27. Joint Commission: Root causes of sentinel events. Retrieved May 1, 2010, from http://www.jointcommission.org/sentinelevents/statistics/
28. American Association of Critical-Care Nurses and AACN Certification Corporation: Safeguarding the patient and the profession: The value of critical care nurse certification. Am J Crit Care 12(2):154–164, 2003
29. Studer Q: Hardwiring Excellence: Purpose, Worthwhile Work, Making a Difference. Gulf Breeze, FL: Fire Starter Publishing, 2004
30. Barden C: Certification: Good for whom? AACN News 20(2):2, 2003
31. Mason D: What's in a letter. Am J Nurs 101(1):7, 2001
32. American Association of Critical-Care Nurses Certification Corporation: History of AACN Certification Corporation. Retrieved May 15, 2010, from http://www.aacn.org/wd/certifications/content/aboutus.pcms?menu=certification
33. Cary AH: Certified registered nurses: Results of the study of the certified workforce. Am J Nurs 101(1):44–52, 2001
34. Fitzpatrick JJ, Campos TM, Graham G, et al: Certification, empowerment, and intent to leave current position and the profession among critical care nurses. Am J Crit Care 19(3):218–226, 2010

35. Ulrich B, Lavandero R, Hart K, et al: Critical care nurses' work environments: A baseline status report. Crit Care Nurse 26(5): 646–657, 2006
36. Klein E: You Are the Leader You've Been Waiting for: Enjoying High Performance and High Fulfillment at Work. Encinitas, CA: Wisdom Heart Press, 2006
37. Collins J: Good to Great. New York, NY: Harper Collins, 2001
38. Nightingale F: Notes on Nursing: What It Is, and What It Is Not. London, UK: Harrison and Sons, 1859
39. American Association of Critical-Care Nurses. Beacon Program Overview. Retrieved May 1, 2010, from http://www.aacn.org/wd/beaconapps/content/about.pcms?menu=beaconapps&lastmenu=divheader_program_overview
40. American Association of Critical-Care Nurses: Beacon Award for Critical Care Excellence-Information and Statistics. Retrieved June 1, 2010, from http://www.aacn.org/wd/beaconapps/content/facts.pcms?pid=1&&menu=beaconapps
41. American Association of Critical-Care Nurses: Practice Alerts. Retrieved May 10, 2010, from http://www.aacn.org/wd/practice/content/practicealerts.pcms?menu=practice
42. American Association of Critical-Care Nurses: AACN Circle of Excellence 2006 Award Recipients. Retrieved August 1, 2006, from http://www.aacn.org/AACN/Memship.nsf/ vwdoc/COE2006Rec
43. Buresh B, Gordon S, Bell N: Who counts in news coverage of health care. Nurs Outlook 39(5):204–208, 1991
44. Sigma Theta Tau International: The Woodhull Study on Nursing and the Media: Health Care's Invisible Partner. Indianapolis, IN, Author, 1998

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

PART 3

Special Populations in Critical Care

10

The Critically Ill Pediatric Patient

Patricia A. Moloney-Harmon

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Analyze anatomical and physiological differences in the infant and child that necessitate the modification of physical assessment parameters and intervention techniques.
2. Describe special considerations in ventilatory management and medication administration for the critically ill child.
3. Evaluate pain assessment tools that can be used for the critically ill child.
4. Examine important aspects of interaction with the critically ill child and family that will enhance interventions.

Many critical care clinicians feel ill equipped to care for children seen in adult intensive care units (ICUs), emergency departments, procedural suites, and recovery rooms. To facilitate smooth and optimal care of the critically ill child, it is wise to adopt a framework for the modification of the adult critical care practice to include the pediatric patient. A comprehensive framework is beyond the scope of this chapter, but readers are referred to the PEDS framework, discussed in more detail elsewhere.[1] This chapter highlights prominent anatomical and physiological differences and related implications, equipment selection, recognition of the decompensating child, and unique challenges in caring for the pediatric patient in a critical care environment.

▲ Prominent Anatomical and Physiological Differences and Implications

Vital Signs

Infants and young children have an age-appropriate, but higher, heart rate and respiratory rate than adults. The higher heart and respiratory rates assist in meeting the need for a higher cardiac output, despite a smaller stroke volume and a higher basal metabolic rate. Blood pressure in children is lower than that of adults. Vital signs (Table 10-1), although important parameters, should not be evaluated in isolation but rather in a trending fashion.

Tachycardia is a nonspecific response to a variety of entities, such as anxiety, fever, shock, and hypoxemia. Although the child is predisposed to bradycardia, tolerance is poor. Persistent bradycardia produces significant changes in perfusion because cardiac output is heart rate dependent. Bradycardia is most often caused by hypoxemia, but any vagal stimuli, such as suctioning, nasogastric tube insertion, and defecation, may precipitate an event.

As for respiratory rate, an infant or child increases his or her respiratory rate to compensate for an increased oxygen demand. Tachypnea is often the first sign of respiratory distress. A slow respiratory rate in a sick child often indicates impending respiratory arrest. Associated conditions, such as fever and seizure activity, which further increase the metabolic rate, also increase oxygen requirements. These conditions can cause rapid deterioration in an already compromised child.

Table 10-1 Pediatric Vital Signs

Age	Heart Rate (beats/min)	Respirations (breaths/min)	Systolic Blood Pressure (mm Hg)
Newborn	100–160	30–60	50–70
1–6 wk	100–160	30–60	70–95
6 mo	90–120	25–40	80–100
1 y	90–120	20–30	80–100
3 y	80–120	20–30	80–110
6 y	70–110	18–25	80–110
10 y	60–90	15–20	90–120
14 y	60–90	15–20	90–130

Unlike the adult, the child's blood pressure is the last parameter to fall in the face of shock. Children can compensate for up to a 25% blood loss before the systolic blood pressure falls. A normal blood pressure should never discourage interventions for the child showing signs of circulatory failure. The pulse pressure is often a more reliable indicator for assessing the adequacy of perfusion. Hypertension is uncommon unless the child has renal disease.

Neurological System

Brain growth occurs at a rapid rate during the first few years of life. Because brain growth is rapid during this time, measurement of head circumference is important in the child until 2 years of age. The circumference of the child's head is related to intracranial volume and estimates the rate of brain growth.

The child's cranial sutures are not completely fused until 18 to 24 months of age. The posterior fontanelle closes by 3 months of age, and the anterior fontanelle closes by 9 to 18 months of age. The fontanelles provide a useful assessment tool in the infant. The characteristics of the fontanelles can be used to assess hydration status or the presence of increased intracranial pressure (ICP). Bulging fontanelles may indicate increased ICP or fluid overload. Sunken fontanelles may be seen with fluid deficit.

Like adults, infants and children have protective reflexes (eg, the cough and gag reflexes). There are also several newborn reflexes (ie, the Moro, rooting, grasp, and Babinski reflexes), which differ from adult reflexes. For example, the Babinski reflex is present until 9 to 12 months of age or until the child starts walking. A positive Babinski reflex response (fanning of the toes and dorsiflexion of the big toe when the lateral aspect of the sole of the foot is stroked) is expected in an infant, yet is considered an abnormal finding in an older child or adult. In-depth discussion of these reflexes is beyond the scope of this chapter; the reader is referred to a developmental anatomy text for further information.

An infant's or child's mental status is assessed the same way as an adult's, by noting the level of consciousness, interaction with the environment, and appropriateness of behavior for age. Level of consciousness is assessed by noting whether the child is arousable and oriented. This can be done by observing for spontaneous arousability or by providing verbal, tactile, or noxious stimuli. Even though the assessment is the same, the assessment techniques must be age appropriate. Specific techniques are provided in the section in this chapter on interaction. An important difference to note when interacting with the child is paradoxical irritability (ie, the inability of the child to be calmed with normal comfort measures, such as cuddling). Paradoxical irritability, when present with meningeal irritability, nuchal rigidity, and positive Brudzinski's and Kernig's signs, may indicate meningitis.

Infants and young children are at high risk for ineffective thermoregulation, resulting in physiological instability from a variety of maturational and environmental factors. Closely monitoring body temperature and providing a temperature-controlled environment help manage temperature regulation. The temperature is measured at regular intervals, and external factors affecting body temperature should be controlled.

Cardiovascular System

Decreased perfusion to the skin is an early and reliable sign of shock. Because a child's skin is thinner than an adult's, skin characteristics change easily and rapidly with changes in perfusion. Skin color, texture, and temperature and capillary refill are of great significance during assessment of the child. Before assessing the skin, it is important to note the room temperature because some findings may be a normal response to the environment (such as mottling in a drafty operating room). Mottling in a bundled infant or warm environment is reason for further investigation. The nurse assesses skin temperature and the line of demarcation between extremity coolness and body warmth. Coolness or the progression of coolness toward the trunk may be a sign of diminishing perfusion.

Peripheral cyanosis is normal in newborns but abnormal in young children and adults. Central cyanosis (circumoral) is always abnormal. Capillary refill time is normally recorded in seconds rather than as "brisk, normal, or slow" and normally is no longer than 2 seconds. Estimated blood volume varies with age; despite a higher volume per kilogram of body weight in children, the overall total circulating volume is small. A small amount of blood loss can be significant in a child.

Respiratory System

The infant's or child's large head (in proportion to body size); weak, underdeveloped neck muscles; and lack of cartilaginous support to the airway lead to an easily compressible or obstructed airway. The nurse must avoid overextending or overflexing the neck because the airways are easily collapsible. Head and neck position alone can facilitate a patent airway. Ideal positioning for the decompensating child is in a neutral ("sniffing") position and can be accomplished by placing a small roll horizontally behind the shoulders (Fig. 10-1).

Infants, until 6 months of age, are obligate nose-breathers, so any obstruction of nasal passages can produce significant airway compromise and respiratory distress. Secretions, edema, inflammation, poorly taped nasogastric tubes, or occluded nasal cannulas can cause obstructed

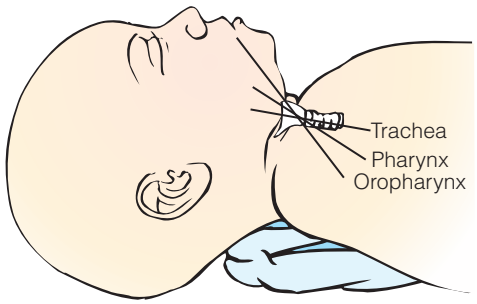


FIGURE 10-1 ▲ The neutral ("sniffing") position can improve airflow in a decompensating child by aligning the oropharynx (O), pharynx (P), and trachea (T) with the mouth.

nasal passages in an infant. The infant's and young child's airways are smaller in diameter and in length, thus requiring smaller artificial airways. Airway compromise can be caused by the slightest amount of inflammation or edema of the natural airway or from a mucus plug in either the natural or artificial airway. The narrowest part of the child's airway (until approximately 8 years of age) is at the level of the cricoid ring, as opposed to the glottic opening in the adult.

The young child's thin, compliant chest wall allows for easy assessment of air entry, which is assessed by observing the rise and fall of the child's chest with adequate ventilatory efforts. Unequal chest movement may indicate the development of a pneumothorax or atelectasis but also may indicate endotracheal tube obstruction or displacement into the right mainstem bronchus. The child's flexible rib cage and poorly developed intercostal muscles offer little stability to the chest wall; therefore, suprasternal, sternal, intercostal, and subcostal retractions may be seen during respiratory distress. The presence and location of retractions should be noted. Accessory muscles also are poorly developed, so an infant or child may use the abdominal muscles to assist with breathing. This gives the appearance of "seesaw" breathing, a paradoxical movement of the chest and abdomen. Seesaw breathing becomes more exaggerated with respiratory distress. As in the adult, the major muscle of respiration is the diaphragm. However, the child is more diaphragm dependent.

Because of the thin chest wall, breath sounds are more audible than in the adult. In addition, obstructed airways often produce sounds that are easily heard during assessment. The nurse listens for expiratory grunting, inspiratory and expiratory stridor, and wheezing. Expiratory grunting is a sound produced in an attempt to increase physiological positive end-expiratory pressure to prevent small airways and alveoli from collapsing. The infant's and child's thin chest wall may allow breath sounds to be heard over an area of pathology when sounds are actually being referred from another area of the lung. The nurse listens for changes in the breath sounds as well as for their presence or absence.

Gastrointestinal System

Children normally have a protuberant abdomen; however, there are numerous causes of abnormal abdominal distention. A nasogastric or orogastric tube should be inserted early rather than later in a critically ill child to minimize the risk for distention. Abdominal distention can interfere with respiratory excursion and may even cause respiratory arrest. Active removal of air with a syringe may be necessary if distention is not relieved by putting the tube to straight drainage. In addition, the abdominal girth is measured every shift or more often if there is a concern about abdominal distention.

Stomach capacity varies with the age of the child. A newborn's stomach capacity is 90 mL, a 1-month-old's is 150 mL, a 12-month-old's is 360 mL, and an adult's is 2,000 to 3,000 mL. Because stomach capacity is smaller, care is taken when formula and other fluids are instilled into the abdomen. Bolus feedings are of an appropriate amount, consistent with the child's stomach capacity.

The infant and young child have a gastric emptying time of 2.5 to 3 hours, which increases to 3 to 6 hours in the older child. An appropriate amount of time to allow for absorption of formula is taken into account when measuring residuals. If the child is receiving chest physiotherapy, the amount of time between therapy and feeding is considered, or the gastric contents are checked to avoid problems with reflux and aspiration.

Renal System

Infants have less ability to concentrate urine and therefore have a normal urine output of 2 mL/kg/h. For children and adolescents, normal urine output is 1 mL/kg/h and 0.5 mL/kg/h, respectively. Because of the infant's limited ability to concentrate urine, a low specific gravity does not necessarily mean that the infant is adequately hydrated. The immaturity of the child's kidney means that the child may not process fluid as efficiently as the adult and is less able to handle sudden large amounts of fluid, leading to fluid overload.

Infants and young children have a larger body surface area in relation to body weight. Maintenance fluid requirements are determined based on body weight (Table 10-2). Children have a higher percentage of total body water, most of which is composed of extracellular fluid (ECF), compared with adults. The ECF makes up 50% of the body weight in infants but 20% in adults. In addition, children have a higher insensible water loss because of a higher basal metabolic rate, higher respiratory rate, and larger body surface area. The child's higher percentage of total-body water and higher insensible water loss increase the risk for dehydration. Sudden weight

Table 10-2 Calculation of Maintenance Fluid

Body Weight (kg)	Fluid Requirements per Day	Fluid Requirements per Hour
Less than 10	100 mL/kg	4 mL/kg
10–20	1,000 mL + 50 mL/kg for each kg above 10	2 mL/kg for each kg above 10
More than 20	1,500 mL + 20 mL/kg for each kg above 20	1 mL/kg for each kg above 20

From Roberts KE: Fluid and electrolyte regulation. In Curley MAQ, Moloney-Harmon PA (eds): Critical Care Nursing of Infants and Children, Philadelphia: WB Saunders Co, 2001, pp 369–392, with permission of Elsevier Science.

Table 10-3 Clinical Assessment of Severity of Dehydration

Patient	Mild Dehydration	Moderate Dehydration	Severe Dehydration
Infant	5%	10%	15%
Adolescent	3%	6%	9%
Infants and young children	Thirsty, alert, restless	Thirsty, restless, or lethargic but irritable to touch or drowsy	Drowsy, limp, cold, sweaty, cyanotic extremities, may be comatose
Older children and adults	Thirsty, alert, restless	Thirsty, alert, postural hypotension	Usually conscious, apprehensive, cold, sweaty, cyanotic extremities, wrinkled skin of fingers and toes, muscle cramps
Signs and Symptoms			
Tachycardia	Absent	Present	Present
Palpable pulses	Present	Present (weak)	Decreased
Blood pressure	Normal	Orthostatic hypotension	Hypotension
Cutaneous perfusion	Normal	Normal	Reduced and mottled
Skin turgor	Normal	Slight reduction	Reduced
Fontanelle	Normal	Sunken	Very sunken
Eyes	Normal	Slightly depressed	Sunken
Tears	Present	Present or Absent	Absent
Mucous membranes	Moist	Dry	Very dry
Respirations	Normal	Deep, may be rapid	Deep and rapid
Urine output	Normal	Oliguria	Anuria and severe oliguria
Estimated fluid deficit (mL/kg)	30–50	60–90	≥100

Adapted from Greenbaum LA. Fluids and electrolytes. In Kliegman RM, Jenson HB, Marcdante KJ, et al (eds): Nelson Essential of Pediatrics, 5th ed. St. Louis: Elsevier Health Sciences, 2006, p 162, with permission from Elsevier Science.

loss or gain may indicate fluid imbalance. Children should be weighed daily at the same time using the same scale.

Signs of dehydration include dry mucous membranes, decreased urine output, increased urine concentration, sunken fontanelles and eyes, and poor skin turgor (Table 10-3). The severity of dehydration varies with the degree of dehydration and the child's fluid and electrolyte status. Circulatory compromise accompanies severe dehydration. Treating a child's dehydration in an adult ICU requires pediatric consultation. Fluid overload is manifested by bulging fontanelles, taut skin, edema (usually periorbital and sacral), hepatomegaly, and other signs of congestive heart failure.

Endocrine System

Infants and young children have smaller glycogen stores and increased glucose demand because of their larger brain-to-body size ratio. The smaller stores and increased demand predispose infants and young children to the development of hypoglycemia. Blood glucose levels are closely monitored, especially when the infant or child is not permitted to have anything by mouth and numerous adjustments are being made to nutritional support.

Immune System

Immunological differences in infants and small children may predispose them to infection. The skin of newborns is thinner; therefore, it provides less of a barrier to outside pathogens. Because infants and young children have fewer stored neutrophils, they are less able to repeatedly replenish white blood cells in the face of an overwhelming infection. The complement levels are lower, which affects the chemotactic activity of phagocytes and the opsonization of bacteria. There is also a relative deficiency of immunoglobulins, making infants and young children more susceptible to infections caused by viruses, *Candida* species, and acute inflammatory bacteria. In addition, infants may not demonstrate fever and leukocytosis in response to an infection. It is important to observe for subtle signs, such as changes in feeding behaviors, altered glucose metabolism, and hypothermia.

Integumentary System

Expected differences in the skin, hair, nails, and glands depend on the age of the child. Infants and children, without exposure to the sun or wind, are expected to have smooth-textured skin without coarse adult terminal hair. Infants, up to about 14 days of age, may be covered with lanugo, a fine, silky-textured hair. Infants also have less developed hypodermal fat and, as a result, are at risk for hypothermia. The sweat glands do not begin to function until 1 month of age and are not fully functional until adolescence.

In the young child, the most noticeable variation may be that of bruising as the child increases activity and play becomes more aggressive. It is very important to attend to

bruising seen in the child because it may be associated with abusive situations. The nurse notes the location and color changes of bruising indicating the stage of healing. Bruising is more common and not unexpected on the lower legs and the face. Bruising on the upper arms, buttocks, and abdomen occurs less often and may indicate abuse.

In the adolescent, the sweat glands and sebaceous glands become fully functional. The adolescent may be expected to experience body odor, increasing axillary perspiration, and acne. The development of axillary and pubic hair is expected related to the increasing levels of circulating androgen levels in both male and female adolescents.

▲ Selected Pediatric Challenges

Ventilatory Issues

The most common cause of cardiopulmonary arrest in children is respiratory in nature. This fact mandates that respiratory distress and failure be recognized early and that airway management interventions be immediate (Table 10-4). Signs of respiratory decompensation include diminished level of consciousness, tachypnea, minimal or no chest movement with respiratory effort, evidence of labored respirations with retractions, seesaw breathing, minimal or no air exchange noted on auscultation, and the presence of nasal flaring, grunting, stridor, or wheezing.

The initial intervention for respiratory decompensation is positioning the child to open the airway. If the child does not respond to position alone, manual ventilation with 100% oxygen using a bag-mask device is initiated. There are several sizes of pediatric manual resuscitation bags; the correct size is determined by noting the child's tidal volume and deciding whether the bag is capable of delivering 1.5 times the child's tidal volume. Even though a pressure manometer may assist in minimizing pressure, the true indicator of delivery of an adequate tidal volume is a clinical one. The adequate amount of tidal volume delivered during a manual resuscitation breath is the amount that causes rise and fall of the child's chest.

If bag-mask ventilation is not successful in restoring the child's ventilatory status, endotracheal intubation is required. Numerous sizes of endotracheal tubes are available for infants and children. To estimate the correct size of endotracheal tube, the size of the child's little finger or the following formula can be used:

$$\text{Internal diameter} = (16 + \text{age in years}) / 4$$

A cuffed tube can be used safely in the in-hospital setting.[2] For cuffed endotracheal tubes, the formula used to estimate the internal diameter is as follows:

$$\text{Internal diameter} = (16 + \text{age in years}) / 4 + 3$$

Because these are both estimations of endotracheal tube size, tubes one-half size smaller and larger should be available for immediate use. Table 10-5 on page 117 provides information regarding endotracheal tube sizes and other equipment issues.

Monitoring the patient during intubation is critical to assess for desaturation or bradycardia. Once the child is intubated, observation of chest movement and auscultation of the lungs help determine correct placement. A radiograph is used to confirm proper placement. When placement is confirmed, the tube is securely taped to avoid accidental displacement. In addition, soft restraints should be used to prevent the child from removing the tube. Adequate sedation and analgesia are provided to increase the child's comfort and manage anxiety during intubation.

Medication Administration

Because a child may differ in weight significantly from the average child in the associated age group, medications are prescribed on a microgram, milligram, or milliequivalent per kilogram of body weight basis rather than on a standard dose according to age. Confirming the weight (in kilograms) that is being used to determine drug dosages is important. This same weight should be used during the child's entire hospitalization unless there is a significant change in the child's weight. Because pediatric dosages may be unfamiliar to the adult clinician, precalculated emergency drug sheets are helpful. The emergency drug sheet should include the recommended resuscitation medication dosages, medication concentration, and final medication dose and volume the individual child is to receive. The recommended dosages should reflect the American Heart Association's Pediatric Advanced Life Support standards.

An important recommendation for medication administration in the pediatric patient is the single-dose system. The single-dose system involves preparing one syringe to contain only the prescribed medication dose. The syringe should be properly labeled with the drug name and dose. The nurse administers the entire volume of the syringe to ensure that the prescribed dose has been given. The single-dose system prevents overmedication or undermedication of the child.

Medication errors have received increased attention since the publication of the 2000 Institute of Medicine report, "To Err Is Human." The most common reason for harm to pediatric patients, medication errors result in a higher risk for death.[3] The prescribing phase is associated with the most errors (dosing errors), and the administration phase results in the second most errors.[4] Nurses are the last potential barrier between an occurrence and an adverse outcome; they are most likely to intercept the error. Preventing medication errors is especially important in children because there is a much smaller margin for error in this patient population. Risk reduction strategies such as ensuring staff competency and computerized physician order entry should be in place as best practice. All providers must use vigilance in ordering and administering medications to a pediatric patient.[5]

Pain Management

Because of the nature of the environment and associated procedures, the critically ill child is at high risk for pain. The first

Table 10-4 Quick Examination of a Healthy Versus Decompensating Child

Assessment	Healthy Child	Decompensating Child
Airway		
Patency	Child requires no interventions; child verbalizes and is able to swallow, cough, gag.	Child self-positions and requires interventions, such as head positioning, suctioning, adjunct airways. Unmaintainable airway requires intubation.
Breathing		
Respiratory rate	Breathing is within age-appropriate limits.	Breathing is tachypneic or bradypneic compared with age-appropriate limits and conditions.* *Note:* Warning parameter: more than 60 breaths/min
Chest movement (presence)	Chest rises and falls equally and simultaneously with abdomen with each breath.	Child has minimal or no chest movement with respiratory effort.
Chest movement (quality)	Child has silent and effortless respirations.	Child shows evidence of labored respirations with retractions. Asynchronous movement (seesaw) is observed between chest and abdomen with respiratory efforts.
Air movement (presence)	Air exchange is heard bilaterally in all lobes.	Despite movement of the chest, minimal or no air exchange is noted on auscultation.
Air movement (quality)	Breath sounds are of normal intensity and duration.	Nasal flaring, grunting, stridor, and/or wheezing are noted.
Circulation		
Heart rate (presence)	Apical beat is present and within age-appropriate limit.*	Heart rate is absent; bradycardia or tachycardia occurs as compared with age-appropriate limits.* *Note*: Warning parameters: Infant: <80 beats/min Child <5 y: more than 180 beats/min Child older than 5 y: more than 150 beats/min
Heart rate (quality)	Heart rate is regular with normal sinus rhythm.	Heart rate is irregular, slow, or very rapid; common dysrhythmias include supraventricular tachycardia, bradyarrhythmias, and asystole.
Skin	Extremities are warm, pink with capillary refill 2 s or less; peripheral pulses are present bilaterally with normal intensity.	Child has pallor, cyanosis, or mottled skin color and cool-to-cold extremities. Capillary refill time is 2 s or more; peripheral pulses are weak or absent; central pulses are weak.
Cerebral perfusion	Child is alert to surroundings, recognizes parents or significant others, is responsive to fear and pain, and has normal muscle tone.	Child is irritable, lethargic, obtunded, or comatose; has minimal or no reaction to pain; and/or has loose muscle tone (floppy).
Blood pressure	Blood pressure is within age-appropriate limits.	Blood pressure falls from age-appropriate limits,* a late sign of decompensation. *Note*: A fall of 10 mm Hg systolic pressure is significant. Lower systolic blood pressure limit: Infant 1 mo or less, 60 mm Hg Infant 1 y or less, 70 mm Hg Child, 70 mm Hg + (2 × age in years)

*All vital signs are interpreted within the context of age, clinical condition, and other external factors, such as the presence of fever.

Adapted from Moloney-Harmon PA, Rosenthal CH: Nursing care modifications for the child in the adult ICU. In Stillwell S (ed): Mosby's Critical Care Nursing Reference. St. Louis, MO: Mosby-Year Book, 1992, pp 588–670, with permission from Elsevier Science.

step in assessing pain in children is to understand the child's response to, and communication of, pain. This is based on a variety of factors, including the child's developmental level, past and present experience with pain, cultural aspects, personality, parental presence, and age, as well as the nature of the illness or injury.[6] For instance, critically ill children may be in severe pain but may be unable to communicate because of sedation, paralytic agents, mechanical ventilation, or coma.

Table 10-5 Recommended Resuscitation Equipment for Infants and Children

Equipment	Child's Weight						
	4–8 kg (8.8–17.6 pounds)	8–11 kg (17.6–24.2 pounds)	11–14 kg (24.2–30.8 pounds)	14–18 kg (30.8–39.6 pounds)	18–24 kg (39.6–52.8 pounds)	24–32 kg (52.8–70.4 pounds)	32+ kg (70.4 + pounds)
Oxygen mask	Newborn	Pediatric	Pediatric	Pediatric	Pediatric	Adult	Adult
Oral airway	Infant	Small child	Child	Child	Child	Small adult	Small adult
Resuscitation bag	Infant	Child	Child	Child	Child	Adult	Adult
Laryngoscope blade	0–1 straight	1 straight	2 straight or curved	2 straight or curved	2 straight or curved	2–3 straight or curved	3 straight or curved
Endotracheal tube (mm)	2.5 preterm; 3.0–3.5 term infant	4.0 uncuffed	4.5 uncuffed	5.0 uncuffed	5.5 uncuffed	6.0 cuffed	6.5 cuffed
Endotracheal tube (cm at the tip)	10–10.5	11–12	12.5–13.5	14–15	15.5–16.5	17–18	18.5–19.5
Stylet	Small	Small	Small	Small	Large	Large	Large
Suction catheter	6–8	8	8–10	10	10	10–12	12–14
Nasogastric tube (F)	5–8	8–10	10	10–12	12–14	14–18	18
Urinary catheter	5–8	8–10	10	10–12	10–12	12	12
Chest tube (F)	10–12	16–20	20–24	20–24	24–32	28–32	32–40
Blood pressure cuff	Newborn or infant	Infant or child	Child	Child	Child	Child or adult	Adult
IV catheter (G)	22–24	22–24	20–22	18–22	18–20	18–20	16–20
Butterfly catheter	23–25	23–25	21–23	21–23	21–23	20–22	18–21
Vascular catheter	3.0 F 5–12 cm	3.0–4.09 F 5–12 cm	3.0–4.0 F 5–12 cm	4.0–5.0 F 5–25 cm	4.0–5.0 F 5–25 cm	4.0–5.0 F 5–25 cm	5.0–8.0 F 5–30 cm
Guide wire (mm)	0.46	0.46–0.53	0.53–0.89	0.53–0.89	0.53–0.89	0.53–0.89	0.89

Data from Hazinski M: PALS Provider Manual. Dallas, American Heart Association, 2002; Slota M: AACN Core Curriculum for Pediatric Critical Care Nursing, Philadelphia, PA: WB Saunders, 2006.

From the AACN Pediatric Critical Care Pocket Reference Card. © 1998 American Association of Critical Care Nurses (AACN). Adapted with permission of the publisher.

Reprinted from Dimens Crit Care Nurs 20(1):23, 2001, with permission.

Pain assessment is multidimensional. Synthesis of a variety of parameters provides information that can be used to make a decision about the level of the child's pain and the most appropriate intervention. Assessment of pain by nurses is influenced by factors such as educational level, skills, experience, personal beliefs, and different strategies adopted for assessment.[6] Infants and young children cannot communicate verbally, which makes pain assessment challenging. This inability to communicate is also an issue in the sedated or chemically paralyzed child. This requires that the nurse assesses pain using different cues, which include physiological and behavioral changes.[7]

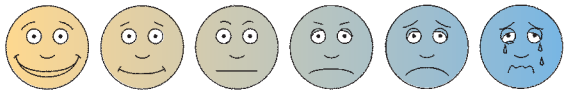
Physiological parameters used in pain assessment include heart rate, respiratory rate, blood pressure, and oxygen saturation. Other parameters described by Anand and Carr[8] include sweating, increased muscle tone, and skin color changes. These parameters return to normal as physiological adaptation occurs. This adaptation can actually occur within minutes, and the nurse must realize that the child may still be in pain. The physical signs are not necessarily specific for pain but may be the only parameter available to the nurse caring for the critically ill child.

Behavioral responses may be helpful for pain assessment, especially in the child who cannot communicate. The next section on interaction with children and families discusses the continuum of responses related to pain and comfort.

Another dimension of pain assessment is self-report. Many tools are available. However, these often require children to interact or use their hands and are not usually helpful in the critical care setting. Examples of self-report tools include the numerical rating scale (see Chapter 5, Fig. 5-1), the FACES scale (Fig. 10-2), and the color scale. If the child is unable or unwilling to give a report, the parent's report of pain is often helpful. Multidimensional scales, such as the COMFORT, Modified Motor Activity Assessment, and FLACC (face, legs, activity, cry, consolability) scales (see Fig. 10-2), are helpful because they combine dimensions of behavioral and physiological distress and do not require interaction or use of the hands.

Pain management interventions are multidimensional whenever possible, including nonpharmacological and pharmacological approaches. However, pharmacological intervention is never withheld when it is appropriate. Opioids are usually the first-line drugs for pain

A



0 1 2 3 4 5

B

	0	1	2
Face	No particular expression or smile	Occasional grimace or frown, withdrawn, disinterested	Frequent to constant frown, clenched jaw, quivering chin
Legs	Normal position or relaxed	Uneasy, restless, tense	Kicking, or legs drawn up
Activity	Lying quietly, normal position, moves easily	Squirming, shifting back and forth, tense	Arched, rigid, or jerking
Cry	No cry (awake or asleep)	Moans or whimpers, occasional complaint	Crying steadily, screams or sobs, frequent complaints
Consolability	Content, relaxed	Reassured by occasional touching, hugging, or "talking to," distractable	Difficult to console or comfort

FIGURE 10-2 ▲ Tools for assessing pain in children. **A:** The FACES scale. This scale may be used in children 3 years and older. Explain that FACE 0 is a very happy face because there is no pain. FACE 1 hurts just a little bit. FACE 2 hurts a little bit more. FACE 3 hurts even more. FACE 4 hurts a whole lot. FACE 5 hurts very much; the pain can make you cry. Ask the child to choose the face that best describes the pain he or she is feeling. **B:** The FLACC (face, legs, activity, cry, consolability) scale. This scale can be used with children younger than 3 years. To use the FLACC scale, assess the child in each category, assigning a score between 0 and 2. Total the score, and then evaluate the total using the 0–10 pain scale parameters. (**A** from Wong DL, Hockenberry-Eaton M, Wilson D, et al: Wong's Essentials of Pediatric Nursing, 7th ed. St. Louis, Mosby, 2005, p 1259. Copyrighted by Mosby, Inc. Reprinted by permission. **B** from Merkel SI, Voepel-Lewis T: The FLACC: A behavioral scale for scoring post-operative pain in young children. Pediatr Nurs 23(3):293–297, 1997. © 2002, The Regents of the University of Michigan.)

management in the critically ill child. A variety of pharmacological drugs are available, and the choice of medication depends on the child's response and the practitioner's preference. Nursing responsibilities include assessing the child's need for the drug, administering the appropriate dose, and monitoring the child's response. Sedation and analgesia are part of the daily management of the critically ill child. However, inherent in the use of these drugs is the risk for adverse responses. The nurse is responsible for vigilant monitoring and for implementing changes based on the child's response and recommendations by the multidisciplinary team.[9]

Other methods of pain control include intravenous patient-controlled analgesia (PCA) and epidural analgesia. PCA helps the child maintain a steady state of pain relief and also gives the child some control over pain. Epidural analgesia is also helpful for a variety of children. Epidural narcotics provide selective analgesia but do have associated side effects, including respiratory depression, nausea and vomiting, pruritus, and urinary retention.

Nurses may consider the use of nonpharmacological methods, such as distraction, relaxation, massage, and hypnosis, in conjunction with pharmacological drugs. The method must be age appropriate, and parental presence is considered. Whatever methods are used, a critical determinant of their effectiveness is the child's response.

▲ Interaction with Children and Families

Interacting with children demands familiarity with their developmental capabilities and psychosocial needs. Categorization of children into groups according to physical and cognitive age can assist the nurse in predicting the child's expected social, cognitive, and physical capabilities. Developmental and psychosocial assessment is beyond the scope of this chapter; therefore, the reader should consult an appropriate growth and development reference. Although each age group of children has common developmental capabilities, tasks, and fears, it is helpful to recognize the common fears of all children despite their age. These fears include loss of control, threat of separation, painful procedures, and communicated anxiety.

Unlike the adult patient, the young child does not consciously screen most behavior and spoken words. The young

Table 10-6 Contrasting Nonverbal Behavioral Cues of the Healthy and the Critically Ill Child

Healthy	Critically Ill
Posture	
Moves, flexes	May be loose, flaccid May prefer fetal position or position of comfort
Gestures	
Turns to familiar voices	Responds slowly to familiar voices
Movement	
Moves purposefully Moves toward new, pleasurable items Moves away from threatening items, people	Exhibits minimal movement, lethargy Shows increased movement, irritability (possibly indicating cardiopulmonary or neurological compromise, pain, or sleep deprivation)
Reactions/Coping Style	
Responds to parent(s) coming, leaving Responds to environment and equipment Cries and fights invasive procedures	Exhibits minimal response to parent presence, absence Exhibits minimal response to presence or absence of transitional objects Displays minimal defensive responses
Facial Expressions	
Looks at faces and makes eye contact Changes facial expressions in response to interactions Responds negatively to face wash Blinks in response to stimuli Widens eyes with fear Is fascinated with own mouth Holds mouth "ready for action"	May not track faces, objects Avoids eye contact or has minimal response to interactions Minimally changes facial expression during face wash Exhibits increased or decreased blinking Avoids eye contact Avoids or dislikes mouth stimulation Drools or displays loose mouth musculature Sucks intermittently or weakly

Taken from Moloney-Harmon P, Rosenthal CH: Nursing care modifications for the child in the adult ICU. In Stillwell S (ed): Critical Care Nursing Reference Book. St. Louis, MO: Mosby-Year Book, 1992, p 590, with permission from Elsevier Science.

child subconsciously communicates behaviorally through verbal, nonverbal (body language, behaviors), and abstract (play, drawing, story telling) cues. Although the child's behavior is more natural in a familiar environment, the cues available to the clinician can suggest how a child is feeling or perceiving an event or the presence of a person. In general, the child's behavior is more activity oriented and more emotional than that of adults. These qualities of a child's behavior should be expected as the norm of average, healthy children and may be used as parameters against which to contrast the behavior of the critically ill child (Table 10-6).

Behavioral responses are particularly helpful during the assessment of pain or comfort. The infant or child may display body movement that spans the entire activity continuum from minimal movement, such as rigidity and guarding, to high activity, such as thrashing and kicking. Assessing various behavioral responses (eg, gestures, posture, movement, and facial expression) and examining the congruency between these responses are particularly helpful.

Interaction with pediatric patients and their families is also facilitated by the appreciation of the child's significant others. The philosophy of family centered care is essential to the optimal care of the pediatric patient. Gone are the days when parents dropped their children for care at the entrance of the hospital. Although there are several components of family centered care, the salient concept is to value, recognize, and support the family in the care of their child. The family is the constant in the child's life and is ultimately responsible for responding to the child's emotional, social, developmental, physical, and health care needs. Appropriate support and incorporation of parents may buffer the threats of the ICU environment on the child. Parents may assist or influence the child's cognitive appraisal of the environment, personnel, and events. The child often uses the reactions of the parent as a barometer in interpreting events in ways ranging from threatening to beneficial.[10]

The tone and manner in which the clinician approaches the bedside of a pediatric patient and his or her family are important. Communicated anxiety refers to the anxious feelings conveyed to the child by the parents, the health care team members, or both. Interventions to relieve the anxiety of parents and fellow health care team members have a direct impact on the child's well-being. Interventions may include assisting parents and staff in anticipating the child's responses to therapy and illness and guiding parents and staff in therapeutic communication techniques.

Parents depend on nurses to humanize the critical care experience for their child. A recent study examined parents' perceptions of nurses' caring practices in the pediatric ICU. Parents reported that nurses used behaviors that demonstrated affection, caring, watching, and protecting. Parents stated that the most desirable nursing behaviors were those that complemented the parental role, which preserved family integrity during a time of crisis.[11] Clinical practice guidelines that support the parents of children in pediatric ICUs facilitate family involvement.[12]

Examples of nursing diagnoses for the pediatric patient in the ICU are given in Box 10-1.

BOX 10-1 EXAMPLES OF NURSING DIAGNOSES

For the Critically Ill Pediatric Patient

- Ineffective Airway Clearance related to obstructed airway
- Anxiety related to environment
- Risk for Imbalanced Body Temperature
- Interrupted Family Processes related to shift in health status of a family member
- Deficient Fluid Volume related to active fluid volume loss, failure of regulatory mechanisms
- Delayed Growth and Development related to separation from significant others

▲ Clinical Applicability Challenges

CASE STUDY

S. is a 2-year-old, 13-kg girl admitted for respiratory distress. On admission, her vital signs are as follows: heart rate, 170 beats/min; respiratory rate, 56 breaths/min; blood pressure, 105/65 mm Hg; axillary temperature, 101.4°F (38.5°C); and oxygen saturation, 90%. Physical examination reveals an agitated, irritable, and crying young girl with intercostal and substernal retractions and nasal flaring. Her expiratory wheezing can be heard across the room. The child's respiratory rate is high for her age, although tachypnea is to be expected for her clinical condition. She has a fever and is anxious, which may also account for the tachypnea. The anxiety, agitation, and increased work of breathing increase the oxygen demand while the lower airway obstruction decreases oxygen transport. The warning parameters of tachypnea, nasal flaring, retractions, and wheezing indicate a child with respiratory distress who is at risk for respiratory failure.

Initial management priorities include positioning S. for comfort and administering oxygen while assessing her response. Albuterol, an inhaled β_2-agonist, is administered. After these interventions, her vital signs are as follows: heart rate, 154 beats/min; respiratory rate, 40 breaths/min; blood pressure, 110/64 mm Hg; and oxygen saturation, 98% on 40% oxygen by face mask.

One hour later, S's vital signs are as follows: heart rate, 80 beats/min; respiratory rate, 20 breaths/min; blood pressure, 90/50 mm Hg; and oxygen saturation, 88%. Retractions are more pronounced, and wheezing is not audible by auscultation. She is extremely lethargic and is not responding to her parents. Arterial blood gas values reveal the following: pH, 7.22; $PaCO_2$, 58 mm Hg; PaO_2, 78 mm Hg; and HCO_3^-, 28 mEq/L on a fraction of inspired oxygen of 0.6. The child is demonstrating signs of fatigue and respiratory failure. Warning parameters include an unmaintainable airway, bradypnea, worsening retractions, lethargy, no air movement (indicated by lack of wheezing), and failure to recognize her parents. Management priorities at this time include bag-mask ventilation and intubation if she does not respond to bag-mask ventilation.

Treatment is directed toward ensuring oxygenation and ventilation while reversing bronchospasm. Medications include inhaled bronchodilators and corticosteroids. Mechanical ventilation is also provided until S. shows improvement, as demonstrated by arterial blood gas values, vital signs, and clinical condition.

1. S. is demonstrating signs of impending respiratory failure. Describe the signs that indicate respiratory failure in this child.

2. Discuss the management priorities for S. related to her impending respiratory failure.

3. Describe assessment parameters to determine S.'s response to intervention.

References

1. Moloney-Harmon P, Rosenthal CH: Nursing care modifications for the child in the adult ICU. In Stillwell S (ed): Critical Care Nursing Reference Book. St. Louis, MO: Mosby-Year Book, 1992, pp 588–670
2. American Heart Association: Pediatric advanced life support. Circulation 112(24):N-167–N-187, 2005
3. Kohn LT, Corrigan JM, Donaldson MS (eds): To Err Is Human: Building a Safer Health System. Washington, DC: National Academy Press, 2000
4. American Academy of Pediatrics: Prevention of medication errors in the pediatric inpatient setting. Pediatrics 112(2):431–436, 2003
5. Wong ICK, Wong LYL, Cranswick NE: Minimising medication errors in children. Arch Dis Child 94(2):161–164, 2009

6. Cohen LL, Lemanek K, Blount RL, et al: Evidence-based assessment of pediatric pain. J Pediatr Psychol 33(9):939–955, 2008
7. Griffin RA, Polit DF, Byrne MW: Nurse characteristics and inferences about children's pain. Pediatr Nurs 34(4):297–307, 2008
8. Anand KJS, Carr DB: The neuroanatomy, neurophysiology, and neurochemistry of pain, stress, and analgesia in newborns and children. Pediatr Clin North Am 36(4):795–821, 1989
9. Anand KJ, Willson DF, Berger J, et al: Tolerance and withdrawal from prolonged opioid use in critically ill children. Pediatrics 125(5):e1208–25, 2010
10. Frazier A, Frazier H, Warren N: A discussion of family-centered care within the pediatric intensive care unit. Crit Care Nurs Q 33(1):82–86, 2010
11. Harbaugh BL, Tomlinson PS, Kirschbaum M: Parents' perceptions of nurses' caring behaviors in the pediatric intensive care unit. Issues Compr Pediatr Nurs 27(3):163–178, 2004
12. Davidson JE, Powers K, Hedayat KM, et al: Clinical practice guidelines for support of the family in the patient-centered intensive care unit: American College of Critical Care Medicine Task Force 2004–2005. Crit Care Med 35(2):605–622, 2007

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

11 The Critically Ill Pregnant Woman

Cathleen R. Maiolatesi

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Summarize normal physiological changes that occur in the cardiovascular, respiratory, renal, and hematological systems during pregnancy.
2. Differentiate the signs and symptoms of preeclampsia and severe preeclampsia.
3. Explain the pathophysiology of severe preeclampsia.
4. Describe parameters of nursing assessment of a severely preeclamptic patient on intravenous magnesium sulfate.
5. Discuss three obstetrical conditions that predispose a woman to development of disseminated intravascular coagulation.
6. Describe the initial care of an obstetrical trauma patient.
7. Summarize the psychosocial support needed for an obstetrical patient in the intensive care unit.

Most women experience a normal pregnancy. However, a small percentage of women experience life-threatening complications that may result from the pregnancy itself or develop as a result of a preexisting (comorbid) condition. Such critically ill pregnant women provide a unique challenge to nurses. The physical assessment of these patients includes the interaction between the maternal host and the fetus. Critical care nurses are not expected to have the knowledge and skills associated with fetal heart rate monitoring, and perinatal nurses may not possess the knowledge and skills required for patients needing ventilator support or hemodynamic monitoring. It is important when a critically ill pregnant woman is in the intensive care unit (ICU) that a collaborative approach be used to provide care.[1]

The general principles of diagnosis and management are similar to those used for other ICU patients. However, physiological changes inherent in pregnancy must be considered to decrease morbidity and mortality.[2] Critical care nurses caring for these patients must understand the physiological changes that occur as the body adapts to pregnancy and distinguish normal from abnormal responses. Table 11-1 outlines these changes.

▲ Physiological Changes in Pregnancy

Cardiovascular Changes

Normal cardiovascular changes that occur during pregnancy affect pulse, blood pressure, cardiac output, and blood volume (see Table 11-1). Maternal blood volume increases 40% to 50% above baseline values. This increase, which consists mostly of plasma, begins in the first trimester and continues throughout pregnancy. The increase is necessary to provide adequate blood flow to the uterus, fetus, and changing maternal tissues and to accommodate blood loss at birth. Red blood cell volume increases by 20% and is disproportionate to the plasma increase, resulting in maternal physiological anemia. Heart rate increases 10 to 15 beats/min as early as 7 weeks of gestation and returns to the prepregnancy level by 6 weeks postpartum.[3] Changes in blood volume and heart rate lead to an increase in cardiac output of 30% to 50% (6 to 7 L/min) during pregnancy.[3] Cardiac output increases slightly more intrapartum as a result of the shunting of blood from the placental–fetal unit. Immediately after birth, a larger increase in cardiac output (59% to 80%) occurs when the empty uterus contracts and shunts approximately 1,000 mL of blood back into the systemic circulation[2] (Table 11-2). A woman loses approximately 500 mL of blood during a vaginal birth and approximately 1,000 mL of blood during a cesarean birth. This is usually well tolerated.

Development of the uteroplacental unit provides a low-resistance network for the expanded blood volume, which reduces cardiac afterload.[4] The pulmonary vascular resistance, or right afterload, also decreases in response to increased blood volume and vasodilation. Under hormonal influence, smooth muscles and vascular beds relax, lowering systemic vascular resistance (SVR). Blood pressure decreases during the first and second trimesters and returns to prepregnancy levels by the third trimester. Blood pressure during pregnancy is affected by maternal position more so than in the nonpregnant state. Supine hypotension occurs when the

Table 11-1 Physiological Changes in Pregnancy

	Change	Pregnancy Levels
Cardiovascular Changes		
Blood volume	>40%–50%	1,260–1,625 mL
Red blood cells	>20%	250–450 mL
Blood pressure Systolic Diastolic	 <5–12 mm Hg <10–20 mm Hg	
Cardiac output	>30%–50%	6–7 L/min
Heart rate	>10%–30%	Increased by 15–20 beats/min
Systemic vascular resistance	<20%–30%	1,210 ± 266 dynes/s/cm^{-5}
Pulmonary vascular resistance	<34%	78 ± 22 dynes/s/cm^{-5}
Colloid osmotic pressure	<10%–14%	<22.4 ± 0.5
Respiratory Changes		
Functional residual capacity	<10%–21%	1,343–1,530
Tidal volume	>30%–35%	600 mL
Renal Changes		
Renal blood flow	>25%–50%	1,500–1,750 mL/min
Glomerular filtration rate	>50%	140–170 mL/min
Creatinine clearance	>50%	100–150 mL/min

Table 11-2 Cardiac Output Changes in Pregnancy and Labor and Delivery

Stage of Pregnancy, Labor, or Delivery	Cardiac Output
By 8 weeks of gestation	22%–30% increase
By 20 weeks of gestation	50% increase
Repositioned from supine to left lateral decubitus	21% increase
Early first stage of labor (dilated <3 cm)	13%–17% increase
Late first stage of labor (dilated 4–7 cm)	23% increase
Second stage of labor (dilated >8 cm)	34% increase
During each contraction	11%–15% increase
Immediately after delivery (10 min)	59%–80% increase (dependent on type of anesthesia)
Within 1 h of delivery	49% increase

mother remains in a flat position. The side-lying position is recommended, but if the patient must be supine, the uterus should be tilted away from the inferior vena cava by using a wedge under the hip.

Respiratory Changes

Respiratory changes as seen in Table 11-1 occur to accommodate the enlarged uterus and the increased oxygen demands of the mother and fetus. Structural changes include the upward shift of the diaphragm, which decreases functional residual capacity, and rib cage volume displacement, which increases tidal volume by 30% to 35%.[5] Airway mucosal changes include hyperemia, hypersecretion, increased friability, and edema. These changes are significant when inserting nasogastric tubes or nasotracheal tubes because of the risk for epistaxis. Respiratory rate remains unchanged, although some women experience tachypnea or shortness of breath at some time during their pregnancy. The exact cause of dyspnea is unknown, but it may be related to hyperventilation, increased oxygen consumption, or decreased partial pressure of arterial carbon dioxide ($PaCO_2$).

Oxygen consumption increases by 15% to 20% during pregnancy and may increase by 300% during labor.[2] This results in an increased partial pressure of arterial oxygen (PaO_2) to 104 to 108 mm Hg. $PaCO_2$ decreases to 27 to 32 mm Hg and allows for the increased diffusion of carbon dioxide from the fetus to the mother.[5] Renal excretion of bicarbonate causes a slight increase in maternal pH, which is usually insignificant.

Renal Changes

Changes in renal function, also outlined in Table 11-1, accommodate the increase in metabolic and circulatory requirements of pregnancy. Renal blood flow increases by 30% and glomerular filtration rate (GFR) by 50%.[3] These increases allow elevations in the clearance of many substances, such as creatinine and urea, and are reflected in lower serum levels.

Gastrointestinal and Metabolic Changes

Gastrointestinal changes in pregnancy occur as a result of the growing uterus. Displacement of the esophageal sphincter into the thoracic cavity allows stomach contents to enter the esophagus passively. The pregnant woman is prone to passive regurgitation and aspiration, especially when under general anesthesia or any time she may be unconscious.[2] Hormonal influences cause delayed gastric emptying and increased gastric acid secretion in the third trimester. Smooth muscle relaxation contributes to nausea, heartburn, and constipation. Pregnancy creates a diabetogenic state because the body becomes increasingly resistant to insulin and hyperinsulinemia occurs. Hepatic and maternal fasting blood glucose levels decrease owing to the constant transfer of glucose to the fetus.

Hematological Changes

Hematocrit laboratory values decrease because of the hemodilution effect of increased plasma volume. Normal hematocrit values are 32% to 40% during pregnancy.[2] The white blood cell count is elevated from the normal range of 5,000–10,000/mm^3 to 6,000–16,000/mm^3.[4] There is an increase in clotting factors VII through X and a decrease in factors XI and XIII, which inhibit coagulation. Fibrinogen increases to 300 to 600 mg/dL. Bleeding and clotting times and platelet counts remain the same in pregnancy.

Fetal and Placental Development Considerations

Clinicians must carefully balance the effects and risks of all treatment decisions on the pregnant woman and her fetus. Maternal circulation and nutrition and exposure to teratogens influence embryonic and fetal development.

There are three stages in fetal development: pre-embryonic (first 14 days), embryonic (day 15 through 8 weeks), and fetal (8 weeks through 40 weeks and delivery). During the embryonic stage, vital organs such as the heart and brain are in development. It is during this stage that the fetus is most vulnerable to teratogens (Fig. 11-1).

Certain medications used in treating the critically ill pregnant woman may cross the placenta and have teratogenic effects on the fetus. For this reason, the clinician must consider the risks and benefits of medication therapy. In 2001, the U.S. Food and Drug Administration revised the five risk categories for labeling drug use in pregnancy (Table 11-3).

The placenta is the organ responsible for the metabolic exchange of oxygen, nutrition, and waste removal between the pregnant woman and the fetus. In early pregnancy, the placenta produces four hormones necessary to maintain the pregnancy. The hormone human chorionic gonadotropin is the basis for pregnancy tests and preserves the function of the corpus luteum. Another hormone, human placental lactogen, stimulates maternal metabolism to supply needed nutrients for fetal growth. This hormone is responsible for the increase in insulin resistance associated with pregnancy. The hormones progesterone and estrogen are eventually produced by the placenta and are responsible for uterine growth and uteroplacental blood flow.

Placental function depends on maternal blood flow. Diseases and conditions that cause vasoconstriction, such as hypertension, cocaine use, or smoking, can diminish blood flow to the placenta and fetus. Even excessive maternal exercise can shunt blood away from the placenta and fetus.

▲ Critical Care Conditions in Pregnancy

During pregnancy, normal physiological changes occur to provide for growth of the fetus and prepare the mother for birth.

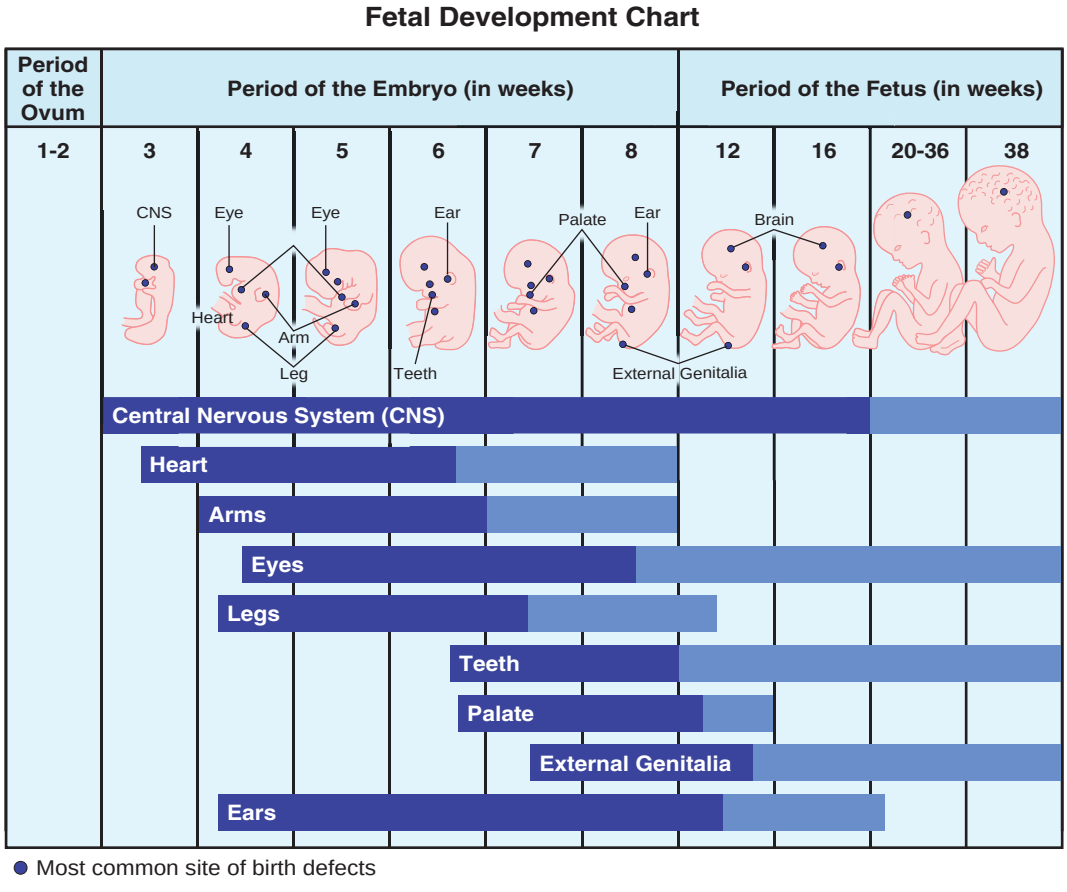


FIGURE 11-1 ▲ Critical periods of development. *Dark blue* denotes highly sensitive periods. (Adapted from the National Organization on Fetal Alcohol Syndrome.)

Table 11-3 Teratogenic Medication Risk Categories

Category	Description
A	Adequate, well-controlled studies in pregnant women have not shown an increased risk for fetal abnormalities.
B	Animal studies have revealed no evidence of harm to the fetus; however, there are no adequate and well-controlled studies in pregnant women. **OR** Animal studies have shown an adverse effect, but adequate and well-controlled studies in pregnant women have failed to demonstrate a risk to the fetus.
C	Animal studies have shown an adverse effect, and there are no adequate and well-controlled studies in pregnant women. **OR** No animal studies have been conducted and there are no adequate and well-controlled studies in pregnant women.
D	Studies, adequate well-controlled or observational, in pregnant women have demonstrated a risk to the fetus. However, the benefits of therapy may outweigh the potential risk.
X	Studies, adequate well-controlled or observational, in animals or pregnant women have demonstrated positive evidence of fetal abnormalities. The use of the product is contraindicated in women who are or may become pregnant.

Medical or obstetrical complications may alter this adaptation and shift an uncomplicated pregnancy into a critical situation. ICU admissions become necessary in 1% to 3% of pregnancies, with the majority of indications being hemodynamic instability, obstetrical hemorrhage, and respiratory failure.[6] The most common obstetrical complications requiring ICU admission are severe preeclampsia, disseminated intravascular coagulation (DIC), amniotic fluid embolus, acute respiratory distress syndrome (ARDS), and trauma. Box 11-1 contains sample nursing diagnoses for patients with high-risk pregnancies.

Severe Preeclampsia

Hypertensive disorders of pregnancy occur in approximately 3% to 7% of all pregnancies.[7,8] They are the third leading cause of maternal death in the United States. Terms used to describe the different types of hypertension that may occur in pregnancy are listed in Box 11-2. Preeclampsia is a hypertensive disorder occurring in 5% to 7% of pregnancies.[9] The etiology of preeclampsia is unknown; however, predisposing risk factors include nulliparity, multiple gestation, diabetes, age younger than 18 years or older than 35 years, and chronic hypertension.

Preeclamptic symptoms include hypertension, edema, and proteinuria. Hypertension in pregnancy is defined as a blood pressure of greater than 140/90 mm Hg.[9] In the past, increases of 30 mm Hg systolically or 15 mm Hg diastolically were used to diagnose hypertension in pregnancy. These criteria have become unreliable and should no longer be used. If the prepregnancy blood pressure is unknown, a blood pressure measurement of 140/90 mm Hg obtained twice at intervals of 6 hours apart in the same position and using the same arm is appropriate to diagnose this complication. In severe preeclampsia, the systolic blood pressure is greater than 160 mm Hg, and the diastolic blood pressure is greater than 110 mm Hg.[8] Edema may be generalized but is more pronounced in the hands and face. Proteinuria is diagnosed by protein concentrations of 5 g or greater in a 24-hour urine specimen. Oliguria may occur and is defined as a urine output less than 30 mL/h or less than 500 mL/24 h. Other symptoms of severe preeclampsia include visual and cerebral disturbances, such as blurred vision and headaches, epigastric pain, impaired liver function, thrombocytopenia, and pulmonary edema.

Physiological Principles

Severe preeclampsia is associated with vascular endothelial damage caused by arteriolar vasospasms and vasoconstriction.[9]

BOX 11-1 EXAMPLES OF NURSING DIAGNOSES

For Critically Ill Pregnant Patients

- Anxiety related to poor/uncertain pregnancy outcomes
- Stress Overload related to poor/uncertain pregnancy outcomes
- Grieving related to threat to self
- Fear related to fetal well-being
- Risk for Injury related to infection
- Interrupted Family Processes related to intensive care hospitalization
- Decreased Cardiac Output related to increased intrathoracic pressures (patient receiving mechanical ventilation)
- Risk for Disturbed Maternal-Fetal Dyad related to maternal position, blood loss, or placental trauma

BOX 11-2 Clinical Terminology: Hypertensive Disorders During Pregnancy

- *Preeclampsia:* a pregnancy-specific syndrome observed after the 20th week of pregnancy with systolic blood pressure of greater than or equal to 140 mm Hg or diastolic blood pressure of greater than or equal to 90 mm Hg, accompanied by significant proteinuria. In women with preeclampsia, blood pressure usually returns to baseline within days to weeks after delivery.
- *Eclampsia:* the occurrence, in a woman with preeclampsia, of seizures that cannot be attributed to other causes. Convulsions usually occur after midpregnancy and may occur postpartum.
- *Gestational hypertension:* a blood pressure elevation detected for the first time after midpregnancy, distinguished from preeclampsia by the absence of proteinuria.
- *Chronic hypertension:* elevated blood pressure in the mother that predated the pregnancy. It can also be diagnosed in retrospect when preeclampsia or gestational hypertension fails to normalize after delivery.

From National Institutes of Health, National Heart Lung and Blood Institute: Report of the Working Group on Research on Hypertension During Pregnancy, April 2001.

Arterial circulation is disrupted by alternating areas of constriction and dilation. Damage to the endothelium results in leakage of plasma into the extravascular space and allows platelet aggregation to occur. Colloidal osmotic pressure decreases as protein enters the extravascular space, and the woman is at risk for hypovolemia and altered tissue perfusion and oxygenation.[10] Physiologic changes in the maternal cardiovascular system such as increased plasma volume, cardiac output, heart rate and capillary permeability, and a decrease in colloid osmotic pressure are exaggerated in preeclampsia and predispose the women to develop pulmonary edema.[11] Symptoms of pulmonary edema include coughing, dyspnea, chest pain, tachycardia, cyanosis, and pink, frothy sputum.[11]

Arterial vasospasm and endothelial damage also decrease perfusion to the kidneys. The decreased kidney perfusion results in a decreased GFR and leads to oliguria. Oliguria may not be an indication of hypovolemia and should not be treated with diuretics. Glomerular capillary endothelial damage permits protein to leak across the capillary membrane and into the urine, resulting in proteinuria, increased blood urea nitrogen, and increased serum creatinine. If vasospasm and hypercoagulability are long lasting, ischemia occurs in the glomeruli. Complete recovery of renal function usually occurs after delivery.[12]

The liver is also affected by multisystem vasospasm and endothelial damage. Decreased perfusion to the liver can cause ischemia and necrosis. The liver may become edematous as a result of inflammatory infiltrates and obstructed blood flow.[12] Liver damage is reflected in elevated liver function study results, such as serum aspartate aminotransferase, lactate dehydrogenase, and serum alanine aminotransferase.[2]

Neurological sequelae may include seizures, cerebral edema, and cerebral hemorrhage. Symptoms associated with neurological progression are headaches, blurred vision, hyperreflexia and clonus, and changes in the level of consciousness. Increased intracranial pressure and decreased perfusion can lead to hypoxia, coma, and death.[10]

Management

The only cure for severe preeclampsia is delivery of the fetus. The decision to deliver the fetus versus continuing expectant management (ie, to maintain the pregnancy and monitor changes) is individualized.[8]

Usually these patients require invasive hemodynamic monitoring, frequent blood pressure measurements, strict intake and output monitoring, laboratory report monitoring, aggressive anticonvulsant and antihypertensive drug therapy, and, if undelivered, fetal surveillance. Management is focused on preventing seizures and respiratory complications, controlling hypertension, monitoring cardiovascular status, and maintaining fluid status. If the woman does not deliver, fetal monitoring is necessary. Critical care and obstetrical staff must collaborate to provide close fetal observation. It is important that they consider the fetus to be another patient.

Hemodynamic monitoring permits accurate assessments of cardiac output and fluid volume status. Normal hemodynamic values in pregnancy are listed in Table 11-4.[13] Elevated pulmonary artery occlusion pressure (PAOP) and pulmonary artery pressure (PAP) values may indicate hypervolemia, thereby placing the woman at risk for cardiogenic pulmonary edema. (See Chapter 17 for a more detailed discussion of hemodynamic monitoring.) Interventions to reduce preload include restricting intravenous (IV) fluids, repositioning the patient on her side, and administering diuretics when fluid overload or pulmonary edema is present. Decreased central venous pressure, PAP, and PAOP values indicate hypovolemia, and the patient may need a fluid challenge.

Table 11-4 Hemodynamic Values in Nonpregnant and Pregnant Women

	Nonpregnant	Pregnant
Central venous pressure	5–10 mm Hg	1.1–6.1 mm Hg
Pulmonary Artery Pressure		
Systolic	20–30 mm Hg	18–30 mm Hg
Diastolic	8–15 mm Hg	6–10 mm Hg
Mean	10–20 mm Hg	11–15 mm Hg
Pulmonary artery occlusion pressure	6–12 mm Hg	5.7–9.3 mm Hg
Cardiac output	4.3–6.0 L/min	5.2–7.2 L/min

Drug therapy is directed at preventing seizures and hypertensive crises. IV magnesium sulfate is the drug of choice for severe preeclampsia to prevent maternal seizures (Box 11-3). Magnesium sulfate blocks the reuptake of acetylcholine at the nerve end synapses and relaxes smooth muscles. Side effects include drowsiness, flushing, diaphoresis, hyporeflexia, hypocalcemia, and respiratory paralysis.[14] A therapeutic serum level of 4 to 7 mg/dL is maintained through a continuous infusion of 1 to 3 g/h. Serum levels higher than 15 mg/dL may result in respiratory arrest.

Hydralazine hydrochloride (Apresoline) is the antihypertensive agent most commonly used during pregnancy. It causes arterial vasodilation and decreases mean arterial pressure and SVR. Hydralazine increases cardiac output, heart rate, and renal blood flow. Doses are commonly given in 5- to 10-mg boluses intravenously every 20 minutes until a satisfactory reduction in blood pressure is achieved.[14] Other antihypertensive agents used include nitroprusside (Nitropress), nifedipine (Procardia), and labetalol hydrochloride (Normodyne). These drugs may be used when hydralazine therapy fails.

Nursing Interventions

The nurse must assess the patient for increased risk for seizures by evaluating neurological symptoms. To reduce the risk for seizures, the nurse can decrease light and sound stimulation to the patient. Treatments and interventions are coordinated to optimize rest periods. If seizures occur, the nurse protects the

BOX 11-3 Magnesium Sulfate Administration

Dose concentration: 20 g in 500 mL normal saline solution or D_5W = 2 g/50 mL
Loading dose: 4 to 6 g intravenous bolus over 10 to 20 minutes
Maintenance dose: 2 to 3 g/h by intravenous infusion

BOX 11-4 NURSING INTERVENTIONS

For Severe Preeclampsia

Strict Bed Rest in Left Lateral Tilt
- Explain rationale and expected benefits.
- Encourage family and friends to visit and provide activities that may prevent boredom.
- Explain seizure precautions.

Medications
- Explain the action of drugs such as magnesium sulfate and antihypertensive drugs.
- Explain the frequency of laboratory tests, vital sign assessment, and urinary output measurement.

Fetal Surveillance
- Explain external fetal monitoring and tests used to monitor fetal well-being, such as the nonstress test, the biophysical profile, Doppler flow studies, and fetal pulse oximetry.
- Explain the rationale used to determine adequate uteroplacental function.

Delivery
- Prepare the patient for the possibility of cesarean delivery.
- Explain the rationale for the need to deliver.
- Explain neonatal intensive care unit (NICU) if unable to physically take the patient on a unit tour.
- Arrange for a discussion with a neonatologist if the infant is premature or expected to be admitted to NICU.

patient from injury, ensures a patent airway, provides adequate oxygenation, and evaluates possible aspiration. After stabilizing the patient, uterine and fetal activities are quickly assessed. In most instances, immediate delivery of the fetus is indicated.

If the patient is receiving magnesium sulfate therapy, the nurse continuously assesses for symptoms of magnesium toxicity, such as respiratory depression and hyporeflexia. Magnesium is excreted in the urine, and prolonged oliguria allows magnesium to accumulate to toxic blood levels.

If the patient has delivered, magnesium sulfate therapy should be continued for 24 hours. The nurse must assess uterine bleeding. The uterus should be firm after delivery, and if not, uterine massage and oxytocin therapy are needed. Box 11-4 summarizes some of the key nursing interventions for patients with severe preeclampsia.

HELLP Syndrome

HELLP syndrome (*h*emolysis, *e*levated *l*iver enzymes, and *l*ow *p*latelets) accompanies severe preeclampsia and eclampsia in approximately 10% to 20% of diagnosed cases. Maternal mortality rates may be as high as 30%.[15] It is often considered a variation of severe preeclampsia. Women in whom HELLP syndrome develops are usually older than 27 years, white, and multiparous. Patients with HELLP syndrome are at an increased risk for developing complications such as renal failure, pulmonary edema, DIC, placental abruption, ARDS, and liver hematoma and rupture.

Signs and symptoms of HELLP syndrome may be similar to those of severe preeclampsia and include epigastric pain, nausea, malaise, and right upper quadrant tenderness. Laboratory results reveal decreased platelets (<100,000/mm^3) and elevated liver enzymes.

Physiological Principles

Hemolysis occurs when red blood cells pass through vasospastic vessels, producing burr cells or schistocytes.[15] Liver enzyme levels become elevated as liver damage occurs resulting from ischemia secondary to vasospasm. Prolonged vasospasm can lead to hepatic necrosis. Platelets are consumed because of the aggregation at endothelial damage sites.

Management

As with severe preeclampsia, delivery is the treatment of choice for HELLP syndrome; however, the timing of delivery remains controversial. Most clinicians recommend expectant management occur no longer than 48 hours after the diagnosis of HELLP is established.[12] If delivery does not occur, management includes bed rest, frequent blood pressure assessments, frequent laboratory evaluation of liver function, coagulation status, and intensive fetal surveillance.[15] The patient is managed in the same manner as severe preeclampsia, and magnesium sulfate and antihypertensive agents are given as needed. Blood products may be given to correct coagulation abnormalities.

HELLP syndrome may mimic other disease entities, and differential diagnosis must be made to rule out autoimmune thrombocytopenic purpura, chronic renal disease, pyelonephritis, cholecystitis, gastroenteritis, hepatitis, pancreatitis, thrombotic thrombocytopenic purpura, hemolytic–uremic syndrome, or acute fatty liver disease of pregnancy.[15]

Monitoring changes in vital signs, bleeding, pain, and laboratory values is necessary when caring for patients with HELLP syndrome. Fetal surveillance is important and should include assessments for fetal heart rate and signs and symptoms of placental abruption. Nurses must be aware of the complications that can occur in patients with HELLP syndrome. Signs of worsening pain, vascular collapse, or shock may indicate liver hematoma or rupture. Accurate monitoring of intake and output must be maintained to assess renal status.

Disseminated Intravascular Coagulation

Several conditions predispose a pregnant woman to DIC because of changes in the coagulation and fibrinolytic systems. These conditions include preeclampsia, abruptio placentae, amniotic fluid embolus, fetal death, and sepsis.[16] Although the incidence of sepsis has decreased because of antibiotic therapy, it is responsible for 3% to 8% of the maternal deaths in the United States.[16] Sepsis during pregnancy is a result of bacterial invasion of the uterine cavity.

Immunosuppression is a normal consequence of pregnancy and thought to occur so that the fetus is not rejected by the maternal immune system. This alteration increases the susceptibility to infection and decreases the body's ability to fight infection. Septic shock may develop in a few days or several hours. Manifestations of septic shock include tachycardia, tachypnea, temperature instability, increased cardiac output, and decreased peripheral resistance.

Abruptio placentae is the premature separation of the placenta from the uterine wall and is one of the most common

causes of DIC. Blood collects between the uterus and placenta, causing consumption of clotting factors. The placental unit contains high concentrations of thromboplastin. When the placenta prematurely separates, thromboplastin continues to be released systemically, activating the clotting and fibrinolytic systems throughout the body. Parallel to the activation of the fibrinolytic system, the hemostatic system initiates clot formation at the site of the separation.[16] Clinical signs of abruption include acute abdominal pain, uterine tenderness, premature contractions, and vaginal bleeding. Abruptions may be subtle, and blood may not be visible.

Intrauterine fetal death can also lead to DIC. Tissue thromboplastin is released from the dead fetus into the maternal circulation activating the procoagulant system. Coagulopathy is gradual and consistent with chronic, low-grade DIC.

Management

Management of patients with DIC includes identifying the underlying condition and initiating appropriate therapy, evaluating and monitoring the coagulation system to restore hemostasis, and preventing further hemorrhage and thrombosis. Management of DIC caused by sepsis includes prompt delivery of the fetus and IV administration of broad-spectrum antibiotics. For abruptio placentae, prompt delivery of the fetus is necessary to control further bleeding.

Nursing care is aimed at preventing further bleeding, monitoring coagulation studies, and assessing the patient for multisystem involvement, altered tissue perfusion, and fluid volume deficits.[16] Nursing care includes monitoring respiratory status, administering IV fluids to prevent hypovolemia, assessing hemodynamic values, and administering and evaluating antibiotics, blood replacement products, and antipyretics.[14] (See Chapter 49 for a more detailed discussion of DIC.)

Amniotic Fluid Embolism

Amniotic fluid embolism (AFE), although rare, is responsible for approximately 10% of maternal deaths in the United States.[10] AFE occurs when amniotic fluid gains entry into the maternal circulation. This entry may occur during cesarean birth or uterine rupture or through small tears in the endocervical veins during a vaginal delivery. Once amniotic fluid enters the maternal circulation, it is rapidly transported to the pulmonary vasculature, resulting in pulmonary emboli. The pulmonary response to AFE is vasospasm, which produces transient pulmonary hypertension and profound hypoxia. The maternal system becomes hemodynamically compromised, similar to anaphylactic shock, with elevated PAP and left ventricular failure.[10] Predisposing factors that may lead to AFE include preeclampsia, multiple gestation, polyhydramnios (excess amniotic fluid), low insertion of placenta, postterm pregnancy, hypertonic contractions during labor, abruptio placentae, uterine rupture, maternal seizures, and umbilical cord prolapse. Clinical manifestations of AFE include sudden onset of dyspnea, cyanosis, and hypotension, followed by cardiopulmonary arrest.

Management

Management of AFE is directed at maintaining left ventricular output and an adequate airway.[10] Interventions include intubation and ventilation with 100% oxygen, IV administration of vasopressors and crystalloid fluids, cardiopulmonary resuscitation (CPR), administration of blood products, and pulmonary artery catheterization. In extreme cases, extracorporeal membrane oxygenation (ECMO) may be used to provide adequate oxygenation and ventilatory support during treatment of the AFE. Potential sequelae include acute pulmonary edema, respiratory distress, DIC, hemorrhage, and multisystem failure.

The nurse must react quickly when AFE is suspected. Following the basic ABCs (airway, breathing, and circulation), the nurse can prioritize interventions needed in this stressful situation. If the patient is not intubated, oxygen must be administered using a face mask, and oxygen saturation assessed using a pulse oximeter. The nurse can anticipate that after intubation, a resuscitation bag or mechanical ventilation will be needed. To maximize venous return, the woman should be positioned on her side or a wedge placed under her hip. A large-bore IV line is needed to administer IV fluids and blood products and to correct hypotension. Assessment focuses on the cardiovascular, pulmonary, hematological, and neurological systems.

Acute Respiratory Distress Syndrome

ARDS is characterized by progressive respiratory distress, severe hypoxemia, low lung compliance, noncardiogenic pulmonary edema, and diffuse infiltrates on chest radiography.[1] Precipitating factors of ARDS associated with pregnancy include abruptio placentae, severe preeclampsia, pyelonephritis, DIC, sepsis, AFE, aspiration, systemic infections, and fetal death in utero. Maternal hypoxemia can lead to spontaneous labor and fetal hypoxia, acidosis, and death; therefore, it should be aggressively managed. Perfusion to vital organs, including the fetus, must be maintained to reduce morbidity and mortality. Adequate fetal oxygenation requires a maternal arterial oxygen saturation (SaO_2) of at least 95%.

Management

Pregnant women with ARDS require cardiovascular support and mechanical ventilation using positive end-expiratory pressure (PEEP). Hemodynamic monitoring is essential for evaluating changes associated with ARDS, such as central hypovolemia and noncardiogenic pulmonary edema. Ventilator settings include a rate of 12 breaths/min, a tidal volume of 12 to 15 mL/kg body weight, 100% oxygen, and a PEEP of 5 cm H_2O.[1]

Nursing care of pregnant women with ARDS is primarily supportive. Interventions are directed at optimizing oxygen transport to tissues and restoring pulmonary capillary integrity. A complete respiratory assessment is made, including evaluation of SaO_2 using pulse oximetry; observation of respiratory rate, character, and effort; and auscultation of lungs. The symptoms of noncardiogenic pulmonary edema are similar to those of cardiogenic pulmonary edema (ie, tachypnea, tachycardia, rales, shortness of breath); however, the nurse should be aware that a decrease in PAOP and PAP (from the obstetrical normal; see Table 11-4, p. 126) may indicate noncardiogenic pulmonary edema. Noncardiogenic pulmonary edema in obstetrical patients can be caused by aspiration of gastric contents, sepsis, blood transfusion reactions, DIC, and AFE.

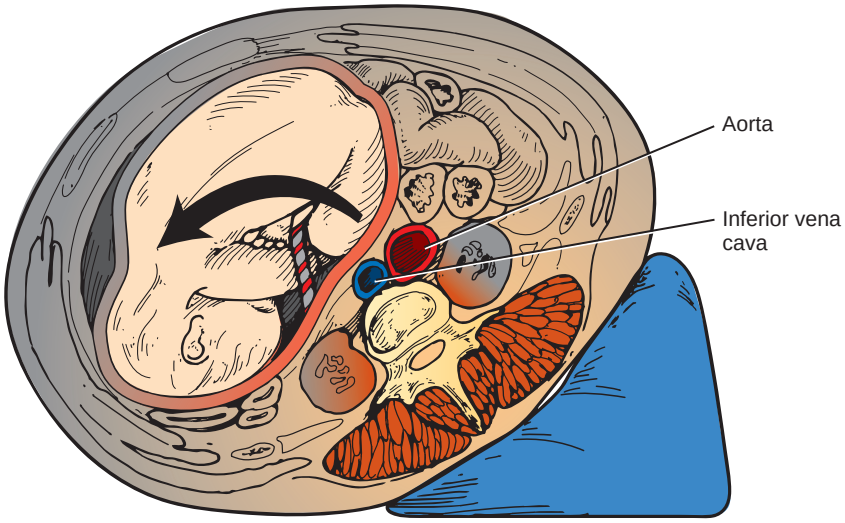


FIGURE 11-2 ▲ Placing a wedge under the woman's right hip decompresses the major abdominal vessels, maximizing the cardiopulmonary resuscitation effort.

Nursing interventions to improve uteroplacental blood flow include positioning the patient on her side and maintaining adequate fluid volumes.[11]

Nursing care for women who are mechanically ventilated includes psychosocial support to help relieve anxiety, fear, and separation from family. Nurses can facilitate communication between the patient and family and keep them informed about maternal and fetal conditions. In extreme cases of ARDS in the postpartum period, ECMO may be used to provide adequate oxygenation and ventilatory support.

Trauma

Accidental injuries occur in 6% to 7% of all pregnancies and are associated with spontaneous abortion, preterm labor, abruptio placentae, and fetal death. Trauma is the leading cause of nonobstetrical maternal death.[2] Common types of trauma include blunt trauma from motor vehicle crashes (49%), falls, and domestic violence (18% to 25%) and penetrating trauma from stab wounds or gunshots (4%).[17] Fetal survival depends on maternal survival, so immediate care and stabilization of the pregnant woman are essential.

Hemodynamic instability may not be initially apparent because of the normal physiological cardiovascular changes during pregnancy. A pregnant woman can lose up to 2,000 mL of blood before becoming hemodynamically unstable.[2]

Management

Management consists of immediate stabilization and care. Immediate stabilization of all trauma patients consists of applying the ABCs of resuscitation: establishment of airway, breathing, and circulation. First an airway is established, and oxygen is provided at a rate of 10 to 12 L/min to produce a PaO_2 level of 60 mm Hg or higher. This PaO_2 level is necessary for optimal fetal oxygenation. A nasogastric tube should be inserted to avoid aspiration.

If CPR is necessary, the anatomical and physiological changes in pregnancy must be considered to maximize efforts. The uterus compresses major abdominal vessels and displaces abdominal contents, which decreases chest compliance. Placing a wedge under the woman's right hip displaces the uterus and decompresses the vessels (Fig. 11-2). This intervention can increase cardiac output up to 30%.[2] Standard advanced cardiac life support procedures are used, including defibrillation and most drugs. The administration of vasopressors should be avoided because the vasoconstrictive action can impair uteroplacental perfusion.[21] Medical antishock trousers or pneumatic antishock garment equipment is rarely used; however, if they are employed, the abdominal compartment should not be inflated.[18]

IV access using large-bore catheters is needed, and aggressive IV infusions are used to increase stroke volume and maintain cardiac output. If hemorrhage occurs, bleeding must be controlled. A decrease in arterial blood pressure may not be an indication of hypovolemia because of low resistance in the uteroplacental system. A 30% to 35% loss can occur with severe consequence to the fetus before hypotension is noted. Fluid replacement therapy must be administered at a higher rate.[2]

Once the pregnant woman has been stabilized, her neurological status is assessed. After this assessment, a fetal assessment, including determination of life, is made. The fetal heart rate can be auscultated using a fetoscope, stethoscope, fetal Doppler, or ultrasonography. Additional assessments can be made on arrival at the hospital or trauma center. These assessments include electrocardiography, a complete physical examination, and laboratory tests, such as arterial blood gases (Table 11-5), complete blood count, platelet count, electrolytes, blood type and crossmatch, and the Kleihauer-Betke test. Used to detect fetomaternal hemorrhage, the Kleihauer-Betke

Table 11-5 Arterial Blood Gas Values in Nonpregnant and Pregnant Women

	Nonpregnant	Pregnant
PaO_2	80–100 mm Hg	87–106 mm Hg
$PaCO_2$	36–44 mm Hg	27–32 mm Hg
pH	7.35–7.45	7.40–7.47
HCO_3^-	24–30 mEq/L	18–21 mEq/L

test identifies red blood cells from the fetus that have entered the maternal circulatory system. This is primarily of concern in the Rh-negative pregnant patient.[2] Assessments should include parameters such as the onset of regular contractions (indicating labor may have begun), vaginal bleeding, and leakage of fluid from the vagina (indicating ruptured membranes).

▲ Providing Emotional Support

Emotional support is very important to all critically ill pregnant women and their families. If the woman labors in the ICU, her coach or significant other should be allowed to remain at the bedside. When she gives birth, breastfeeding and bonding can be encouraged when feasible. The mother needs access to her newborn and family during this time. If the newborn is not able to be at the bedside, frequent updates about the newborn are important and can be provided by the staff. Providing a flexible and individualized atmosphere to a new family is a challenge in the ICU. The importance of coordinating obstetrical and critical care cannot be overemphasized. Box 11-5 outlines strategies for promoting emotional well-being in high-risk pregnancies.

BOX 11-5 Strategies for Promoting Emotional Well-Being in High-Risk Pregnancies

- Shift orientation from the health care team to family centered care.
- Incorporate cultural beliefs into the environment. Maintain family rituals when possible.
- Understand the role of the pregnant woman and her family members and assist them with their tasks to optimize family function.
- Provide names of family support groups.
- Provide information and education to family members.
- Encourage the family when they are coping well.
- Validate the family's emotions.

If the fetus dies as a result of maternal complications, grief support may be needed. The nurse may collaborate with Labor and Delivery staff (who may have additional grief training), psychiatric liaison nurses, social workers, psychologists, psychiatrists, or clergy to offer emotional support to a grieving mother and family.

▲ Clinical Applicability Challenges

CASE STUDY

Ms. W. is a 41-year-old para 2032 (two previous full-term pregnancy, two children are living, three miscarriages) admitted at 37 weeks and 4 days of gestation for an induction of labor due to oligohydramnios (very low amniotic fluid). Until recently, she received routine prenatal care, and the pregnancy was uneventful. Her obstetrical history is significant for a cesarean section for breech presentation, a vaginal birth, and three spontaneous miscarriages. Once Ms. W. was admitted, an external fetal monitor was applied, an intravenous (IV) line was inserted, and IV infusion of Pitocin was begun for induction of labor. Her vital signs were BP 121/74, respiration rate 20, pulse rate 79, and temperature 37°C. Several hours later, it was decided that Ms. W. would need a cesarean section for failure to dilate. After the baby was delivered by cesarean section, excessive bleeding was noted on the right side of the uterine incision. Despite attempts to control hemostasis by suturing various areas that were extensions of the surgical incision, the uterus became atonic and a cesarean hysterectomy was necessary. The patient continued to bleed with an estimated 7-liter blood loss. Ms W.'s hematocrit was 23.1%, hemoglobin was 8.3 g/dL, platelet count was 49, fibrinogen was 113 mg/dL, prothrombin time was 15.1 seconds, and activated partial thromboplastin time was 59.4. She received 12 units of packed red blood cells (PRBCs), 7 units of fresh frozen plasma, 1 pack of platelets, and factor VII in the operating room. Packing with Gelfoam was performed and the patient was transferred immediately to the surgical intensive care unit (SICU).

On admission to the unit, Ms. W. was tachycardic (145 beats/min), hypotensive (BP 80/40), and tachypneic (36 breaths/min). Her urine output was less than 30 mL in an hour. She was unconscious and intubated. Her lab results did not show improvement initially and additional fresh frozen plasma, platelets, and factor VII were administered. She was diagnosed with DIC. Her vital signs and lab results improved over the first 24 hours and by postoperative day 3, she was transferred to the postpartum unit. On postoperative day 4, she was ambulating, voiding, and tolerating a regular diet. She was discharged home on postoperative day 5.

1. What signs and symptoms led to the diagnosis of DIC?
2. How can the SICU staff provide a family centered environment for this patient?
3. What outcomes would be expected following the successful response to the therapy provided?

References

1. Price L, Slack A, Nelson-Piercy C: Aims of obstetric critical care management. Best Pract Res Clin Obstet Gynecol 22(5):775–799, 2008
2. Ruffolo D: Trauma care and managing the injured pregnant patient. J Obstet Gynecol Neonatal Nurs 38(6):704–714, 2009
3 Criddle L: Trauma in pregnancy. Am J Nurs 109(11):41–47, 2009
4. Carlin A: Physiological changes of pregnancy and monitoring. Best Pract Res Clin Obstet Gynecol 22(5):801–823, 2008
5. Madappa T: Alterations in pulmonary physiology during pregnancy. Pulmonary Disease and Pregnancy. WebMD, 2009
6. Madan I, Puri I, Jain N, et al: Characteristics of obstetric intensive care unit admissions in New Jersey. J Matern Fetal Neonatal Med 22(9): 785–790, 2009
7. Lykke J, Langhoff-Roos J, Sibai B, et al: Hypertensive pregnancy disorders and subsequent cardiovascular morbidity and type 2 diabetes mellitus in the mother. Hypertension 53:944–951, 2009
8. Haddad B, Sibai B. Expectant management in pregnancies with severe pre eclampsia. Semin Perinatol 33:143–151, 2009
9. Yoder S, Thornburg L, Bisognana, J: Hypertension in pregnancy and women of childbearing age. Am J Med 122:890–895, 2009
10. Williams J, Mozurkewich E, Chilimigras J, et al: Critical care in obstetrics: pregnancy related conditions. Best Pract Res Clin Obstet Gynecol 22(5):825–846, 2008
11. Bauer S, Cleary K: Cardiopulmonary complications of pre-eclampsia. Semin Perinatol 33(3):158–165, 2009
12. Habli M, Sibai B: Hypertensive disorders of pregnancy. In Gibbs R, Karlan B, Haney A (eds): Danforth's Obstetrics and Gynecology, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008
13. Bridges E, Womble S, Wallace M, et al: Hemodynamic monitoring in high risk obstetrics patients: I. Cardiovasc Med 23(4):53–62, 2003
14. Mcoy S, Baldwin K: Pharmacotherapeutic options for the treatment of preeclampsia. Am J Health Syst Pharm 66:337–344, 2009
15. Haram K, Svendsen E, Abildgaard U: The HELLP syndrome: Clinical issues and management. A review. BMC Pregnancy Childbirth 9(8):1–15, 2009
16. Thacil J, Toh C: Disseminated intravascular coagulation in obstetric disorders and its acute haematological management. Blood Rev 23:167–176, 2009
17. Mirza F, Devine P, Gaddipati S: Trauma in pregnancy: A systematic approach. Am J Perinatol 27(7):579–586, 2010
18. Hak D, Smith W, Susuki T: Management of hemorrhagic in life threatening pelvic fractures. J Am Acad Orthop Surg 17(7):447–457, 2009

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

12 The Critically Ill Older Patient

Barbara Resnick

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Explain general physical changes that occur as a result of the normal aging process.
2. Describe the developmental tasks of the older person.
3. Discuss specific conditions that affect the major body systems of the older person.
4. Explain cognitive changes that may occur in the older patient.
5. Compare and contrast delirium and dementia in the older patient.
6. Describe assessment indicators of potential abuse or neglect of the older person.
7. Describe why the principle *start low, go slow* is important for the older patient in regard to the absorption, distribution, metabolism, and excretion of medications.

America is growing older. Between 2002 and 2030, the older population will more than double, increasing to 71.5 million from 35.6 million (Fig. 12-1). Almost one in five people will be 65 years old or older,[1] and more people will seek medical care for chronic disease, the leading cause of disability among older adults. When older adults have acute exacerbations of their diseases, they often require hospitalization in an intensive care unit (ICU).

As a result, critical care nurses need to understand the many physiological changes that occur normally with aging. These alterations are progressive and usually are not apparent or pathological. However, these age-related changes put the critically ill older adult at increased risk for complications. Preventive nursing care, focused on avoiding potential problems, is necessary.

The leading causes of death among older patients are heart disease, malignant neoplasms, cerebrovascular accidents (CVAs), influenza, and chronic obstructive pulmonary disease. Chronic conditions (eg, arthritis, hearing and visual deficits) are prevalent among older people. With advancing age, these conditions become more common and result in increased hospitalizations. A longer life span has been the single most important cause of the increased numbers of older people with multiple chronic and acute illnesses.

▲ Normal Psychobiological Characteristics of Aging

Biological Issues

Intrinsic aging refers to characteristics and processes that occur universally with all older adults. Changes resulting from the aging process must be distinguished from those resulting from a particular disease process, disuse, or environmental factors, such as ultraviolet radiation. Extrinsic aging is composed of factors that influence aging to varying degrees in different people. Extrinsic factors include such things as lifestyle or exposure to environmental influences. Normal aging is defined as the sum of intrinsic aging, extrinsic aging, and idiosyncratic or individual genetic factors specific to each individual.[2]

In most physiological systems, the normal aging processes do not result in significant impairment or dysfunction in the absence of disease and under resting conditions. It is only in response to stress that an age-related reduction in physiological reserves causes a loss of homeostatic balance. The following are some examples of intrinsic age changes:

- Reduced resistance to stress
- Poor tolerance of extremes of heat and cold because of hypothalamic and skin changes
- Reduced sensory perceptions
- Greater fluctuation in blood pH

Aging, in one organ or the entire body, may be premature or delayed in relation to actual chronological age. The effect of aging on cellular tissues is asymmetrical. For example, the changes resulting from aging in relation to the brain, bone, cardiovascular, and lung tissues may be fairly obvious, whereas changes affecting the liver, pancreas, gastrointestinal tract, and muscle tissues are less obvious. Several organic changes that result from aging are listed in Box 12-1.

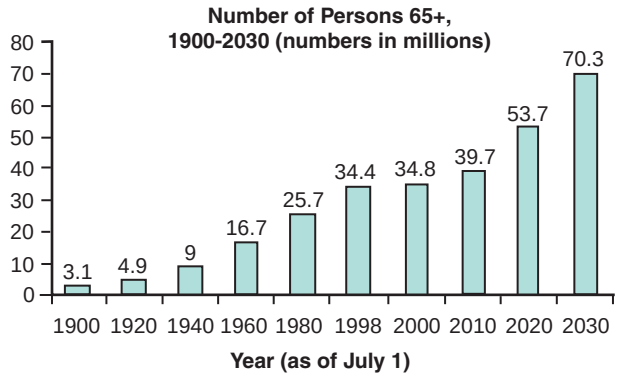


FIGURE 12-1 ▲ Profile of Americans age 65 years and older (based on data from the United States Bureau of the Census). Data from the year 1900 to present were used to predict the number of Americans aged 65 years and older in the year 2030. (From www.aoa.gov/aoa/stats/profile/default.htm.)

Psychosocial Issues

In addition to physical signs of aging, nurses caring for acutely ill older patients must be aware of the older person's normal developmental tasks and the specific dreams or wishes of a particular senior. Developmental tasks of older people are listed in Box 12-2.

The need for support and meaningful relationships continues throughout life. Support can be described as a feeling of belonging or a belief that one is an active participant in the surrounding world. The feeling of mutuality with others in the environment lends strength and helps decrease the sense of isolation. Support by family, friends, and the community can provide an older patient with a greater sense of stability and security.

Self-worth and perceived well-being are feelings that usually coincide in older adults. The perception of well-being arises from the satisfaction of meeting an acceptable proportion of one's life goals. It can be described as an inner contentment one has in life as a whole. Related to this, a feeling of self-worth is derived not only from a sense of well-being but also from satisfaction with one's image or acceptance by others. Self-worth also reflects the quality of interactions with family and friends.

Family environment for the older adult includes, among others, dimensions of interpersonal relationships, personal growth, integrity of the family unit, and adaptation to stress. As family members age, all these areas of concern intensify because of changes in roles of family members, alterations in the family power structure, and changes in financial and decision-making dynamics. Acute illness increases the urgency for effective cooperation among all family members as the traditional family structure is suddenly challenged.

When older patients are admitted to intensive care, issues of family cohesion and adaptability often surface. Frequently, families face immediate changes in roles, with adult children and grandchildren assuming the roles of caretakers and nurturers for the older family members. The family must suddenly adjust to dramatically different demands. Frequent visits to the hospital; dialogues with nurses, physicians, and social workers; and efforts to support and communicate with the patient become primary tasks. Amid these activities, family members (particularly those who have been given power of attorney) find themselves being pressed for decisions about immediate and long-term care. At this time, the issue of the person's end-of-life care preferences, competency, and ability to be involved in treatment decisions may arise. Effective communication and a willingness to listen to and respect the wishes of the older patient become foremost. If this is achieved, the stress on families is reduced because of increased acceptance of the plan of care by all family members.

BOX 12-2 Developmental Tasks of the Older Person

- Deciding where and how to live for his or her remaining years
- Preserving supportive, intimate, and satisfying relationships with spouse, family, and friends
- Maintaining an adequate and satisfying home environment relative to health and economic status
- Providing sufficient income
- Maintaining a maximum level of health
- Attaining comprehensive health and dental care
- Maintaining personal hygiene
- Maintaining communication and adequate contact with family and friends
- Maintaining social, civic, and political involvement
- Initiating new interests (in addition to former activities) that increase status
- Recognizing and feeling that he or she is needed
- Discovering the meaning in life after retirement and when confronted with illness of self or spouse and death of spouse and other loved ones; adjusting to death of loved ones
- Developing a significant philosophy of life and discovering comfort in a philosophy or religion

BOX 12-1 Organic Changes With Aging

- The amount of connective and collagen tissue is increased.
- Cellular elements in the nervous system, muscles, and other vital organs disappear.
- The number of normally functioning cells is reduced.
- The amount of fat is increased.
- Oxygen use is decreased.
- During rest, the amount of blood pumped is decreased.
- Less air is expired by the lungs.
- Excretion of hormones is decreased.
- Sensory and perceptual activity is decreased.
- Absorption of lipids, proteins, and carbohydrates is decreased.
- Presbyesophagus occurs.
- The arterial lumen thickens.

▲ Physical Challenges

Chronic changes in one organ system may be associated with changes in other systems. Moreover, there is individual variation in age-related changes. Therefore, each person must be evaluated based on the age-related changes actually present rather than on those that are "normal" for a particular age.

It is equally important to distinguish age-related changes from those associated with a chronic disease or acute illness and to avoid prematurely attributing some findings to age if they are caused by illness. A discussion of the effects of aging on various body systems follows; age-related changes and the clinical implications of the changes are summarized in Table 12-1.

Table 12-1 Summary of Age-Related Changes, Clinical Implications, and Key Nursing Interventions

System	Clinical Implications	Key Nursing Interventions
Cardiovascular		
• Atrophy of muscle fibers that line the endocardium • Atherosclerosis of vessels • Increased systolic blood pressure • Decreased compliance of the left ventricle • Decreased number of pacemaker cells • Decreased sensitivity of baroreceptors	• Increased blood pressure • Increased emphasis on atrial contraction with an S_4 heard • Increased dysrhythmias • Increased risk for hypotension with position change • Valsalva maneuver may cause a drop in blood pressure • Decreased exercise tolerance	• To prevent falls related to positional hypotension, make sure the person changes position slowly and waits before ambulating.
Neurologic		
• Decreased number of neurons and increase in size and number of neuroglial cells • Decline in nerves and nerve fibers • Atrophy of the brain and increase in cranial dead space • Thickened leptomeninges in spinal cord	• Increased risk for neurologic problems: CVA, parkinsonism • Slower conduction of fibers across the synapses • Modest decline in short-term memory • Alterations in gait pattern: wide based, shorter stepped, and flexed forward • Increased risk for hemorrhage before symptoms are apparent	• To compensate for the decline in short-term memory, provide more time to complete memory-associated tasks.
Respiratory		
• Decreased lung tissue elasticity • Thoracic wall calcification • Cilia atrophy • Decreased respiratory muscle strength • Decreased partial pressure of arterial oxygen (PaO_2)	• Decreased efficiency of ventilatory exchange • Increased susceptibility to infection and atelectasis • Increased risk for aspiration • Decreased ventilatory response to hypoxia and hypercapnia • Increased sensitivity to narcotics	• To prevent infection and atelectasis, encourage deep breathing and coughing.
Integumentary		
• Loss of dermal and epidermal thickness • Flattening of papillae • Atrophy of sweat glands • Decreased vascularity • Collagen cross-linking • Elastin regression • Loss of subcutaneous fat • Decreased melanocytes • Decline in fibroblast proliferation	• Thinning of skin and increased susceptibility to tearing • Dryness and pruritus • Decreased sweating and ability to regulate body heat • Increased wrinkling and laxity of skin • Loss of fatty pads protecting bone and resulting in pain • Increased need for protection from the sun • Increased time for wound healing	• To prevent damage to fragile skin, avoid shearing forces. • To counteract dryness, immerse skin in water daily and apply emollients. • To minimize pain, pad thinned areas with additional layers (eg, extra socks for feet). • Encourage sunscreen use.
Gastrointestinal		
• Decreased liver size • Less efficient cholesterol stabilization and absorption • Fibrosis and atrophy of salivary glands • Decreased muscle tone in bowel • Atrophy of and decrease in number of taste buds • Slowing in esophageal emptying • Decreased hydrochloric acid secretion • Decreased gastric acid secretion • Atrophy of the mucosal lining • Decreased absorption of calcium	• Change in intake due to decreased appetite • Discomfort after eating related to slowed passage of food • Decreased absorption of calcium and iron • Alteration of drug effectiveness • Increased risk for constipation, esophageal spasm, and diverticular disease	• Encourage small, frequent meals to avoid discomfort and improve intake. • Encourage fluids and fiber to improve bowel function.

(continued on page 135)

Table 12-1 **Summary of Age-Related Changes, Clinical Implications, and Key Nursing Interventions (continued)**

System	Clinical Implications	Key Nursing Interventions
Urinary		
• Reduced renal mass • Loss of glomeruli • Decline in number of functioning nephrons • Changes in small vessel walls • Decreased bladder muscle tone	• Decreased GFR • Decreased sodium-conserving ability • Decreased creatinine clearance • Increased BUN • Decreased renal blood flow • Altered drug clearance • Decreased ability to dilute urine • Decreased bladder capacity and increased residual urine • Increased urgency	• To prevent complications from medical therapy, monitor drug clearance and alter dosing as necessary. • Monitor for UTI
Reproductive		
• Atrophy and fibrosis of cervical and uterine walls • Decreased vaginal elasticity and lubrication • Decreased hormones and reduced oocytes • Decreased seminiferous tubules • Proliferation of stromal and glandular tissue • Involution of mammary gland tissue	• Vaginal dryness and burning and pain with intercourse • Decreased seminal fluid volume and force of ejaculation • Reduced elevation of the testes • Prostatic hypertrophy • Connective breast tissue is replaced by adipose tissue, making breast examinations easier	• To compensate for vaginal dryness or pain, encourage the use of lubricating creams, estrogen cream, or both. • Monitor for urinary retention in men.
Musculoskeletal		
• Decreased muscle mass • Decreased myosin adenosine triphosphatase activity • Deterioration and drying of joint cartilage • Decreased bone mass and osteoblastic activity	• Decreased muscle strength • Decreased bone density • Joint pain and stiffness • Loss of height • Increased risk for fracture • Alterations in gait and posture	• Encourage resistive exercises to reverse a decline in muscle strength. • Encourage exercise and intake of calcium and vitamin D. • Encourage activity and exercise.
Sensory		
Vision		
• Decreased rod and cone function • Pigment accumulation • Decreased speed of eye movements • Increased intraocular pressure • Ciliary muscle atrophy • Increased lens size and yellowing of the lens • Decreased tear secretion	• Decreased visual acuity, visual fields, and light/dark adaptation • Increased sensitivity to glare • Increased incidence of glaucoma • Distorted depth perception with increased falls • Less able to differentiate blues, greens, and violets • Increased eye dryness and irritation	• Provide materials with large print. • Make sure there is adequate lighting without glare. • Use contrasting colors for print material.
Hearing		
• Loss of auditory neurons • Loss of hearing from high to low frequency • Increased cerumen • Angiosclerosis of ear	• Decreased hearing acuity and isolation (specifically, decreased ability to hear consonants) • Difficulty hearing, especially when there is background noise, or when speech is rapid • Cerumen impaction may cause hearing loss.	• Make sure to face the person; use touch and visual cues to facilitate communication. • Evaluate for cerumen impaction, and remove cerumen as necessary.
Smell		
• Decreased number of olfactory nerve fibers	• Inability to smell noxious odors • Decreased fowod intake	• Present odors the patient can smell. The ability to smell fruity odors, for example, are retained. Also use spices in foods to augment the sense of taste.

(continued on page 136)

Table 12-1 **Summary of Age-Related Changes, Clinical Implications, and Key Nursing Interventions (continued)**

System	Clinical Implications	Key Nursing Interventions
Taste		
• Altered ability to taste sweet and salty foods; bitter and sour tastes remain		• Use alternative seasonings.
Touch		
• Decreased sensation	• Safety risk with regard to recognizing dangers in the environment: hot water, fire, small objects on floor that may result in tripping	• Avoid a cluttered environment.
Endocrine		
• Decreased testosterone, GH, insulin, adrenal androgens, aldosterone, and thyroid hormone • Decreased thermoregulation • Decreased febrile response • Increased nodularity and fibrosis of thyroid • Decreased basal metabolic rate	• Decreased ability to tolerate stressors such as surgery • Decreased sweating and shivering and temperature regulation • Lower baseline temperature; infection may not cause an elevation in temperature • Decreased insulin response, glucose tolerance • Decreased sensitivity of renal tubules to ADH • Weight gain • Increased incidence of thyroid disease	• Monitor the temperature of the room. • Provide adequate clothing and blankets to keep the patient warm. • Closely monitor blood glucose levels of patients with diabetes.
Immune		
• Decline in both T-cell and B-cell function • The decreased numbers of B cells secreting immunoglobulin G • The thymus gland involutes, and there is a decrease in thymic hormone levels. • The number of autoantibodies increases.	• Poor immune response and risk for infection	• There is some evidence that exercise helps to boost the immune system and should be encouraged. Also, meticulous use of universal precautions and infection control techniques should be encouraged.

Auditory Changes

With age, there is a change in the shape of the ear: the auricle becomes elongated and broader, the cartilage is less elastic and less flexible, and tophi may appear on the pinna. The hairs on the external ear canal become longer and coarser, the tympanic membrane is thicker and more fixed, and there are fewer cerumen glands (leading to thicker, drier cerumen). In the cochlea, hair cells, neuron-supporting cells, ganglion cells, and fibers are decreased, causing decreased hearing and balance. An estimated 7 million people older than 65 years have significant hearing loss, and continuation of current trends indicates that in 2010, more than 11 million people have this problem.[3] Specifically, the aging process affects hearing in two critical ways: reduction in threshold sensitivity and reduction in the ability to understand speech. Threshold elevations that occur between 8,000 and 20,000 Hz are not detectable with a routine hearing test. Therefore, hearing loss because of aging or other factors is not documented clinically until frequencies are at or below 8,000 Hz.

Presbycusis is a sensorineural hearing loss and is the most common form of hearing loss in older adults. Presbycusis is characterized by a gradual, progressive, bilateral, symmetrical, high-frequency sensorineural (perceptive) hearing loss with poor speech discrimination. Sensorineural hearing loss is due to degeneration or changes in the neural receptors in the cochlea, cranial nerve VIII (the acoustic nerve), and central nervous system. Treatment may vary dramatically from simple removal of impacted earwax to surgical removal of an auditory nerve tumor. Thirteen percent of people 65 years of age and older, if tested, would show signs of presbycusis.[3]

Conductive hearing loss is due to the blockage of sound transmission from the external ear through the tympanic membrane and small bones in the middle ear. Like presbycusis, conductive hearing loss is commonly found in older adults, and it is not unusual for older people to have both sensorineural and conductive hearing loss.

Findings on Physical Examination and Management

The ear canal of the older adult should be evaluated at regular intervals (every few months) because of the tendency to have thicker, drier cerumen, which can occlude the canal and affect hearing. The older patient may retain the ability to hear pure tones, but if these pure tones are grouped to form words, the ability to understand and perceive these sounds as intelligible speech may be lost. This loss is known as impairment of discrimination ability. The patient has increased difficulty hearing high-frequency, stimuli-sibilant sounds

(-f-, -s-, -th-, -ch-, and -sh-). Noisy environments further hamper the ability to hear certain sounds. Therefore, the person may respond inappropriately to questions, withdraw, or frequently ask for the speaker to repeat what is said. Eliminating background noise, speaking lower and louder, and using multiple means of getting information across (eg, verbal as well as written formats) can facilitate communication. People with impaired discrimination may also have problems with balance during transfers and ambulation, and they experience frequent falls. Activity and exercise interventions should be implemented as soon as possible to strengthen muscles and bones and improve balance.

Visual Changes

Like all other body systems, the eye is affected by aging. Structural and functional changes occur slowly and gradually. Visual perception depends on the integration of various neurosensory systems and structures that age at different rates.

Normal changes associated with aging may include a loss of elasticity in the eyelids and subsequent wrinkling, ptosis (upper eyelid drooping), and "pouches" resulting from changes in the tissues beneath the eyelid skin and the subsequent formation and accumulation of fatty tissue. The conjunctiva may develop a yellowish or discolored membrane or become thickened as a result of environmental hazards, such as dust and exposure to drying and irritating pollutants. Arcus senilis, which is a white or gray ring around the limbus (junction of the cornea and sclera), may be related to a high blood level of fatty substances accumulated with advancing age. Although there is a decrease in the amount of lacrimation with age, overflow of tears may occur because of impaired drainage of the ductal system.

The iris loses its ability to accommodate rapidly to light and dark and develops an increased need for light. With age, the pupil becomes smaller and fixed. The lens becomes inflexible with less complete accommodation for near and far vision. The vitreous humor behind the lens may pull on the retina, producing holes or tears and predisposing the older person to retinal detachment. The ciliary muscle becomes stiff, which contributes to the problems of accommodating to distances. By the age of 60 years, presbyopia may develop. Presbyopia is the inability to shift focus from far to near. A possible rationale for this loss is that the older, aging lens, which is less flexible, cannot easily change shape from the action of the focusing muscle to which it is attached.

In older adults, dark–light adaptation slows as the pupillary response slows and rods degenerate. As the lens yellows with increasing age, color discrimination becomes less acute, especially in the blue-green tones. Peripheral vision may decline because of decreased extraocular muscle strength, and depth perception may decline because of a thickening lens. Therefore, time must be provided for the person to adapt when moving between dark and light environments and when getting out of bed.

In addition to normal changes in vision, there is an increased incidence of cataracts, glaucoma, senile macular degeneration, and diabetic retinopathy. These diseases must be studied in relation to normal aging of the eye structure. A cataract is a clouding of the normally clear and transparent lens of the eye. When a cataract interferes with the transmission of light to the retina, some loss in visual acuity may result. The older patient may complain of increased sensitivity to glare, a blurring of vision, halo images, cloudiness, decreased visual acuity, and decreased contrast sensitivity. Risk factors for cataract formation include diabetes mellitus, heredity, ultraviolet-B radiation exposure, smoking, corticosteroid drugs, alcohol use, and insufficient ingestion of antioxidant vitamins. The visual changes can progress to complete loss of vision. Cataracts account for one sixth of all cases of visual impairment in the United States and mostly occur in people older than 50 years.

Glaucoma is one of the major causes of blindness and is especially prevalent in the older adult. Glaucoma is due to increased intraocular pressure. This pressure may result in compression of the optic disc of the eye and damage to cranial nerve II (the optic nerve). This results in loss of peripheral vision and visual acuity. Risk factors for glaucoma include African American race, a family history of glaucoma, ocular hypertension, advanced age, myopia, retinal vascular disturbance, corticosteroid drug use, diabetes mellitus, and vascular crisis (elevation in blood pressure). Age-related changes in the canal of Schlemm, infection, injury, swollen cataracts, and tumors are also etiological factors for glaucoma. Glaucoma is classified based on whether the angle of the anterior chamber is open or narrow and whether the glaucoma is primary or secondary. Primary, open-angle glaucoma is the most common type found in older adults. This type progresses slowly. Primary, angle-closure glaucoma is less common and is characterized by a sudden and marked increase in intraocular pressure with accompanying redness and pain in the eye, headache, nausea or vomiting, corneal edema, and decreased vision. Secondary glaucoma is characterized by an anatomical or functional blockage of the outflow channels. These types of glaucoma can be open angle (such as those that occur from corticosteroid-induced pressure increases) or closed angle (such as those caused by a swollen cataract). Early diagnosis is important because the earlier that treatment is started, the easier it is to control the disease.

Retinal degeneration, or macular degeneration, is the third major source of visual disability in the older adult. Macular degeneration is a pigmentary change of the macular area of the retina caused by small hemorrhages. People see a gray shadow in the center of the visual area but can see well at the outer border. This condition rarely results in total blindness; however, visual loss can progress to legal blindness. Early symptoms include a slight blurring of vision, followed by a blind spot. Compensation techniques include wearing sunglasses or visors, looking to the side, and using magnifiers.

Diabetic retinopathy is the leading cause of blindness in the United States. It is caused by the deterioration of the blood vessels nourishing the retina at the back of the eye. Microaneurysms and small hemorrhages in the eye may leak fluid or blood and cause swelling of the retina. If this leaking blood or fluid damages or scars the retina, the image sent to the brain becomes blurred, and the condition eventually can progress to blindness.

Findings on Physical Examination and Management

The older adult is likely to have smaller pupils, decreased visual acuity, difficulty with depth perception, decreased peripheral vision, and dry eyes. Ectropion and entropion

BOX 12-3 NURSING INTERVENTIONS

For Visually Impaired Patients

- Identify yourself on approach.
- Approach blind patients from the front.
- Assess impact of failing vision and patient's ability to adapt during hospitalization and after discharge.
- Assess stress level because increased stress can necessitate higher dosages of eye medication for patients with glaucoma.
- Be alert to side effects that other medications may have on the eyes (ie, medications containing antihistamines, caffeine, and atropine-like substances).
- Provide eye lubrications when eyes are dry.
- Instill all prescribed medications.

are commonly noted with age. Ectropion is eversion of the eyelid (usually the lower lid) resulting in exposure of the lid, thickening and keratinization, and chronic irritation. Entropion is inversion of the eyelid and results in the eyelashes rubbing against and scratching the cornea. Entropion can result in corneal trauma and scarring and ultimately may result in decreased vision. When cataracts are present, there is opacity of the lens, and the red reflex may be absent during funduscopic examination. The older adult with cataracts presents with dimming of his or her vision and complains that everything appears clouded. People who have glaucoma present with complaints of blurred vision, halos around lights, or decreased peripheral vision. Funduscopic examination in these people shows cupping of the optic disc and atrophy of the optic nerve. Last, older adults with macular degeneration present with a gradual decline in vision, particularly central vision, without a change in peripheral vision. Good lighting, avoiding glare, and using contrasting colors (eg, black letters on white paper) and large print can facilitate vision. As with interventions for hearing changes, providing information in various ways is effective for facilitating vision and compensating for losses. Selected nursing interventions for people with impaired vision are listed in Box 12-3.

Other Sensory Changes

Although hearing and vision changes are the most researched sensory changes occurring in older people, there also may be declines in other senses. The number of taste buds is reported to decrease with age, in conjunction with a decline in the ability to taste substances. Sweet and salty substances are less detectable as one ages; therefore, many older adults complain that food tastes bitter or sour. There is very little information on smell sensation, but it is thought that a decrease in the sense of smell can result from atrophy of the olfactory organ and increased hair in the nostrils.[4] The loss of taste and smell affects the older person's ability to identify food and make odor discriminations.

The threshold of touch varies with the part of the body stimulated. There is a loss of tactile sensation as one ages, although this varies individually. Older adults may not feel the effects of lying too long in one position. A key nursing intervention is to vary the positions of the immobile older patient. The older adult also has decreased kinesthetic sense, which is the person's awareness of his or her body in space. Decreased kinesthetic sense results in postural instability and difficulty reacting to bodily changes in space.

Findings on Physical Examination and Management

With age, the lips tend to become thin and pale, and the tissue of the oral mucosa is thinner, paler, and less elastic. Small yellow sebaceous glands may be seen in the buccal mucosa. The dorsum and margins of the tongue may have decreased number and size of papillae and may be coated with a thin white film. There may also be increased fissures on the dorsal aspect of the tongue, whereas the undersurface is smooth with a bluish-purple hue from increased varicosities. Taste buds and the submaxillary, pituitary, and salivary glands atrophy. The gums are thinner and receded, and there is decreased tooth enamel, dry and less translucent dentin, decreased dental pulp, and diminished perfusion and sensitivity of the gums. Decreased taste sensitivity (especially for sweets and salt) and increased difficulty swallowing food (due to less saliva production) may cause the older adult to present with weight loss. To facilitate taste and improve oral intake, the nurse provides frequent mouth care (before meals), offers foods in a pleasant setting using liberal seasonings to stimulate taste, and encourages the use of interventions, such as sugar-free candies, to stimulate salivation.

Older adults with decreased sensation may present with complaints of increased difficulty performing fine motor activities such as buttoning clothes or picking up objects. They may also have pressure sores and decreased balance. Frequent position changes of older patients are essential and should be instituted every 30 minutes.

Sleep Changes

It is estimated that sleep disturbances occur in more than half of those older than 65 years.[5] An important aspect of the critical care nurse's assessment is to determine whether sleep problems are the result of normal aging, sleep disorders, or sleep disturbances due to the acute care environment.

Although some age-related changes in sleep patterns are the normal consequences of aging, the prevalence and potential for severe sleep disorders calls for increased clinical awareness and evaluation. Such complaints as habitual snoring, frequent awakening, nocturnal sweating, and awakening with anxiety may be signs of a genuine sleep disorder.

The loss of neurons in the brain may be responsible for the normal age changes in the sleep cycle. These include the following:

- A longer time to fall asleep
- Increased time spent in the lighter stages of sleep (stages 1 and 2)
- Decreased time spent in the deeper stages of sleep (stages 3 and 4) and in rapid eye movement sleep
- Increased and shorter repetitions of the sleep cycle

The amount of sleep needed for each person does not change with age. However, there is an increased tendency for older adults to sleep less at night, to be somnolent late in the day

or early evening, and to awaken early in the morning. This has been referred to as the advanced sleep phase syndrome.[6] Older adults also have shortened sleep latency, resulting in daytime napping. Daytime napping further compounds the problem because it reduces the need for nighttime sleep. Common complaints (eg, anxiety, waking up due to choking, headaches, sweating at night, nocturia, and snoring) are not normal age-related changes and should be assessed more thoroughly.

The most prevalent and most serious age-related sleep disorder is sleep apnea. There is evidence of an association between sleep apnea and circulatory disorders, including hypertension, stroke, and angina pectoris. There also may be a link between sleep apnea and reduced life expectancy. The prevalence of disordered breathing in the older patient is high. Moreover, there may be an association among habitual snoring, stroke, and angina pectoris in older men.[6]

Findings on Physical Examination and Management

Older adults with sleep disorders present with an inability to fall asleep, an inability to stay asleep, or both. They may exhibit daytime napping, and fall asleep during activities. Conversely, there may be evidence of sleep deprivation with altered mental status being the major presenting sign. Loud snoring with multiple apnea–hypopnea events is indicative of sleep apnea. These people may have daytime hypersomnolence, fatigue, irritability, and decreased cognitive function because of impaired nighttime sleep patterns.

Normal aging, chronic illness, and drug therapy increase the older person's susceptibility to insomnia. Treatment depends on the problem. Before drug therapy is considered, poor sleep hygiene habits should be addressed. Good sleep hygiene includes the following:

- Avoiding daytime naps that are longer than 30 minutes
- Maintaining regular bedtimes and rising times
- Avoiding heavy evening meals, excessive fluids, alcohol, and caffeine
- Increasing daytime activity, even if this is as simple as sitting up out of bed for extended periods of time
- Keeping the sleep environment quiet, sufficiently dark, at a comfortable temperature, and safe
- Maintaining a day–night schedule such that the period of time in bed for sleep is separate from daytime activities (although this is difficult in the acute care setting when the person is acutely ill; however, as recovery progresses, the person should be out of bed except for brief rest periods and sleeping at night).

Behavior modification has been used successfully for many sleep problems. The conservative use of medications may be indicated in more problematic sleep disorders, periodic movements of sleep, and dementia-related illness.[7] Drug treatment is best when accompanied by improved sleep hygiene and patient education about age-related changes in sleep.

Care should be taken in dispensing sedative-hypnotics for people with risk factors for sleep apnea. Nursing interventions include encouraging older patients with disordered breathing to sleep on their sides and to lose weight if obese. Other interventions include giving supplemental oxygen if hypoxemia, caused by chronic lung disease or hypoventilation, is present.

Skin Changes

Although a variety of cutaneous changes have been associated with age, some of these changes are due to normal or intrinsic age factors, whereas others are due to chronic solar exposure.[8] Photoaging is the combined effect of repeated sun exposure and intrinsic aging on the skin, and it is the cause of what is generally associated with the clinical (and histological) changes that are consistent with "aging." With age, there is a thinning of the skin and a decrease in skin flexibility. This puts the older adult at risk for epidermal tearing from shearing forces. Likewise, there is a loss of elasticity resulting in fine wrinkling, looseness, and sagging. Over time, there is a decrease in the number of dermal blood vessels, and these blood vessels become thinner and more fragile. These changes result in the hemorrhaging (known as senile purpura) commonly seen in older adults, in impaired body temperature management and healing of wounds, and in decreased absorption of topical treatments. With age, there is decreased density and activity of the eccrine and apocrine glands and decreased sebum production.

Overall, because of a combination of skin changes in older adults, there tends to be a quicker breakdown in the skin barrier and a slower recovery of skin integrity. Common interventions to maintain skin integrity are shown in Box 12-4.

Findings on Physical Examination and Management

The skin of older adults tends to sag, especially on the hands and forearms, and causes underlying tissue injury. The person may appear pale and may not be able to correctly perceive surface temperature (eg, how hot water is). The hair becomes gray and is coarser, and the nails may break and become more brittle. Additional hairs develop on the eyebrows, nose, and ears. Wound healing is prolonged, and there is increased risk for contact dermatitis due to increased skin sensitivity. Xerosis, or dry skin, is a common problem for the older adult and is the most common cause of pruritus in this group. The treatment of dry skin focuses

BOX 12-4 NURSING INTERVENTIONS

For Maintaining Skin Integrity in the Older Adult

- Avoid shearing forces when turning the patient.
- Turn the patient frequently.
- Keep the patient appropriately covered for warmth.
- Bathe the patient daily, preferably with total immersion in water 32.2°C to 40.5°C (90°F to 105°F).
- Apply oil-based emollient to the patient's skin after bathing.
- Monitor responses from transdermal medications.
- Monitor wounds closely for healing and signs and symptoms of infection.

primarily on the replacement of water, which is the major cause of dryness.

Older adults should be encouraged to do the following:

- Maintain a sufficient oral intake of fluid, approximately 2,000 mL of liquids daily.
- Increase bathing time so that there is total-body water immersion 10 minutes daily, with water temperature ranging from 90°F to 105°F (32.2°C to 40.5°C).
- Avoid the use of soaps.
- Use an emollient after bathing.

Skin lesions are more common in the older adult, and certainly any change in a skin growth or any lesion that does not heal in a reasonable time should be suspect for malignancy. Malignant lesions tend to occur in sun-exposed areas but may also be present in other areas.

Cardiovascular Changes

A number of cardiovascular changes occur with aging (see Table 12-1). These age-related changes, overt and occult cardiovascular disease, and reduced physical activity all affect cardiovascular function in elderly people. With age, there is a loss of myocytes in both the left and right ventricles, with a progressive increase in myocyte cell volume per nucleus in both ventricles.[9] With age, there is also a progressive reduction in the number of pacemaker cells in the sinus node, with only 10% of the number of cells at age 20 years still remaining at age 75 years. Aging changes in the heart have an impact on afterload, preload, contractility, diastolic function, and the cardiovascular response to exercise.

Afterload is the resistance to the ejection of blood by the left ventricle and is composed of (1) peripheral vascular resistance and (2) characteristic aortic impedance. With age, the large elastic arteries become dilated, with a reduction in compliance. Progressive thickening of the aortic media and intima is associated with aortic enlargement. There is an age-associated increase in arterial stiffness resulting from changes in the arterial media (eg, thickening of the smooth muscle layers, increased fragmentation of elastin, an increase in the amount and characteristics of collagen, and increased calcification). These structural changes are associated with a reduction in aortic distensibility due to increased aortic stiffness with an increase in pulse wave velocity. The reduction in arterial compliance contributes more to the age-related increase in afterload than does the loss of peripheral vascular beds.

The decrease in vessel compliance affects large and small arteries. As a result, even a small increase in intravascular volume can be accompanied by a substantial rise in aortic pressure (and, in turn, systolic blood pressure), which may lead to pressure-produced ventricular hypertrophy.[10]

Circulating levels of catecholamines increase with age, especially related to stress. Specifically, β-adrenergic vasodilation of vascular smooth muscle decreases with aging, and α-adrenergic vasoconstriction of vascular smooth muscle does not change with aging. The impaired vasodilator response to β-adrenergic stimulation with age is particularly important during exercise.

With aging, there is an increase in systolic blood pressure and a widened pulse pressure. A slight reduction in diastolic blood pressure occurs after the sixth decade.[11] The increase in systolic blood pressure is due to an interaction of many factors, with age being only one of them.

Posterior left ventricular wall thickness increases (ie, left ventricular hypertrophy develops) with age, and this is mediated by an increase in systolic blood pressure.[11] This hypertrophy is due to volume, not the number of cardiac myocytes. Fibroblasts undergo hyperplasia, and collagen is deposited in the myocardial interstitium. Increased afterload causes an increase in left ventricular systolic stress and the addition of sarcomeres. These changes result in an increased left ventricular wall thickness with a normal or decreased left ventricular chamber size and an increased relative wall thickness.

Preload is the filling volume of the left ventricle and is determined by numerous factors that influence blood return to the heart. Resting preload does not change with age[12] although left ventricular early diastolic filling is reduced with age. With age, left ventricular stiffness is increased, left ventricular compliance is decreased, left ventricular wall thickness is increased, left ventricular relaxation is impaired, and left ventricular diastolic filling is decreased. An age-related increase in systolic blood pressure also impairs left ventricular early diastolic filling, leading to hypotension if preload is reduced. Despite this age-related reduction, preload is maintained because left atrial contraction becomes more vigorous and thereby increases late diastolic filling of the left ventricle.[12]

An age-related increase in left atrial size from increased wall stress counteracts the effects of decreased left ventricular compliance with aging. Left atrial contraction can contribute up to 50% of left ventricular filling in a poorly compliant left ventricle. Consequently, in older adults, development of atrial fibrillation may cause a marked reduction in cardiac output because of the loss of left atrial contribution to left ventricular late diastolic filling.

The intrinsic ability of the heart to generate force does not change with age, although the duration of contraction and relaxation is prolonged in older adults. There is no reduction of resting left ventricular ejection fraction or circumferential fiber shortening in healthy older adults.

Aging is associated with prolongation of isovolumic relaxation time, a reduction in early diastolic filling of the left ventricle, and augmentation of the late diastolic filling of the left ventricle.[12] Also with aging, there is a slowing of the rate at which calcium enters the sarcoplasmic reticulum after myocardial excitation, and a subsequent decrease in relaxation of the left ventricle.[13] Reduced oxidative phosphorylation and cumulative mitochondrial peroxidation occurring with aging may also impair left ventricular diastolic function.

The maximum amount of oxygen uptake (VO_2max) decreases with age, although the degree to which oxygen uptake decreases is affected by physical conditioning, subclinical coronary artery disease (CAD), smoking, and body weight. With exercise, older adults have a decrease in heart rate, cardiac index, and left ventricular ejection fraction and increases in the left ventricular end-diastolic and end-systolic volume indices.[14]

Findings on Physical Examination and Management

In the absence of vascular disease, these changes should not interfere with normal tissue perfusion. In the older patient, however, the likelihood of atherosclerosis increases. The narrowing of vessels, coupled with their decrease in compliance, may produce tissue ischemia. These changes, along with bed rest, contribute to tissue injury and pressure ulcer formation.

The increased vascular stiffness of aging causes the upstroke of the arterial pulse to appear brisker than usual, potentially masking the slowly rising carotid pulse of aortic stenosis. Older adults commonly present with an early-peaking basal systolic murmur of aortic stenosis, typically accompanied by an S_4 sound at the cardiac apex as evidence of reduced ventricular compliance. These people report and demonstrate a limited ability to tolerate physical activity. Older patients may also have a greater amount of pooling in the lower extremities because of decreased muscle mass and poor venous return. If the patient is placed on bed rest, this fluid pool is redistributed and may cause an overload in the cardiovascular system. The nurse must be alert for vascular overload and congestive heart failure.

Another factor to consider is the shifting of fluids when a patient arises after having been on bed rest. The sudden shift in fluid to the lower extremities and the lowered fluid volume that results from bed rest can produce extreme lightheadedness. This is further complicated by a decrease in baroreceptor sensitivity with age. A slow progression of head elevation and dangling the legs before moving the patient to sitting or standing is necessary to prevent syncope and possible injury from falling.

Although chest pressure is the classic symptom of angina in older adults, there is an increased incidence of silent ischemia in these people. If angina or a myocardial infarction is suspected, a comprehensive history, vital signs, and electrocardiogram (ECG) should be obtained, and a laboratory workup (including cardiac enzymes) should be ordered. If at all possible, it is important to obtain a prior ECG for comparison.

About half of all older adults have abnormalities on the resting ECG, most commonly PR- and QT-interval prolongation, intraventricular conduction abnormalities, reduction in QRS voltage, and a leftward shift of the frontal plane QRS axis. Common age-related changes to the ECG are shown in Table 12-2. Elderly men more frequently have major ECG abnormalities than do elderly women, and these abnormalities increase with age.[15]

Respiratory Changes

It is particularly difficult to distinguish age-related changes in the structure and function of the lungs from those changes caused by disease because the lungs are continually exposed to environmental stressors. However, some commonly identified lung changes with physiological aging include dilation of alveoli, enlargement of airspaces, a decrease in exchange surface area, and loss of supporting tissue for peripheral airways. These changes result in decreased static elastic recoil of the lung and increased residual volume (RV) and functional residual capacity (FRC). Chest wall compliance decreases and thereby increases the work of breathing for the older adult. The most important age-related change in the large airways is a reduction in the number of glandular epithelial cells. This results in reduced production of protective mucus and thus impaired defense against respiratory infection. There are few changes in the bronchi, but the area of the alveoli decreases and the alveoli and alveolar ducts enlarge. The FRC and RV increase, and compliance decreases. There is deposition of amyloid in lung vasculature and alveolar septa, although the significance of this is unclear. Small airways change so that there is dilation of the alveolar ducts and airspaces and an increased tendency for the small airways to collapse during expiration. There is as much as a 20% reduction in alveolar surface area with consequent reduction in respiratory reserve. The major age-related change in the respiratory muscles is a reduction in the proportion of type II A fibers, with consequent impairment of strength and endurance.

Lung volumes fall gradually with age. Specifically, the forced expiratory volume in 1 second/forced vital capacity (FEV_1/FVC) ratio falls by approximately 0.2% per year.[16] The FEV_1/FVC ratio declines less rapidly in older men than in older women, and maximal expiratory flow and maximum voluntary ventilation decline less rapidly in women. The ventilation/perfusion ratio heterogeneity increases, and carbon monoxide transfer decreases with age.

The most clinically important functional changes in the aging respiratory system are as follows:

- The increased tendency for small airways to collapse sooner during expiration
- Reduction in respiratory muscle strength and endurance
- Changes in the monitoring and control of breathing

Table 12-2 Common Age-Related Changes in the ECG

ECG Variable	Age under 30 y	Age 30–39 y	Age 40–49 y	Age over 49 y
R-wave amplitude (mm)	10.4	10.5	9.0	9.3
S-wave amplitude (mm)	15.2	14.2	12.2	12.4
Frontal plane axis (degrees)	48.9	48.1	36.5	38.8
PR duration (ms)	15.9	16.2	16.0	16.2
QRS duration (ms)	7.6	7.5	7.4	8.0
QT duration (ms)	37.8	37.5	37.9	39.6
T-wave amplitude (ms)	5.2	4.6	4.3	4.4

Data from, Bachman S, Sparrow D, Smith LK. Age-related changes in electrocardiographic variables. Am J Cardiol 48:513, 1981.

There is a tendency for older adults to have relative inefficiency in control and monitoring of ventilation. This is especially true with regard to responses to both hypoxia and hypercapnia at rest. Elderly people tend to have increased ventilatory response to exercise-induced carbon dioxide production.[14]

The VO_2max declines with age owing to a decrease in the diffusing capacity and alveolar capillary volume along with ventilation–perfusion mismatch. Mucociliary clearance is reduced with age, although there is some evidence to suggest that the cough reflex is unaffected by aging.[17] However, older adults tend to have more frequent and severe bacterial, viral, and fungal infections, but this is due to pathological processes commonly seen in older people, changes in other body systems, and some of the functional and structural changes identified.

Pulmonary function studies in healthy older adults have shown an increase in the RV and a decline in the total lung capacity. The actual declines in FVC and maximum expiratory flow rates are attributed more to changes in body weight and strength than to changes in the pulmonary tissue. There tends to be an increase in the partial pressure of arterial carbon dioxide ($PaCO_2$) and a decrease in the partial pressure of arterial oxygen (PaO_2) with age. The gradual decline in PaO_2 is caused by a loss of elastic recoil and a subsequent reduction in airway caliber, early airway closure, and maldistribution of ventilation.

However, despite all these changes, the respiratory system remains capable of maintaining adequate gas exchange at rest and during exertion throughout the life span. Aging does tend to result in diminished reserve of the respiratory system in cases of acute disease. Specifically, decreased sensitivity of respiratory centers to hypoxia or hypercapnia results in a diminished ventilatory response in cases of heart failure, infection, or aggravated airway obstruction. Moreover, decreased perception of bronchoconstriction and diminished physical activity may result in less awareness of the disease and delayed diagnosis.[18]

Findings on Physical Examination and Management

Older adults commonly present with barrel chest (an increased anteroposterior diameter), which has a significant impact on appearance as well as chest wall compliance (particularly in patients who are supine) and may result in lung sounds being more distant and less discernible. It may be helpful to use a pediatric diaphragm to listen for breath sounds in older adults with prominent ribs. This allows for a more firm application of the stethoscope between the interspaces.

Dyspnea is a common complaint, and the causes may be cardiac, pulmonary, metabolic, mechanical, or hematological, or due to deconditioning. Increased lung sounds are also common with age and may be due to deconditioning and age-related fibrotic changes rather than acute disease, such as congestive heart failure. When evaluating the older adult, it is particularly important to match clinical signs and symptoms of disease with objective diagnostic findings, such as chest radiography and laboratory testing.

Pulmonary infections are more common with age because of the changes described previously. However, expectorated tissue specimens in older adults are often unreliable because of pharyngeal colonization. To prevent infection, careful attention to nutrition, especially to sufficient calories, protein, and fluid intake, is needed. Moreover, frequent position changes assist in clearing secretions and aid ventilation and perfusion of the lungs.

Renal Changes

Age-associated kidney changes can be categorized as anatomical or functional. Anatomical changes include the loss of renal glomeruli, decreased kidney size, renal tubular changes, and renal vascular changes. There are also functional kidney changes, as described in Box 12-5.

The total number of glomeruli decreases by 30% to 40% by the eighth decade. The loss of glomeruli, in conjunction with decreased kidney perfusion, results in a decreased glomerular filtration rate (GFR). However, one longitudinal study revealed that not all people exhibit a decline in GFR.[19] Changes are likely due to a combination of lifestyle factors and associated chronic illness. The decrease in filtration may result in decreased clearance of substances normally excreted. An increase in blood urea nitrogen (BUN) or creatinine may indicate the extent to which the GFR is diminished. However, creatinine levels from muscle breakdown may be less than in younger patients and could mask an elevated creatinine level. Creatinine clearance is a more accurate measure of renal function in the older patient. Corrected GFRs can easily be calculated for older adults using the Modification of Diet in Renal Disease formula:

$$\begin{aligned} \text{mL/min/1.73 m}^2 = {} & 175 \times (\text{SCr})^{-1.154} \times (\text{age})^{-0.203} \\ & \times 0.742 (\text{if female}) \\ & \times 1.210 (\text{if black}) \end{aligned}$$

where SCr equals serum creatinine (see also Chapter 31, Box 31-3).

GFR calculators are available to assist with the calculations at http://kidney.org or http://nephron.com/cgi-bin/MDRD.cgi. Evaluation of renal function is extremely important if the patient is receiving drugs normally excreted by the kidney. Table 12-3 provides an overview of normal laboratory values, including renal function, and how these change with age.

BOX 12-5 Age-Associated Functional Kidney Changes

- Decreased GFR
- Decreased mean creatinine clearance rate
- Increased mean creatinine concentrations
- Decreased blood flow through the kidneys
- Decreased tubular transport capacity
- Decreased functional nephrons
- Decreased concentrating ability
- Decreased diluting ability
- Decreased plasma renin activity
- Impaired sodium conservation

Table 12-3 Normal Laboratory Values and Age-Related Changes

Laboratory Test	Normal Values	Age-Related Changes
Urinalysis		
Protein	0–5 mg/100 mL	Rises slightly
Glucose	0–15 mg/100 mL	Glycosuria appears after high plasma levels and is very unreliable
Specific gravity	1.005–1.020	Lower maximum of 1.016–1.022
Sedimentation rate	Men 0–20 mm/h Women 0–30 mm/h	Increases with age; no clinical significance
Iron	50–60 mcg/dL	Slight decrease
Iron binding	230–410 mcg/dL	Decrease
Hemoglobin	Men 13–18 g/100 mL Women 12–16 g/100 mL	No normal decline with age
Hematocrit	Men 45%–52% Women 37%–48%	No normal decline with age
Leukocytes	4,300–10,800/mm^3	No normal decline with age
Lymphocytes	500–2,400 T cells/mm^3 50–200 B cells/mm^3	Both T- and B-cell levels decrease
Platelets	150,000–350,000/mm^3	No change with age
Albumin	3.5–5.0 g/100 mL	Declines because of a decrease in liver size and enzymes
Globulin	2.3–3.5 g/100 mL	Slight increase
Serum protein	6.0–8.4 g/100 mL	No change with age
Blood urea nitrogen (BUN)	Men 10–25 mg/100 mL Women 8–20 mg/100 mL	Can increase with age
Creatinine	0.6–1.5 mg/100 mL	Increases, although this is related to lean body mass
Creatinine clearance	104–124 mL/min	Decreases by 10% per decade after age 40 y
Glucose	<200 mg/dL fasting	Slight increase in glucose tolerance of 10 mg/dL per decade after age 30 y
Triglycerides	40–150 mg/100 mL	20–200 mg/100 mL
Cholesterol	120–220 mg/100 mL	Increases with age, more so in women than in men
Thyroxine (T_4)	4.5–13.5 mcg/100 mL	No change
Triiodothyronine (T_3)	90–220 ng/100 mL	Decreases 25%
Thyroid-stimulating hormone (TSH)	0.5–5.0 mcg/mL	No significant change with age
Alkaline phosphatase (AP)	13–39 IU/L	Increases by 8–10 IU/L, although elevations >20% are likely due to disease
Prostate-specific antigen (PSA)	4 ng/mL	No change with age, although elevated levels may be seen in nonmalignant disease
Uric acid	Men 44–76 mg/L Women 23–66 mg/L	Slight increase with age

With age, there is also a decrease in renal blood flow, decreased tubular function, and decreased ability to concentrate urine. Basal renin is diminished by 30% to 50% in older adults. Renin alterations and other renal changes diminish the capability of the older adult to maintain sodium and water balance, especially in the presence of stress. There may also be a lessened response to antidiuretic hormone (ADH), which can result in a decreased ability to concentrate urine. This decreased ability may lead to problems of fluid–electrolyte balance as sodium, potassium, and water are lost. Loss of hydrogen ions may also make the acid–base balance more difficult to maintain.

Findings on Physical Examination and Management

Older adults tend to have decreased sensations of thirst and consequently drink less fluid. This change leaves them vulnerable to dehydration, especially when medications

with diuretic actions are administered. To prevent renal damage, care must be taken to ensure that the hospitalized older patient has adequate fluid intake by oral, enteral, or parenteral routes. Fluid balance may also be precarious because disease states such as diabetes can produce diuresis. Further, potassium and sodium levels may already be low when the patient arrives in the unit. Care must be taken to ensure that electrolyte balance remains stable or is restored. Confusion, dysrhythmias, coma, and death can occur quickly in the older patient with altered electrolyte balance.

As bladder muscle tone is lost, incomplete emptying with retention can foster the development of urinary tract infections (UTIs) that can ascend and become renal infections. Hypertrophy of the prostate gland also places older men at risk for UTI because the enlarged gland interrupts urine flow. Loss of muscle tone, retention with overdistention, and loss of sphincter control lead to incontinence in the older man or woman. For older patients, this loss of control is embarrassing and disconcerting.

If any type of incontinence or retention develops in an older patient during the stay in the ICU, the nurse should perform a comprehensive evaluation to determine the underlying cause of the urinary problem. Specifically, consideration should be given to drugs, particularly anticholinergic drugs, metabolic and neurological problems, and bladder inflammation as potential causes (Table 12-4). If an indwelling (Foley) catheter is necessary during acute illness, it should be removed as soon as the primary reason for inserting it (eg, hourly urine measurements) has passed. Early removal may prevent deterioration of bladder function and UTI. See Evidence-Based Practice Highlight 12-1 for information about how to prevent catheter-associated UTIs.

Gastrointestinal Changes

The gastrointestinal system undergoes many age-related changes (see Table 12-1). The mechanical and chemical processes of digestion that begin in the mouth may be impaired because of loss of teeth, poor hygiene, and a decrease in salivary secretions. Many older adults experience diminished senses of taste and smell, which may lead to decreased food intake.[20]

Flattening of the gastric mucosae and secretory changes have been noted in normal older adults. This influences active transport mechanisms for calcium, iron, and vitamin B_{12} absorption. The gastrointestinal fluid pH, gastric emptying time, intestinal transit rates, and gastrointestinal blood flow and surface area are all altered in the older adult.

Slowing of peristalsis may interfere with swallowing, gastric emptying, and passage through the bowel. The decrease in hydrochloric acid, digestive enzymes, and bile may contribute to incomplete digestion of nutrients. In some older adults, diminished intrinsic factor and decreased vitamin B_{12} synthesis may produce pernicious anemia.

Data are insufficient to make assumptions regarding changes in absorption in the large and small bowel. Some evidence indicates that absorption is somewhat impaired. Given that the eating patterns of older adults may not include all food groups, deficiencies may arise from lack of intake rather than malabsorption.

The decreased motility of the large bowel is probably not sufficient to produce constipation in the active adult. However, older adults who are on bed rest, have decreased intake of food and fluids, and are exposed to multiple medications may experience constipation and fecal impaction. Dependence on or misuse of laxatives must be assessed when

Table 12-4 Causes of Incontinence

Cause	Explanation
Drug Side Effects	
Diuretics	Urgency
Caffeine and alcohol	Diuretic effect and irritation
Sedatives	Inhibition of micturition, functional changes
Anticholinergics	Constipation causing obstruction
Calcium channel blockers	Constipation causing obstruction, smooth muscle relaxation
Nonsteroidal anti-inflammatory drugs (NSAIDs)	Blockage of prostaglandin receptors causing decreased force of contraction
Physiological Changes	
Hypoxemia	Depressed function of brain
Delirium	Depressed function of brain
Hyperglycemia	Diuretic effect of glycosuria
Hypercalcemia	Diuretic effect of hypercalciuria
Functional impairment	Inability to get to the toilet in time
Bladder Inflammation	
Infection	Uninhibited bladder contractions
Atrophic vaginitis	Uninhibited bladder contractions

EVIDENCE-BASED PRACTICE HIGHLIGHT 12-1

Catheter-Associated Urinary Tract Infections

△ Expected Practice

- Prior to placement of any indwelling urinary catheter, assess patient for accepted indications and alternatives. (Level C)
- Adhere to aseptic technique for placement, manipulation, and maintenance of indwelling urinary catheters. (Level E)
- Document all instances of indwelling urinary catheters, including insertion date, indication, and removal date. (Level C)
- Promptly discontinue indwelling urinary catheters as soon as indications expire. (Level C)

△ Supporting Evidence

- Prolonged catheterization is the major risk factor for catheter-associated urinary tract infections (CAUTIs).[7,8]
- Twenty-five percent of inpatients and up to 90% of patients in an ICU have a urinary catheter during hospitalization, often without an appropriate indication.[4] Indwelling urinary catheters are placed without sufficient rationale, and/or remain in place after indications expire.[9]
- CAUTIs can be decreased by interventions that facilitate removal of unnecessary catheters.[10,11]
- Most hospitals have not implemented effective strategies for preventing CAUTIs.[12,13]

AACN Levels of Evidence

Level A Meta-analysis of quantitative studies or metasynthesis of qualitative studies with results that consistently support a specific action, intervention, or treatment
Level B Well-designed, controlled studies with results that consistently support a specific action, intervention, or treatment
Level C Qualitative studies, descriptive or correlational studies, integrative reviews, systematic reviews, or randomized controlled trials with inconsistent results
Level D Peer-reviewed professional organizational standards with clinical studies to support recommendations
Level E Multiple case reports, theory-based evidence from expert opinions, or peer-reviewed professional organizational standards without clinical studies to support recommendations
Level M Manufacturer's recommendations only

Excerpted from American Association of Critical-Care Nurses Practice Alert. Available online at http://aacn.org. All references cited in this alert are available with the associated resources related to this chapter. Visit: http://thepoint.lww.com

the history is taken because this may further exacerbate the constipation and management of bowel function.

Findings on Physical Examination and Management

Examination of the mouth in an older adult commonly reveals a wearing of dental enamel and gum recession, thereby exposing more of the teeth and increasing the likelihood of decay. Oral care is critical given the associated risk for pneumonia with poor oral care. The oral mucous membranes also tend to be very dry, making eating difficult. Swallowing disorders are common and can be due to functional abnormality of the oral, pharyngeal, or esophageal stage of swallowing.

Older adults commonly report constipation, and stool may be palpable in the large bowel. In addition to an abdominal examination, a rectal examination must be performed to determine the consistency of stool and the anal tone, and to establish an appropriate treatment plan. Heartburn (which may present as chest pain) is also a common complaint and is often associated with epigastric tenderness. Aortic aneurysms are more common in older adults and present as a pulsatile mass in the abdomen.

Acute abdominal pain in this population is a particularly challenging complaint, and causes include diverticulitis, bowel obstruction, appendicitis, pancreatic disease, infarction, and cancer. However, identifying older adults with acute abdominal problems is even more challenging because these people often present without pain or any other significant signs and symptoms of an acute problem (eg, fever, anorexia). Careful evaluation of the abdomen in older adults is essential to monitor for acute changes.

Nursing interventions for gastrointestinal changes begin with a careful history. Eating habits, including time and frequency of food intake, food preferences, usual intake, food intolerances, and taste and smell changes must be assessed. Use of laxatives, enemas, and vitamin supplementation also should be explored. Evaluation of the teeth and gums helps to establish how well food can be handled mechanically.

When planning care, the nurse must consider that bed rest slows peristalsis and aggravates any preexisting condition related to motility. Adequate fluid intake, bulk in the diet, use of natural laxatives (eg, prune juice, warm liquids), and as much active or passive exercise as the condition of the patient allows may help maintain a normal pattern of bowel movements. Laxatives may need to be included in the regimen once constipation does occur.

Hospitalized patients of any age can become rapidly malnourished secondary to the stress of acute illness, an increased demand for energy, and lack of nourishment. Therefore, it is important to look for indicators of nutritional risk. Indicators include a history of recent weight loss, a diet lacking in protein and calories, an albumin level less than 3.5 g/dL, and a lymphocyte count less than 1,500/mm^3. The older patient who enters the hospital already mildly to moderately malnourished and who has a poor intake of protein and calories may quickly become severely malnourished. This malnourished state can markedly compromise the immune response and increase the incidence and severity of infection. Therefore, it is crucial to see that critically ill older patients maintain adequate nutritional intake.

Musculoskeletal Changes

An estimated 43 million Americans (one in every 7 persons) have some form of diagnosed arthritis or other rheumatic condition, and this number is expected to rise to 60 million

during the next 20 years. Arthritis affects people of all ages, although it is more prevalent among older adults and women. The disease causes pain, stiffness, and tenderness around the joints and typically affects the hands, feet, knees and hips. Symptoms can range from mild to severe. While arthritis rarely kills, it is a chronic disease that causes significant disability and reduces quality of life. A large percentage of older adults have some form of arthritis, with this becoming more prevalent if the person is female and older. Arthritis is not a normal age change, although it does contribute to mobility problems. Mobility also is often limited among hospitalized older adults and results in a decrease in muscle protein synthesis, strength, and lower extremity and whole body mass. Muscle mass may be lost also because of a reduction in the number and size of muscle fibers or an increase in connective tissues. These changes result in less muscle tension and decreased strength of the contraction. The decrease of lean muscle mass and the loss of elasticity contribute to lost flexibility and increased stiffness.

Skeletal calcium losses are a universal concomitant of aging and reflect an imbalance of bone remodeling, with osteoclastic bony resorption exceeding osteoblastic new bone formation. After approximately 30 years of age, when peak skeletal bone mass has been achieved, resorption begins to exceed formation with subsequent skeletal calcium losses and a decline in bone density. The rate of bone loss is approximately 0.5% to 1% yearly from age 60 years onward. The bone changes that occur are likely due to a multitude of factors in addition to normal age-related changes: lack of exercise, poor nutrition, and calcium malabsorption. Known age-related changes in factors influencing bone homeostasis occur in calcium, vitamin D, and gonadal hormone status. Dietary intake of calcium, gastrointestinal absorption of calcium, and vitamin D synthesis are all decreased with age. Decreased intake of vitamin D_2 and decreased skin absorption of 7-dehydrocholesterol as a result of inadequate sunlight exposure also may contribute to vitamin D deficiency with age. The patient on bed rest may rapidly lose bone mineral concentration. The calcaneus (heel) and spine are most susceptible, with a loss of approximately 1% per week. This loss is related to lack of weight bearing.

Musculoskeletal function is dictated largely by the size of the muscle mass that is contracting, and to a lesser extent by changes in surrounding connective tissue in the joint and neural recruitment, conduction velocities, and fatigue. Sedentary people lose large amounts of muscle mass over time. Unfortunately, muscle mass cannot be maintained into old age even with habitual aerobic activities in either normal or athletic adults.[21] Only loading of muscle with weight-lifting exercise has been shown to reverse loss of muscle mass and strength in older adults.[22,23] There is also a decrease in oxidative and glycolytic enzyme capacity with age, a decrease in total number of muscle fibers, selective atrophy of type 2 (fast-twitch) fibers, and shortening of tendons and ligaments with decreased tissue elasticity. Bone changes, as evidenced by osteoporosis, present with decreased height, kyphosis, and scoliosis.

Findings on Physical Examination and Management

Older adults with osteoporosis may have spontaneous fractures, occurring simply from moving in bed. In general, older adults have a decrease in overall muscle strength and an increased tendency to have muscle cramping. Crepitus and pain with range of motion of the joints is common, particularly in the weight-bearing joints (eg, the knee). Gait and posture changes are frequently present, and older adults tend to have a stooped posture with a slow, shuffling gait.

Forced fasting of the critically ill hospitalized patient may further accelerate muscle loss through catabolism and gluconeogenesis. The added burden of bed rest leads to a rapid loss of mobility, strength, and energy in the older patient. Maintaining nutrition, changing position frequently, active and passive exercise, and getting out of bed as much as permitted by condition are essential to preserving strength, energy, and bone mass. If the patient is comatose or has suffered loss of function, proper positioning and splinting can help prevent permanent deformity.

Endocrine Changes

The equilibrium concentrations of the principal hormones are not necessarily altered with age; however, for older adults, there may be a change in how hormonal equilibrium is achieved. Therefore, with advancing age, some alterations in hormone production, metabolism, and action occur. Subtle changes are noted in pituitary dynamics, adrenal gland physiology, and thyroid function; however, the changes in glucose homeostasis, reproductive function, and calcium metabolism are more apparent.[24]

Most of the principal neuroendocrine nuclei in the hypothalamus are structurally intact in old age, although there is some loss of morphologic integrity of the suprachiasmatic nucleus. Morphometric variables associated with increased cellular functional activity have been measured in several hypothalamic nuclei. Certain neurons in the human paraventricular nuclei seem activated, and the neurons that produce arginine vasopressin (AVP) increase in size, and the number of neurons that express both AVP and corticotropin-releasing hormone increases with age.

The anterior pituitary shows unchanged output of stimulating hormones, although the peripheral levels of target hormones decrease. For example, circulating levels of daytime and nighttime thyroid-stimulating hormone and growth hormone (GH) are greatly diminished in old age.[25] In contrast, prolactin and melatonin are decreased only at night. Age-related decreases in hormonal levels are associated with a decrease in the amplitude but not the frequency of secretory pulses.

The decline in GH with age is believed to be associated with the decrease in lean body mass, increase in body fat (especially in the visceral and abdominal compartment), adverse changes in lipoproteins, and reduction in aerobic capacity commonly noted in older adults. Research is ongoing to determine whether replacement of GH in healthy older adults can reverse these changes.[25]

Normal aging is associated with insulin resistance and reduced β-cell function, but it is not known whether changes in proinsulin and the proinsulin/immunoreactive insulin ratio are also related to reduced β-cell function.[26] Glucose tolerance decreases with age. An increase in blood glucose to 200 mg/dL occurs in about half of people older than 70 years.[26] Interpretation of this glucose intolerance requires

the use of age-adjusted parameters to avoid the inappropriate diagnosis and treatment of diabetes mellitus. Evaluating glycosylated hemoglobin (HbA_{1c}) or glycosylated albumin may help establish the presence or absence of diabetes mellitus in the older patient with elevated blood glucose levels. Because the renal threshold for reabsorption of glucose increases with age, higher degrees of hyperglycemia must be present before glucose spills into the urine. Therefore, monitoring for hyperglycemia with urine testing should be avoided.

Throughout life, the adrenal cortex shows significant morphogenic and steroidogenic changes. There is a subtle decline in aldosterone with age and a subtle increase in cortisol; however, the adrenal androgens dehydroepiandrosterone and dehydroepiandrosterone sulfate decline with age in a process similar to menopause. This decline is believed to aggravate some age-related diseases.[27]

The thyroid gland undergoes a progressive decrease in size with aging, although enlargement because of nodules is not uncommon. The concentration of thyroid hormones found in the blood of older adults is variable and influenced by disease. There may, however, be an alteration in the responsiveness of target tissues. Specifically, there may be an age-related reduction in the ability of aged tissues to increase receptor numbers in response to a reduction in hormone levels.[28]

Findings on Physical Examination and Management

The reduction in thyroid hormone levels with increasing age is correlated with many physiological and pathological sequelae: changes in cholesterol metabolism, heart rate, cardiac output, and strength of cardiac contraction and alterations in basal metabolic rate and thermoregulation. The symptoms of thyroid disease, such as apathy, weakness, and weight loss, may not be as pronounced in older adults as they are in younger people. Moreover, these symptoms are often attributed to old age rather than to hyperthyroidism or hypothyroidism. The older patient with hyperthyroidism is likely to present with atrial tachycardia and is more likely to be anorexic than hyperphagic; this person usually does not experience heat intolerance. The hypothyroid older adult may present with increased susceptibility to hypothermia if exposed to cold, a change in cognitive status, fatigue, dizziness, and a tendency to fall.

Being aware of the atypical presentation of thyroid disease in older adults leads the critical care nurse to recognize endocrine imbalance. Once identified, the imbalance can easily be corrected by replacing thyroid hormone or changing the dosage of thyroid replacement.

Diabetes mellitus is frequently seen with acute illness, trauma, or surgery. The end-organ damage of diabetes mellitus is a factor in stroke, myocardial infarction, decreased renal function, and peripheral vascular disease. Long-standing non–insulin-dependent diabetes may be diagnosed only when the patient presents with a stroke or acute myocardial infarction. Therefore, it is important to distinguish among the impaired glucose tolerance of aging, a transient rise in glucose related to acute illness, and the disease process of diabetes.

Recognition of the underlying diabetes and possible end-organ damage may alter the course of the acute illness. For example, knowing that the incidence of congestive heart failure after myocardial infarction is higher in diabetic than in nondiabetic patients, the nurse can be alert for early signs of fluid retention.

BOX 12-6 NURSING INTERVENTIONS

For Preventing Problems in the Older Adult With Diabetes

Skin Alterations
- Monitor for decreased circulation and skin breakdown.
- Provide foot care to maintain skin integrity. Bathe the feet daily and apply emollient.

Hyperglycemia
- Maintain a controlled diet.
- Monitor blood glucose levels.
- Monitor for urinary frequency.
- Monitor for hyponatremia.
- Monitor for dry mouth.
- Monitor for changes in cognition.

Hypoglycemia
- Monitor for acute changes in cognition.

Hydration Status
- Monitor hydration.
- Encourage the intake of 2,000 mL of fluid daily.

End-Organ Disease
- Monitor kidney function.
- Monitor for visual changes (eg, blurred or decreased vision).

Older people with diabetes are, for the most part, not insulin dependent. Therefore, even if they have extremely high blood glucoses, they are rarely ketoacidotic. In fact, the coma of this age group is usually hyperglycemic, hyperosmolar, and nonketotic (HHNK). Managing this state requires a delicate balance of hydration and rapid reduction of blood glucose without massive brain edema and death. The critical care nurse must be aware that HHNK coma can be triggered by acute illness or surgery. Common problems found in older adults with diabetes, and nursing interventions to prevent these problems, are shown in Box 12-6. It should be particularly recognized that the most prevalent sign of either hypogylcemia or hyperglycemia among these people is a change in cognitive status.

Immunological Changes

With age, there is a decline in immune function. Specifically, there is a decline in both T-cell and B-cell function, with a dramatic effect on cell-mediated immunity. The decline in B-cell function may be indirectly related to a decline in T-cell function. The decreased numbers of B cells secreting immunoglobulin G result in a generally poor humoral immune response. With aging, the thymus gland involutes and there is a decrease in thymic hormone levels, and the number of autoantibodies increases.[29] With age, there is also a decrease in the production of antibodies by cells located in mucosal tissues further increasing risk of infection. Lastly, with age, there is a decrease in the production of IgE, the antibody associated with allergic responses, and thus, these responses decrease in older individuals.

Findings on Physical Examination and Management

The usual symptoms of infection such as chills, fever, leukocytosis, or tachycardia may be absent or blunted in the older adult. Instead, these people may present with an acute change in cognition, function, or behavior. Delirium, for example, may be the only sign or symptom of a UTI in an older adult. The most common areas of infection in older adults are the lungs, urinary tract, and skin, and when subtle changes are noted in an older patient, consideration should be given to each of these areas.

▲ Psychological Challenges

Cognitive Changes

Cognition refers to the process of obtaining, storing, retrieving, and using information. The neuroanatomical and neurophysiological underpinnings of cognitive change are unclear. Studies[30,31] have shown that younger people have larger ventricular volumes and smaller gray and white matter volumes compared with older people. There is also greater prefrontal cortex activity in younger adults compared with older adults in the dorsolateral area during memory retrieval. These changes are believed to account for the changes in working memory[31] and executive abilities[32] associated with normal aging. As age increases, there is some decline in perceptual motor skills, concept formation, complex memory tasks, and quick-decision tasks. However, age itself is not the criterion for making decisions about a patient's cognitive functions. Each person's abilities must be judged individually rather than against a norm. Moreover, interventions such as exercise (aerobic and resistive) can improve executive control processes in older adults, particularly older women.[32]

Cognitive function should be assessed and described on admission and monitored routinely over time and whenever the patient's condition changes. While assessing cognitive functions during the patient's stay in intensive care, it is important to remember that physiological deficits, some medications, and internal and external stress, such as environmental stressors, affect cognitive skills. In older adults, acute physical changes frequently initially present as changes in cognitive status. For example, an older adult with pneumonia may not have symptoms such as fever or cough. Rather, this person may present with changes in cognitive status.

The Mini-Cog,[33] which is available for free use, provides the practitioner with a consistent assessment tool to compare responses and monitor results over time. The main drawback to the use of a questionnaire is that some critically ill patients may not be able to hear, see, talk, or write well enough to respond to the questions. Longer, more sensitive tools may also be more fatiguing for the critically ill older patient. When completing the Mini-Cog, if the patients are unable to recall any of the three words, they are categorized as "probably demented." If they can recall all three words, then they are categorized as "probably not demented." People who can recall one or two words are categorized based on their clock drawing test. If the patient draws a clock that is in any way abnormal, he or she is considered "probably demented." If the clock is normally constructed, then he or she is considered "probably not demented."

Several common syndromes cause cognitive impairment, including dementia, delirium, and depression (discussed later). Dementia is based on impairment of memory plus at least one of the following: a personality change or impairment in abstract thinking, judgment, or higher cortical functions. Delirium is the abrupt onset of clouding of consciousness and is a medical emergency. Table 12-5 identifies factors to differentiate dementia from delirium. Reversible causes of dementia and delirium are listed in Box 12-7. Tools to evaluate memory and differentiate between dementia and delirium are available at http://www.geronurseonline.org. See Evidence-Based Practice Highlight 12-2 for the assessment and management of delirium.

Learning

Older adults may take longer to respond to and assimilate new material. They may also be hesitant to take on new tasks. Motivation continues to be an important aspect of learning new material. If the material is irrelevant or meaningless, motivation is decreased, which is often interpreted as an inability to learn. The person's sensory and cognitive abilities are taken into account when teaching older patients. It may be necessary to present information in small segments using varied stimuli, including touching, seeing, hearing, and (if vision permits) writing. If movements are slowed, allow time for the completion of motor tasks, such as manipulating equipment or carrying out exercises.

Memory

The older person's memory decline involves short-term memory rather than long-term and remote memory. Recall of memory from the past is least impaired by age. Remote memory recall (items learned many years ago) can be a positive therapeutic strategy for older patients. Reminiscence is an adaptive mechanism that helps the nurse learn about the patient and increases the patient's feelings of self-worth and competence.

Depression

Depression disorders are among the most common complaints of older adults and the leading cause of suicide in later life. Symptoms of depression are listed in Box 12-8 on page 151. Based on the diagnostic criteria for major depression, at least five of these symptoms should occur almost daily for at least 2 weeks. These symptoms of depression in the older adult can be masked by normal age-related changes or disease states. For example, difficulty sleeping, early morning awakening, and lethargy are common physical complaints of the normal aging person. Alternatively, depression in the older adult may more commonly present with pseudohypochondriasis, preoccupation with past life events, and changes in cognitive ability. In some patients, the dominant emotional mood may not be sadness but anger, anxiety, or irritability.

Causes of depression are multifaceted and include multiple losses associated with aging, underlying illness, or drugs. Box 12-9 on page 151 lists drug groups that may cause depression. Screening tools, such as the Geriatric Depression Scale,[34] shown in Box 12-10 on page 152, are useful to identify people who are depressed. Once depression is identified, appropriate interventions, including drug therapy, behavioral modification, and counseling, can be initiated.

Table 12-5 Summary of Differences Between Dementia and Delirium

	Dementia		Delirium
	Alzheimer's Disease (AD)	Vascular (Multi-Infarct) Dementia	
Etiology	Familial (genetic [chromosomes 14, 19, 21]) Sporadic	Cardiovascular (CV) disease Cerebrovascular disease Hypertension	Drug toxicity and interactions; acute disease; trauma; chronic disease exacerbation Fluid and electrolyte disorder
Risk factors	Advanced age; genetics	Preexisting CV disease	Preexisting cognitive impairment
Occurrence	50%–60% of dementias	20% of dementias	6%–56% of hospitalized older people
Onset	Slow	Often abrupt Follows a stroke or transient ischemic attack	Rapid, acute onset A harbinger of acute medical illness
Age of onset (y)	Early-onset AD: 30s–65 Late-onset AD: 65+ Most commonly: 85+	Most commonly 50–70 y	Any age, but predominantly in older persons
Gender	Males and females equally	Predominantly males	Males and females equally
Course	Chronic, irreversible; progressive, regular, downhill	Chronic, irreversible Fluctuating, stepwise progression	Acute onset Hypoalert–hypoactive Hyperalert–hyperactive Mixed hypo–hyper
Duration	2–20 y	Variable; years	Lasts 1 d to 1 mo
Symptom progress	Onset insidious. *Early*—mild and subtle *Middle and late*—intensified Progression to death (infection or malnutrition)	Depends on location of infarct and success of treatment; death due to underlying CV disease	Symptoms are fully reversible with adequate treatment; can progress to chronicity or death if underlying condition is ignored
Mood	Early depression (30%)	Labile: mood swings	Variable
Speech/language	Speech remains intact until late in disease *Early*—mild anomia (cannot name objects); deficits progress until speech lacks meaning; echoes and repeats words and sounds; mutism.	May have speech deficit/ aphasia depending on location of lesion	Fluctuating; often cannot concentrate long enough to speak May be somnolent
Physical signs	*Early*—no motor deficits *Middle*—apraxia (70%) (cannot perform purposeful movement) *Late*—dysarthria (impaired speech) *End stage*—loss of all voluntary activity; positive neurologic signs	According to location of lesion: focal neurologic signs, seizures Commonly exhibits motor deficits	Signs and symptoms of underlying disease
Orientation	Becomes lost in familiar places (topographic disorientation) Has difficulty drawing three-dimensional objects (visual and spatial disorientation) Disorientation to time, place, and person—with disease progression		May fluctuate between lucidity and complete disorientation to time, place, and person
Memory	Loss is an early sign of dementia; loss of recent memory is soon followed by progressive decline in recent and remote memory		Impaired recent and remote memory; may fluctuate between lucidity and confusion
Personality	Apathy, indifference, irritability *Early disease*—social behavior intact; hides cognitive deficits *Advanced disease*—disengages from activity and relationships; suspicious; paranoid delusions caused by memory loss; aggressive; catastrophic reactions		Fluctuating; cannot focus attention to converse; alarmed by symptoms (when lucid); hallucinations; paranoid
Functional status, activities of daily living	Poor judgment in everyday activities; has progressive decline in ability to handle money, use telephone, function in home and workplace		Impaired
Attention span	Distractable; short attention span		Highly impaired; cannot maintain or shift attention
Psychomotor activity	Wandering, hyperactivity, pacing, restlessness, agitation		Variable; alternates between high agitation, hyperactivity, restlessness, and lethargy
Sleep–wake cycle	Often impaired; wandering and agitation at nighttime		Takes brief naps throughout day and night

From, Smeltzer SC, Bare BG, Hinkle JL, et al: Brunner and Suddarth's Textbook of Medical–Surgical Nursing, 12th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, pp 216.

BOX 12-7 Reversible Causes of Dementia and Delirium

Drugs
Emotional illness (including depression)
Metabolic/endocrine disorders
Eye/ear/environment
Nutritional/neurological disorders
Tumors/trauma
Infection
Alcoholism/anemia/atherosclerosis

The nurse must also be aware of cardiovascular side effects of antidepressants. The tricyclic antidepressive drugs are less commonly used owing to the risk for side effects. For example, tricyclic antidepressants can result in ST-segment and T-wave changes, although these are not necessarily indicative of myocardial damage. Ventricular dysrhythmias and disturbances in cardiac conduction are potential serious side effects and may result in the drug being reduced or discontinued. Anticholinergic effects, especially in patients with Alzheimer's disease, benign prostatic hypertrophy, or CAD, may also be seen. The selective serotonin reuptake

EVIDENCE-BASED PRACTICE HIGHLIGHT 12-2
Delirium Assessment and Management

△ Expected Practice

- Implement delirium assessment for all critically ill patients using validated tools such as the Confusion Assessment Method for the ICU (CAM-ICU) or Intensive Care Delirium Screening Checklist (ICDSC) (Level B)
- Create strategies to decrease delirium risk factors, including early exercise (Level B)
- Be cautious with benzodiazepine use, giving only what is needed (Level C)
- Consider whether to adopt a core bundle like the ABCDE bundle (Level E)

△ Supporting Evidence

- In the absence of a validated tool, delirium goes undetected by both doctors and nurses in more than 65% of ICU patients.[16–19] The reports underscore the need for systematic utilization of standardized assessment tools, which is in concert with the recommendations from national (Society of Critical Care Medicine) and international guidelines.[20–23] Systematic use of validated assessment tools is necessary to detect delirium, which would otherwise go undetected and consequently untreated.
  - Two tools with robust validity and reliability are the Confusion Assessment Method for the ICU (CAM-ICU)[4,24–26] and the Intensive Care Delirium Screening Checklist (ICDSC).[27,28]
  - Implementation of both tools has been described in the literature; both have high accuracy and favorable compliance, and require minimal education.[29–33]
- Risk factors for ICU delirium have been understudied and underreported, with few available studies and little consensus among them.[34]
  - The following baseline risk factors are the only ones reported as significant in two or more multivariate analyses: preexisting dementia, history of baseline hypertension, alcoholism, and admission severity of illness.[2,35–37,38,39]
  - Although age has been identified as one of the most significant risk factors for delirium development in non-ICU literature, there is conflicting evidence in critical care literature to support this claim.[34] Thus, further research is required to verify age as a risk factor for delirium in the ICU.
  - Although immobility has not been reported as a risk factor for the development of delirium in the ICU, it has been reported in non-ICU cohorts.[40] Recent studies have reported that early mobility in critically ill patients results not only in improved physical functions, but improved cognitive function as well, reducing delirium duration by 2 days.[41–43] "Early" is defined by these protocols as within the first 3 days of the ICU stay and focuses on progressive mobility pathways, starting with passive range of motion and progresses to active range of motion, sitting on the side of the bed, and ambulating as tolerated.[44] Early exercise is a primary nonpharmacologic intervention shown to reduce delirium duration in critical care patients and should be considered a cornerstone of any delirium-reduction protocol.
- Iatrogenic risk factors are often modifiable and are referred to as precipitating factors.[45] Sedatives have been the only consistently identified ICU delirium risk factor and are discussed here[3,35,36,38,46]
  - Benzodiazepines. Studies have reported benzodiazepines to be an independent risk factor for the transition to delirium.[3,36]
  - Dexmedetomidine. Two recent studies, "Maximizing Efficacy of Targeted Sedation and Reducing Neurological Dysfunction (MENDS)" and "Safety and Efficacy of Dexmedetomidine Compared With Midazolam (SEDCOM)," reported a significant reduction in delirium duration in patients receiving dexmedetomidine when compared to benzodiazepines (lorazepam and midazolam, respectively).[47–49] Both studies utilized dexmedetomidine at higher doses and for longer durations than the current Food and Drug Administration (FDA) labeling approval, which is a maximum dose of 0.7 mcg/kg/h for a 24-hour duration. These studies suggest that a benzodiazepine-sparing sedation strategy using an alternative sedative, such as dexmedetomidine, may result in better outcomes, including reduced duration of delirium.
  - Opioids and propofol. The data concerning opioids are difficult to interpret, because some studies show a dose-dependent relationship, while other studies indicate there is no relationship between the use of these drugs and delirium development in the ICU.[3,36–39] Only one study has explicitly addressed propofol, and it reports no significant relationship with the drug and ICU delirium.[34,36] More research is needed with both propofol and opioids to fully understand their relationship to the development and duration of delirium.
- **Management of ICU Delirium**
  - **No drug has been approved by the FDA to treat delirium**. In fact, the FDA has issued an alert that atypical antipsychotic medications are associated with mortality risk among older patients, and another analysis has reported that haloperidol had an even higher mortality risk in non-ICU older patients than atypical antipsychotics.[50–53]
  - Clinical practice guidelines traditionally recommended antipsychotics as the medication class of choice for delirium, yet very little evidence exists to support this internationally adopted treatment.[20–23,54–57] Currently, there are only two placebo-controlled pilot studies involving antipsychotics and delirium treatment in the ICU. The "Modifying the Incidence of Delirium (MIND)" study compared

(continued on page 151)

EVIDENCE-BASED PRACTICE HIGHLIGHT 12-2

Delirium Assessment and Management (continued)

haloperidol, ziprasidone, and placebo and reported no differences in regard to delirium resolution or any other outcomes or safety concerns in the three treatment groups.[58] Another study compared quetiapine to placebo in patients already determined to be delirious who had an as-needed haloperidol order and found that the patients who received quetiapine experienced a faster resolution of delirium, less delirium, less agitation, and more somnolence.[59]

- These two studies are the first steps in understanding the best pharmacologic treatment; however, larger trials are needed to confirm these findings in order to systematically direct the choice for delirium treatment.
- All patients receiving antipsychotics (haloperidol or any of the atypical antipsychotics) should be routinely and systematically monitored for side effects, especially QT prolongation.[20]
- Rivastigmine, a cholinesterase inhibitor, has not been shown to be superior to placebo for the treatment of ICU delirium. A large European trial was stopped prematurely because of increased mortality in the rivastigmine group.[60]
- The Society of Critical Care Medicine suggests identification of causes as the first step in delirium management. The following THINK pneumonic may be helpful in determining the cause when delirium is found to be present in ICU patients:
  - Toxic situations
    - CHF, shock, dehydration
    - Deliriogenic meds (tight titration of sedatives)
    - New organ failure (eg, liver, kidney)
  - Hypoxemia
  - Infection/sepsis (nosocomial)
  - Immobilization
  - Nonpharmacologic interventions (Are these being neglected?)
    - Hearing aids, glasses, sleep protocols, music, noise control, ambulation K+ or electrolyte problems
- Putting it all together: ABCDE bundle. Several recent reviews have described the idea of implementing a core model of care combining multiple evidence-based practice strategies subsequently incorporated into routine daily care for the purpose of improving overall patient outcomes and allowing a systematic reduction in the modifiable risk factors for delirium.[61–63] The ABCDE bundle includes spontaneous awakening and breathing trial coordination, careful sedation choice, delirium monitoring, and early progressive mobility and exercise. The intent of combining and coordinating these individual strategies is to "(1) improve collaboration among clinical team members, (2) standardize care processes, and (3) break the cycle of oversedation and prolonged ventilation, which appear causative to delirium and weakness."[61] The ABCDE bundle is a helpful paradigm for critical care nurses to consider when focusing on implementing strategies to improve patient care and reduce the impact of modifiable delirium risk factors.
  - Awakening and Breathing Trial Coordination (the Wake Up and Breathe Protocol)
  - Choice of Sedative
  - Delirium Detection
  - Early Progressive Mobility and Exercise

AACN Levels of Evidence

Level A Meta-analysis of quantitative studies or metasynthesis of qualitative studies with results that consistently support a specific action, intervention, or treatment

Level B Well-designed, controlled studies with results that consistently support a specific action, intervention, or treatment

Level C Qualitative studies, descriptive or correlational studies, integrative reviews, systematic reviews, or randomized controlled trials with inconsistent results

Level D Peer-reviewed professional organizational standards with clinical studies to support recommendations

Level E Multiple case reports, theory-based evidence from expert opinions, or peer-reviewed professional organizational standards without clinical studies to support recommendations

Level M Manufacturer's recommendations only

Excerpted from American Association of Critical-Care Nurses Practice Alert. Available online at http://aacn.org. All references cited in this alert are available with the associated resources related to this chapter. Visit: http://thepoint.lww.com

inhibitors are much more commonly used now to treat depression. Side effects to monitor include changes related to sleep, appetite, personality or behavior, and blood pressure readings.

Untreated depression may result in suicide, which is a serious problem among older adults. Of all suicides committed in this country annually, 25% involve people older than age 65. White men older than age 85 are at particular risk.[35] Because of their many losses and changes, older adults may view suicide as a means of fulfilling a fantasy of "reunion" with a dead spouse or significant other. The nurse must monitor signs and symptoms of depression, explore the causes of depression, facilitate treatment, and watch for suicide attempts or warnings.

BOX 12-8 *PATIENT SAFETY*

Symptoms of Depression

- Depressed mood
- Decreased interest in activities
- Weight changes
- Sleep changes
- Psychomotor changes
- Fatigue
- Feelings of worthlessness or guilt
- Decreased concentration
- Suicidal ideation

BOX 12-9 **Drug Groups That May Cause Depression in the Older Person**

Analgesics/anti-inflammatory drugs
Anticonvulsants
Antihistamines
Antihypertensives
Antimicrobials
Antiparkinsonian drugs
Hormones
Immunosuppressive drugs
Tranquilizers

BOX 12-10 Geriatric Depression Scale

Patient _____ Examiner _____ Date _____

Directions to Patient: Please choose the best answer for how you have felt over the past week.
Directions to Examiner: Present questions VERBALLY. Circle answer given by patient. Do not show to patient.

1.	Are you basically satisfied with your life?	yes	**no** (1)
2.	Have you dropped many of your activities and interests?	**yes** (1)	no
3.	Do you feel that your life is empty?	**yes** (1)	no
4.	Do you often get bored?	**yes** (1)	no
5.	Are you hopeful about the future?	yes	**no** (1)
6.	Are you bothered by thoughts you can't get out of your head?	**yes** (1)	no
7.	Are you in good spirits most of the time?	yes	**no** (1)
8.	Are you afraid that something bad is going to happen to you?	**yes** (1)	no
9.	Do you feel happy most of the time?	yes	**no** (1)
10.	Do you often feel helpless?	**yes** (1)	no
11.	Do you often get restless and fidgety?	**yes** (1)	no
12.	Do you prefer to stay at home rather than go out and do things?	**yes** (1)	no
13.	Do you frequently worry about the future?	**yes** (1)	no
14.	Do you feel you have more problems with memory than most?	**yes** (1)	no
15.	Do you think it is wonderful to be alive now?	yes	**no** (1)
16.	Do you feel downhearted and blue?	**yes** (1)	no
17.	Do you feel pretty worthless the way you are now?	**yes** (1)	no
18.	Do you worry a lot about the past?	**yes** (1)	no
19.	Do you find life very exciting?	yes	**no** (1)
20.	Is it hard for you to get started on new projects?	**yes** (1)	no
21.	Do you feel full of energy?	yes	**no** (1)
22.	Do you feel that your situation is hopeless?	**yes** (1)	no
23.	Do you think that most people are better off than you are?	**yes** (1)	no
24.	Do you frequently get upset over little things?	**yes** (1)	no
25.	Do you frequently feel like crying?	**yes** (1)	no
26.	Do you have trouble concentrating?	**yes** (1)	no
27.	Do you enjoy getting up in the morning?	yes	**no** (1)
28.	Do you prefer to avoid social occasions?	**yes**	(1) no
29.	Is it easy for you to make decisions?	yes	**no** (1)
30.	Is your mind as clear as it used to be?	yes	**no** (1)

TOTAL: Please sum all bolded answers (worth one point) for a total score. _____

Scores: 0–10 Normal **11–20 Moderate Depression** **21–30 Severe Depression**

From Yesavage JA, Brink TL: Development and validation of a geriatric depression screening scale: A preliminary report. J Psychiatr Res 17:37–49, 1983. Available also at http://en.wikipedia.org/wiki/Geriatric_Depression_Scale

Abuse of the Older Person

Mistreatment of older people is a problem that affects more than 4% of the older adults in the United States.[36] Abuse of older adults occurs in homes and institutions and takes many forms. Abuse may be blatant or subtle; it may be physical, psychological, or material (eg, financial). Abuse may involve neglect (by others or by self), exploitation, or abandonment. The abused older person is often physically or mentally frail and unable to report the abusive situation. Abuse can also happen to emotionally and intellectually stable older people who are unable to stop the abuse or report it because of their financial or emotional dependence on the abuser. They may also be afraid of being abandoned.

Abuse can occur because of lack of knowledge about the older person's basic needs, a lack of resources to help, or a desire to protect an inheritance. People who may or may not live with the older person may be responsible for the abuse. Care givers who are extremely stressed may become abusive. In some situations, the abused older adult is the caretaker.

The nurse must be alert to the signs and symptoms of elder abuse as outlined in Box 12-11. Any suggestion by the patient or family that things are not well at home is pursued. A statement such as, "My son hasn't been here yet. He sometimes forgets his commitments," should open the door

BOX 12-11 PATIENT SAFETY

Signs and Symptoms of Elder Abuse

- Lack of compliance with management of health problems
- Unexplained injuries, such as fractures, bruises, lacerations
- Burns
- Poor personal hygiene
- Sexually transmitted disease
- Altered mood
- Depression
- Failure to thrive (underhydration/impaired nutritional status)
- Impaired skin integrity/fungal rashes

BOX 12-12 The HEAT Screening Method for Indications of Alcohol Abuse

How do you use alcohol?
Have you ever thought you used alcohol to **E**xcess?
Has **A**nyone else ever thought you used too much?
Have you ever had any **T**rouble resulting from your use of alcohol?

for further conversation. It might uncover a mother who is worried about her son's drinking and perhaps about the way he treats her when he has been drinking. Attempts are made to compare the history given by the patient with that given by the family. Inconsistencies need to be explored further. Likewise, it is helpful to ask caretakers if they are able to give the care they feel is needed. Indications that the patient is "getting to be a handful" may be a clue to mismanaged care or a caretaker in need of support and assistance. In either situation, the nurse can provide information and support and refer the patient and care giver to a social worker or mental health nurse for further assistance. All health care workers, including nurses, must know their responsibility under state law for reporting abuse of the older patient.

Alcohol Abuse

Alcohol abuse occurs in the aging population. Clinical studies estimate that 15% of men and 12% of women are high-risk drinkers based on information provided by the National Institute on Alcohol Abuse and Alcoholism.[37] Problem drinking in older adults occurs for similar reasons as it does in younger adults. However, smaller amounts of alcohol create larger problems for older people, and they may be more susceptible to alcohol-induced disease. Differences in metabolism of alcohol in older people, the smaller volume of body water, and the decrease in lean body tissue may increase the propensity to alcoholism or alcohol problems. In addition, older adults are high consumers of psychotropic drugs and are at risk for drug–alcohol interactions.

Nursing interventions include screening the older adult for alcohol use. The HEAT screening method,[38] shown in Box 12-12, is useful for screening purposes. A positive response on any item is a reason to obtain a more detailed history of alcohol use. When alcohol abuse is suspected, the immediate goal is to stabilize physiological and psychological responses to alcohol withdrawal and determine the impact of alcohol abuse on whatever other diagnoses have resulted in the need for critical care. As soon as possible, the nurse should refer the patient to a social worker, psychiatric liaison nurse, or alcohol counselor.

▲ Challenges in Medication Use

The rule for giving therapeutic medications to the older patient is *start low, go slow*. In other words, be patient. Changes related to aging can have a great impact on drug response. Changes in renal function, gastrointestinal secretions and motility, and cell receptor sites and concurrent disease states can alter the absorption, distribution, and excretion of drugs. These changes are summarized in Table 12-6.

Before admission to the ICU, older patients may have been taking many different medications, including over-the-counter (OTC) medications such as vitamins, tonics, herbals (eg, Saint John's wort, glucosamine), laxatives, antacids, and pain relievers. They may also have a history of heavy alcohol intake. Any of these drugs can cause problems if combined with medications administered in the hospital.

The nurse needs to elicit a careful history of drug use from the patient and family. The family can be asked to bring in all medications the patient has been using; these include OTC medications and herbal remedies. Although alcohol use may be a sensitive topic, establishing the pattern of use can be essential in preventing untoward drug interactions and anticipating problems with liver damage or withdrawal.

Special considerations concerning administration of drugs to the older patient include knowing the drugs the patient has been taking; assessing renal, hepatic, endocrine, and digestive systems; and evaluating lean body mass. Impaired body systems may affect the absorption, metabolism, and excretion of drugs. Additional considerations are listed in Box 12-13 on page 155. A decrease in lean body mass and an increase in total body fat may alter the distribution of the drug in the body.

Drug Absorption

Drug absorption is affected by the following age-related changes: decreased gastric acid, decreased gastrointestinal motility, decreased gastric blood flow, changes in gastrointestinal villi, and decreased blood flow and body temperature in the rectum. The increased pH of gastric secretions and delayed stomach emptying time can alter the degradation, and thus the absorption, of drugs. Drugs that are not stable in an acid medium can be severely reduced in bioavailability if they remain in the stomach for long periods. Drugs that are designed to be acted on in the small intestine may be affected by the higher pH of the aging stomach. A coated, pH-sensitive medication, such as erythromycin, may lose its coating in the stomach and be degraded before reaching the absorption sites in the small intestine. Coated gastric irritants may lose their coatings and cause bleeding or nausea and vomiting.

Some drugs are eliminated from the body before they enter the systemic circulation by a process called *first-pass metabolism*. In general, the enzymes responsible for this first-pass effect are decreased in the elderly so that bioavailability of drugs with high hepatic extraction may be increased with age. These drugs require dosage reduction in older adults.

Drug Distribution

Distribution of drugs in the body can be affected by a decrease in lean body mass, an increase in total body fat, or a decrease in total body fluid, all of which may accompany aging. Drugs that bind to muscle (eg, digoxin) become more bioavailable as lean body mass diminishes, increasing the risk for toxicity. Fat-soluble drugs (eg, flurazepam [Dalmane], chlorpromazine [Thorazine], phenobarbital) can be deposited in fat and result in cumulative effects of oversedation. In patients with a volume deficit, drugs that are water soluble (eg, gentamicin

Table 12-6 **Altered Drug Responses in Older People**

Age-Related Changes	Effect of Age-Related Change	Applicable Medications
Absorption		
Reduced gastric acid; increased pH (less acid)	Rate of drug absorption—possibly delayed	Vitamins
Reduced gastrointestinal motility; prolonged gastric emptying	Extent of drug absorption—not affected	Calcium
Distribution		
Decreased albumin sites	Serious alterations in drug binding to plasma proteins (the unbound drug gives the pharmacologic response); highly protein-bound medications have fewer binding sites, leading to increased effects and accelerated metabolism and excretion	Selected highly protein-binding medications: Oral anticoagulants (warfarin) Oral hypoglycemic drugs (sulfonylureas) Barbiturates Calcium channel blockers Furosemide (Lasix) Nonsteroidal anti-inflammatory drugs (NSAIDs) Sulfonamides Quinidine Phenytoin (Dilantin)
Reduced cardiac output	Decreased perfusion of many bodily organs	
Impaired peripheral blood flow	Decreased perfusion	
Increased percentage of body fat	Proportion of body fat increases with age, resulting in increased ability to store fat-soluble medications; this causes drug accumulation, prolonged storage, and delayed excretion	Selected fat-soluble medications: Barbiturates Diazepam (Valium) Lidocaine Phenothiazines (antipsychotics) Ethanol Morphine
Decreased lean body mass	Decreased body volume allows higher peak levels of medications	
Metabolism		
Decreased cardiac output and decreased perfusion of the liver	Decreased metabolism and delay of breakdown of medications, resulting in prolonged duration of action, accumulation, and drug toxicity	All medications metabolized by the liver
Excretion		
Decreased renal blood flow; loss of functioning nephrons; decreased renal efficiency	Decreased rates of elimination and increased duration of action; danger of accumulation and drug toxicity	Selected medications with prolonged action: Aminoglycoside antibiotics Cimetidine (Tagamet) Chlorpropamide (Diabinese) Digoxin Lithium Procainamide

From, Smeltzer SC, Bare BG, Hinkle JL, et al: Brunner and Suddarth's Textbook of Medical–Surgical Nursing, 12th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 213.

[Garamycin]) may have a higher concentration and may reach toxic levels rapidly.

Drug Metabolism

The liver is the major organ for biotransformation and detoxification of medications. Drug-metabolizing reactions are classified as phase I reactions, which involve adding or unmasking a polar chemical group to increase water solubility, and phase II or conjugation reactions, which involve linking the drug to another molecule such as glucose, acetate, or sulfate. In older adults, phase I metabolism is often impaired, whereas phase II metabolism is usually unaffected. In the older patient, there may be some decrease in the metabolism of drugs requiring hepatic enzymes for transformation. This results in an increased plasma level and prolonged half-life of the drug. The benzodiazepines (eg, diazepam [Valium], flurazepam), for example, have a half-life increase from 20 to 90 hours in the older patient.

BOX 12-13 Considerations for Medication Use in Older People

- Drug dosage guidelines are usually based on studies in younger people, and recommended adult dosage guidelines may not be appropriate for older patients.
- Older people may be taking numerous prescription drugs and may self-medicate with borrowed, old, and OTC drugs.
- The effects of alcohol use must be considered.
- The potential for drug interactions and adverse reactions is increased because of the effects of aging on drug absorption, distribution, metabolism, and excretion.
- Drug toxicities are different from those in younger people. Fewer symptoms may be identified, and they may develop more slowly but be more pronounced once they occur.
- Behavioral side effects are more common in older people because the blood–brain barrier becomes less effective. When there is an acute change in mental status, medication should always be considered as the cause.

Hepatic oxidation of these drugs can further be affected by alcohol-induced changes in the liver. There may be a decrease in drug metabolism with occasional alcohol use. In chronic alcohol use, however, drug metabolism is increased, and excretion is accelerated.

Drug Excretion

The kidney is the primary excretory organ for clearing drugs. Drugs that are excreted unchanged (eg, digoxin, cimetidine, antibiotics) or have renally excreted active metabolites require dosage reduction in the older adult to avoid accumulation and toxicity. Serum creatinine alone is not a good determinant of renal function in older people. A creatinine clearance study reflects a more accurate estimation for drug clearance.

▲ Clinical Applicability Challenges

CASE STUDY

Mrs. Z., an 85-year-old white woman, is admitted to the acute care unit with wheezing, increased shortness of breath, decreased physical function (eg, activities of daily living such as bathing and dressing) and increased confusion. She lives in an assisted living facility and over the past few days was noted as "not being herself" in that she was less responsive, ate and drank less, fell twice trying to ambulate to the bathroom, and was less engaged in usual activities. She denied any specific complaints of difficulty with breathing, cough, or bowel or bladder changes. Her past medical history includes degenerative joint disease, Parkinson's disease, dementia associated with Parkinson's (baseline Mini-Cog showed 1/3 recall and an inability to draw a clock, thus screening her as "probably demented"), hypertension, coronary artery disease, overactive bladder, decreased hearing, gastroesophageal reflux disease, depression, vascular insufficiency, and irritable bowel syndrome. Her physical function at baseline was described as "minimum assistance with activities of daily living with help including verbal encouragement and cueing for bathing and dressing upper extremity, and moderate assistance to reach feet for bathing and dressing" She ambulated with a walker and supervision due to balance problems. Current medications included acetaminophen (Tylenol) 1,000 mg PO three times per day PRN for pain; namenda 10 mg PO twice a day; nitroglycerin patch 0.4 mg/h; vitamin D_3 1,000 units daily, olanzapine (Zyprexa) 5 mg daily, aspirin 81 mg PO daily, bupropion (Wellbutrin XL) 300 mg PO daily, losartan (Cozaar) 100 mg PO daily, duloxetine (Cymbalta) 60 mg PO daily; folic acid 1 mg PO daily, and furosemide (Lasix) 40 mg PO daily.

On admission, Mrs. Z denied any specific symptoms. She was pleasant when addressed and answered simple questions appropriately although she initiated no conversation and passively waited for others to address her or intervene. On physical examination, she was febrile with a temperature of 100.1°F and pulse oximetry score of 85% on room air at rest; blood pressure was 128/70 and heart rate 102 bpm and regular. She was noted to have decreased breath sounds in both lung bases and expiratory wheezes throughout. Her respiratory rate was 18. She responded well to oxygen at 2 l via nasal cannula, and her pulse oximetry score improved to 92%. Blood samples were drawn for laboratory testing, and results included white blood cell (WBC) 13.0, hemoglobin 13.4, hematocrit 41.0, platelet count 236.0, neutrophils 10,413, lymphocytes 1,599, creatinine 1.17, BUN 21, calcium 9.3, glucose 200 (nonfasting), sodium 146, potassium 3.9, chloride 109, and carbon dioxide 17; estimated GFR was 46. Mrs. Z.'s chest radiograph showed a normal heart size. There was haziness seen in the right lung base consistent with an early infiltrate. Neurological findings, per Mini-Cog, indicated that she probably has some dementia. She had a bilateral pill-rolling tremor that was worse at rest and when she was stressed to perform. She required moderate assistance to stand, and bradykinesia and retropulsion were noted. Using her rolling walker, she was able to ambulate 10 ft with moderate assistance. She was admitted for pneumonia, and treatment was initiated with ceftriaxone (Rocephin) 1 g IV every 12 hour.

1. What is your immediate concern related to Mrs. Z.'s pulmonary status and underlying complaints?
2. How do you answer Mrs. Z.'s daughter's questions in terms of explaining the laboratory work?

(continued on page 156)

CASE STUDY *(Continued)*

3. How would you encourage Mrs. Z.'s daughter to help her mother optimize her hospital experience and prevent complications?
4. What nursing care interventions are essential to help Mrs. Z. maintain and restore functional ability?
5. What nursing care interventions are appropriate related to pulmonary status and comorbidities?
6. Specifically, what nursing care interventions would you implement related to preventing falls?

References

1. Administration on Aging. Aging Statistics available at: http://www.aoa.gov/AoARoot/Aging_Statistics/index.aspx
2. Grimes A, Chandra SB: Significance of cellular senescence in aging and cancer. Cancer Res Treat 41(4):187–195, 2009
3. Torre P III, Barlow JA: Age-related changes in acoustic characteristics of adult speech. J Commun Disord 42(5):324–333, 2009
4. Laplante-Lévesque A, Hickson L, Worrall L: Rehabilitation of older adults with hearing impairment: A critical review. J Aging Health 22(2):143–53, 2010
5. Smith W, Murphy C: Epidemiological studies of smell: discussion and perspectives. Ann N Y Acad Sci 1170:569–573, 2009
6. Silva GE, An MW, Goodwin JL, et al: Longitudinal evaluation of sleep-disordered breathing and sleep symptoms with change in quality of life: the Sleep Heart Health Study (SHHS). Sleep 32(8):1049–1057, 2009
7. Fetveit A: Late-life insomnia: A review. Geriatr Gerontol Int 9(3): 220–234, 2009
8. Farage MA, Miller KW, Berardesca E, et al: Clinical implications of aging skin: cutaneous disorders in the elderly. Am J Clin Dermatol 10(2):73–86, 2009
9. Ljubicic V, Menzies KJ, Hood DA: Mitochondrial dysfunction is associated with a pro-apoptotic cellular environment in senescent cardiac muscle. Mech Ageing Dev 20(2), 3–9, 2009
10. Okura H, Takada Y, Yamabe A, et al: Age- and gender-specific changes in the left ventricular relaxation: A Doppler echocardiographic study in healthy individuals. Circ Cardiovasc Imaging 2(1):41–46, 2009
11. Mulcahy J, Johnson P, James M: Electrocardiogram QT interval increases in acute stroke. Cerebrovasc Dis 29(2):178–180, 2010
12. Kuznetsova T, Herbots L, López B, et al: Prevalence of left ventricular diastolic dysfunction in a general population. Circ Heart Fail 2(2):105–112, 2009
13. Palmieri V, Russo C, Palmieri EA, et al: Changes in components of left ventricular mechanics under selective beta-1 blockade: insight from traditional and new technologies in echocardiography. Eur J Echocardiogr 10(6):745–752, 2009
14. Soto PF, Herrero P, Schechtman KB, et al: Exercise training impacts the myocardial metabolism of older individuals in a gender-specific manner. Am J Physiol Heart Circ Physiol 295(2):H842–H850, 2008
15. Miyajima H, Nomura M, Nada T, et al: Age-related changes in the magnitude of ventricular depolarization vector: Analyses by magnetocardiogram. J Electrocardiol 33(1):31–35, 2000
16. Forman DE, Clare R, Kitzman DW, et al; HF-ACTION Investigators: Relationship of age and exercise performance in patients with heart failure: The HF-ACTION study. Am Heart J 158(4 Suppl):S6–S15, 2009
17. Nasra J, Belvisi MG: Modulation of sensory nerve function and the cough reflex: Understanding disease pathogenesis. Pharmacol Ther 124(3):354–375, 2009
18. Kemp JP: Exercise-induced bronchoconstriction: The effects of montelukast, a leukotriene receptor antagonist. Ther Clin Risk Manag 5:923–933, 2009
19. Vlassara H, Torreggiani M, Post JB, et al: Role of oxidants/inflammation in declining renal function in chronic kidney disease and normal aging. Kidney Int Suppl 1(114):S3–S11, 2009
20. Bhutto A, Morley JE: The clinical significance of gastrointestinal changes with aging. Curr Opin Clin Nutr Metab Care 11(5):651–660, 2008
21. Schaap LA, Pluijm SM, Deeg DJ, et al; Health ABC Study: Higher inflammatory marker levels in older persons: Associations with 5-year change in muscle mass and muscle strength. J Gerontol A Biol Sci Med Sci 64(11):1183–1189, 2009
22. Steib S, Schoene D, Pfeifer K: Dose-response relationship of resistance training in older adults: A meta-analysis. Med Sci Sports Exerc 45(5):902–914, 2010
23. Srinivas-Shankar U, Wu FC: Frailty and muscle function: role for testosterone? Front Horm Res 37:133–149, 2009
24. Pitroda AP, Harris SS, Dawson-Hughes B: The association of adiposity with parathyroid hormone in healthy older adults. Endocrine 36(2):218–223, 2009
25. Münzer T, Harman SM, Sorkin JD, et al: Growth hormone and sex steroid effects on serum glucose, insulin, and lipid concentrations in healthy older women and men. J Clin Endocrinol Metab 94(10):3833–3841, 2009
26. Kawamoto R, Kohara K, Tabara Y, et al: Insulin resistance and prevalence of prehypertension and hypertension among community-dwelling persons. J Atheroscler Thromb 17(2):148–155, 2010
27. Dharia S, Slane A, Jian M, et al: Effects of aging on cytochrome b5 expression in the human adrenal gland. J Clin Endocrinol Metab 90(7):4357–4361, 2005
28 Todd CH: Management of thyroid disorders in primary care: Challenges and controversies. Postgrad Med J 85(1010):655–659, 2009
29. Aspinall R, Andrew D: Thymic involution in aging. J Clin Immunol 20(4):250–256, 2000
30. Walther K, Birdsill AC, Glisky EL, et al: Structural brain differences and cognitive functioning related to body mass index in older females. Hum Brain Mapp 31(7):1052–1064, 2010
31. Mistur R, Mosconi L, Santi SD, et al: Current challenges for the early detection of Alzheimer's disease: Brain imaging and CSF studies. J Clin Neurol 5(4):153–166, 2009
32. Venkatraman VK, Aizenstein H, Guralnik J, et al: Executive control function, brain activation and white matter hyperintensities in older adults. Neuroimage 49(4):3436–3442, 2010
33. Borson S, Scanlan J, Hummel J, et al: Implementing routine cognitive screening of older adults in primary care: Process and impact on physician behavior. J Gen Intern Med 22(6):811–817, 2007
34. Yesavage JA, Brink TL, Rose TL, et al: Development and validation of a geriatric screening scale: A preliminary report. J Psychiatr Res 17(1): 37–49, 1982–1983
35. Manthorpe J, Iliffe S: Suicide in later life: Public health and practitioner perspectives. Int J Geriatr Psychiatry 25(12):1230–1238

36. Acierno R, Hernandez MA, Amstadter AB, et al: Prevalence and correlates of emotional, physical, sexual, and financial abuse and potential neglect in the United States: The National Elder Mistreatment Study. Am J Public Health 100(2):292–297, 2010
37. Sacco P, Bucholz KK, Spitznagel EL: Alcohol use among older adults in the National Epidemiologic Survey on Alcohol and Related Conditions: A latent class analysis. J Stud Alcohol Drugs 70(6):829–838, 2009
38. Resnick B: Alcohol use in a continuing care retirement community. J Gerontol Nurs 29(10):22–29, 2003

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

13 The Postanesthesia Patient

Nathaniel M. Apatov and E. Jane McCarthy

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. List anesthetic techniques used for surgery and interventional procedures.
2. Describe nursing interventions and assessment strategies for the patient recovering from anesthesia.
3. Explain common complications encountered during the immediate postanesthetic period and the necessary nursing interventions.
4. Compare and contrast moderate IV sedation and general anesthesia.

The time immediately after surgery is the most crucial period in the patient's recovery from anesthesia. Patients are taken to the postanesthesia care unit (PACU) to receive nursing care from a PACU nurse or directly to the intensive care unit (ICU) to receive nursing care from an ICU nurse. The purpose of this chapter is to describe anesthetic techniques used during surgery and the complications that can occur during the immediate postoperative time to help the critical care nurse better understand the nursing care needs of the immediate postanesthetic patient. To help better understand anesthesia techniques used intraoperatively, common clinical terms related to the use of anesthesia are listed in Box 13-1.

▲ Preoperative Anesthesia Patient Assessment

The anesthesia provider interviews and examines the patient before surgery. From this preanesthetic examination and discussions with the patient and surgeon, the anesthesia provider decides the anesthetic technique. This decision is based on the patient's age, anesthetic history, medical history, and the operation to be performed. The anesthesia provider's options range from conscious sedation with the use of regional or intravenous (IV) agents to general anesthesia with the use of IV or inhalational anesthetic agents. Whenever possible, the patient and family are part of the decision-making process. These options are illustrated in Table 13-1 and Figure 13-1.

▲ Postanesthesia Report to PACU or ICU Nurse

What happens in the operating room (OR) affects the patient's immediate postoperative care and overall recovery. To convey what has occurred during surgery, the anesthesia provider gives a detailed report to the nurse who is assuming immediate postoperative care of the patient. Information given in the report is listed in Box 13-2. Initial assessment parameters reported to the anesthesia provider by the nurse are the patient's blood pressure, pulse rate, respiratory rate, temperature, oxyhemoglobin saturation (SaO_2), and level of consciousness. The anesthesia provider gives a report on intraoperative hemodynamic parameters, anesthetic technique, surgical procedure, urine output, blood loss, and fluid replacement. While receiving the report from the anesthesia provider, the nurse simultaneously assesses the patient and develops a nursing plan of care. In the PACU or ICU, vital signs are monitored every 15 minutes or more often if the patient's condition warrants. Box 13-3 on page 161 provides collaborative care guidelines for the postanesthesia patient. The American Society of Peri Anesthesia Nurses, endorsed by the American Society of Anesthesiologists and the American Association of Nurse Anesthetists, recommends that all assessment data be collected and documented on the patient's postoperative record.[1]

In addition, while assessing the patient and taking vital signs, the nurse uses the "stir-up" regimen that involves encouraging the patient to deep-breathe, cough, and move as allowed by the surgical procedure or intervention. The nurse also assesses pain levels and implements appropriate interventions to assist the patient in participating in the "stir-up" regimen. This regimen also allows the nurse to identify changes in the patient's cognitive function.

▲ Complications in the Postanesthesia Patient

The nurse must be able to assess and treat common complications in the postanesthesia patient. Postanesthesia patient care focuses primarily on cardiopulmonary assessment and management. The following are the common complications seen in the nursing care of the immediate postoperative patient.

BOX 13-1 Clinical Terminology

Sedation: An induced state of quiet, calm, or sleep by means of a medication. The degree of sedation ranges from anxiolysis to anesthesia.
Minimal sedation: The patient responds normally to verbal stimuli. Impairment to cognition and coordination may exist.
Moderate sedation: A drug-induced depression of consciousness during which the patient responds purposefully to verbal commands either alone or in conjunction with tactile stimulation. There is some alteration of mood, drowsiness, and sometimes analgesia. The patient's protective reflexes remain intact.
Deep sedation: A drug-induced depression of consciousness during which the patient cannot be easily aroused but responds purposefully after repeated or painful stimulation. Spontaneous ventilation and the ability to maintain a patent airway may be impaired. The patient may require assistance in maintaining a patent airway.
General anesthesia: A drug-induced loss of consciousness during which a patient cannot be aroused, even by painful stimulation. The ability to independently maintain ventilatory function may be impaired. The patient may require assistance in maintaining a patent airway and positive-pressure ventilation may be required. Cardiovascular function may be depressed.
Monitored anesthesia care (MAC): A specific anesthesia service in which an anesthesia provider has been requested to participate in the care of a patient undergoing a therapeutic or diagnostic procedure. It does not describe the depth of sedation.
Regional anesthesia: Regional anesthesia is achieved by placing local anesthetics close to appropriate nerves to achieve a conduction block that provides analgesia and numbness.
Spinal anesthesia: A local anesthetic is injected into the lumbar intrathecal space. The anesthetic blocks conduction in spinal nerve roots and dorsal ganglia. Anesthesia and analgesia usually occur below the level of injection.
Epidural anesthesia: A local anesthetic is injected via a catheter into the epidural space. The effects are similar to spinal analgesia.
Peripheral nerve block: A local anesthetic is injected at a specific nerve site to achieve a defined area of anesthesia.

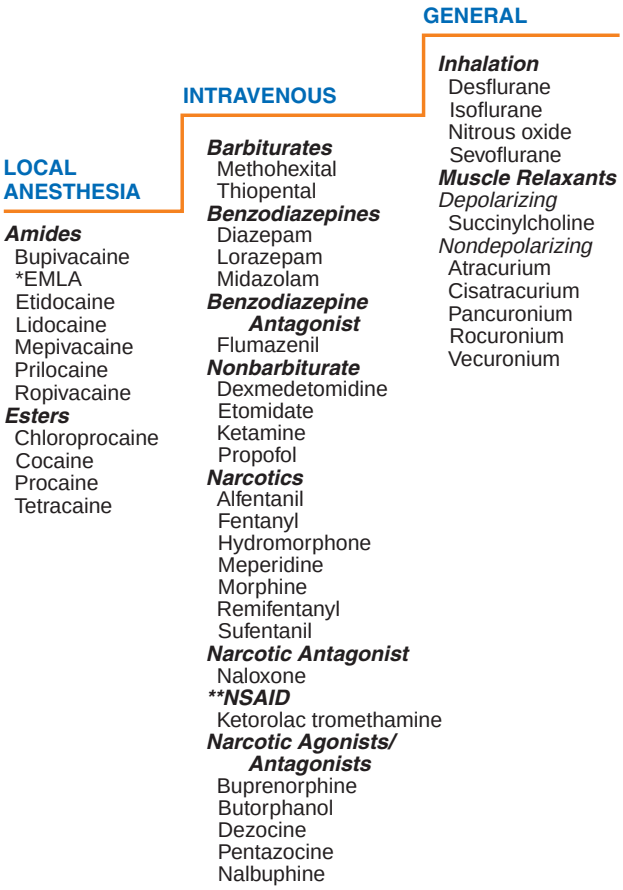
Hypoxemia

Hypoxemia characterized by an oxyhemoglobin saturation (SaO_2) of less than 90% or a partial pressure of arterial oxygen (PaO_2) of less than 60 mm Hg is life threatening. General anesthetic drugs can cause hypoxemia in the immediate postoperative period as a result of ventilation–perfusion mismatch.[2] Signs and symptoms of hypoxemia include tachycardia, cardiac dysrhythmias, dyspnea, tachypnea, disorientation, agitation, and cyanosis. Hypoxemia and hypoventilation may also occur postoperatively following the use of a spinal or epidural regional anesthetic that has blocked the spinal nerves innervating the muscles of respiration and weakening the respiratory effort.[3]

When nitrous oxide is used for general anesthesia, 100% oxygen is administered in the OR for 3 to 4 minutes at the discontinuance of this anesthetic. These patients are transported receiving oxygen to prevent diffusion hypoxia in the PACU. Patients who receive a general anesthetic or sedation receive supplemental oxygen in the immediate postoperative period because general anesthesia and sedation cause a compromise in respiratory function. Patients in the immediate postoperative period are routinely monitored for adequate oxygenation by using the pulse oximeter, a noninvasive device that continuously monitors oxygen hemoglobin saturation (SaO_2). This device has been used in the OR since the 1980s and more recently in the PACU, resulting in a significant decrease in anesthesia and postanesthesia respiratory mishaps.[4]

Table 13-1 Anesthetic Options for Surgical and Interventional Procedures

Conscious State	Sedated State	Unconscious State
Modalities		
Conscious sedation Monitored anesthesia care (MAC) Local anesthesia Regional anesthesia	MAC Local anesthesia Regional anesthesia	Regional anesthesia General anesthesia
Medications		
Local anesthetics Intravenous (IV) medications	Local anesthetics IV medications	Local anesthetics IV medications Inhalation anesthetics Muscle relaxants
Effect on Patient		
Patient cooperative Follows commands Maintains protective reflexes	May follow commands Usually maintains protective reflexes	Unconscious Diminished or loss of protective reflexes Alterations in cardiopulmonary dynamics



* EMLA, Eutectic mixture local anesthetics
** NSAID, Nonsteroidal anti-inflammatory drugs

FIGURE 13-1 ▲ Medication choices for anesthetic options

Reversal agents are used intraoperatively to reverse the effects of muscle relaxants, sedatives, and opioids. The effects of muscle relaxants, sedatives, and opioids may last longer than the reversal agent resulting in hypoventilation and hypoxemia even though a reversal agent has been administered. For this reason, it is important to know the onset and duration of action of drugs used to reverse the effects of the sedative, muscle relaxant, or narcotic. This knowledge allows for appropriate assessment and intervention when a change in the patient's condition occurs.

BOX 13-2 Anesthesia Provider-to-Nurse Report: Information to Convey

Name of patient
Surgical procedure
Type of anesthetic (agents and reversal agents used)
Estimated blood loss
Fluid and blood replacement
Intraoperative blood pressure and heart rate range
Intraoperative complications
Preoperative patient history
Language barrier

Note: The anesthesia provider should not leave the patient until the nurse is satisfied with the patient's immediate postoperative condition.

Hypoventilation

Hypoventilation leading to hypercarbia ($PaCO_2$ > 45 mm Hg) may result from the following:

- Inadequate respiratory drive secondary to the effects of residual anesthesia, such as opioids, sedatives, and general anesthesia inhalational agents
- Inadequate functioning of respiratory muscles causing inadequate tidal volume due to residual neuromuscular blockade
- Chronic pulmonary disease, such as chronic obstructive pulmonary disease, which may require postoperative ventilatory support
- Laryngospasm or upper airway obstruction resulting from residual general anesthesia (Box 13-4)

The nurse institutes the "stir-up" regimen in the immediate postoperative phase to stimulate the patient, especially if opioids or sedatives were used during surgery. The nurse considers the length of time since reversal agents were administered to antagonize neuromuscular blockade. The patient may not be fully reversed and may exhibit signs of residual neuromuscular blockade assessed by the inability to maintain a head lift for 5 seconds. Moreover, inappropriate use of chest and abdominal wall muscles will result in air hunger, anxiety, and tachycardia. Neuromuscular blocking agents are summarized in Box 13-5. The respiratory effect of neuromuscular nondepolarizing drugs is prolonged with hypothermia. Other conditions that increase the effects of nondepolarizing muscle relaxants are listed in Box 13-6 on page 163.

Hypotension

The most common cardiovascular complication seen in the postoperative period is hypotension due to decreased blood volume resulting from preoperative fasting and intraoperative blood loss. Intervention is indicated if the pressure decreases by more than 30% of the baseline blood pressure.[5] Risk factors for postanesthesia hypotension are listed in Box 13-7 on page 163.

General and regional anesthetics also decrease blood pressure. Spinal and epidural regional anesthetic techniques cause vasodilation and decrease blood pressure resulting from the sympathetic blockade. Opioids cause histamine release and vasodilation resulting in lowered blood pressure. Droperidol and chlorpromazine hydrochloride produce sympathetic blockade and hypotension. Barbiturates and anesthetic inhalational agents, such as isoflurane, sevoflurane, and desflurane, cause myocardial depression. Often, postoperative orthostatic hypotension is due to hypovolemia and decreased venous return to the heart because of inadequate intraoperative volume replacement, blood loss, loss of fluid into the interstitial space, and diuretics. The patient is evaluated for orthostatic hypotension by measuring blood pressure and heart rate in the supine position and in a position with the head of the bed raised 60 degrees, if not contraindicated by the surgical procedure or the patient's status. With orthostatic hypotension, there will be a significant decrease in blood pressure with the head of the bed raised. Cardiac dysrhythmias, such as supraventricular tachycardia and marked bradycardia, can cause a decrease in cardiac output leading to hypotension. Other causes of postoperative

BOX 13–3 *COLLABORATIVE CARE GUIDE for the Postanesthesia Patient*

Outcomes	Interventions
Oxygenation/Ventilation	
Adequate respiration after extubation SaO_2 returns to the preoperative value without supplemental O_2. Airway maintained with intact protective reflexes. No evidence of aspiration.	• Monitor respiratory rate and breathing pattern every 15 min and PRN. • Assess weaning parameters before extubation. • Monitor end-tidal CO_2 and pulse oximetry of mechanically ventilated patients. • Encourage patient to cough and deep-breathe. • Elevate head of bed if not contraindicated. • Use jaw thrust, head tilt, or oral, oropharyngeal, or nasopharyngeal airway to maintain airway. • Stimulate patient every few minutes (eg, call name, touch). • Administer antiemetic as indicated. • Position patient on side; suction and maintain airway if patient is vomiting.
Circulation/Perfusion	
Heart rate and blood pressure returned to preoperative values within 1–2 h after anesthesia. Body temperature within normal limits. No evidence of malignant hyperthermia.	• Monitor vital signs every 15 min and PRN. • Assess pulse quality and regularity. • Monitor for dysrhythmias. • Monitor for hypotension related to bleeding. • Monitor for hypotension related to warming and vasodilation. • Administer IV solution and blood products as ordered. • Anticipate hypothermia; have warming devices readily available. • Measure temperature on admission and PRN until normal. • Warm patient at 1°C–2°C/h. • Monitor for malignant hyperthermia and immediately notify anesthesia provider of temperature increase of 0.5°C. • Administer dantrolene and initiate cooling measures. • Assist with malignant hyperthermia protocol.
Fluids/Electrolytes	
Patient has stable blood pressure and heart rate. Urine output will be 0.5–2 mL/kg/h. No evidence of hypervolemia or hypovolemia.	• Maintain patient IV. • Monitor intake and output. • Assess skin and mucous membranes for signs of hypovolemia. • Measure specific gravity if indicated. • Assess for signs of hypervolemia (eg, pulmonary crackles, neck vein distention). • Measure serum electrolytes if indicated.
Mobility/Safety	
Patient will arouse easily and respond appropriately to commands. Patient will move all extremities purposefully and with normal strength.	• Assess level of consciousness every 15 min and PRN. • Monitor motor and sensory function to assess reversal of neuromuscular blockade. • Assess level of regional block, epidural, or spinal anesthesia.
Skin Integrity	
Skin will remain intact.	• Assess skin immediately postoperatively for pressure areas and burns.
Nutrition	
Nutritional intake will be reestablished without nausea or vomiting.	• Resume enteral feeding with return of bowel sounds. • Begin oral fluids with return of protective airway reflexes.
Comfort/Pain Control	
Pain will be <4 on numeric pain scale or visual analog.	• Assess location, type, and severity of pain. • Administer opioids as indicated. • Monitor response to analgesics. • Institute nonpharmacological pain relief strategies and comfort measures. • Evaluate patient-controlled analgesia IV or epidural as postoperative pain management option.

(continued on page 162)

BOX 13-3 COLLABORATIVE CARE GUIDE for the Postanesthesia Patient (continued)

Outcomes	Interventions
Psychosocial	
Personal support systems will be used to reduce anxiety.	• Encourage significant other visits in early postoperative phase. • Validate patient's significant other's understanding of surgery and illness. • Initiate referrals to social services, clergy, and so forth.
Teaching/Discharge Planning	
Discharge from postanesthesia care phase will occur within 1–2 h. Exercises to prevent postoperative pulmonary complications will be demonstrated. Patient or significant other will state understanding of surgical procedure and outcome of surgery.	• Orient patient frequently. • Explain procedures and pain management treatment plan. • Teach coughing, deep breathing, and incentive spirometer use. • Teach early mobilization. • Teach pain control strategies. • Provide information regarding the procedure, and discuss probable outcomes.

hypotension include sepsis, pulmonary embolism, and transfusion reaction.

Deliberate, controlled hypotensive intraoperative anesthetic techniques are used during surgical procedures, such as neurosurgery, shoulder arthroscopy, and maxillofacial surgery. This hypotensive technique minimizes blood loss, decreases intraoperative blood transfusions, decreases oozing, and minimizes hematoma formation. These patients must be monitored closely postoperatively until blood pressure returns to normal.

Treatment of immediate postoperative hypotension depends on the underlying cause. A priority is to ensure adequate oxygenation and ventilation of the patient while the blood pressure is addressed. Anesthetic drugs may require reversal, including muscle relaxant reversal with anticholinesterase agents, opioid reversal with naloxone (Narcan), and benzodiazepine reversal with flumazenil (Romazicon). IV fluids, blood products, plasma expanders,

BOX 13-4 Managing Laryngospasm and Airway Obstruction

Laryngospasm

Laryngospasm is often caused by blood, mucus, or other oral secretions irritating the vocal cords. Suctioning of the oropharynx before extubation helps to prevent laryngospasm. Laryngospasm is treated with positive-pressure ventilation using 100% FiO_2 via a bag-valve mask with a tight seal. If this does not break the spasm, a small dose of depolarizing muscle relaxant (succinylcholine) may be given IV.

Upper Airway Obstruction

Upper airway obstruction must be identified and treated promptly and effectively. Airway obstruction may range from minimal to complete. Signs of obstruction include

- Paradoxical breathing
- Stridor
- Lack of, or change in, breath sounds
- Hypoxemia
- Change in level of consciousness

Treatment to relieve obstruction must be provided in a systematic fashion.

1. Tilt head/lift chin.
2. Thrust jaw.
3. Call for assistance.
4. Insert an oropharyngeal or nasopharyngeal airway. (An oropharyngeal airway may not be tolerated by the partially anesthetized patient.)
5. Apply positive-pressure ventilation.
6. Perform endotracheal intubation if necessary.

BOX 13-5 Neuromuscular Blocking Agents

Muscle Relaxants

- Muscle relaxant drugs used in anesthesia paralyze patients but provide no sedation or analgesia.
- Muscle relaxants facilitate endotracheal intubation, relax muscles for surgical procedures, terminate laryngospasm, eliminate chest wall rigidity, and provide for ease of mechanical ventilation if indicated.
- Depolarizing and nondepolarizing muscle relaxants are used in anesthesia and work at the myoneural junction by blocking nicotinic acetylcholine receptors.

Depolarizing Agent (Succinylcholine)

- Succinylcholine combines with acetylcholine receptors at the myoneural junction and mimics the action of acetylcholine.
- Onset of action is 1 to 2 minutes and duration of action is 4 to 6 minutes.
- The enzyme pseudocholinesterase removes succinylcholine from plasma, so in conditions involving a decrease in pseudocholinesterase, the length of action of succinylcholine increases, keeping patients paralyzed for longer periods.
- Decreased pseudocholinesterase enzyme may be seen in pregnancy, liver disease, malnutrition states, severe anemia, cancer, and with other pharmacological agents, such as quinidine, phospholine eye drops, and propranolol.

Nondepolarizing Agents

- Nondepolarizing agents (atracurium, cisatracurium, pipecuronium, vecuronium, pancuronium, doxacurium, rocuronium) compete with acetylcholine at the myoneural junction for muscle membrane receptors.
- Onset of action is within 2 to 3 minutes, depending on dose.
- Duration of action ranges from 20 minutes to 2 hours, depending on the drug and dosage.
- Muscle relaxant reversal agents (neostigmine, edrophonium) duration of action may be shorter than the duration of action of muscle relaxants, resulting in respiratory muscle weakness and respiratory insufficiency. Anticholinesterases cause muscarinic side effects including bradycardia and increased salivary secretions. These side effects are counteracted with the administration of an anticholinergic drug (atropine, glycopyrrolate) in conjunction with the anticholinesterase.

BOX 13-6 Conditions and Drugs That Increase the Effects of Nondepolarizing Muscle Relaxants

Local anesthetics
General anesthetics
Antibiotics: aminoglycosides, polypeptides, polymyxin
Antiarrhythmics: quinidine, procainamide
Furosemide (Lasix)
Acid–base status: respiratory acidosis, metabolic alkalosis
Electrolyte imbalance: hypokalemia, hypocalcemia, dehydration, magnesium administration
Hypothermia

crystalloids, and vasopressor drugs are administered to increase blood pressure caused by intraoperative blood loss. The nurse also inspects wound dressings, drains, and surgical sites for postoperative bleeding. If there is significant postoperative bleeding, the surgeon must be notified and the patient may have to return to the OR.

When assessing and treating a patient with hypotension, the nurse must also rule out the possibility of a technical problem rather than physiological problems. Is the blood pressure cuff the correct size and is it positioned correctly? Is the stethoscope positioned correctly? Is the patient's position a factor? If an arterial line is present, is the patient peripherally constricted or does the patient have significant peripheral vascular disease? Is the transducer correctly calibrated? Troubleshooting for device errors occurs simultaneously with patient assessment.

Hypothermia

Heat loss during surgery is due to reduced basal metabolism and the anesthetized patient's inability to shiver to produce heat to maintain body temperature. Inhalational anesthetic agents depress the thermoregulatory center resulting in hypothermia. Vasodilation occurs with regional anesthesia because of the sympathetic blockade resulting in heat loss and hypothermia. Other intraoperative causes of hypothermia include heat loss through radiation, convection, and conduction because of prolonged skin exposure, saturated surgical drapes, cold antiseptic prepping solutions, cold irrigation solutions, and cold IV solutions. Geriatric patients are at greater risk for hypothermia because of alterations in their hypothalamic function. Neonates are more at risk for hypothermia because of their immature thermoregulatory center and their high body surface to volume ratio. Hats, warm blankets, forced air warming devices, and IV fluid warmers are used to prevent hypothermia preoperatively, intraoperatively, and postoperatively.

Patients admitted to the PACU or ICU with hypothermia have prolonged postoperative recovery time and higher incidence of postoperative complications such as wound infection.[6] Rewarming of the postoperative patient is done immediately using heated blankets, warm IV fluids, and warming devices such as the forced air warmer.

BOX 13-7 *PATIENT SAFETY*

Factors That May Cause Postoperative Hypotension

Drug and Conditional Factors
- Epidural or spinal anesthetic blockade
- Inhalation anesthetic agents
- Hypovolemia
- Hypothermia
- Myocardial depression
- Sepsis
- Transfusion reaction
- Increased intrathoracic pressure from mechanical ventilation
- Dysrhythmias (supraventricular tachycardia)
- Myocardial infarction
- Congestive heart failure
- Bradycardia
- Pain
- Bladder/abdominal distention

Technical Factors
- Blood pressure cuff size and position
- Tight abdominal dressing
- Transducer balance and calibration
- Stethoscope position

Postoperative Nausea and Vomiting

Postoperative nausea and vomiting (PONV) is one of the more common problems in the PACU and is a frequent cause of postoperative hospital admission.[7] Although not life threatening, PONV leaves the patient with a lasting unpleasant memory and may have an impact on future surgical and anesthetic decisions. Frequent causes include use of preoperative and intraoperative opioids, increased gastric secretions, spinal anesthesia, and surgical procedures involving manipulation of eye muscles, abdominal muscles, or genitourinary muscles. Laparoscopic techniques and procedures involving the breast are also associated with an increase in PONV.

Vomiting is regulated by the vomiting center located in the medulla and receives stimuli from the gastrointestinal tract, the chemoreceptor trigger zone, the labyrinthine apparatus (motion sickness), and cortical and visual input. Examples of stimuli that cause vomiting are gastric distention, opioids, anesthetic drugs, hypoxemia, postoperative pain, and hypotension. PONV can usually be relieved by identifying the causative factor and making the appropriate intervention. Antiemetic drugs given to treat PONV in the immediate postoperative period may have a synergistic effect on opioids, and decreasing the dose of narcotic is indicated. Patients at increased risk for PONV are treated using a multimodal pharmacologic approach perioperatively beginning in the preoperative period. The anesthetized patient is not only prone to vomiting but, because of the unprotected reflexes, there is a greater chance of regurgitation and pulmonary aspiration. Proper positioning of patients with altered levels of consciousness is essential. The ideal position is on the side with the head and neck extended. If the surgical procedure precludes turning the patient on the side or the patient is unable to comply, then the patient must not be left unattended until consciousness is regained. A Yankaeur suction device attached to wall suction should be immediately available.

Postoperative Pain

Postoperative surgical pain is caused by several factors including the surgical incision, tissue manipulation, the

BOX 13-8 Factors Influencing Pain

Surgical procedure: site and nature of the operation
Anxiety level: fear of surgery, disfigurement, death, loss of control, previous experiences
Patient expectations: effectiveness of preoperative teaching, adequately prepared for outcome
Pain tolerance: prior use of medications, including analgesics, individual differences
Anesthesia technique: analgesics used during the intraoperative period, use of naloxone

psychological state of the patient, and the anesthetic technique used intraoperatively. Adequate pain relief is important during the postoperative period because it allows the patient to cough, deep-breathe, and ambulate sooner, thus reducing the incidence of postoperative complications such as atelectasis pneumonia. If the anesthetic was an inhalation anesthetic agent without opioids or local anesthetics, a patient may have more pain than a patient who received intraoperative opioids or a regional anesthetic. Patients who have been given analgesics during surgery and who then receive naloxone, an opioid antagonist, at the end of surgery may experience severe pain because the opioid antagonist reversed the analgesic effects of any prior opioid drugs. Because these patients may experience hypoventilation again, the nurse must wait 15 to 45 minutes after the administration of naloxone before giving an analgesic drug. Box 13-8 lists factors that influence the patient's response to pain. The following are types and methods of administration of analgesic drugs commonly used for the immediate postoperative patient.

Intravenous Opioid Drugs

Intravenous (IV) titration of opioids, such as morphine, fentanyl, or hydromorphone, in the immediate postoperative period offers the fastest and most effective method of pain relief. Because the patient's basal metabolic rate decreases during surgery and the patient may be hypothermic, the uptake of intramuscular medication is often difficult to predict.

Toradol

Tordadol (ketorolac tromethamine) may also be administered during surgery and has been found effective in treating immediate mild to moderate postoperative pain. Torodol is a nonsteroidal anti-inflammatory drug that exhibits analgesic, anti-inflammatory, and antipyretic activity. Peak analgesia occurs in 45 to 60 minutes after intramuscular or IV injection, and the analgesic effect lasts 6 to 8 hours. This drug is given in doses up to 30 mg and is not used for more than 5 days postoperatively. The drug is contraindicated in patients with active peptic ulcers, recent gastrointestinal bleeding, or renal insufficiency.

Patient-Controlled Analgesia

The use of patient-controlled analgesia (PCA) in the PACU allows the patient to administer his own analgesic IV using a PCA device. Clinical studies show that patients report less pain when they are able to control the administration of opioids for their pain relief.[8] PCA analgesia may be used to inject analgesic drugs IV; alternatively, it may be used to inject analgesic drugs into the epidural space. Both modalities are effective ways for patients to control their own analgesic needs.

Epidural Opioid Drugs

Epidural opioid analgesia is effective in treating acute postoperative pain.[9] Patients who receive epidural opioids are less sedated, able to ambulate sooner, and have improved respiratory function. Epidural opioids are administered as a bolus injection at the end of surgery or by a continuous epidural infusion via an epidural catheter postoperatively. When administering continuous epidural infusions, an infusion pump is used. Epidural drugs must be preservative free. To ensure patient safety during epidural opioid infusion, infusion sets are used that have no injection ports. The infusing pump, infusion bag, and infusion tubing are clearly labeled with the word *epidural*. The reason for these safeguards is that accidental infusion of other drugs with preservatives could cause neural damage resulting in paralysis or death. Frequently used preservative-free epidural drugs include morphine, hydromorphone, and fentanyl. The duration of analgesia varies with the narcotic administered. Frequently used opioids for epidural administration are preservative-free morphine, which has a duration of action from 2 to 24 hours; hydromorphone, which has a duration of 10 to 14 hours; and fentanyl, which has a duration of 4 to 6 hours.

Epidural Local Anesthetic Drugs

Dilute local anesthetic solutions using lidocaine, ropivacaine, bupivacaine, or etidocaine are given epidurally for postoperative pain either with opioids or alone. The combination of local anesthetics and opioids has been used to optimize analgesia and minimize the side effects of local anesthetics and opioids using minimal doses for both. Nurses are responsible for recognizing and treating the side effects (Box 13-9) of patients who are receiving epidural analgesia.

Hypertension

The most common causes of postoperative hypertension are pain and hypothermia. Hypertension is seen with hypothermia because of the peripheral vasoconstriction and shivering. Hypertension also occurs with hypoxemia and hypercarbia because of the endogenous catecholamine release. Ketamine, a nonbarbiturate drug, which is used as a dissociative anesthetic, stimulates the sympathetic nervous system and may cause tachycardia and hypertension. Naloxone, if given too rapidly, may cause hypertension leading to pulmonary edema or cerebral hemorrhage. Other causes of hypertension include anxiety, urinary bladder distention, fluid overload, a too-narrow blood pressure cuff, and withholding antihypertensive drugs before surgery. Unless instructed otherwise, patients should take their antihypertensive drugs up to the day of surgery.

Hypertensive patients require reassurance and close observation. Mild to moderate hypertension in the immediate postoperative period is usually treated with IV vasoactive drugs such as hydralazine (Apresoline) and labetalol (Trandate). Hypertensive crises do occur in the immediate postoperative period, and continuous IV infusions of nicardipine, sodium nitroprusside, or nitroglycerin are used to keep the blood pressure in a safe range. When hypertension

BOX 13-9 Side Effects of Epidural Analgesia and Possible Remedies

Urinary Retention
- Catheterize as needed.

Postural Hypotension
- Give fluid (volume) replacement.
- Administer ephedrine 5 mg or phenylephrine 50 mcg IV as ordered.

Pruritus (Itching of Face, Head, and Neck)
- Treat with diphenhydramine (Benadryl) 25 mg PO, IM, IV.
- Treat with naloxone (Narcan) 0.1 mg IV.
- Treat with propofol 10 mg IV.

Nausea and Vomiting
- Administer metoclopramide (Reglan) 10 mg IV.
- Administer scopolamine patch.
- Administer a 5HT-3 antagonist

Respiratory Depression
- Administer naloxone 0.1 mg up to a maximum of 0.4 mg IV.
- Monitor for 30 minutes after naloxone administration because opioid half-life may be longer

accompanies emergent delirium, IV sedatives may be required. If the patient is hypertensive because of anxiety and verbal reassurance is ineffective, benzodiazepines, such as midazolam (Versed), may be necessary. If the hypertension results from fluid overload during surgery, the patient may require urinary catheterization and diuretics, such as furosemide (Lasix).

Cardiac Dysrhythmias

The cardiac dysrhythmias covered in this chapter are those induced by anesthetic agents and complications frequently seen in the immediate postoperative period (Table 13-2). Chapter 17 provides detailed information on cardiac dysrhythmias. The most common causes of cardiac dysrhythmias in the immediate postoperative period are hypoventilation, electrolyte imbalances, hypoxemia, hypovolemia, fluid overload, hypothermia, and pain (Box 13-10).

Malignant Hyperthermia

Malignant hyperthermia (MH) is an autosomal dominant, pharmacogenetic disorder of skeletal muscle characterized by a hypermetabolic response to an anesthetic triggering agent resulting in skeletal muscle damage, hyperthermia, and death if untreated (see Spotlight on Genetics 13-1). With an MH episode, not every patient responds the same and not every anesthetic will cause MH in an MH susceptible patient. Most MH episodes occur in the OR during general anesthesia and less frequently in the immediate postoperative period. MH is triggered in susceptible individuals by halogenated inhalational anesthetic agents and the depolarizing muscle relaxant succinylcholine. Other anesthetic agents such as nitrous oxide, local anesthetics, opioids, propofol, sodium thiopental, and the nondepolarizing muscle relaxants do not trigger MH episodes.

The prevalence of MH is unknown and can vary by geographic location. The states with the highest incidence are Michigan, West Virginia, and Wisconsin.[10] MH is more prevalent in people with muscular abnormalities, such as

Table 13-2 Cardiac Dysrhythmias Associated With Anesthetics

Anesthetic Agent	Dysrhythmia
Local anesthesia with epinephrine	Tachycardia
Spinal and epidural	Bradycardia secondary to vagal response
Barbiturates	
Sodium pentothal	Bradycardia, AV dissociation, occasional PVCs
Nonbarbiturate etomidate	Sinus tachycardia
Morphine sulfate	Transient brachycardia
Meperidine hydrochloride	Transient tachycardia
Fentanyl	Bradycardia
Opioid antagonist	PVCs, ventricular tachycardia, occasional ventricular fibrillation
Ketamine	Tachycardia
Isoflurane	Tachycardia
Enflurane, Sevoflurane, Desfluran1e	AV dissociation, tachycardia
Muscle relaxants	
Succinylcholine	Sinus bradycardia, junctional rhythms, PVCs Patients with burns, trauma, paraplegia or quadriplegia prone to ST-segment depression, peaked T waves, widening QRS complex leading to ventricular tachycardia, ventricular fibrillation, or asystole
Pipecuronium bromide	Atrial fibrillation, ventricular extrasystole
Pancuronium	Tachycardia and nodal rhythms
Anticholinesterases	Bradycardia, slowed AV conduction, PVCs
Anticholinergics	Tachycardia

AV, atrioventricular; PAC, premature atrial contraction; PVC, premature ventricular contraction.

BOX 13-10 Causative Factors for Dysrhythmias

- Hypoxemia (sinus bradycardia, sinus tachycardia, premature ventricular contractions [PVCs], supraventricular tachycardia)
- Hypoventilation/hypercarbia (sinus tachycardia, PVCs, sinus bradycardia)
- Hypovolemia (sinus tachycardia)
- Fluid overload (PVCs, supraventricular tachycardia, premature atrial contractions [PACs], atrial fibrillation/flutter)
- Hyperthermia (sinus tachycardia, PVCs)
- Hypothermia (sinus bradycardia, atrial fibrillation, atrioventricular nodal blocks)
- Pain (sinus tachycardia, PVCs)

muscular dystrophy. The incidence of MH has been estimated to be as low as 1 in 250,000 patients receiving anesthetics to as high as 1 in 4,200 patients receiving anesthetics using triggering agents. MH was first observed in the 1960s with the introduction of inhalational anesthetic agents with resultant mortality of 80%. Today, with the use of intraoperative monitoring of end-tidal CO_2 ($ETCO_2$), SaO_2, and dantrolene sodium treatment, the mortality has decreased to about 5%.

The pathophysiology of MH occurs when the MH susceptible skeletal muscle is exposed to an MH triggering agent, and calcium is released at an abnormally high rate resulting in excessive muscle contraction, increased metabolism, high oxygen consumption, high CO_2 production, and heat production. This leads to generalized muscle rigidity including masseter muscle rigidity, increased end tidal CO_2 ($ETCO_2$), acidosis, hypoxemia, tachycardia, and myoglobinuria.

The classic presentation of MH includes a rapid rise in $ETCO_2$, generalized muscle rigidity including masseter muscle rigidity of the jaw, unexplained tachycardia, hyperthermia, myoglobinuria, acidosis, and hyperkalemia. Masseter muscle rigidity after the administration of succinylcholine may be the earliest sign of MH. Temperature elevation is a late sign of MH and is preceded by an elevation of $ETCO_2$. If these symptoms occur, the anesthetic is discontinued and treatment rapidly instituted.

SPOTLIGHT ON GENETICS 13-1

Malignant Hyperthermia Susceptibility (MHS)

- Is a rare reaction that occurs with the use of volatile inhalation agents such as isoflurane, enflurane, sevoflurane, and desflurane.
- Is due to the genetic mutation of the *RYR1* gene that codes for the ryanodine receptor that regulates calcium release
- The mutation of the *RYR1* gene, in the presence of the inhalation agent, affects the skeletal muscle, leading to the sustained release of calcium from the sarcoplasmic reticulum causing the hypermetabolic state to occur in the tissue.
- Can now be identified via genetic testing of those patients and/or family members who have experience complications during or immediately postoperative

Genetic Home Reference-http://ghr.nlm.nih.gov—Accessed July 14, 2011

Anderson-Pompa K, Foster A, Parker L, et al: CE Article: Genetics and susceptibility to malignant hyperthermia. Crit Care Nurs 28(6):32–36, 2008.

BOX 13-11 Contents of Malignant Hyperthermia Kit

- Methylprednisolone
- Furosemide
- Sodium bicarbonate
- Dextrose (50%)
- Sterile water
- Insulin
- Mannitol
- Refrigerated intravenous (IV) fluids
- Dantrolene sodium
- New oxygen tubing and delivery devices
- Foley catheter tray
- Nasogastric tubes
- Blood specimen tubes
- Arterial blood gas kits
- MHAUS guidelines and contact information booklet

MHAUSv, Malignant Hyperthermia Society of the United States.

MH is treated by discontinuance of the anesthetic triggering agent, 100% inspired oxygen, hyperventilation, correction of acid–base imbalances, and cooling measures with a cooling blanket and cool IV fluids. Dantrolene sodium, 2.5 mg/kg IV, is given and may be repeated up to 10 mg/kg as necessary to control MH signs and symptoms. Dantrolene sodium is reconstituted with preservative-free sterile water and is labor intensive because of its difficulty in reconstituting with an aqueous solution. Most ORs and PACUs have an MH kit, and Box 13-11 lists the common contents of an MH kit.

Recrudescence or return of the signs and symptoms of MH can occur in the ICU or PACU hours after resolution of the initial event.[11] For that reason, after an episode of MH, the patient is observed in the ICU for 24 hours after the temperature has returned to normal. Dantrolene sodium is administered 1 mg/kg every 6 hours as needed. During this time, monitoring is maintained including temperature. Upon discharge, patients are referred to the Malignant Hyperthermia Association of the United States (MHAUS) for support and continued education about this disorder.[11,12]

▲ Moderate IV Sedation Administered by an Registered Nurse Versus Monitored Anesthesia Care

Moderate sedation using midazolam (Versed) and small doses of opioids IV provides a drug-induced depression of consciousness with the patient responding to verbal commands, either alone or associated with light tactile stimulation. No interventions are required to maintain airway patency or spontaneous ventilation. In addition, cardiovascular function is maintained. A patient who has been given moderate sedation has the ability to maintain a patent airway, retain protective airway reflexes, and respond to verbal commands. If these three conditions are not met, the patient is not receiving moderate sedation. The advantage of moderate

Table 13-3 Comparison of Moderate Sedation and Monitored Anesthesia Care

Characteristics	Moderate Sedation	Monitored Anesthesia Care (MAC)
Responsiveness	Purposeful response to repeated or painful verbal or tactile stimulation	May have purposeful response after stimulation
Airway	No intervention required	Intervention may be required
Spontaneous ventilation	Adequate	May be inadequate
Cardiovascular function	Usually maintained	Usually maintained

sedation is that it allows the patient to respond to verbal commands and physical stimulation. Moderate sedation is used for ambulatory surgical, therapeutic, and diagnostic procedures. The regimen usually consists of a narcotic, a sedative, and a local anesthetic.[13]

The goal of moderate sedation is to decrease patient anxiety associated with the surgical procedure or intervention using the least amount of drug necessary. Moderate sedation enhances patient cooperation, maintains stable vital signs, elevates the pain threshold, provides amnesia, and allows for rapid postoperative recovery.[13]

Propofol is used for sedation for surgical procedures and interventional procedures, such as endoscopies because of its short onset time and fast recovery time. A disadvantage of propofol is the risk for rapid loss of consciousness and apnea resulting in cardiopulmonary compromise. In a clinical study by Schilling et al,[14] propofol was found to cause a greater incidence of decline in SaO_2 and blood pressure compared to midazolam in patients requiring moderate sedation. Because propofol is an IV general anesthetic, those administering propofol must be trained in the administration of general anesthesia and airway management.[14] Several entities regulate standards for the administration of moderate sedation including state boards of nursing, the Joint Commission, and health care facilities. The following skills are required of the registered nurse (RN) who is administering the sedation and managing the care of patients receiving moderate sedation:

- Demonstrate knowledge of anatomy, physiology, pharmacology, cardiac dysrhythmia recognition, and complications related to moderate sedation and sedative drugs.
- Assess patient care requirements during moderate sedation and recovery. Physiological measurements include respiratory rate and effort, oxyhemoglobin saturation, blood pressure, heart rate and rhythm, and patient's level of consciousness.
- Understand the principles of oxygen delivery and respiratory and cardiovascular physiology and demonstrate the ability to use oxygen delivery and monitoring devices.
- Anticipate and recognize complications of moderate sedation related to the sedative drugs administered.
- Assess, diagnose, and intervene in undesired outcomes of moderate sedation in compliance with orders (including standard orders) or institutional protocols.
- Demonstrate skills in airway management.
- Demonstrate knowledge of the legal responsibilities of administering IV moderate sedation and monitoring patients receiving IV moderate sedation, including the RN's liability for any untoward reactions or life-threatening complications.

Monitored anesthesia care (MAC) describes a service in which an anesthesia provider has been requested to provide anesthesia care for a patient undergoing a therapeutic or diagnostic procedure usually requiring sedation. The difference between moderate sedation and MAC is that the anesthesia provider is present to recognize and treat the patient who becomes anesthetized following IV sedation and who develops apnea or an obstructed airway. Sedative drugs including propofol and fentanyl are given by the anesthesia provider and local anesthetics are injected into the surgical site by the surgeon. Postoperative care of the patient who has received moderate sedation or MAC is similar, although the patient who has received MAC may require more intervention in the postanesthesia phase. Table 13-3 provides a comparison of moderate sedation and MAC.

▲ Clinical Applicability Challenges

CASE STUDY

Mrs. K. is a 69-year-old woman who was taken to the emergency room after falling at home.

A hip arthroplasty had to be performed and she is admitted to the PACU from the OR. She has a history of congestive heart failure, chronic ethanol abuse, asthma, and hypertension. Induction of anesthesia was uneventful with the exception of a labile blood pressure intraoperatively. The anesthesia provider's report yields the following information:

- Patient 5 ft, 5 inches tall, weight 66 kg
- Hemoglobin 46 g/100 mL; hematocrit 15%
- Medications include hydrochlorothiazide, captopril, albuterol

(*continued on page 168*)

CASE STUDY *(Continued)*

- No known drug allergies
- OR time 1 hour, 35 minutes
- General endotracheal anesthesia
- Heart rate 90s, systolic blood pressure 120 to 80 mm Hg, diastolic blood pressure 55 to 75 mm Hg intraoperatively
- Estimated blood loss of 650 mL
- Total crystalloid administered: 1,450 mL of lactated Ringer's solution
- Urine output of 0.25 mL/kg/h
- Midazolam 2 mg was given IV as a premedicant/anxiolytic.

The patient received a total of 350 mcg of fentanyl. Neostigmine and glycopyrrolate were given to antagonize rocuronium. Further, the patient received cefazolin 1 g before incision and had bilateral lower extremity sequential compression stockings on at all times. The patient's initial PACU blood pressure was 90/57 mm Hg with a heart rate of 107 bpm. After the patient was medicated for pain with morphine sulfate 4 mg IV, her blood pressure decreased to 74/43 mm Hg, and her heart rate increased to 115 bpm. Following the drop in blood pressure, the patient complains of intense nausea.

1. What is the most likely cause of the hypotension?
2. What are other factors that may be contributing to the hypotension?
3. What is the initial intervention to treat hypotension in this patient?
4. What would be the pharmacologic agent of choice for treating the hypotension?
5. What is the likely cause of the patient's nausea?

References

1. American Society of PeriAnesthesia Nurses: Standards of PeriAnesthesia Nursing Practice. Thorofare, NJ: American Society of PeriAnesthesia Nurses, 2006
2. McCarthy EJ: Ventilation perfusion relationships. AANA J 55(5), 1987
3. Nagelhout J, Zaglaniczny K: Nurse Anesthesia, 4th ed. Philadelphia, PA: WB Saunders, 2010
4. MacRae MG: Closed claims studies in anesthesia: a literature review and implications for practice. AANA J 75(4):267–275, 2007
5. Reich DL, Hossain S, Krol M, et al: Predictors of hypotension after induction of general anesthesia. Anesth Analg 101(3):622–628, 2005
6. Burger L, Fitzpatrick J: Prevention of inadvertent perioperative hypothermia. Br J Nurs 18(18):1114, 1116–1119, 2009
7. Philip BK, Cheng Y-T, Gan TJ, et al: Postoperative nausea/vomiting after high risk ambulatory surgeries. Anesthesiology 107:A40, 2007
8. Hudcova J, McNicol E, Quah C, et al: Patient controlled opioid analgesia versus conventional opioid analgesia for postoperative pain. Cochrane Database Syst Rev 4:CD003348, 2006
9. Viscusi ER: Patient-controlled drug delivery for acute postoperative pain management: A review of current and emerging technologies. Reg Anesth Pain Med 33:146–158, 2008
10. Malignant Hyperthermia Association of the United States: ABCs of managing malignant hyperthermia. 2006 Malignant Hyperthermia Association of the United States. Retrieved August 4, 2007, from http://medical.mhaus.org
11. Burkman JM, Posner KL, Domino KB: Analysis of the clinical variables associated with recrudescence after malignant hyperthermia reactions. Anesthesiology 106(5):901–906, 2007
12. Litman R, Rosenberg H: Malignant hyperthermia: Update on susceptibility testing. JAMA 293(23):2918–2924, 2005
13. Kost M: Moderate Sedation/Analgesia: Core Competencies for Practice, 2nd ed. Philadelphia, PA: WB Saunders, 2004
14. Schilling D, Rosenbaum A, Schweizer S, et al: Endoscopy 41(4): 295–298, 2009

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

PART 4

SPECIAL SITUATIONS IN CRITICAL CARE

14

Rapid Response Teams and Transport of the Critically Ill Patient

Dennis W. Jones and Christine N. Lynch

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Explain the indications for a rapid response team (RRT).
2. Discuss the role of the RRT and considerations before establishing an RRT system.
3. Describe the indications for interfacility transport of the critically ill patient.
4. Compare and contrast the advantages and disadvantages of air versus ground transport.
5. Discuss the specific considerations and implications for care for air transport.
6. Explain the Emergency Medical Transfer Active Labor Act requirements for an appropriate interhospital transfer.
7. Describe the indications and key factors necessary for an effective interfacility transfer plan.
8. Analyze the role of the registered nurse in the five phases of the interfacility transport of the critically ill.

The patients being cared for in hospitals today are older, sicker, and have more comorbidities. A large percentage of these patients experience an adverse event during their hospitalization. Many of these events are preceded by warning signs in the form of hemodynamic instability.[1] A system to reach these patients at the first sign of instability could prevent deterioration before cardiac arrest and improve patient outcomes. The need for early detection and rapid treatment of unstable patients led to the development of the rapid response team (RRT).

▲ Rapid Response Teams

The first RRTs, called medical emergency teams (METs), were introduced at Liverpool Hospital in Australia in 1990. The METs were established to enable early identification and aggressive management of the seriously ill patient before cardiac arrest.[2] Since 1990, RRTs have been growing in popularity not only in Australia but also around the world.

Rationale for the Need

The National Registry of Cardiopulmonary Resuscitation (NRCPR), the largest registry of its kind, is an American Heart Association–sponsored, prospective, multisite, observational study of in-hospital resuscitation.[3] The NRCPR showed that despite advances in resuscitation practices and outcomes in the prehospital settings, little progress has been made with regard to improving in-hospital survival following cardiac arrest. This study found in-hospital survival following cardiac arrest to be a dismal 17%, essentially unchanged for the past 40 years.[3]

Most hospitals treat hundreds of patients each day, and there is a great deal of variability in the care provided. In addition, the level of monitoring may vary because those providing care have varying levels of skill. The performance of health care providers and of the system in which they work has a direct impact on patient care and outcomes. The variability in the care provided not only has an impact on both quality and safety, but also contributes to the differences seen in hospital mortality rates. In a review of the literature, the Institute of Healthcare Improvement (IHI), as part of its 5 Million Lives campaign (2006), found that three main systemic issues contribute to the differing hospital mortality rates. These include:

- Failure in planning (including assessments, treatments, and goals)
- Failure to communicate (eg, patient-to-staff, staff-to-staff, staff-to-physician)
- Failure to recognize the deteriorating patient condition

These fundamental problems often lead to a failure to rescue.[4]

The challenge for hospitals is to create a 24-hour system to recognize the seriously ill early and respond rapidly with personnel skilled in advanced resuscitation. It is estimated that 15% to 20% of all hospitalized patients develop serious adverse events, including cardiac arrest.[1] These adverse events are rarely sudden or unforeseen. In fact, they are usually preceded by at least one sign or symptom of physiological deterioration that occurs in the hours before the critical change in status (Box 14-1).[1]

The Role of the Rapid Response Team

The RRT brings critical care expertise to the bedside. In an effort to promote the implementation of RRTs in all hospitals in the United States, the IHI has written a *Rapid Response Teams: How-To Guide* (http://www.ihi.org). The members of the RRT perform the following functions:

- Assess the patient
- Stabilize the patient using RRT protocols (Fig. 14-1)
- Collect data (eg, vital signs, radiographic and laboratory data)
- Communicate with the health care team
- Provide education and support to the nurse initiating the call
- Assist with triage decisions
- Assist with transfer to a higher level of care, if needed[5]

The early intervention of RRTs has been shown to decrease the incidence of cardiac arrest and improve mortality rates.[5]

BOX 14-1 Rapid Response Team Calling Criteria

- Threatened airway
- Respiratory rate less than 8 breaths/min or more than 28 breaths/min
- SpO_2 less than 90%
- Heart rate less than 40 beats/min or more than 130 beats/min
- Systolic blood pressure less than 90 mm Hg
- Urine output less than 50 mL in 4 hours
- Acute mental status change
- Any patient about whom you are worried or concerned

The RRT system is based on three major components: early recognition of the deteriorating patient, rapid response with swift provision of care by clinical staff trained in advanced resuscitation, and a means by which to measure the system. When establishing an RRT system, several factors must be considered (Box 14-2, p. 172; Box 14-3, p. 173).[6]

▲ Interfacility Transport

Critically ill patients often must be transported between health care facilities. To ensure safe and expeditious transport, it is important to consider both the method of transport and the people involved in the transport process.

Typically, transport is indicated when the patient's need for complex diagnostic procedures or sophisticated medical and nursing expertise exceeds what can be provided at a facility. Family requests may also initiate a patient transport. For example, a family may want their family member transferred to a hospital closer to home.

Outcomes of evolving health care reform also have increased the demands for interfacility transport of critically ill patients. Third-party payers may require patients to be transported to a facility that is a member of their network. In addition, many hospitals vie for fewer patients and have developed their own transport teams to provide a flow of patients to their particular facility.

Whatever the reason for transporting a patient, a risk–benefit analysis of the transport should always be performed. Risks for the patient range from physical safety to physiological compromise to emotional distress.[7] When the benefits for the patient exceed the risks, an interfacility transport is warranted. Figure 14-2 on page 174 provides an algorithm for interfacility transfer.

The American College of Emergency Physicians (ACEP) has outlined physician responsibilities at this point. These responsibilities are as follows:

- The sending physician performs the patient assessment and determines the appropriate level of care during transfer.
- The receiving physician ensures that his or her facility is capable of providing necessary patient services to care for the patient.

The medical director of the critical care transportation agency provides medical direction during transport as well as all medical oversight of the transportation operation, which includes, but is not limited to, determining minimal team composition and equipment requirements, education, and practice.[8]

Modes of Interfacility Transport

Once the decision has been made to transport, the method of transport must be determined. The two primary methods of interfacility transport are ground and air.

Ground Transport

Ground transport includes ambulances and mobile intensive care units (ICUs). Air transport occurs by either a

Union Memorial Hospital

Patient Label

Rapid Response Team Protocols

DATE	TIME	Auto fill date and time ordered	Rapid Response Team Protocols (Check all that apply.)
			1. Initiate pulse ox and cardiac monitoring. 2. Start oxygen therapy with 2L/min nasal cannula. 3. Titrate oxygen to SpO_2 92% (88 to 92% if history of COPD). May use Venturi mask if necessary. 4. ☐ ABGs ☐ EKG ☐ Portable CXR ☐ Transcutaneous pacing
			5. ☐ Initiate Decreasing Level of Consciousness Protocol: ☐ Start IV. If hemodynamically unstable[a] or orthostatic[b] give 250 ml bolus NS. ☐ EKG ☐ CBC ☐ BMP ☐ ABGs ☐ Blood Glucose
			6. ☐ Initiate Respiratory Distress Protocol: ☐ Start IV ☐ ABGs ☐ CBC ☐ Portable CXR ☐ Albuterol neb 2.5 mg
			7. ☐ Initiate Chest Pain Protocol: ☐ STAT EKG ☐ Start IV ☐ Cardiac Enzymes
			8. ☐ Initiate Seizure Protocol: ☐ Start IV ☐ Blood Glucose ☐ Pad Bednails ☐ Anti-seizure drug level, if applicable. ☐ Page senior resident for possible anti-seizure medication order and for urgent evaluation.
			9. ☐ Initiate GI Bleed Protocol: ☐ Large bore IV ☐ STAT, CBC, BMP, PT/INR and PTT, Type and screen if hemodynamically unstable or orthostatic ☐ Start a second large bore IV. ☐ Page senior resident for urgent evaluation. ☐ EKG ☐ Type and crossmatch for 2 units PRBCs. ☐ Page attending physician regarding desire for GI consult and/or initiation of H_2 blockers or PPI.
			10. ☐ Initiate Hypotension Protocol: ☐ Start IV ☐ STAT CBC, BMP, PT/INR and PTT, ABGs, portable CXR. ☐ Give 500 ml bolus NS ☐ Assess surgical site. ☐ D/C any sedation and blood pressure medications.
			Definitions: [a]Hemodynamically unstable; SBP < 90 OR HR > 130. [b]Orthostatic: 20 mm Hg drop in SBP, 10 mm Hg drop in DPB, or a 20 bpm increase in HR upon upright positioning for 3 min.

RRT Leader Signature: _____

Date / Time: _____ Beeper: _____

FIGURE 14-1 ▲ Example of rapid response team (RRT) protocols used with a nurse-led team. (Courtesy of Union Memorial Hospital, Baltimore, MD.)

rotary-wing vehicle (helicopter) or a fixed-wing vehicle (airplane). When selecting the mode of transport, the following factors must be considered:

- Distance
- The safety of the transport environment
- Patient "out of hospital" time
- The patient's condition and the potential for complications
- The patient's need for critical or time-sensitive intervention (eg, rescue angioplasty)
- Traffic conditions
- Weather conditions[9]

In addition, the advantages and disadvantages of ground versus air transport must be considered when selecting the mode of transport. Table 14-1 on page 175 summarizes the advantages and disadvantages of ground versus air transport.

Air Transport

Table 14-2 on page 175 summarizes the special considerations for air transport. It is important for the nurse who may be caring for a patient to have a basic understanding of these considerations to aid in patient preparation prior to flight team arrival and promote a smooth transfer of care upon the team's arrival.

The environment in which the patient is transported differs greatly from the in-hospital setting. Because the patient is transported at a higher altitude, where the barometric pressure is reduced, the possibility of hypoxia increases. However, the flight team will assess the patient upon arrival and determine how much oxygen the patient will need during the transport based on the patient's clinical condition and the flight plan.

Any air-filled cavity in the patient's body, such as the stomach, lungs, or containers (air splint, glass intravenous bottle),

BOX 14-2 Considerations When Implementing a Rapid Response Team System

Gaining leadership support. The support of senior leadership is essential for the success of the proposed RRT system. Advantages of an RRT system include:

- Marketing advantage in a competitive health care environment
- Greater medicolegal protection and decreased liability
- Decreased patient and family complaints
- Improved identification of patients in need of palliative care
- Avoidance of unnecessary intensive care unit (ICU) admissions
- Decreased job-related stress for nurses and residents
- Decreased number of in-hospital arrests

Determining team structure. The structure of the RRT varies according to facility size, level of patient acuity, availability of resources, and the frequency of adverse events and cardiac arrest. Examples of different models include:

- ICU registered nurse (RN) and respiratory therapist (RT)
- ICU RN, RT, and nurse practitioner, clinical nurse specialist, or physician assistant
- ICU RN, RT, and intensivist or hospitalist

Establishing communication tools and protocols. Communication tools provide the RRT leader with a template for gathering pertinent information, facilitating communication with the physician, and facilitating triage decision making. If the RRT is led by a nurse, the use of protocols is essential for the quick delivery of indicated therapies and tests (see Fig. 14-1).

Training for responders. Members of the RRT must receive the proper training. Areas to be reviewed include:

- The benefits of early rescue
- Teamwork with non–critical care staff
- Protocols available to guide RRT therapy
- Triage skills and advanced cardiac life-support certification
- The importance of the linkage to palliative care
- What is expected of RRT members when responding to a call
- The use of communication tools, such as SBAR (see Box 14-3)
- The chain of command for nurse-led teams

Training for staff. Staff members must be made aware that the RRT exists, educated about the role of the RRT, and taught how to activate the RRT system. Methods of raising staff awareness include the following:

- Formal teaching and in-service training
- Newsletters
- Posters with the RRT calling criteria
- Pocket cards and badge holder with calling criteria
- Brochures with RRT concepts and calling criteria
- Inclusion of RRT education in employee orientation sessions

Calling criteria and the mechanism for activating the RRT system. Many studies have been done to establish those physiological signs that are often displayed before an adverse event or cardiac arrest.[5] When considering the RRT calling criteria that will be used in your institution, these evidence-based data should be considered. (See Box 14-1 for examples of calling criteria.) The mechanism for activating the RRT system should be clear, quick, and easy so that the staff will use it and the team will respond rapidly.

Feedback mechanisms. Feedback promotes continued improvement of the RRT system and can be used to drive educational programs. Feedback can be obtained by:

- Tracking patient outcomes
- Conducting satisfaction surveys with the staff

Evaluation of effectiveness. A means by which to measure the success of the RRT system is imperative. Three key measures that are used include

- Codes per 1,000 discharges
- Codes outside the ICU
- Utilization of the RRT system

Adapted from Institute for Healthcare Improvement: 5 Million Lives Campaign. Getting Started Kit. Rapid Response Teams: How-To Guide, 2006. Available at: http://www.ihi.org/IHI/Programs/Campaign/.

can be affected physiologically by changes in barometric pressure. The degrees to which any deleterious effects may occur to the patient during transport depend on the type of aircraft and altitude flown. The flight team screens the patient carefully and takes preventive measures to ensure a safe and uneventful transport.

Other environmental factors affecting the patient during the transport include changes in temperature and humidity as well as the presence of noise and vibration. The mode of transport will dictate the degree to which each of these factors occurs—that is, whether the vehicle is a fixed-wing or rotary-wing aircraft—and the type of aircraft. The flight crew takes the necessary steps to either prevent or decrease the effects of each of these factors on the patient.

If the critically ill patient is conscious and aware of the need for air transport, the sending nurse screens the patient for fear or anxiety related to flying and a history of motion sickness while in a moving vehicle. Medical consultation is indicated when any of these factors exist since treatment with an anxiolytic or antiemetic medication could aid in preventing clinical problems during the flight. Again, the flight crew screens the patient for these factors during the preflight assessment.

Transfer Guidelines and Legal Implications

To facilitate the appropriate transfer of patients, the ACEP has developed guidelines. These principles of appropriate patient transfer are listed in Box 14-4 on page 176.

Legislation exists that provides guidelines, regulations, and penalties for patient transfer. One such law, the Consolidated Omnibus Reconciliation Act (COBRA) of 1985, contains provisions addressing the transfer of patients from hospital to hospital. The purpose of the legislation is to prevent inappropriate transfers of patients who seek emergency department care. As a result, this legislation has become known as the "antidumping" law. The following provisions of the COBRA legislation prevent any patient from being denied an initial screening in an emergency department, transferred to another hospital, or discharged without receiving care:

1. Hospitals must provide screening examinations for every person who comes to the emergency department and requests care.
2. If the patient has an emergency medical condition, the hospital must provide stabilizing treatment or transfer the patient to another medical facility. The physician must document that the medical benefits outweigh the risks of the transfer.

BOX 14-3 SBAR Communication Tool

SBAR is an acronym for Situation, Background, Assessment, and Recommendation. Communication tools such as the SBAR provide the RRT leader with a template for gathering pertinent information, facilitating communication with the physician, and facilitating triage decision making.

Situation:	State your name and unit. State the name of the patient you are calling about. State the problem for which the team was consulted.
Background:	State the admission diagnosis and the date of admission. State the pertinent medical history. Provide a brief synopsis of the patient's hospital course. State the code status of the patient.
Assessment:	Most recent vital signs BP: _____; pulse: _____; respirations: _____; temp: _____; Any change from prior assessments: Mental status: Quality of respirations: Pulse rate/rhythm change: Pain: Skin color: Neurologic changes: Nausea and vomiting, output:
Recommendation:	State your triage recommendations. For example: Transfer to coronary care unit Have the physician come see the patient at this time Arrange for specialist to see the patient now Arrange for tests (eg, chest x-ray, arterial blood gas, ECG, complete blood cell count)

Adapted from Duncan KD: Nurse-led medical emergency teams: A recipe for success in community hospitals. In DeVita MA, Hillman K, Bellomo R (eds): Medical Emergency Teams: Implementation and Outcome Measurement. New York, NY: Springer, 2006, pp 122–133.

3. The receiving medical facility agrees to accept the patient and provide appropriate medical treatment. The receiving medical facility must have adequate space and qualified personnel to care for the patient.
4. The transfer is conducted by qualified personnel, and appropriate equipment needed to provide care during the transfer is available.[10]

Situations in which a patient is not stabilized, yet conditions are appropriate for transfer occur when:

1. The risks of remaining at the initial facility are outweighed by the benefits of transfer.
2. The patient or family requests the transfer.
3. A physician is not present at the initial facility but a qualified medical person certifies that the benefits outweigh the risks.
4. The transfer occurs with appropriate equipment and qualified personnel.[10]

Figure 14-3 on page 177 presents the requirements for evaluating a patient's suitability for transfer, as outlined by the Emergency Medical Transfer Active Labor Act (EMTALA). In addition, the Air and Surface Transport Nurses Association (ASTNA, formerly known as the National Flight Nurses Association, or NFNA) developed nursing standards for transport of the critically ill patient by rotary-wing transport.[11]

Phases of Interfacility Transport

Five phases of transport have been identified: (1) notification and acceptance by the receiving facility, (2) preparation of the patient by the transport team, (3) the actual transport, (4) turnover of the patient to the receiving hospital, and (5) continuous quality improvement monitoring after transport. Each phase will be described in detail as it applies to the transport.

Phase One: Notification and Acceptance by the Receiving Facility

The first phase of transport requires contacting the receiving facility and confirming that it is willing to accept the patient and provide an accepting physician and an appropriate patient care unit to receive the patient. Additionally, the mode of transport is determined at this time. Communication is a key element in this phase of the process. All personnel (sending, transporting, and receiving) must have the necessary information to make the appropriate transport decision. Standards of care and/or protocols are necessary for the transport process to be carried out in an organized way. A transfer checklist is helpful to ensure that no steps in the transfer process are missed. In addition, an awareness of the policies and procedures of the transporting agencies used in an area is needed for a smooth transport process.[7]

The identification of a responsible physician is essential so that a medical provider is available for consultation while en route and on arrival. The ACEP has described medical direction for interfacility transfers as a shared responsibility. The transferring physician ensures that the transport team is composed of professionals appropriate to the needs of the patient and that an appropriate vehicle and equipment are used for transport.[8] If the local emergency medical system is not providing medical direction en route, then the responsible physician must be identified as being part of the hospital-based or private ambulance program.

The Commission of Air Medical Transport Services, which is the certifying body for the air medical industry, maintains the staffing standard on critical care transports; either a registered nurse (RN) or a licensed physician must be present as the primary care provider.[12] Transport teams should have standard orders or protocols if they are unable to contact a physician during transport. The transport flight team has practice protocols that guide their practice; however, they may also receive medical direction from the medical director of the transport program.

Once the specific transporting agency is determined, an overview of the patient's condition and specific clinical needs is communicated. Necessary information includes the patient's

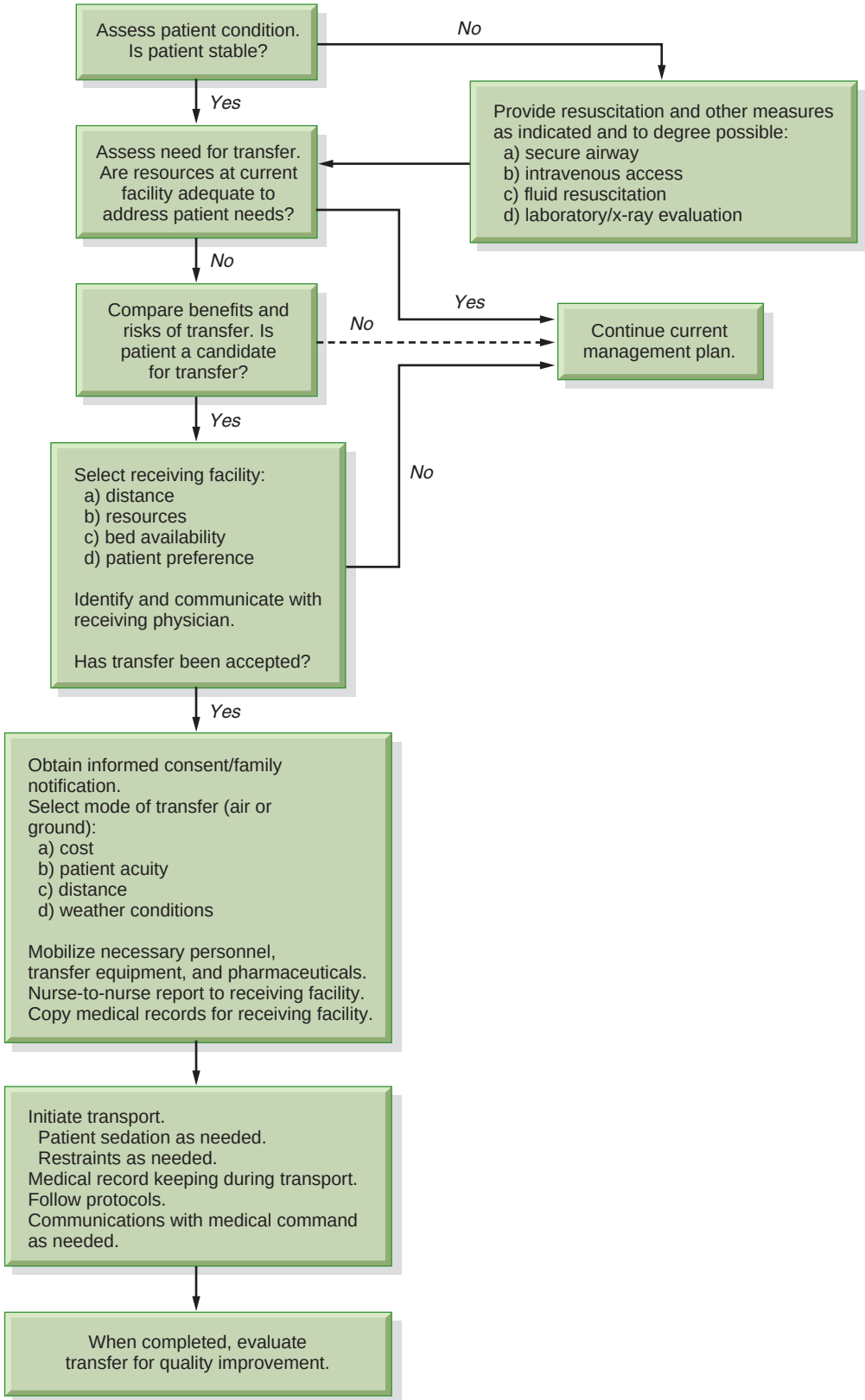


FIGURE 14-2 ▲ Interfacility transfer algorithm. (From Warren J, Fromm RE, Orr RA, et al: Guidelines for the inter- and intrahospital transport of critically ill patients. Crit Care Med 32(1):256–262, 2004.)

Table 14-1 Advantages and Disadvantages of Ground Versus Air Transport

Mode of Transport	Advantages	Disadvantages
Ground	Adequate work space for personnel and equipment Sensitive monitoring equipment may work better No weight restrictions Adequate lighting Able to travel in most types of weather	Longer transport time Unfavorable road conditions may make transport uncomfortable for patient Interventions difficult to perform in a moving vehicle Ambulance unavailable for other calls in the community
Air	May shorten "out of hospital" time Crew generally composed of advanced-level care providers Improved communication capability Ground emergency medical services remain available in the community	Weather conditions restrict availability of the vehicle Potentially more costly Limited space (helicopters) Weight limitations Physiological impact on patient and crew Psychological impact on patient (eg, fear of flying)

From Holleran R: Prehospital Nursing: A Collaborative Approach. St. Louis, MO: CV Mosby, 1994.

name, age, diagnosis, reason for transfer, vital signs, intravenous and special monitoring devices, continuous infusion medications, airway and oxygenation or ventilation status, and special equipment needs (eg, intra-aortic balloon pump). This information assists in determining the composition of the transport team as well as the equipment and medications needed. The American Association of Critical-Care Nurses (AACN), the American College of Critical Care Medicine, and the Society of Critical Care Medicine (SCCM) offer guidelines for accompanying personnel for interfacility transfers[7] (Box 14-5, p. 178).

Interhospital critical care transport is a highly specialized nursing practice area which requires specific knowledge and

Table 14-2 Special Considerations for Air Transport

Stressors	Effect	Nursing Interventions
Altitude change	Hypoxia is due to the following: Decrease in the partial pressure of oxygen Decrease in the diffusion gradient for oxygen molecules to cross the alveolar membrane Decrease in oxygen availability	Provide supplemental oxygen. Use pulse oximeter and end-tidal CO_2 monitor.
Barometric pressure (atmospheric pressure) change	With increasing altitude, the barometric pressure decreases and gases expand. Expansion of gases affects eardrums, sinuses, gastrointestinal tract, pleural spaces, and hollow organs. Expansion of gases affects air splints, pressure bags or cuffs, balloon cuffs on endotracheal tubes, intravenous fluid bags and bottles, pneumatic antishock garments.	Insert a nasogastric tube to decompress the stomach. If possible, fill cuffs with water or saline solution rather than air. Monitor equipment and decompress with higher altitudes. Vent glass bottles and wrap to protect against breakage. Apply pressure cuffs to intravenous solution bags.
Thermal change	As altitude increases, temperature decreases. Oxygen demand increases as the body tries to maintain warmth.	Use blankets to keep the patient warm.
Humidity change	As air is cooled, it loses moisture. Mucous membranes dry.	Humidify supplemental oxygen. Provide adequate fluid intake.
Gravitational change	Gravitational change affects acceleration and deceleration forces. Transient increase in venous return occurs for patients positioned with head at the back of the aircraft. Potential exists for motion sickness.	Use a head-forward position for patients with fluid overload or increased intracranial pressure. To minimize motion sickness, provide oxygen, cool cloth to face, cool air to face. Administer medications, such as transdermal scopolamine patches and promethazine.
Noise	It is difficult to monitor blood pressure, breath sounds, endotracheal tube air leak.	Explain sounds to patient. Monitor blood pressure by Doppler device. Provide continuous airway assessment. Wear head sets or ear plugs.
Vibration	Vibration may distort readings on equipment. Equipment may loosen or move.	Secure all equipment. Check equipment function frequently.

From Harrahill M: Interfacility transfer. In Kitt S, Selfridge-Thomas J, Proehl J, et al (eds): Emergency Nursing: A Physiologic and Clinical Perspective, 2nd ed. Philadelphia, PA: WB Saunders, 1995, pp 12–18.

BOX 14-4 Principles of Appropriate Patient Transfer

- The health and well-being of the patient must be the overriding concern when any patient transfer is considered.
- Emergency physicians and hospital personnel should comply with state and federal regulations regarding patient transfer. A medical-screening examination should be performed by a physician or by properly trained ancillary personnel according to written policies and procedures.
- The patient should be transferred to another facility only after medical evaluation and, when possible, stabilization.
- The physician should inform the patient or responsible party of the reasons for and the risks and likely benefits of transfer, and document this in the medical record.
- The hospital and medical staff should identify individuals responsible for transfer decisions and clearly delineate their duties regarding the patient transfer process.
- The patient should be transferred to a facility appropriate to the medical needs of the patient, with adequate space and personnel available.
- A physician or other responsible person at the receiving hospital must agree to accept the patient before transfer.
- The patient transfer should not be refused by the receiving hospital when the transfer is medically indicated and the receiving hospital has the capability and/or responsibility to provide care for the patient.
- Communication to exchange clinical information between responsible persons at the transferring and receiving hospitals must occur before transfer.
- An appropriate medical summary and other pertinent records should accompany the patient to the receiving institution.
- The patient should be transferred in a vehicle that is staffed by qualified personnel and contains appropriate equipment.
- When transfer of patients is part of a regional plan to provide optimal care of patients at specified medical facilities, written transfer protocols and interfacility agreements should be in place.

Adapted from American College of Emergency Physicians: Principles of appropriate patient transfer. Ann Emerg Med 19(3):337–338, 1990.

skills. Nurses who have not received training or are inexperienced in this practice area must be aware of possible regulations governing their practice, should they be requested to accompany a patient during interhospital transport. Practice standards or regulations promulgated by a state board of nursing may exist, which the RN must be aware of prior to agreeing to transport the patient.

The AACN and the SCCM have developed a transfer curriculum and competencies for accompanying staff, which may assist in identification of issues related to the role of the nurse in the field.[7] Current regulations require that the patient or a legally authorized representative give informed consent for the transport. If consent cannot be obtained, documentation of the indications for the transport and the reason that consent was not obtained must appear in the medical record.[7]

Phase Two: Preparation of the Patient by the Transport Team

Phase two begins upon arrival of the transport team. A thorough patient report is provided to the arriving team and should include chief complaint, allergies, medical history, reason for transport, patient's age, vital signs, treatments already provided, and their outcomes. Copies of the chart and all radiographs are sent with the patient. To avoid duplication of efforts, the sending and transporting nurses decide who will provide a patient report to the receiving hospital. If the transport is arranged by the sending nurse, the transport nurse updates the receiving nurse as needed.

The transport team performs an assessment of the patient and compares its findings to the previous assessment and plan of care. If interventions are indicated prior to transport, the transport team and the referral facility personnel determine who assumes responsibility for the interventions. It is important that all stabilization procedures, for example, endotracheal intubation, are completed before departure from the referring facility to ensure the procedures are completed successfully. Completion of these procedures in the less-controlled transport environment increases the risk for error related to unpredictable lighting, movement, and vibration. Although resuscitation and stabilization are initiated at the referral hospital, full stabilization may not be achieved until the patient arrives at the receiving hospital.[10]

The psychosocial preparation of the patient and family for transport is an important step before transport begins. The sending nurse ensures that the patient and family understand all facets of the transport process including the reason, transport mode, time of transport, and transport destination. Information about the family is also communicated at this time and includes identification of a family spokesperson and the family's plan for getting to the receiving hospital. If the transport team is unable to meet with the family, the sending nurse supplies information about how to contact the family. The transport team, particularly the flight team, in order to allay any further anxiety related to flying, explains all procedures, safety precautions, and the need for preflight medications (eg, antiemetics) to the patient and family before departure.

Physical preparation of the patient is the next important step to ensure a safe transport. The ABCs of care (airway, breathing, and circulation) are the top priority. Adequate oxygenation and ventilation are ensured before transport begins. As noted earlier, procedures deemed necessary, such as intubation are performed prior to departure. Most intubated patients are sedated to prevent them from dislodging the endotracheal tube and to decrease fear and discomfort during transport. Further, a nasogastric tube may be inserted to prevent aspiration of stomach contents into the airway. Because auscultation of breath sounds is difficult en route, end-tidal carbon dioxide levels and oxygen saturation are used to monitor respiratory status. Supplemental oxygen is almost universally administered during transport to maintain adequate oxygenation.

The patient's circulatory and hemodynamic statuses are stabilized before transport. Any bleeding is controlled, and adequate intravenous access is established and well secured. For a patient with an unstable volume status, several large-bore intravenous lines are indicated. If the patient is already on intravenous drips, the transport team transfers the drips over to its own equipment and will remix drips as needed. The patient's circulatory status is continuously assessed through blood pressure and cardiac monitoring during the entire transport process. Cardiac arrest medications and a defibrillator should be easily accessible.

Patients with actual or potential spinal injuries should have immobilization devices in place prior to transport. The transport team may request that the staff of the sending

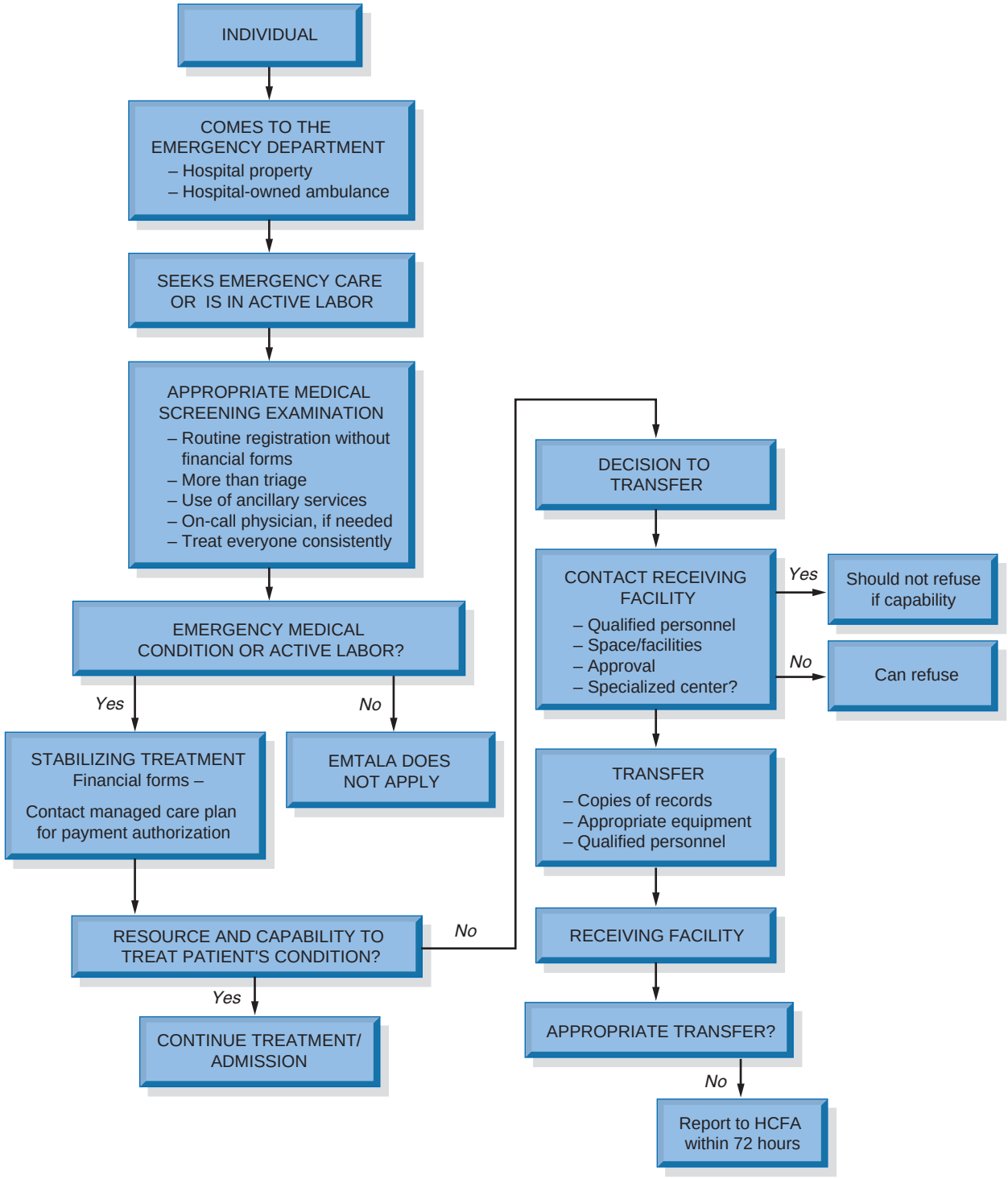


FIGURE 14-3 ▲ Emergency Medical Transfer Active Labor Act (EMTALA) flow chart. HCFA (formerly Health Care Finance Administration) is now Center for Medicare and Medicaid Services. (Reprinted from Lee NG: Legal Concepts and Issues in Emergency Care. Philadelphia, PA: WB Saunders, 2001, p 140, with permission.)

facility facilitate this before the arrival of the transport team. Patients with skeletal long-bone fractures must have the fractured limb immobilized before departure to prevent pain and further complications.

Pain control during transport is also addressed through transport orders or medical protocols. The best agents for pain management are those with a rapid onset, short duration, and ease of administration and storage.

Phase Three: The Transport Process

Phase three is the actual transport of the patient. The time spent in careful planning of the transport and stabilization of

BOX 14-5 Guidelines for Accompanying Personnel for Interfacility Transfer

- A minimum of two people in addition to the vehicle operator should accompany the patient.
- At least one of the accompanying personnel should be a RN, physician, or advanced emergency medical technician.
- When a physician does not accompany the patient, there should be a mechanism available to communicate with the physician any changes in the patient status and obtain additional orders. If an accompanying physician is not possible, advanced authorization by standing orders to perform acute life-saving interventions must be established.

From Warren J. Fromm RE, Orr RA, et al: Guidelines for the inter- and intra-hospital transport of critically ill patients. Crit Care Med 32(1):256–262, 2004.

the patient eases the transport process. The transport vehicle must contain the essential equipment needed for transporting a critically ill patient. Box 14-6 lists the minimal necessary equipment.[9]

The ABCs of care continue to be the primary focus of the transport team. Box 14-7 lists the minimum and recommended standards for monitoring the critically ill patient during the transport. Each member of the transport team must have a clear understanding of their role in the continuous assessment, planning, and intervention that takes place when caring for the patient. Throughout the transport, the team also provides explanations and reassures the patient because transport can be very stressful. The transport team is responsible for documenting all facets of care provided during transport as well as the patient's response to the care.

Before arriving at the receiving facility, if able, the transport nurse calls either a full or updated report to the

BOX 14-6 Minimally Essential Equipment Necessary for Transport

- Airway and ventilatory management equipment:
  - Resuscitation bag and mask of proper size and fit for the patient
  - Oral airways, laryngoscopes, and endotracheal tubes of proper size for the patient
  - Oxygen source with a quantity sufficient to meet the patient's anticipated consumption with at least 1-hour reserve in addition

  Suction apparatus and catheters
- Cardiac monitor/defibrillator/transcutaneous pacemaker
- Blood pressure cuff and stethoscope
- Materials for intravenous therapy and devices for regulation of infusion
- Drugs
  - For advanced cardiac resuscitation
  - For the management of acute physiological derangements
  - For special needs of the patient
- Spinal immobilization devices
- Communication equipment

From Warren J. Fromm RE, Orr RA, et al: Guidelines for the inter- and intra-hospital transport of critically ill patients. Crit Care Med 32(1):256–262, 2004.

BOX 14-7 Standards for Monitoring Patient Transport

Minimal Standard for Monitoring Parameters During Interhospital Transport

- Blood pressure
- Continuous pulse oximetry
- Electrocardiographic monitoring
- Respiratory rate

Recommended Monitoring Parameters for Select Patients

- Intra-arterial blood pressure
- Central venous pressure
- Pulmonary artery pressure
- Intracranial pressure
- Capnography

From Warren J. Fromm RE, Orr RA, et al: Guidelines for the inter- and intra-hospital transport of critically ill patients. Crit Care Med 32(1):256–262, 2004.

RN on the receiving unit. This report includes an estimated time of arrival at the receiving facility. The nurse also communicates any special needs, changes in patient status, and unchanged but pertinent findings. In some situations, a flight nurse may not be able to provide a patient update; therefore, an updated bedside report is provided on arrival.

Phase Four: Turnover of the Patient to the Receiving Facility

Phase four of transport involves handing over the patient to the receiving unit staff at the receiving facility. Backup plans on how to handle an acutely deteriorating patient in transit between the transport vehicle and the ICU should also be identified. This plan may include stopping at the emergency department to stabilize the patient; it is essential for the emergency department staff to be aware of this possibility. Once the patient arrives safely in the receiving unit, the transport team and the receiving staff determine when the receiving staff will take over the responsibility for the patient's care. A final verbal update and all medical documents and patient belongings are given to the receiving staff. The written report of the transport is also completed.

Phase Five: Posttransport Continuous Quality Improvement Monitoring

The final phase of transport is very important and involves continuous quality improvement monitoring. Ideally, the referring facility, the transport team, and the receiving facility are involved in the review process. The first phase of the quality improvement monitoring involves evaluation of the current transport, including any quality indicators developed by the transporting agency. These indicators may include appropriateness of the transfer, accompanying personnel, timeliness of the transfer, patient outcome, management of complications, and transfer outcome. The second phase entails the ongoing review of the transport system. Such reviews focus on system functioning. Indicators may include complications, deaths in transport, and deaths after transport.[7]

The multidisciplinary continuous quality improvement monitoring team scrutinizes the collected data for patterns and trends, identifies solutions to patient care problems, initiates corrective action, and communicates such action to all involved in the transport process. Through a quality improvement plan, the transport process is improved and results in optimal care of the critically ill patient during the transport process.[7]

▲ Intrafacility Transport

The transport of a critically ill patient for diagnostic evaluation or treatment within the hospital is often necessary for optimal care. When this occurs, associated risks are involved.[13] It is imperative that the benefits of the transport outweigh the risks. Although a complete discussion regarding this topic is beyond the scope of this chapter, important factors should be considered when planning a transfer of a critically ill patient outside the ICU. One factor, for example, is the rate of adverse events that occur during intrahospital transport, which ranges from 5.9% to 70%; these events usually fall into two categories.[13] Category one involves monitoring equipment and its use (eg, equipment failure, disconnected leads, depleted oxygen). Category two involves physiological changes in the patient, which may include changes in blood pressure, hypoxia, dysrhythmias, or increased intracranial pressure.[14]

Because of the inherent risks during intrahospital transport, some hospitals are forming specially trained transport teams organized with two specific goals: to reduce risk and to improve patient safety.[13] Justification for these transport teams may be based on inadequate ICU staffing and an inability to effectively manage a clinically complex patient while out of the ICU setting.[13]

In one retrospective study it was demonstrated that having a specialized transport team reduces adverse clinical events during transport.[14]

To provide safe and effective intrahospital transport of a critically ill patient, the critical care nurse prepares for the transport and communicates[5] with the receiving location when necessary. Appropriate personnel and equipment must accompany the patient. The SCCM guidelines recommend that a competent critical care nurse and at least one other person accompany the patient. The additional person may be a respiratory therapist, RN, or critical care technician. The decision should be based on the patient's needs. The SCCM strongly recommends that a physician with training in airway management, advanced cardiac life support, and critical care or equivalent accompany any unstable patient.[9] Essential equipment that must be sent with the patient includes a blood pressure monitor, a pulse oximeter, a cardiac monitor–defibrillator, airway management equipment, an oxygen source, basic resuscitation drugs, and appropriate intravenous fluids and infusion pumps.

▲ Clinical Applicability Challenges

CASE STUDY

Mr. J., a 60-year-old African American male, was admitted to the coronary care unit (CCU) an hour ago from the emergency department with the diagnosis of anteroseptal myocardial infarction, congestive heart failure, and cardiogenic shock. His wife and family are present. The patient is intubated and sedated; placed on a ventilator; and given an infusion of dopamine, dobutamine, and amiodarone. Shortly after arrival in the CCU, the cardiologist decides to insert an intra-aortic balloon catheter and place the patient on an intra-aortic balloon pump.

The hospital where Mr. J. is being treated is a large community hospital, but it does not have the capability to perform interventional cardiac procedures. The cardiologist decides to transfer the patient to the closest tertiary care facility, which is 40 miles away. Transfer is indicated for cardiac catheterization; percutaneous transluminal coronary angioplasty with possible stent placement; and the need for cardiac surgery, which cannot be performed in the facility currently treating Mr. J.

The cardiologist at the community hospital has contacted the attending cardiology interventionalist, who has agreed to accept Mr. J. This physician has agreed that Mr. J. should be transferred via air medical service. The patient will be transferred directly to the cardiac catheterization laboratory and then to an awaiting bed in the CCU. The air medical service that will be transporting Mr. J. is staffed by a critical care flight RN and paramedic. They will bring all necessary equipment for the transport, including but not limited to a transport ventilator, intravenous infusion pump, and intra-aortic balloon pump.

The attending cardiologist for Mr. J. obtains consent for transport from Mr. J.'s wife. The consent form explains reasons for, as well as the risks and benefits associated with, the transfer.

1. What are the indications for transfer of Mr. J. to a tertiary facility?

2. Why is air medical transport indicated for Mr. J.?

3. What is the most appropriate nursing diagnosis for Mr. J.?

References

1. Jones D, Bellomo R, Goldsmith D: General principles of medical emergency teams. In DeVita MA, Hillman K, Bellomo R (eds): Medical Emergency Teams: Implementation and Outcome Measurement. New York, NY: Springer Press, 2006, pp 80–90
2. Kerridge RK, Saul WP: The medical emergency team, evidence-based medicine and ethics. Med J Aust 179:313–315, 2003
3. Peberdy MA, Kaye W, Ornato JP, et al: Cardiopulmonary resuscitation of adults in the hospital: A report of 14720 cardiac arrests from the National Registry of Cardiopulmonary Resuscitation. Resuscitation 58:297–308, 2003
4. Institute for Healthcare Improvement: 5 Million Lives Campaign. Getting Started Kit. Rapid Response Teams: How-To Guide, 2006. Available at: http://www.ihi.org/IHI/Programs/Campaign/
5. Buist M, Bernard S, Nguyen TV, et al: Association between clinically abnormal observations and subsequent in-hospital mortality: A prospective study. Resuscitation 62:137–141, 2004
6. Duncan KD: Nurse-led medical emergency teams: A recipe for success in community hospitals. In DeVita MA, Hillman K, Bellomo R (eds): Medical Emergency Teams: Implementation and Outcome Measurement. New York, NY: Springer, 2006, pp 122–133
7. American Association of Critical-Care Nurses Transfer Guidelines Task Force and the Guidelines Committee, American College of Critical Care Medicine, Society of Critical Care Medicine: Guidelines for the Transfer of Critically Ill Patients. Aliso Viejo, CA, American Association of Critical-Care Nurses, 1998
8. American College of Emergency Physicians: Position Paper: Interfacility Transportation of the Critical Care Patient and Its Medical Direction, 2005. Available at: http://acep.org
9. Warren J, Fromm RE, Orr RA, et al: Guidelines for the inter- and intrahospital transport of critically ill patients. Crit Care Med 32(1): 256–262, 2004
10. Glass DL, Rebstock J, Handberg E: Emergency Treatment and Labor Act (EMTALA) avoiding the pitfalls. J Perinat Neonat Nurs 18(2): 104–105, 2004
11. Arndt K (ed), for the Air and Surface Transport Nurses Association. Standards for Critical Care and Specialty Rotor-Wing Transport. Lexington, KY: Myers Printing, 2003
12. Commission on Accreditation of Medical Transport Systems: Accreditation Standards: Critical Care Staffing, 7th ed. Anderson, SC: Author, 2010
13. Fanara B, Manzon C, Barbot O, et al: Recommendations for the intrahospital transport of critically ill patients. Crit Care 14(3):R87, 2010
14. Kue R, Brown P, Ness C, et al: Adverse clinical events during intrahospital transport using a specialized transport team: A preliminary report. Am J Crit Care 20(2):153–131; quiz 162, 2011

15 Disaster Management: Implications for the Critical Care Nurse

Nancy Blake and Jeffrey S. Upperman

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Describe the nurse's role in mass casualty incidents.
2. Explain the nurse's role in triage.
3. Describe how a radiological attack can occur and how to treat patients who have experienced a radiological attack.
4. Discuss how a chemical attack can occur and how to treat patients who have experienced a chemical attack.
5. Describe how a biological attack can occur and how to treat patients who have experienced a biological attack.

Communities rely on hospitals particularly in times of disaster. When recent disasters and terrorist attacks in the United States exposed weaknesses in emergency planning in hospitals, lawmakers passed legislation for federal funding for hospital-based disaster preparedness planning and resources. In 2005, the catastrophic hurricane Katrina surprised and shocked many hospital administrators and health care workers, revealing potentially how vulnerable the nation's health care system is. The Oklahoma City bombing and the attacks on the World Trade Center and the Pentagon alerted the nation to the major terrorist threats that exist around the world and that could easily overwhelm hospitals if hundreds or thousands of survivors need emergency treatment. The devastating earthquake in Haiti and the earthquake and tsunami in Japan demonstrated that in the event of widespread destruction, there may be numerous patients with traumatic injuries needing hospitalization and care in intensive care units (ICU). A similar large-scale disaster in the United States, involving thousands of casualties would stretch resources beyond capacity.

▲ Fundamentals of Disaster Science

The critical care nurse's role is crucial in all phases of the disaster planning and response cycle. Critical care nurses should participate in their facility's disaster planning process. When the disaster strikes, the job of critical care nurses depends on the impact of the disaster on the facility's structures, its surrounding environment, and the available staffing. For instance, if the hospital's electricity is out and the generators do not work, important equipment in the ICU, such as ventilators and monitors, will not function. In other disaster circumstances, critical care nurses may need to evacuate patients from the ICU when structures are unstable and resources are scarce. At this critical time, these nurses will cap intravenous (IV) lines for use at a later time. After the initial crisis, resources may be scarce, and vital medications may be unavailable and thus need to be rationed; in this case, critical care nurses must work closely with physicians and pharmacists to determine alternative methods or medications. Under austere conditions, critical care nurses caring for the sickest patients face a daunting task. These nurses need to consider altered standards of care and work with the health care team when faced with futile care decisions. During the recovery phases, critical care nurses are a crucial resource in leading and participating in the transitional delivery of care in their facilities. A Pediatric and Emergency Mass Critical Care Task Force published guidelines for triage and treatment, supplies and equipment, neonatal and pediatric regionalized systems, education, community preparedness, legal considerations, family-centered care, ethical issues and the reality of pediatric and mass critical care in the developing world in September of 2011.[1]

In catastrophic disaster situations, such as Haiti after the earthquake and Japan after the earthquake and tsunami, in which entire communities are devastated, the level of care for the community depends on the equipment, supplies, and facilities on hand. For example, if all buildings have been destroyed, physicians and nurses must care for patients in alternative care sites, such as tents or shelters. In these situations, the goal becomes providing the highest care possible, with limited resources and equipment. Wall oxygen and suction may not be available. Portable oxygen tanks become a vital resource. Nurses may need to attach a syringe to a suction catheter to provide manual suction. Portable or disposable handheld short-term ventilators may be all that is available. In major disasters, one must not forget the psychological and social distress that health care workers are experiencing. In cases of severe disruption, psychosocial resources are needed to assist the critical care nurses because many critically ill patients may die.

As a valued health resource during a disaster response, critical care nurses dictate how and when patients move in the facility for internal evacuation and surge capacity (see Triage, on pages 184–186). Nurses perform best with adequate training and preparation; therefore, as this chapter discusses, plans must delineate the appropriate deployment of nurses during a disaster and the resources needed for them to be successful.

▲ Response to Mass Casualty Incidents

By definition, a mass casualty incident (MCI) is characterized by large numbers of patients needing medical treatment that exceeds the capabilities of local emergency and health care personnel. To be integrated as part of the community's plan for emergency preparedness in MCIs, nurses must have a basic level of skills and education to appropriately respond and protect themselves and others, particularly during chemical, biological, radiological, nuclear, and explosive events. Critical care nurses may be among the first responders to large MCIs because public health teams and the emergency medical system (EMS) are overwhelmed. However, every nurse must have sufficient knowledge and skill to understand the potential for an MCI, identify when such an incident may have occurred, and protect himself or herself while caring for victims. The trauma and public health system in the United States is continuously improving its ability to respond to MCIs and critically ill patients who have suffered multiple traumas. During an MCI, nurses must perform the primary and secondary surveys as with any other trauma patient. They must know how to recognize their own roles and limitations as well as where to seek additional information and resources. A group initially known as the International Nursing Coalition for Mass Casualty Education (INCMCE) has developed basic competencies for entry-level registered nurses related to MCIs (Box 15-1). This group is now known as the Nursing Emergency Preparedness Education Coalition.[2]

▲ Response to Terrorism

Terrorism is the unlawful use of force or violence against persons or property to intimidate civilians or coerce government or a civilian population while promoting political and social objectives.[3] Ultimately, terrorists aim to make people fearful. Terrorism can overwhelm a nation's health care system.

In 2002, the American Association of Critical-Care Nurses (AACN) released a Statement of Commitment to Mass Casualty and Bioterrorism Preparedness recognizing that critical care nurses will be called on to respond to disaster and mass casualty situations. This statement includes the following:

"Bioterrorism and the potential of mass casualties is a significant public health threat facing the United States. The nation's capacity to respond to this threat depends in part on the ability of the health care professionals and public health officials to rapidly and effectively detect, manage, and communicate during an event resulting in mass casualties."[4] The AACN works closely with the Red Cross to help support critical care nurses during times of disaster.

In August 2003, a report to the Congressional Committee from the United States General Accounting Office (GAO) regarding Hospital Preparedness for a Bioterrorist Incident was published. Some of the GAO's findings were alarming. Although most urban hospitals across the country reported that they participated in basic planning and coordination of activities for bioterrorism response, they did not have the medical equipment, especially ventilators, to handle the number of people who would likely require care following a bioterrorist incident. Most hospitals stated they lacked the necessary resources to handle a large influx of patients.[5] Because many facilities would not be prepared to handle an attack, even fewer would be able to handle a large influx of critically ill patients who needed critical care. The GAO report also recounted many projected scenarios surrounding a possible influenza pandemic suggesting that the United States would be severely short of ventilators and trained staff to care for patients on ventilators.

In 2004, a working group in Pittsburgh, Pennsylvania, made recommendations to hospital and clinical leaders regarding the delivery of critical care services in the wake of a bioterrorist attack resulting in hundreds or thousands of critically ill patients. In these situations, traditional hospital and clinical care standards in general, and critical care standards in particular, likely could no longer be maintained. The study group came up with no clinical guidelines to deal with these situations. However, it did develop the following six planning assumptions regarding the current critical care medicine response for bioterrorism:

1. Future bioterrorist attacks may be covert and could result in hundreds, thousands, or more, critically ill victims.
2. Critical care will play a key role in decreasing morbidity and mortality rates after a bioterrorist attack.
3. Mass critical care cannot be provided without substantial planning and new approaches to providing critical care.
4. A hospital has limited ability to divert or transfer patients to other hospitals in the aftermath of a bioterrorist attack.
5. Currently deployable medical teams of the federal government have a limited role in increasing a hospital's immediate ability to provide critical care to large numbers of victims of a bioterrorist attack.
6. Hospitals may need to depend on nonfederal sources or reserves of medications and equipment necessary to provide critical care for the first 48 hours following discovery of a bioterrorist attack.[6]

▲ Role of Hospital Emergency Incident Command System

Hospitals respond to MCIs using the Hospital Emergency Incident Command System (HEICS), which is now referred to as the Hospital Incident Command System (HICS). HICS is an incident management system based on the Incident Command System (ICS) that assists hospitals in improving their emergency management planning as well as response and recovery capabilities for unplanned and planned events. HICS is consistent with ICS and the National Incident Management System principles, which allow for multiagency response to events. The HICS organizational chart is shown in Figure 15-1 on page 185. Critical care nurses need to be aware of how they specifically fit into their hospital disaster plan; they may be called on to take on a major role in the HICS organizational system.

BOX 15-1 Competencies for Entry-Level Registered Nurses Related to Mass Casualty Incidents

Core Compentencies

I. Critical Thinking

1. Use an ethical and nationally approved framework to support decision making and prioritizing needed in disaster situations.
2. Use clinical judgment and decision-making skills in assessing the potential for appropriate, timely individual care during a mass casualty incident (MCI).
3. Use clinical judgment and decision-making skills in assessing the potential for appropriate, individual ongoing care after an MCI.
4. Describe at the predisaster, emergency, and postdisaster phases the essential nursing care for
   a. individuals,
   b. families,
   c. special groups (eg, children, elderly, and pregnant women), and
   d. communities.
5. Describe accepted triage principles specific to MCIs, for example, the START or Simple Triage and Rapid Treatment System.

II. Assessment

A. General

1. Assess the safety issues for self, the response team, and victims in any given response situation in collaboration with the incident response team.
2. Identify possible indicators of a mass exposure (ie, clustering of individuals with the same symptoms).
3. Describe general signs and symptoms of exposure to selected chemical, biological, radiological, nuclear, and explosive (CBRNE) agents.
4. Demonstrate the ability to access up-to-date information regarding selected nuclear, biological, chemical, explosive, and incendiary agents.
5. Describe the essential elements included in an MCI scene assessment.
6. Identify special groups of patients that are uniquely vulnerable during an MCI (eg, very young, aged, and immunosuppressed).

B. Specific

1. Conduct a focused health history to assess potential exposure to CBRNE agents.
2. Perform an age-appropriate health assessment including
   a. airway and respiratory assessment;
   b. cardiovascular assessment, including vital signs and monitoring for signs of shock;
   c. integumentary assessment, particularly a wound, burn, and rash assessment, and pain assessment;
   d. injury assessment from head to toe;
   e. gastrointestinal assessment, including stool specimen collection and basic neurological assessment;
   f. musculoskeletal assessment; and
   g. mental status, spiritual, and emotional assessment.
3. Assess the immediate psychological response of the individual, family, or community following an MCI.
4. Assess the long-term psychological response of the individual, family, or community following an MCI.
5. Identify resources available to address the psychological impact (eg, Critical Incident Stress Debriefing [CISD] teams, counselors, Psychiatric/Mental Health Nurse Practitioners [P/MHNPs]).
6. Describe the psychological impact on responders and health care providers.

III. Technical Skills

1. Demonstrate the safe administration of medications, particularly vasoactive and analgesic agents, via oral (PO), subcutaneous (SC), intramuscular (IM), and intravenous (IV) administration routes.
2. Demonstrate the safe administration of immunizations, including smallpox vaccination.
3. Demonstrate knowledge of appropriate nursing interventions for adverse effects from medications administered.
4. Demonstrate basic therapeutic interventions including
   a. basic first aid skills,
   b. oxygen administration and ventilation techniques,
   c. urinary catheter insertion,
   d. nasogastric tube insertion,
   e. lavage technique (ie, eye and wound), and
   f. initial wound care.
5. Assess the need for and initiate the appropriate CBRNE isolation and decontamination procedures available, ensuring that all parties understand the need.
6. Demonstrate knowledge and skill related to personal protection and safety, including the use of personal protective equipment (PPE), for
   a. level B protection,
   b. level C protection, and
   c. respiratory protection.
7. Describe how nursing skills may have to be adapted while wearing PPE.
8. Implement fluid and nutrition therapy, taking into account the nature of injuries and/or agents exposed to and monitoring hydration and fluid balance accordingly.
9. Assess and prepare the injured for transport, if required, including provisions for care and monitoring during transport.
10. Demonstrate the ability to maintain patient safety during transport through splinting, immobilization, monitoring, and therapeutic interventions.

IV. Communication

1. Describe the Incident Command System (ICS) during an MCI.
2. Identify your role, if possible, within the ICS.
3. Locate and describe the emergency response plan for the place of employment and its role in community, state, and regional plans.
4. Identify one's own role in the emergency response plan for the place of employment.
5. Discuss security and confidentiality during an MCI.
6. Demonstrate appropriate emergency documentation of assessments, interventions, nursing actions, and outcomes during and after an MCI.
7. Identify appropriate resources for referring requests from patients, media, or others for information regarding MCIs.
8. Describe principles of risk communication to groups and individuals affected by exposure during an MCI.
9. Identify reactions to fear, panic, and stress that victims, families, and responders may exhibit during a disaster situation.
10. Describe appropriate coping strategies to manage self and others.

Core Knowledge

I. Health Promotion, Risk Reduction, and Disease Prevention

1. Identify possible threats and their potential impact on the general public, emergency medical system (EMS), and the health care community.

(continued on page 184)

BOX 15-1 Competencies for Entry-Level Registered Nurses Related to Mass Casualty Incidents (continued)

2. Describe community health issues related to CBRNE events, specifically limiting exposure to selected agents, contamination of water, air, and food supplies, and shelter and protection of displaced persons.

II. Health Care Systems and Policy

1. Define and distinguish the terms *disaster* and *MCI* in relation to other major incidents or emergency situations.
2. Define relevant terminology, including
   a. CBRNE,
   b. WMD,
   c. triage,
   d. Incident Command Service (ICS),
   e. personal protective equipment,
   f. scene assessment, and
   g. comprehensive emergency management.
3. Describe the four phases of emergency management: preparedness, response, recovery, and mitigation.
4. Describe the local emergency response system for disasters.
5. Describe the interaction between local, state, and federal emergency response systems.
6. Describe the legal authority of public health agencies to take action to protect the community from threats, including isolation, quarantine, and required reporting and documentation.
7. Discuss principles related to an MCI site as a crime scene (eg, maintaining integrity of evidence and chain of custody).
8. Recognize the impact MCIs may have on access to resources and identify how to access additional resources (eg, pharmaceuticals and medical supplies).

III. Illness and Disease Management

1. Discuss the differences and similarities between an intentional biological attack and that of a natural disease outbreak.
2. Assess, using an interdisciplinary approach, the short-term and long-term effects of physical and psychological symptoms related to disease and treatment secondary to MCIs.

IV. Information and Health Care Technologies

1. Demonstrate use of emergency communication equipment that you will be required to use in a MCI response.
2. Discuss the principles of containment and decontamination.
3. Describe procedures for decontamination of self, others, and equipment for selected CBRNE agents.

V. Ethics

1. Identify and discuss ethical issues related to CBRNE events:
   a. Rights and responsibilities of health care providers in MCIs, for example, refusing to go to work or report for duty, refusal of vaccines.
   b. Need to protect the public versus an individual's right for autonomy, for example, right to leave the scene after contamination.
   c. Right of the individual to refuse care, informed consent.
   d. Allocation of limited resources.
   e. Confidentiality of information related to individuals and national security.
   f. Use of public health authority to restrict individual activities, require reporting from health professionals, and collaborate with law enforcement.
2. Describe the ethical, legal, psychological, and cultural considerations when dealing with the dying and or the handling and storage of human remains in an MCI.
3. Identify and discuss legal and regulatory issues related to
   a. abandonment of patients,
   b. response to an MCI and one's position of employment, and
   c. various roles and responsibilities assumed by volunteer efforts.

VI. Human Diversity

1. Discuss the cultural, spiritual, and social issues that may affect an individual's response to an MCI.
2. Discuss the diversity of emotional, psychosocial, and sociocultural responses to terrorism or the threat of terrorism on one's self and others.

Professional Role Development

1. Describe these nursing roles in MCIs:
   a. Researcher
   b. Investigator/epidemiologist
   c. EMT or First Responder
   d. Direct care provider, generalist nurse
   e. Direct care provider, advanced practice nurse
   f. Director/coordinator of care in hospital/nurse administrator or emergency department nurse manager
   g. On-site coordinator of care/incident commander
   h. On-site director of care management
   i. Information provider or educator, particularly the role of the generalist nurse
   j. Mental health counselor
   k. Member of planning response team
2. Identify the most appropriate or most likely health care role for oneself during an MCI.
3. Identify the limits to one's own knowledge, skills, abilities, and authority related to MCIs.
4. Describe essential equipment for responding to an MCI, for example, stethoscope, registered nurse license to deter imposters, packaged snack, change of clothing, bottles of water.
5. Recognize the importance of maintaining one's expertise and knowledge in this area of practice and of participating in regular emergency response drills.
6. Participate in regular emergency response drills in the community or place of employment.

From Nursing Emergency Preparedness Education Coalition, July 2003. Available at: http://www.nursing.vanderbilt.edu/incmce/competencies.html.

▲ Triage

Triage is a system for rationing resources in response to an overwhelming medical emergency event. Effective triage is one of the first procedures that the critical care nurse uses in responding to a disaster. Patients are quickly categorized into minimal, delayed, immediate, and expectant or morgue categories (see Box 15-2).

In addition to triage occurring outside the facility, health care workers should also perform triage inside the facility and ensure that salvageable patients are appropriately categorized. As patients are triaged, personnel should send them to the area where they will receive the appropriate level of care

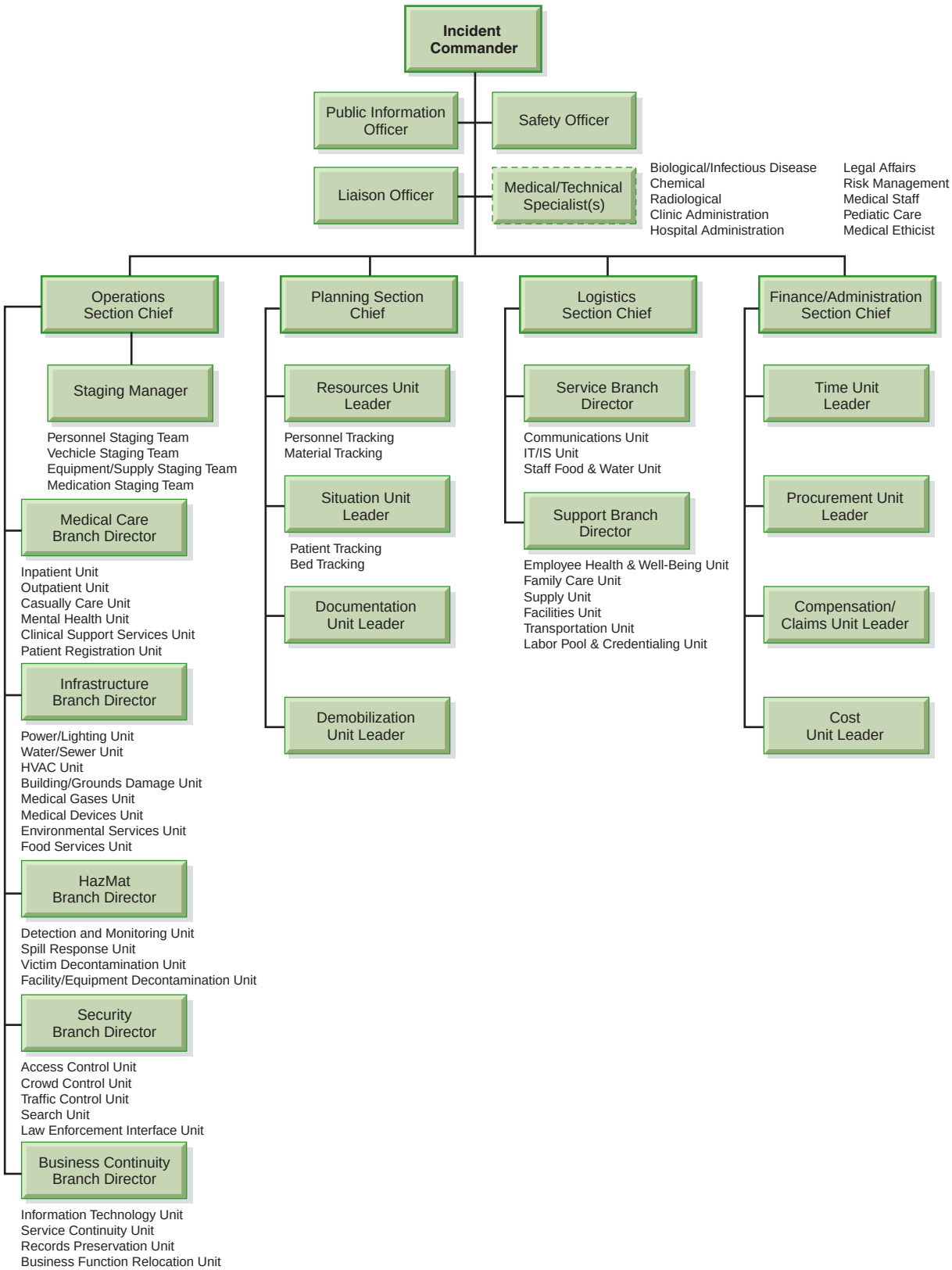


FIGURE 15-1 ▲ Hospital Incident Command System organizational chart. (From the Hospital Incident Command System Training Manual. Available at: http://www.emsa.ca.gov.)

BOX 15-2 START: Triage Categories

In disaster situations, the Simple Triage and Rapid Treatment (START) system offers a quick and efficient way to prioritize victims' treatment needs based on respiratory, perfusion, and mental status observations. Categories include

- Minor: Victims who can get up and walk are determined to be in less need and care may be delayed for up to 3 hours.
- Delayed: Victims who are breathing have an irregular or absent pulse but who can respond to commands, such as "open your eyes; close your eyes" may be considered for care that can be delayed up to 1 hour
- Immediate: Victims whose breathing indicates impending shock or possible suffocation need immediate care
- Dead: Victims whose breathing, perfusion, and mental status are absent and/or unresponsive to stimuli may be considered dead, and therefore, not in need of urgent care.

Source: Community Emergency Response Team Unit, LAFD Disaster Preparedness Section, www.cert-la.com.

for their triage category. The latter form of triage requires policy, education, and practice for desirable results.

An emerging concept as a result of triage is surge capacity. Surge capacity is a health care system's ability to expand quickly above and beyond normal services to meet an increased demand for medical care in the event of large-scale health emergencies. Nursing responsibility and patient care ratios will also adjust during a disaster event, and surge capacity depends on nurses caring for more patients. In some instances, disasters or surge capacity needs may require staff to move patients inside facilities to safer zones or to temporary clinical care areas.

Nurses serve as a focal point for assessing patients for movement and assembling the necessary materials for continuing the care of these patients at the new site. It is important to practice the movement of patients for internal evacuation periodically.

▲ Unnatural Disasters

Unnatural disasters include terrorist acts. Terrorism can take many forms.

Explosions and Blast Attacks

Explosives and bombs are the weapons of choice for terrorists. As a result, blast injuries occur and victims typically require evaluations by hospital personnel. Although some injured victims do not need hospital admission, more severely injured victims will need critical care.

The injuries can result from primary, secondary, or tertiary blast effects. Primary blast injuries are the result of sudden changes in atmospheric pressure caused by an explosion. Examples of primary blast injuries are as follows:

- Ear injuries, such as perforated eardrums
- Pulmonary injuries, including hemorrhagic contusion and hemopneumothorax
- Gastrointestinal hemorrhage or bowel perforation or rupture

Secondary blast injury occurs when victims are struck by flying objects and debris. Tertiary blast injuries occur when the body is hurled through the air and struck by another object.

Nuclear or Radiological Attacks

The medical consequences of a nuclear or radiological attack or accident depend on the nuclear or radiation source. Radiation accidents or attacks can occur as the result of problems with nuclear reactors, industrial sources, and medical sources. It is important that clinicians understand how a radiological attack can occur and how to treat patients. A radiological or nuclear attack can occur in one of five ways:

1. *Simple radiological device* (SRD): An SRD is a device designed to spread radioactive material without the use of explosives, thereby exposing many people to various levels of radiation.
2. *Radiological dispersal device* (RDD): An RDD is a device formed by combining an explosive agent with radioactive materials. The initial explosion kills or injures those closest to the bomb, and the radioactive material remains to expose and contaminate survivors and possibly emergency responders.
3. *Nuclear reactor sabotage:* This type of incident is low because of sophisticated shielding, but it could occur with an attack on a nuclear reactor.
4. *Improvised nuclear device* (IND): An IND is any device designed to cause a nuclear detonation. It is not easy to make such a weapon detonate correctly. This type of incident is actually an RDD. Although an IND is unlikely because of the necessary engineering sophistication, a stolen device would generate high levels of radiation.
5. *Nuclear weapon:* This method of exposure could occur if a weapon were stolen. This is another example of a remote radiological attack, but it could happen.[7]

There are two categories of radiation incidents: (1) *External exposure* is irradiation from a source distant or in close proximity to the body. External irradiation can be divided into *whole-body* exposure or *local* exposure. (2) *Contamination* is unwanted radioactive material in or on the body.[8]

Response to Attack

Based on the type of incident, the response is different. Most external exposures result in irradiation of the victim. Once the person is removed from the source of radiation, the irradiation ceases. A person exposed to external radiation does not become radioactive and poses no hazard to people nearby.

Contamination incidents require an entirely different approach to dealing with the victim than external radiation. Care givers and support personnel must be careful not to spread the contamination to uncontaminated parts of the victim's body, to themselves, or to the surrounding area. Internal contamination can result from inhalation, ingestion, direct absorption through the skin, or penetration of radioactive materials through open wounds. Serious or significant medical condition treatment should always take precedence over radiological assessment or patient decontamination.

After radiation exposure, individuals may develop acute radiation syndrome (ARS). Strong, healthy people are often

Table 15-1 Phases of Effects of Radiation Exposure

Phase	Time of Occurrence	Signs and Symptoms
Prodromal phase (presenting symptoms)	48–72 h after exposure	Nausea, vomiting, loss of appetite, diarrhea, fatigue High-dose radiation: fever, respiratory distress, increased excitability
Latent phase (a symptom-free period)	After resolution of prodromal phase; can last up to 3 wk With high-dose radiation, latent period is shorter	Decreasing lymphocytes, leukocytes, thrombocytes, red blood cells
Illness phase	After latent period phase	Infection, fluid and electrolyte imbalance, bleeding, diarrhea, shock, and altered level of consciousness
Recovery phase *Or* Death	After illness phase After illness phase	Can take weeks to months for full recovery Increased intracranial pressure is a sign of impending death

From Smeltzer SC, Bare BG, Hinkle JL, et al (eds): Brunner & Suddarth's Textbook of Medical-Surgical Nursing, 12th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 2007.

resistant to minimal exposure. The factors that determine whether ARS occurs include exposure to high-dose radiation (minimum, 100 cGy) and rate of radiation with whole-body exposure and penetrating-type radiation. There are several stages of radiation exposure (Table 15-1).

Management

Treatment priorities after a radiological attack:

1. *Treat and stabilize life-threatening injuries.* It is essential to stabilize the patient and treat life-threatening injuries first. Once that is done, a health care provider with radiological health training should perform a radiological assessment. Use a Geiger counter for radiological measurements.
2. *Prevent and minimize internal contamination.* Time is critical to prevent radioactive uptake. Administration of potassium iodide within 2 hours of contamination prevents radioiodine from accumulating in the thyroid gland (Table 15-2).
3. *Assess internal contamination and decontamination.* This information is covered in the following section on Chemical Attacks. Contaminated patients who are not seriously injured should be decontaminated before treatment.
4. *Contain contamination and decontamination.*
5. *Minimize external contamination to medical personnel.* Staff should wear personal protective clothing. Respirators are necessary if the patient is highly contaminated.
6. *Assess local radiation injuries/burns and flush them if contaminated.*
7. *Follow up on patients with significant whole-body irradiation or internal contamination.*
8. *Counsel patient and family about the potential for long-term risks and effects.*[9]

Table 15-2 Potassium Iodide (KI) Administration

Patient	KI Dose (mg)
Adults	130
Women who are breastfeeding	130
Children 3–18 y	65
Infants and children 1 mo to 3 y	32
Newborns to 1 mo	16

From Pediatric Preparedness for Disaster and Terrorism: A Natural Consensus Conference. National Center for Disaster Preparedness Mailman School of Public Health, Columbus University, March 2007.

Chemical Attacks

Chemical warfare agents are hazardous chemicals designed to irritate, incapacitate, injure, or kill.[10] Although several of these agents were used during wartime, many of the most recent uses have been in terrorist attacks. For example, the sarin gas attacks in Japan in the mid-1990s resulted in few deaths but an overwhelming influx of contaminated patients to medical facilities. Combinations of chemical attacks with explosions and blast attacks are sometimes called "dirty bombs." To have chemical contamination associated with explosions, the victims need to be in close proximity to the explosion or blast attack.

Chemical agents pose a genuine threat for many reasons. First, they are readily accessible; for example, tear gas is sold in stores. Second, they are easy to find and easy to transport without being considered unusual; for example, transport of many nerve agents occurs on a daily basis by truck or rail. By the time a chemical attack is completed, the terrorist or criminal can be gone.

These toxic chemicals can be absorbed through the eyes, skin, airways, or a combination of these routes. Table 15-3 summarizes the common types of chemical agents, their mechanisms of action, signs and symptoms of exposure, and treatment for exposure.

Types of Chemical Agents

NERVE AGENTS. Nerve agents are the most toxic of all weaponized military agents. Examples of these agents are tabun, sarin, soman, and VX. Nerve agents inhibit cholinesterase, and they can cause sudden loss of

Table 15-3 Common Chemical Agents and Antidotes

Agent	Action	Signs and Symptoms	Decontamination and Treatment
Nerve agents: sarin, soman, organophosphates	Inhibition of cholinesterase	Increased secretions, gastrointestinal motility, diarrhea, bronchospasm	Soap and water Supportive care Benzodiazepine Pralidoxime Atropine
Blood agent: cyanide	Inhibition of aerobic metabolism	Inhalation—tachypnea, tachycardia, coma, seizures; can progress to respiratory arrest, respiratory failure, cardiac arrest, and death	Sodium nitrate Sodium thiocyanate Amyl nitrate Hydroxocobalamin
Vesicant agents: lewisite, sulfur mustard, nitrogen mustard, phosgene	Blistering agents	Superficial to partial-thickness burn with vesicles that coalesce	Soap and water Blot; do not rub dry
Pulmonary agents: phosgene, chlorine, ammonia	Separation of alveoli from capillary bed	Pulmonary edema, bronchospasm	Airway management Ventilatory support Bronchoscopy
Skin and eye irritants: mace (CN), tear gas (CS)	Local reaction to skin and eyes; may cause respiratory difficulties	Tearing of the eyes; burning of the skin; possible respiratory difficulty	Irrigate eyes—water only Soap and water to skin

From Slota M (ed): Core Curriculum for Pediatric Critical Care Nursing. St. Louis, MO: Elsevier, 2006; and Smeltzer SC, Bare BG, Hinkle JL, et al (eds): Brunner & Suddarth's Textbook of Medical-Surgical Nursing, 12th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 2203.

consciousness, seizures, apnea, and death. Diagnosis is usually made on the basis of clinical signs and symptoms.

VESICANTS. Vesicants cause blistering. The most commonly used vesicants are sulfur, mustard, and lewisite. These agents injure the eyes, skin, airways, and some internal organs. Vesicants may be commonly used as a mass casualty weapon because they are potent and difficult to detect, have delayed effect, produce prolonged disability, are stable in storage, can be transported easily, and are inexpensive to produce. They remain active in an area for up to 1 week. Some may have an odor of mustard or garlic.

CYANIDE. Cyanide is a widely used chemical in the United States. Terrorists use cyanide in confined spaces, such as subway cars, shopping centers, convention centers, and small buildings. Shortly after inhaling cyanide, victims may become anxious and hyperventilate. Inhalation of cyanide can cause convulsions, asystole, and death. Antidotes must be administered immediately.

PULMONARY INTOXICANTS. Pulmonary intoxicants cause severe life-threatening lung injury after inhalation. The effects are generally delayed for several hours. Examples are phosgene, perfluoroisobutylene (PFIB), ammonia, and chlorine. In 1984, an industrial accident, not a terrorist attack, involving a pulmonary intoxicant occurred at the Union Carbide plant in Bhopal, India. The released chemicals, for example, methyl isocyanate, caused great morbidity and death. To this day, this industrial accident remains one of the worst to occur in India. Pulmonary intoxicants are irritating to the eyes and respiratory tract. These agents can cause severe pulmonary edema of a noncardiac nature. The pathophysiology involves a permeability defect in the alveolar capillary membrane, and there may be a clinical latency period following exposure.

RIOT CONTROL AGENTS. Riot control agents, which have an immediate effect, irritate the eyes, nose, mouth, skin, and respiratory tract. They stimulate tear production by the lacrimal glands. The effect lasts about 30 minutes. Examples of these agents, which are routinely used by police, are chloroacetophenone (CN; Mace), oleoresin capsicum (OC; pepper spray), and chlorobenzylidenemalononitrile (CS; tear gas).[10]

Management

In the event of chemical exposure, decontamination is necessary. Staff need to be trained to decontaminate patients appropriately and should have access to appropriate personal protective equipment (PPE). With most chemical agents, only a splash-resistant gown, much like that used everyday in hospitals, and an N95 mask used for respiratory isolation in hospitals are necessary. Some of the more potent chemicals also require a self-contained breathing apparatus and a chemical-resistant suit. People who use these specialty suits and breathing devices should receive training in putting on the suit and have a respiratory evaluation annually.

As required by the Joint Commission hospitals should have decontamination protocols and procedures in place. Decontamination shelters can either be a room designated at the entrance to the emergency department with appropriate drainage and shower capability or a trailer; the latter appears to be the most popular choice in the United States. Decontamination trailers are advantageous because they are mobile and can be moved to the area designated for decontamination. Decontamination shelters should:

1. Have a water connection. Most are compatible with the facility's water lines.
2. Have the ability to collect and contain large quantities of water.

3. Have something to mix with the water to remove the various chemical agents. Most chemical agents respond to soap and water.
4. Have adequate lighting in the shelter.
5. Have some connection to electricity, whether through the hospital or a generator.
6. Have a conveyor system for nonambulatory patients.
7. Allow for patient privacy.
8. Have room for about two or three personnel. These personnel are preferably not health care providers because all clinical staff will be needed to provide medical care.
9. Have sufficient room for families. It may be necessary to decontaminate parents as well as children. (Parents can also help decontaminate their children.)[11]

Decontamination of children involves some special considerations. Because children may not understand what is happening, they may be uncooperative or even combative. Size may be an issue. Children are lower to the ground, which means that they may be exposed to more of the contaminant. Children have a large surface-to-volume ratio, which places them at higher risk for absorption and exposure to the contaminant. In addition, because even a small dose may be lethal in a child, children need to be decontaminated as quickly as possible. Children are also at a higher risk for cold stress from a rapid drop in temperature or fever from exposure to very warm temperatures. It is necessary to get them into a neutral thermal environment and out of extreme heat or cold. Finally, whenever possible, it helps to keep the family unit together so that the parents can keep the smaller children safe. If the parents are not present, appropriate arrangements must be made for supervision.

Biological Attacks

A biological attack is referred to as bioterrorism or the deliberate release of microorganisms (bacteria, viruses, fungi, or microbial toxins) into a community to produce death, disease, or poisoning.[10] Humans, animals, or plants may be affected. Biological weapons are often called the "poor man's bomb" because they are relatively inexpensive to produce and disseminate. A bioterrorist attack is a real threat, as evidenced by the anthrax attacks of 2001. Letters with anthrax spores arrived by post at some media and legislative offices, closing down many post offices and federal buildings for long periods. The attack resulted in 22 cases of anthrax, 5 deaths, and a nation on high alert.

Many biological substances result in specific signs and symptoms, and every nurse should know the basics of caring for affected patients. In 1999, the Association of Professionals in Infection Control developed a template, the *Bioterrorism Readiness Plan: A Template for Healthcare Facilities*, for hospitals to follow when dealing with bioterrorism.[12] In 2002, this group made some minor modifications to this plan. Table 15-4 summarizes the diseases associated with bioterrorism and management protocols for victims of biological attacks. Many bioterrorist agents are readily accessible. For example, anthrax can be found on some farms, and terrorists can pick it up and haul it away without being noticed.

Smallpox, a viral disease, is a strong bioterrorism threat because it has a high morbidity in an otherwise healthy population. Figure 15-2 on page 191 shows the smallpox rash. In 1980, the World Health Organization (WHO) declared smallpox eradicated.[10] In 2003, a smallpox vaccine became available for health care workers, but the U.S. Food and Drug Administration recalled it, and few people received the vaccine because of issues related to the exclusion criteria and the occurrence of some cardiac problems after administration of the vaccine.

The plague has a negative historical connotation because people still remember references to biblical references and deaths caused by bubonic plague. Animals, such as squirrels and other rodents that live on farmland, city parks, and campgrounds, still carry the bacteria that cause plague. Humans bitten by infected animals can become ill.

Botulism still occurs today in developed countries. A person can contract the disease from eating food that has been infected with the botulism toxin. Treatment is available. Children who develop infantile botulism can take an antidote, commonly known as "Baby Big," if the disease is diagnosed early and verified by a laboratory approved by the Centers for Disease Control and Prevention (CDC). At the time of diagnosis, the local health department sells the hospital the necessary dosage. The main issue is that the botulism is not always diagnosed promptly enough.

One of the more common viral hemorrhagic fevers is the Ebola virus. This virus is found more commonly in Third World countries. It is treatable but carries a negative connotation.

▲ Natural Disasters

A natural disaster is a result of the combination of a natural event (eg, earthquake, extreme heat, flood, hurricane, landslide, tornado, tsunami, volcanic eruption, wildfire, winter storm) and human involvement. By definition, a natural disaster does not occur without human involvement; therefore, an earthquake that destroys an uninhabited island is technically not a natural disaster. Another important feature of a natural disaster is that an unprepared or ill-prepared population is vulnerable, and therefore, a lack of preparation amplifies the unfortunate outcomes as result of a natural disaster.

Hospitals and other health care facilities play a unique role in the community because these facilities already contain an unhealthy, weak population and yet are expected to receive casualties resulting from disasters. Therefore, hospital personnel and administrators must actively prepare for any event that threatens the structure, function, and recovery of their organization and practice the appropriate responses to a hazardous event.

Some experts consider pandemic influenza a potential natural disaster, suggesting that it is a real possibility. Currently, the health care and corporate industries are working together to address the problem; and new vaccines and antiviral agents have been developed. Nurses are critical to the disaster response because they have direct patient care roles and will be present if an unforeseen event arises.

The H1N1 virus outbreak in 2009 increased the census in hospitals and ICUs across the country. Within 3 months of the first identified H1N1 case, the WHO raised it to a level of pandemic because there were community level outbreaks in at least one country other than where it was first identified. Cases spread quickly, especially in the younger population (25 years and younger).[13] The H1N1 outbreak helped address a potential outbreak that became a global emergency.

Table 15-4 Diseases Associated With Bioterrorism

Disease	Etiology	Mode of Transmission (Route of Exposure)	Incubation Period	Clinical Features	Treatment	Prophylaxis
Anthrax	*Bacillus anthracis*	Direct contact with bacterium or spores	1–7 d	**Cutaneous:** itchy papular lesions that turn into vesicles and within 2–6 d form black eschar; lesions most often seen on head, chest, and forearms	Antibiotic therapy **Adults:** ciprofloxacin, 500 twice daily, or doxycycline, 100 mg twice daily for at least 60 d **Children:** ciprofloxacin, 15 mg/kg twice daily, or doxycycline: more than 8 y and <45 kg: 100 mg twice daily more than 8 y and 45 kg or less: 2.2 mg/kg twice daily 8 y or less: 2.2 mg/kg twice daily	None
		Inhalation of spores	2–60 d	**Pulmonary:** nonspecific flu-like symptoms followed by the abrupt onset of respiratory failure (2–4 d after initial symptoms) and hemodynamic collapse	**Adults:** ciprofloxacin, 400 mg every 12 h *and* one or two additional microbials; switch to oral when appropriate. Ciprofloxacin, 50 mg, or doxycycline, 100 mg twice daily **Children:** Ciprofloxacin, 10 mg/kg every 12 h *or* doxycycline, same as for cutaneous except every 12 h instead of twice daily Switch to oral therapy when appropriate.	
		Ingestion of contaminated food, usually meat	1–7 d	**Gastrointestinal:** abdominal pain, nausea, fever, hematemesis, and bloody diarrhea		
Smallpox	Variola virus	Airborne	7–17 d (average, 12 d)	Nonspecific flu-like symptoms, fever, myalgia, skin lesions that progress from macules to papules to vesicles and then scab over in 1–2 wk	Supportive care, after 3 d can try variola immune globulin, negative isolation	Vaccination before exposure or within 3 d of exposure
Plague	*Yersinia pestis*	Flea-borne (bubonic plague), airborne (pneumonic plague)	2–8 d (flea-borne); 1–2 d (airborne)	Fever, cough, chest pain, hemoptysis, mucopurulent or watery sputum with Gram-negative rods on Gram stain, bronchopneumonia on x-ray	Streptomycin, 1 g IM twice daily, *or* gentamycin, 5 mg/kg IM or IV daily, or 3 mg/kg loading dose followed by 1.7 mg/kg IM or IV three times a day; droplet precautions in effect until patient has completed 72 h of antimicrobial therapy	Doxycycline, 100 mg PO twice daily, *or* ciprofloxacin, 500 mg PO twice daily
Tularemia	*Francisella tularensis*	Contact with infected animals (rabbits, deer) or vectors (fleas, ticks, mosquitos); airborne	2–14 d	Fever, chills, headache, muscle pain, nonproductive cough, pneumonia; regional lymphadenopathy if ingested	Same as for plague	Same as for plague

(continued on page 191)

Table 15-4 Diseases Associated With Bioterrorism (*continued*)

Disease	Etiology	Mode of Transmission (Route of Exposure)	Incubation Period	Clinical Features	Treatment	Prophylaxis
Botulism	*Clostridium botulinum*	Foodborne (most common); airborne	**Foodborne:** 12–36 h **Airborne:** 24–72 h	Gastrointestinal symptoms, drooping eyelids, jaw clench, difficulty swallowing or speaking, descending paralysis, blurred vision, no sensory deficits	Supportive care; antitoxin may halt progression of symptoms but is unlikely to reverse them	None
Viral hemorrhagic fevers	Diverse group of viruses (eg, Ebola virus, yellow fever)	Variable, may be person-to-person, via flea or animal bite; or airborne	2–22 d	Variable; usually a nonspecific illness lasting <1 wk with high fever and headache followed by flushing maculopapular rash and conjunctival infection, progressing to diffuse hemorrhagic disease and multiorgan system failure	Supportive care, ribavirin may be helpful in some cases	None

Data from Los Angeles County EMS and Public Health Agencies: Terrorism Agent Information and Treatment Guidelines for Clinicians and Hospitals, 2003.

Together, hospitals and government agencies set up plans to identify the issues and plan strategies to protect the public. The most common symptoms of H1N1 virus are fever, cough, and sore throat. A small percentage of patients required hospitalization and the patients who ended up in the critical care units were put on extracorporeal membrane oxygenation (ECMO), as pulmonary bypass, to treat respiratory failure. A small percentage of the patients treated in the critical care units died. Excellent resources are available for H1N1 and are noted on the CDC and the WHO Web sites. The Association of Infection Control Practitioners (APIC) also developed a reference document for infection prevention in alternate care sites; it can be found on their Web site at www.apic.org.

▲ Psychological Effects of Terrorism

It is natural to be fearful during and after a major disaster, whether or not it is a terrorist incident. People exhibit various responses to stress and stressful events, such as a terrorist attack or a major natural disaster. The responses include fear, grief, and deep sadness. People may complain that they feel "sick to their stomach" and have no appetite. Their sleep pattern and conduct in daily activities may change. Several weeks to months may pass before they feel normal and stable again.

In severe cases, the psychological stress remains months after the event. Multiple factors affect how people respond. The perception of numerous losses generally has a negative impact and may be related inversely to disaster recovery. Research has found that when there are large numbers of deaths and high levels of symptoms, the presence of long-term psychiatric disorders may be quite high.[14] These people should undergo screening for posttraumatic stress disorder (PTSD). PTSD is an intense emotional and physical response to thoughts and reminders of the event that last for many weeks or months after the traumatic event. Victims of PTSD may complain of nightmares, flashbacks, and severe emotional and physical reactions to thoughts of the event.

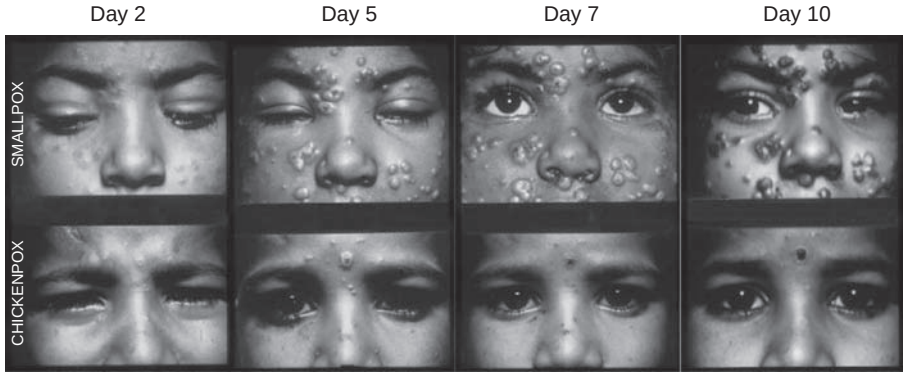


FIGURE 15-2 ▲ Progression of smallpox rash. (From World Health Organization: WHO slide set on the diagnosis of smallpox, 2001. Reproduced by permission of the World Health Organization.)

▲ Clinical Applicability Challenges

CASE STUDY

It is a holiday weekend, and several rock bands are playing in concert in the local amphitheater. The emergency department has been notified by the paramedics that an explosion has occurred in the theater and they are expecting to transport at least 20 critical victims to area hospitals. Your hospital has responded that it can take five critically injured victims. As the casualties arrive and the emergency department begins to triage these patients, many more begin to arrive unescorted by the paramedics. Some of the patients arriving by themselves appear to have serious injuries including severe shortness of breath, confusion, bleeding from the ears, and objects impaled in their chest and torso.

1. You are the critical care charge nurse. What responsibilities might you have in this situation?
2. The Emergency Department staff is overwhelmed and needs assistance in triaging the critical care patients. What are some examples of secondary blast injuries? What are some examples of primary blast injuries?
3. The police department has just notified your emergency department that this is a terrorist attack and an SRD was used. How does the initial treatment change for these patients? Is decontamination necessary?

References

1. Kissoon, N. Task force for pediatric emergency mass critical care. Pediatr Crit Care Med 12(6 Suppl), S103–S108, 2011
2. Daily E, Pajden P, Birnbaum M. A review of competencies developed for disaster healthcare providers: Limitations of current processes and applicability. Prehosp Disaster Med 25(5), 387–395, 2010
3. Available at: http://www.bioterrorism.slu.edu
4. American Association of Critical Care Nurses: Statement of commitment on mass casualty and bioterrorism preparedness. Available at: http://www.aacn.org
5. General Accounting Office (GAO): Report to the Congressional Committees. Disaster Preparedness: Preliminary observations on the evacuation of vulnerable populations due to hurricanes and other disasters. GAO- 06-790T, May 2006
6. Rubinson L, Nuzzo J, Talmor D, et al: Augmentation of hospital critical care capacity after bioterrorist attacks or epidemics: Recommendations of the Working Group in Emergency Mass Critical Care. Crit Care Med 33(10):E1–E13, 2005
7. American College of Radiology: ACR Disaster Planning Task Force—2002. Disaster Preparedness for Radiology Professional Response to Radiological Terrorism, Version 2.0
8. American College of Radiology: ACR Disaster Planning Task Force—2002
9. Linnemann RE: Managing Radiation Medical Emergencies. Philadelphia, PA: Radiation Management Consultants, 2001
10. Los Angeles County EMS and Public Health Agencies: Terrorism Agent Information and Treatment Guidelines for Clinicians and Hospitals, 2003
11. Hudson T, Reilly K, Dulagh J: Considerations for chemical decontamination shelters. Disaster Management and Response 1(4):110–113, 2003
12. Association for Professionals in Infection Control and Epidemiology (APIC) and Centers for Disease Control and Prevention (CDC): Chemical-biological readiness plan: A template for healthcare facilities. ED Manag 11:1–16, 1999
13. Blake N, Stevenson K, England D: H1N1 Pandemic: Life Span Considerations. AACN Adv J Crit 20(4):334–341, 2009
14. Mitchell AM, Sakraida TJ, Zalice KK: Disaster care: Psychological considerations. Nurs Clin North Am 40(3):535–550, 2005

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

PART 5

CARDIOVASCULAR SYSTEM

16

Anatomy and Physiology of the Cardiovascular System

Patricia Gonce Morton

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Briefly describe the characteristics of cardiac muscle cells.
2. Differentiate the electrical events from the mechanical events in the heart.
3. Explain depolarization and repolarization.
4. Describe the normal conduction system of the heart.
5. Explain the formula for calculating cardiac output.
6. Compare and contrast the role of the parasympathetic and sympathetic nervous systems in the regulation of heart rate.
7. Explain the three factors involved in the regulation of stroke volume.
8. Describe the coronary artery blood source for the cardiac chambers and conduction system.
9. Discuss the influence of blood volume and blood pressure on peripheral circulation.

During the 70 years in the life of the average person, the heart will pump approximately 70 times per minute, 24 hours a day, 365 days a year. The heart pumps about 5 quarts of blood a minute, 75 gallons an hour, and 1,800 gallons in a day.[1] Although the work accomplished by this organ is out of proportion to its size, for most people, the heart functions normally throughout the life span. The pumping action of the heart moves blood, a vital substance, throughout the body, supplying oxygen and nutrients to cells and removing waste. Without this action, cells die. For people in whom cardiac problems develop, the results may be dramatic and the outcome drastic. This chapter reviews the principles of cardiovascular anatomy and physiology.

▲ Cardiac Microstructure

Microscopically, cardiac muscle contains visible stripes, or striations, similar to those found in skeletal muscle (Fig. 16-1). The ultrastructural pattern also resembles that of striated muscle. The cells branch and connect freely and form a three-dimensional, complex network. The elongated nuclei, like those of smooth muscle, are found deep in the interior of the cells and not next to the cell membrane as they are in striated muscle.

Cardiac muscle (myocardial) cells are endowed with extraordinary characteristics, most of which belong to the cell membrane or sarcolemma. To pump effectively, the heart muscle must begin contraction as a single unit. To contract myocardial cells simultaneously, cell membranes must depolarize at the same time. The heart does this, without using much neural tissue, by rapidly conducting impulses from cell to cell through intercalated disks. At each end of every myocardial cell, adjacent cell membranes are folded elaborately and attached strongly. These areas comprise the intercalated disks, where depolarization is conducted extremely rapidly from one cell to the next (see Fig. 16-1).

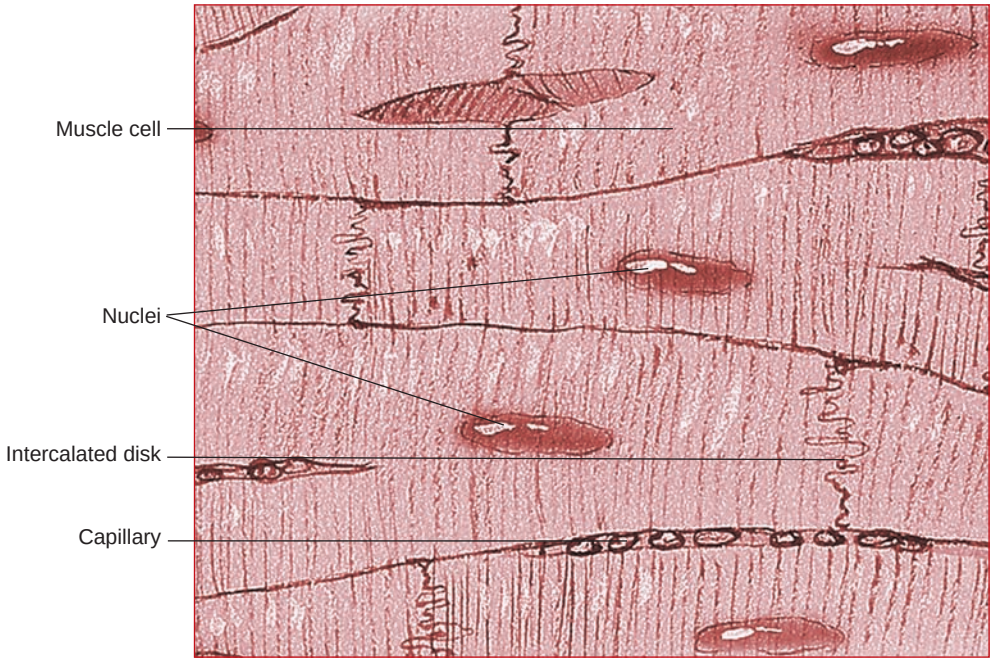


FIGURE 16-1 ▲ Cardiac muscle fibers, showing the branching structure and intercalated disks. (From Anatomical Chart Company: Atlas of Human Anatomy. Springhouse, PA: Springhouse, 2001, p 167.)

Another extraordinary characteristic of myocardial cells, seen mainly in cell membranes, is automaticity. Selected groups of cardiac cells are capable of initiating rhythmic action potentials, and thus waves of contraction, without any outside humoral or nervous intervention. Automaticity and other terms used to describe cardiac tissue functions are listed in Box 16-1.

Within each cardiac cell lie thousands of contractile elements, the overlapping actin and myosin filaments. Figure 16-2 illustrates these elements and the changes seen during diastole and systole. Not shown are the many cross-bridges that extend like rows of oars from the surface of the thicker myosin filaments. During diastole, these bridges are unattached to other filaments. The arrangement of actin and myosin filaments gives cardiac muscle its banded or striated appearance. One grouping of actin and myosin filaments is called a *sarcomere*.

BOX 16-1 Terms Used to Describe Cardiac Tissue Function

Automaticity: the ability of specialized cells in the heart known as pacemaker cells to spontaneously generate an action potential, thus causing depolarization
Conductivity: the ability of cardiac cells to conduct action potentials, thus transmitting the electrical signal from one cell to another
Contractility: the ability of cardiac muscle to shorten in response to depolarization
Excitability: the ability of cardiac tissue to respond to a stimulus and generate an action potential
Rhythmicity: the ability of cardiac cells to spontaneously generate an action potential at a regular rate

▲ Mechanical Events of Contraction

Before mechanical contraction, an action potential travels quickly over each cell membrane and down into each cell's sarcoplasmic reticulum. When an action potential causes depolarization of the sarcoplasmic reticulum, calcium ions move from the sarcoplasmic reticulum into the myocardial cell cytoplasm and bind to troponin molecules on actin filaments. Calcium-bound troponin moves slightly to uncover binding sites on the actin, to which myosin filaments then attach. With a release of energy stored in adenosine triphosphate (ATP), these binding sites move so that actin and myosin slide past each other and new couplings between actin and myosin occur. Rapid, successive uncoupling of cross-bridges and their reattachment to new actin-binding sites lead to rapid and dramatic shortening of the sarcomere (see Fig. 16-2). This shortening is the essence of myocardial contraction (systole). Contraction ceases when the calcium ions return to their storage sites on the sarcoplasmic reticulum, thereby causing the binding sites on the actin filaments to be covered again. The separated actin and myosin filaments then slip past each other in the reverse direction, lengthening the sarcomere to its relaxed state.

Contraction requires calcium and energy. The presence of adequate ATP stores and the movement of calcium provide the essential link between the electrical events of depolarization and the mechanical events of contraction in the heart.

▲ Electrical Events of Depolarization

Membranes of all the cells in the human body are charged, that is, they are polarized and therefore have electrical

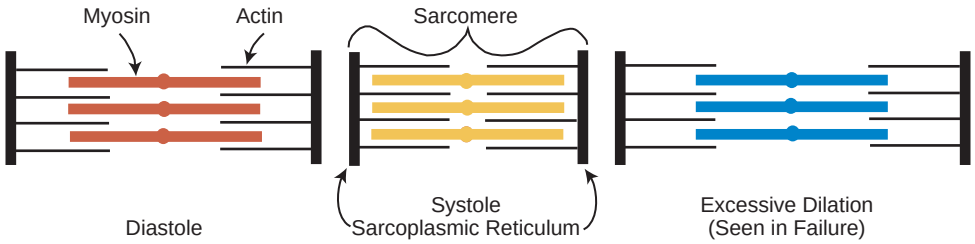


FIGURE 16-2 ▲ Contractile elements lying inside a single sarcomere of a myocardial cell.

potentials. The charges are separated at the membrane. In humans, all cell membranes, regardless of type, are positively charged at rest, with more positively charged particles at the outer surface of the cell membrane than at the inner surface. Figure 16-3A illustrates this "resting stage."

In the depolarized state, the cell membrane is negatively charged, with more negatively charged particles at the outer surface of the cell membrane than at the inner surface. Figure 16-3B illustrates this "depolarized stage." *Excitability* is the term used to describe the ability of a cell to depolarize in response to a given stimulus.

Cardiac muscle membranes are polarized, and the electrical potential can be measured, as it can in any of the cells in the human body. The potential results from the difference between intracellular and extracellular concentrations of electrolytes. When salt compounds of various elements are dissolved in aqueous solutions, they dissociate into their charged particles, called *ions*.

In the resting myocardial cell, there are more potassium ions inside than outside the cell and more sodium and unbound calcium ions outside than inside the cell. All three of these cations (positively charged ions) may diffuse through pores, or channels, in the cell membrane. If each ion freely obeyed the law of diffusion, however, potassium would diffuse out of the cell, whereas sodium and calcium would diffuse into it. Very soon there would be equal concentrations of each ion between the intracellular and extracellular fluids, and no resting potential would exist. It is through selective regulation of the concentrations of these ions on either side of the membrane that the resting membrane potential is maintained. Several factors contribute to this regulation. The first factor is the presence of sodium–potassium "pumps" in the cell membrane. These pumps move sodium out of the cell and potassium into the cell, with both movements occurring against the concentration gradients for each of these ions. The second factor is the active movement of calcium out of the cell against the concentration gradient in response to the passive diffusion of sodium into the cell. The third factor is the regulation of membrane channels, whereby calcium ions can enter the resting myocardial cell. The fourth factor is the presence of intracellular anions (negatively charged particles) that are too large to exit from the cell.

▲ Physiological Basis of the Resting Potential

The cardiac cell contains large anions that cannot exit the cell. These anions attract sodium and potassium cations, which diffuse through membrane channels into the cell. The anions would attract the calcium cation also, except that the membrane channels for the entry of this ion are closed when the cell is at rest. The potassium ions remain within the cell, but the sodium ions are pumped out of the cell almost as fast as they can enter by the sodium–potassium pumps located in the cell membrane. While forcing sodium out of the cell, these pumps actively transport potassium ions into the cell against their concentration gradients. This increase in intracellular potassium still is insufficient to offset all the intracellular anions. Thus, the inside of the myocardial cell remains negative with respect to the outside—as long as the pumps are operative. As a result, the resting potential is approximately −80 mV. For each molecule of an ion pumped from the cell, one molecule of ATP is required to provide the energy necessary to effect the chemical bond between ion and carrier. Maintaining a resting potential thus requires energy. Factors that maintain resting membrane potential of myocardial cells are listed in Box 16-2.

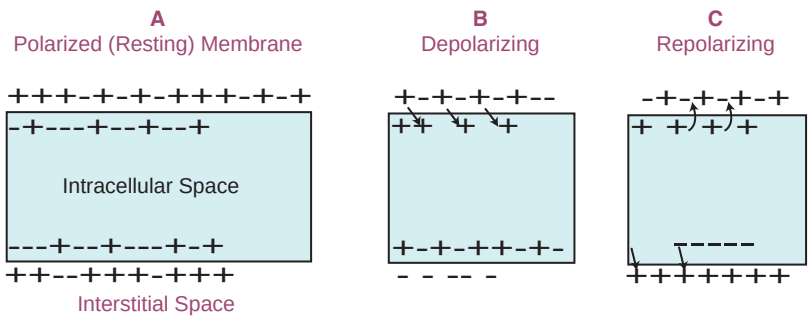


FIGURE 16-3 ▲ Electrical events at rest (diastolic) and preceding contraction (systolic).

BOX 16-2 Factors That Maintain Resting Membrane Potential of Myocardial Cell

- Sodium–potassium pumps within the cell membrane
- Active movement of Ca^{2+} out of cell against its concentration gradient in response to passive diffusion of Na^{+} into cell
- Regulation of membrane channels so that Ca^{2+} ions can enter resting myocardial cell
- Presence of intracellular anions too large to exit from cell

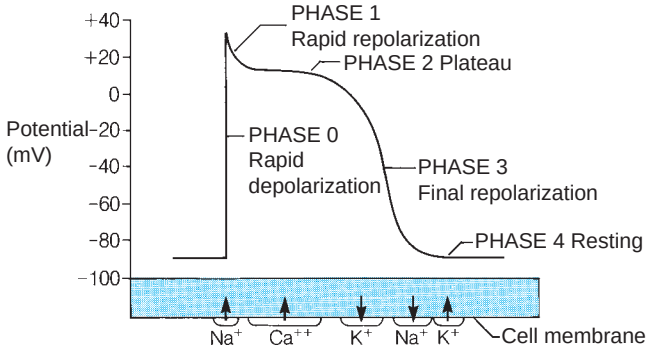


FIGURE 16-4 ▲ Cardiac action potential. Phase 0 is the rapid depolarization phase. During this phase, the fast sodium channels in the cell membranes are stimulated to open, resulting in the rapid influx of sodium. Contraction of the myocardium follows depolarization. Phase 1 is the rapid repolarization phase and occurs at the peak of the action potential. This phase indicates the inactivation of the fast sodium channels with an abrupt decrease in sodium permeability. Phase 2 represents the plateau of the action potential. During this phase, potassium permeability is low, allowing the membrane to remain depolarized throughout phase 2. The influx of calcium that occurs during the plateau phase is much slower than that of sodium and lasts for a longer time. Phase 3 is the final repolarization phase and begins with the downslope of the action potential curve. During this phase, the influx of calcium and sodium ends, and there is a rapid outward movement of potassium. By the end of phase 3, sodium and potassium return to their normal resting state. Phase 4 is the resting membrane potential and corresponds to diastole. During this phase, the sodium–potassium pump is activated, resulting in the active transport of sodium out of the cell, and potassium is moved back into the cell. The *arrows* below the diagram indicate the approximate time and direction of movement of each ion influencing membrane potential. The phase of calcium moving out of the cell is not well defined but is thought to occur during phase 4.

▲ Physiological Basis of the Action Potential

When a stimulus is applied to the polarized cell membrane, the membrane that ordinarily is only slightly permeable to sodium permits sodium ions to diffuse rapidly into the cell. This rapid diffusion occurs because of inactivation of the sodium active transport enzymes (pumps). The result is a reversal of net charges. The outer surface is now more negative than positive, and the membrane is said to be depolarized (see Fig. 16-3B).

When the sodium influx changes the polarity from −80 mV to approximately −35 mV, the electrical change opens the previously closed "calcium channels" in the myocardial cell membrane. Once opened, these channels permit the influx of calcium. The entry of this cation, together with the continued entry of sodium, is responsible for the remainder of the depolarization, which continues until the polarity of the extracellular side equals approximately +30 mV. Such a maximal depolarization inactivates sodium–potassium pumps in nearby membranes. This can cause depolarization in these areas. When the original depolarization becomes self-propagating in this way, it is termed an *action potential*. In a myocardial cell, an action potential triggers the release of intracellular calcium from its storage sites on the sarcoplasmic reticulum. This release plus the calcium influx across the sarcolemma elevates intracellular calcium levels, thereby initiating muscular contraction, as previously described.[1]

If the depolarization remains below a certain critical (threshold) point, it dies out without having opened any calcium channel or inactivated any adjacent sodium–potassium pumps. Because it does not become self-propagating and remains localized, such a depolarization is termed a *local depolarization*.

During depolarization, the elevated intracellular sodium concentration frees potassium ions to diffuse out of the cell in accordance with their concentration gradient. Just as this potassium efflux gains some momentum, however, the sodium–potassium pumps automatically reactivate (they can be inactivated only temporarily). Once reactivated, the pumps begin to restore the original resting potential, a process termed *repolarization* (see Fig. 16-3C). During the initial phase of repolarization, the efflux of potassium and sodium ions exceeds their influx, but as the intracellular sodium ions are removed from the cell, potassium ions remain as the major cation to be electrostatically held within the cell by the intracellular anions. This halts the potassium efflux. The remainder of repolarization consists of pump activity that increases intracellular potassium and decreases intracellular sodium; thus, the resting potential is re-established. The electrical events at the start of repolarization also reclose the calcium entry channels, thereby halting calcium influx. Intracellular calcium levels are reduced when the diffusion of sodium into the cell causes a movement of calcium out of the cell against the latter's concentration gradient.[1] The phases of the action potential are shown in Figure 16-4.

▲ Cardiac Macrostructure

The heart is about the size of a clenched fist and lies in the chest between the lungs in the mediastinal space of the intrathoracic cavity. The right side of the heart is almost entirely in front of the left side of the heart, and the right ventricle occupies most of the anterior cardiac surface (Fig. 16-5). Only a small portion of the left ventricle is in the frontal plane of the heart. The left ventricle forms the left lateral margin of the heart with a tapered inferior tip that is often termed the *cardiac apex*.[2]

The heart is made up of four layers: the endocardium, the myocardium, the epicardium, and the pericardium. The inner layer, known as the *endocardium*, consists of endothelial

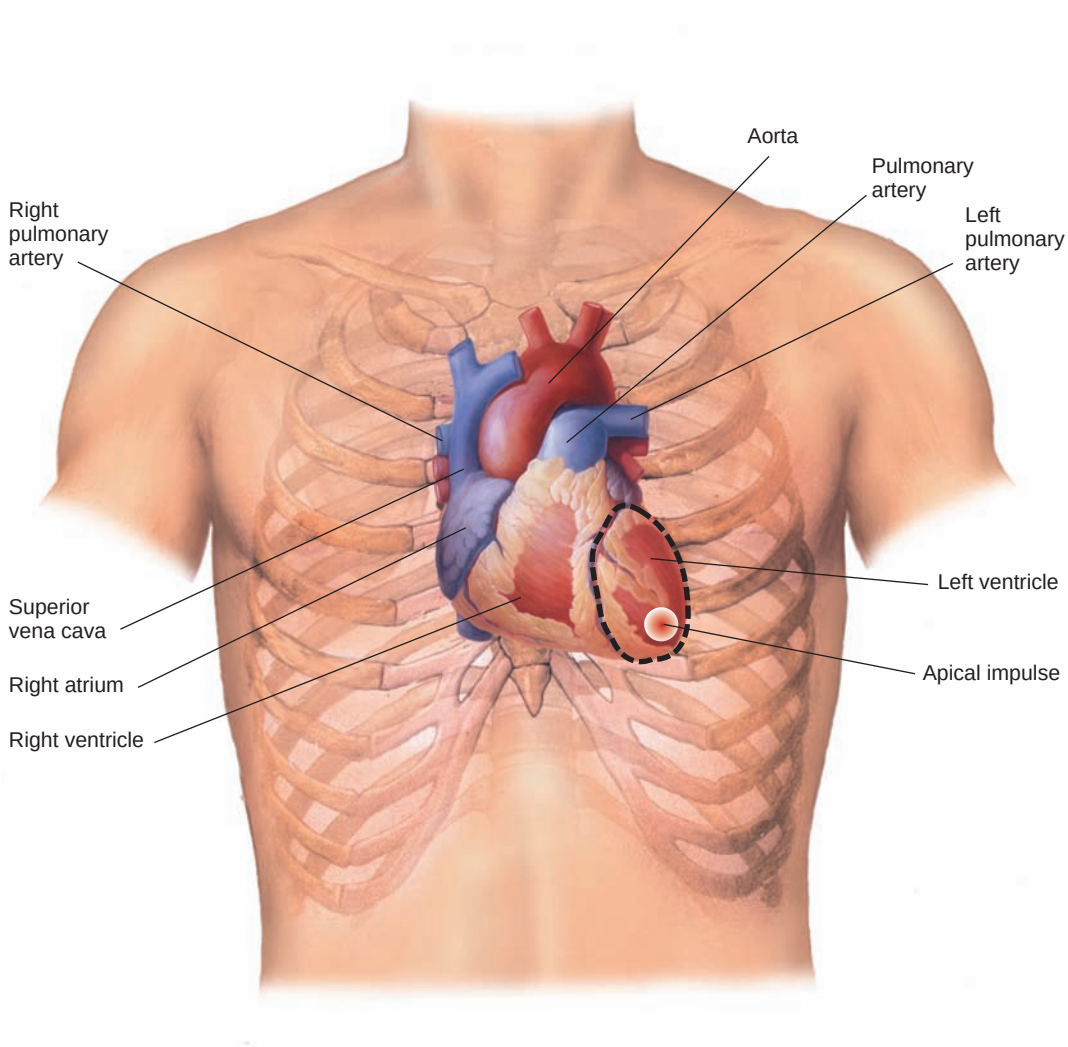


FIGURE 16-5 ▲ Structure of the heart. (From Bickley LS: Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 324.)

tissue that lines the inner surface of the heart and the cardiac valves. The middle layer, known as the *myocardium*, is composed of muscle fibers that enable the heart to pump. The outer layer, known as the *epicardium*, is tightly adherent to the heart and the base of the great vessels. A thin, fibrous, double-layered sac, known as the *pericardium*, surrounds the heart. This structure has two parts: an outer layer called the *parietal pericardium* and the inner layer called the *visceral pericardium*. Between these two layers is a small amount of pericardial fluid (30 to 50 mL) that serves as a lubricant between the two layers.[1]

The heart consists of four chambers: right and left atria, and right and left ventricles. The atria are smaller, thinner-walled, low-pressure chambers. Approximately 30% of blood flow to the ventricles is the result of atrial contraction, also known as *atrial kick*. The remaining 70% of blood that reaches the ventricles is the result of pressure differences between the atria and the ventricles. The ventricles are larger, higher-pressure chambers with thicker walls than the atria. The walls of the left ventricle are thicker than the right ventricle because the left ventricle must generate a large amount of force to eject blood into the aorta. Deoxygenated blood enters the right atrium from the superior and inferior venae cavae. The blood passes through the tricuspid valve into the right ventricle, which then pumps the blood through the pulmonic valve into the pulmonary circulation. After gas exchange in the lungs, oxygenated blood returns to the left atrium, passes through the mitral valve, enters the left ventricle, passes through the aortic valve, and finally enters the aorta (Fig. 16-6).

The cardiac valves are composed of fibrous tissue and allow blood to flow in one direction. The valves open and close as a result of blood flow and pressure differences. The tricuspid and mitral valves are known as the *atrioventricular (AV) valves* because they are located between the atria and the ventricles. The chordae tendineae and the papillary muscles attach to the AV valves and help maintain closure and prevent eversion of the valve leaflets during ventricular contraction so that blood does not move into the atria. The pulmonic and aortic valves are known as the *semilunar valves* because each has three leaflets shaped like half-moons.

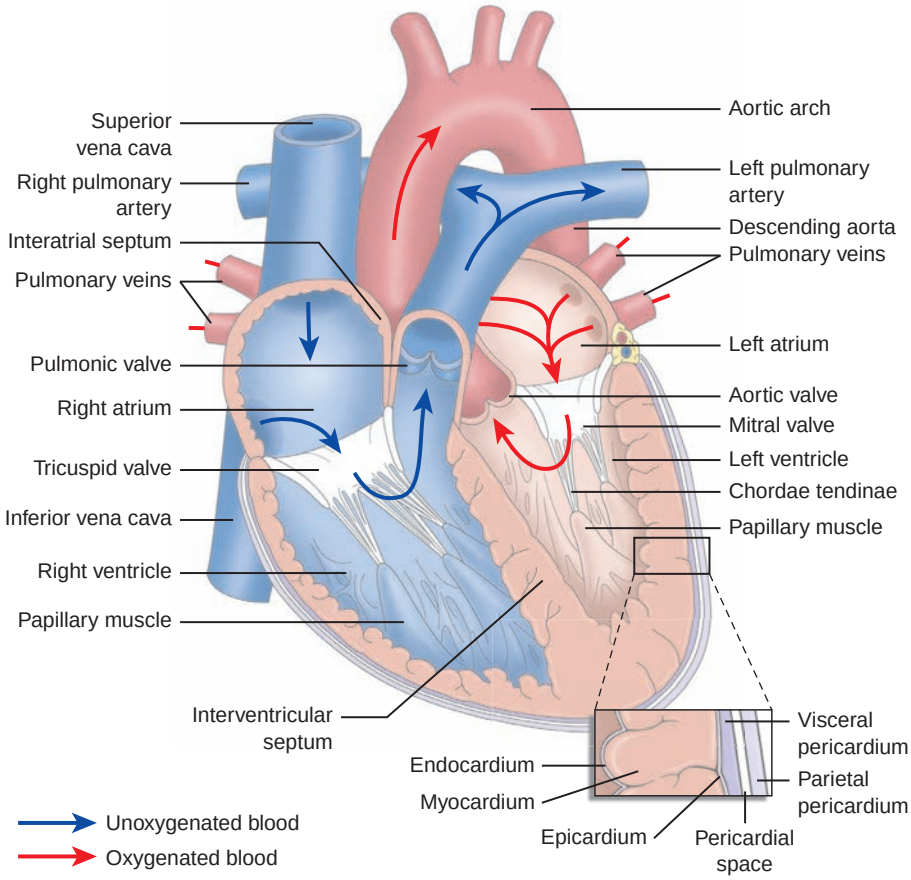


FIGURE 16-6 ▲ Structure of the heart. *Arrows* show course of blood flow through the heart chambers. (From Smeltzer SC, Bare BG, Hinkle JL, et al: Textbook of Medical–Surgical Nursing, 12th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 685.)

▲ Cardiac Conduction

To pump effectively, large portions of cardiac muscle must receive an action potential nearly simultaneously. Special cells that conduct action potentials extremely rapidly are arranged in pathways through the heart. All these cells have automaticity (see Box 16-2, p. 196).

The heart chambers and specialized tissues are diagrammed in Figure 16-7. The sinoatrial (SA) node is located between the opening of the inferior and superior venae cavae in the right atrial wall. The cells of the SA node have the property of automaticity. Because the SA node normally discharges faster than any other heart cell with automaticity (60 to 100 beats/min), this specialized tissue acts as a normal cardiac pacemaker. Atrial action potentials travel through atrial cells by intercalated disks, although some specialized conductive tissue in the atria has been discovered.

In the lower right portion of the interatrial septum is the AV node, also known as the AV junction. This tissue conducts, yet delays, the atrial action potential before it travels to the ventricles. Action potentials reach the AV node at different times. The AV node slows conduction of these action potentials until all potentials have exited the atria and entered the AV node. After this slight delay, the AV node passes the action potential all at once to the ventricular conduction tissue, allowing for nearly simultaneous contraction of all ventricular cells. This AV node delay also allows time for the atria to eject fully their load of blood into the ventricles in preparation for ventricular systole.

From the AV node, the impulse travels down the bundle of His in the interventricular septum into either a right or left bundle branch and then through one of many Purkinje fibers to the ventricular myocardial tissue itself. An action potential can traverse this conducting tissue three to seven times more rapidly than it can travel through the ventricular myocardium. Thus, the bundle branches and Purkinje fibers enable a near-simultaneous contraction of all portions of the ventricle, thereby allowing a maximal unified pump action to occur.[1]

Electrocardiograms

Conduction of an action potential through the heart can be shown by an electrocardiogram (ECG; Fig. 16-8). Because ECGs are extensively covered in Chapter 17, discussion here is brief. An ECG does not show mechanical events of the heart, but in the normal heart, coupling of electrical and mechanical events can be assumed (see Chapter 17).

In Figure 16-8, point 1 shows early ventricular diastole, when the atria and ventricles are at rest. Blood from the large veins is passively filling both atria. As the atria fill, the pressure in the atria exceeds the pressure in the ventricles, and the AV valves open in response to the pressure gradient. The blood from the atria now passively fills the ventricles.

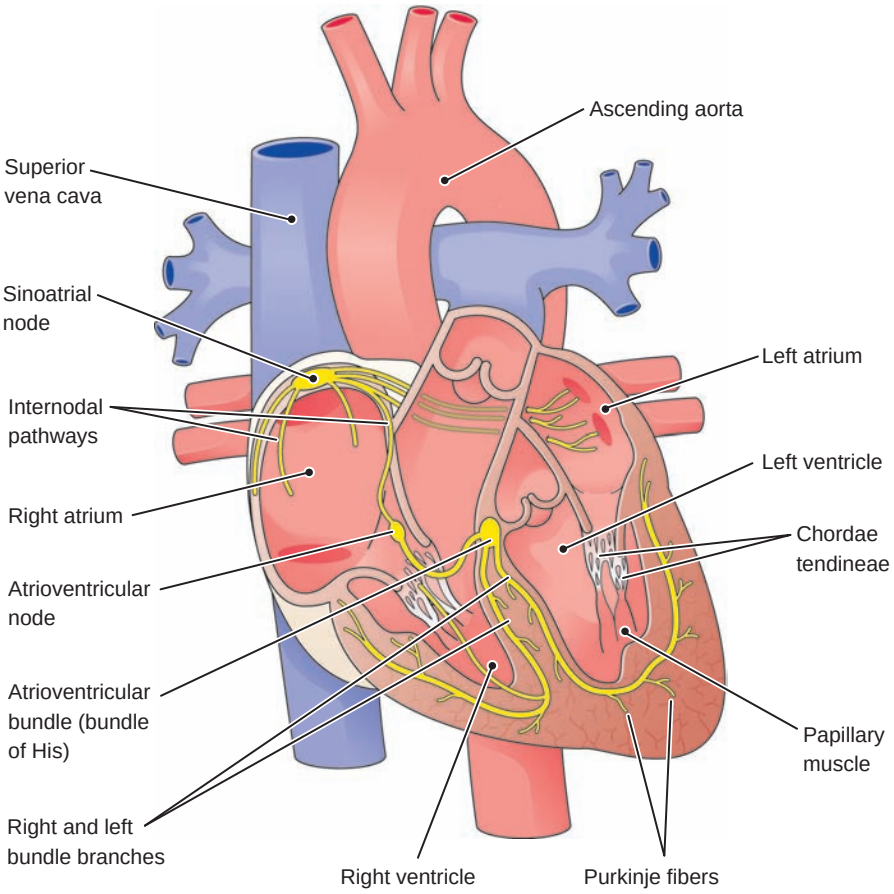


FIGURE 16-7 ▲ The electrical conduction system of the heart begins with impulses generated by the sinoatrial node (*yellow*) and circuited continuously over the heart. (From Weber J, Kelley K: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, 352.)

At point 2, the beginning of late ventricular diastole, both ventricles remain relaxed and are about three fourths full. The SA node fires spontaneously (due to automaticity), and both atria depolarize, generating a P wave. The atria contract, and blood is actively moved from the atria into the ventricles: this "atrial kick" supplies approximately 20% to 30% of the ventricular blood volume.

At point 3, late in the PR interval, the action potential begun in the SA node is being delayed and "collected" in the AV node and travels to the bundle of His. The atria and ventricles are at rest.

At point 4, the action potential moves to the septum, which depolarizes and leads to the Q wave. Septal depolarization is rapidly followed by action potential movement down the right and left bundles into the Purkinje fibers to all cardiac muscle cells. These electrical events are seen as the RS wave on the ECG and are followed rapidly by mechanical contraction of both ventricles. The AV valves close, and the aortic and pulmonic valves open.

At point 5, the heart returns to early ventricular diastole, and the ventricles repolarize. This repolarization shows as a large, wide T wave. The aortic and pulmonic valves close about midway through repolarization.[1]

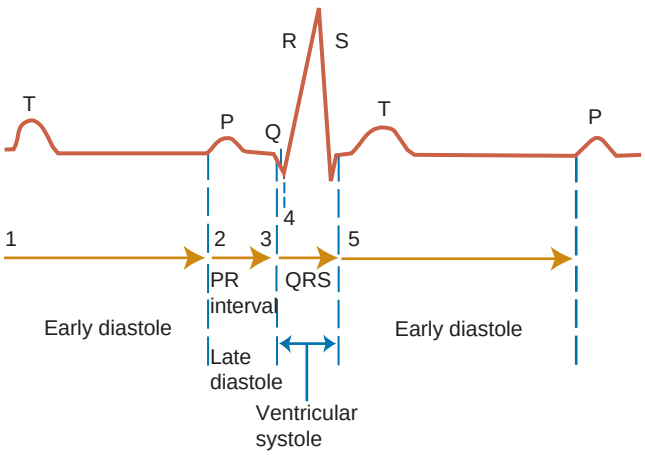


FIGURE 16-8 ▲ Comparison of electrical and mechanical events during one cardiac cycle, using a normal electrocardiogram tracing.

Rhythmicity and Pacing

Automaticity is an inherent property of myocardial conduction cells and occurs as a result of a spontaneous and rhythmic inactivation of the sodium pumps. Under abnormal conditions, cardiac muscle cells also gain automaticity and can produce their own rhythmic series of action potentials and thus their own stimulus for contraction. Coordination of automaticity is important for rhythmic cardiac contraction and is achieved through the varying rates of automaticity found in different cardiac tissues.

The SA node discharges normally in an adult at a resting rate of 60 to 100 times per minute. The remainder of the conduction system and ventricles have progressively slower rates of firing.

The AV node discharges at a rate of 40 to 60 times per minute. The conduction tissues in the ventricles fire about 20 to 40 times per minute. The group of cells with the fastest rate of automaticity paces the heart. Normally, this is the SA node.

If the SA node should fail to fire, a pacemaker site lower in the heart should take control because of the automaticity of cardiac tissue. This new pacemaker site is usually the AV node; however, the heart rate (HR) will most likely be slower. If conduction from the SA node is blocked (unable to pass through the AV node), the fastest pacemaker tissue on both sides of this interruption will govern their respective areas, and the ECG may show independent atrial and ventricular rhythms. Atrial systole is not needed for the ventricle to fill with blood because most ventricular filling is passive and occurs in early diastole. The clinically important rhythm is that of the ventricles; they are the chambers that supply the lungs and the rest of the body with blood. Their systolic rate helps determine true perfusion. The slower the rate, the less able are the ventricles to meet the perfusion needs of the body during exercise or activities of daily living. A very rapid ventricular rhythm also compromises perfusion needs because the shorter the diastole, the less time for filling of the chambers. Decreased ventricular filling reduces cardiac output (CO).

▲ Cardiac Output

A traditional measure of cardiac function, CO is the amount of blood, in liters, ejected from the left ventricle each minute. Cardiac output (CO) is the product of heart rate (HR) and stroke volume (SV), which is the volume of blood ejected per ventricular contraction:

$$CO = HR(\text{beats/min}) \times SV(\text{L/beat})$$

Normal CO for an adult ranges from 4 to 8 L/min. The output can be altered to meet changing bodily demands for tissue perfusion, but the CO equation does not account for differences in body size. An output of 5 L/min may be adequate for a 50-kg man but insufficient for a 120-kg man. Because perfusion is a function of body size, a more accurate measure of cardiac function is cardiac index (CI), which represents the amount of blood, in liters, ejected each minute from the left ventricle (or CO) per square meter of body surface area. CI typically averages 3.0 ± 0.2 L/min and ranges from 2.8 to 4.2 L/min/m^2:

$$CI = CO\ (\text{L/min}) / \text{body surface area}\left(m^2\right)$$

Regulation of Heart Rate

Although the heart has the ability to beat independently of any extrinsic influence, cardiac rate is under autonomic and adrenal catecholamine influence. Parasympathetic and sympathetic fibers innervate the SA and AV nodes. In addition, some sympathetic fibers terminate in myocardial tissues.

Parasympathetic stimulation releases acetylcholine near the nodal cells and decreases the rate of depolarization, thereby slowing cardiac rate. Stimulation of sympathetic fibers causes the release of norepinephrine. This chemical increases the rate of nodal depolarization and has inotropic effects on myocardial fibers, which are discussed later. Thus, sympathetic stimulation increases HR (Table 16-1). The adrenal medulla also releases norepinephrine and epinephrine into the bloodstream. These circulating catecholamines act on the heart in the same way as sympathetic stimulation.

Table 16-1 α and β Effects of Autonomic Nervous System on the Heart and Vascularity

		Noradrenergic Impulses	
Effector Organ	**Cholinergic Impulses Response**	**Receptor Type**	**Response**
Heart			
Sinoatrial (SA) node	Decrease in heart rate (HR); vagal arrest	β_1	Increase in HR
Atria	Decrease in contractility and (usually) increase in conduction velocity	β_1	Increase in contractility and conduction velocity
Atrioventricular (AV) node and conduction system	Decrease in conduction velocity; AV block	β_1	Increase in conduction velocity
Ventricles	—	β_1	Increase in contractility and conduction velocity
Arterioles			
Coronary, skeletal muscle, pulmonary, abdominal viscera, renal	Dilation	α β_2	Constriction Dilation
Skin and mucosa, cerebral, salivary glands	—	α	Constriction
Systemic Veins			
	—	α β_2	Constriction Dilation

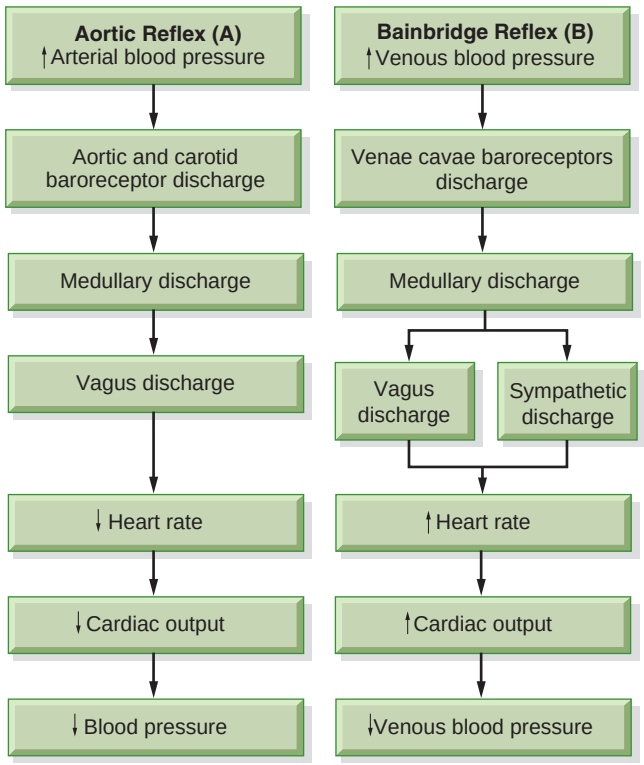


FIGURE 16-9 ▲ Effects of aortic reflex (**A**) and Bainbridge reflex (**B**) on heart rate.

Two reflexes adjust HR to blood pressure: the aortic reflex and the Bainbridge reflex. In the aortic reflex (Fig. 16-9A), a rise in arterial blood pressure stimulates aortic and carotid sinus baroreceptors to fire sensory impulses to the cardioregulatory center in the medulla. The result is an increase in parasympathetic stimulation or a decrease in sympathetic stimulation to the heart. Thus, a rise in arterial blood pressure reflexively causes a slowing of cardiac rate. The decrease in HR results in a decrease in output, which can decrease arterial blood pressure. Conversely, a fall in arterial blood pressure, such as in shock, reflexively increases HR. This aortic reflex is an ongoing regulatory mechanism for homeostasis of arterial blood pressure.

The Bainbridge reflex (see Fig. 16-9B) uses receptors in the venae cavae. An increase in venous return stimulates these receptors, which then fire sensory impulses that travel to the cardioregulatory center. These reflexively cause a decrease in parasympathetic cardiac stimulation and an increase in sympathetic cardiac stimulation, thereby increasing cardiac rate. A fall in venous return causes a decrease in HR. Thus, the Bainbridge reflex adjusts cardiac rate to handle venous return.

Regulation of Stroke Volume

SV is the amount of blood ejected by the left ventricle during systole. Normal values range from 60 to 100 mL/beat. Three factors are involved: preload, afterload (or wall tension), and inherent inotropic myocardial contractility.

Preload

Preload is the amount of stretch placed on a cardiac muscle fiber just before systole. Usually, the amount of stretch in any chamber is proportional to the volume of blood the chamber contains at the end of diastole, before systole. However, in some situations, the chamber can hold a large amount of volume with little change in pressure.

The concept of preload is related to the Frank-Starling law of the heart, which states that the force of myocardial contraction is determined by the length of the muscle cell fibers (Fig. 16-10). Within a certain range, increasing myofibril stretch increases the force of systole. Beyond optimal fibril length, it is hypothesized that too few actin–myosin binding sites overlap to provide an adequate contraction. Below optimal shortening, there is little room for filaments to slide, and cell walls limit further sliding. Also, actin filaments may have begun to overlap, decreasing the number of binding sites available to myosin fibers.

When the force of systole decreases, the chamber pumps poorly and does not empty properly. Excessive blood is left in the chamber at the end of systole. During diastole, when the chamber fills, this extra blood causes overfilling of the chamber and increases stretch. The next systole will be even weaker, as preload increases during every diastole.

Because preload is affected by the volume at the end of diastole, it often is equated with end-diastolic volume or pressure. Thus, left ventricular preload is represented by left ventricular end-diastolic pressure.

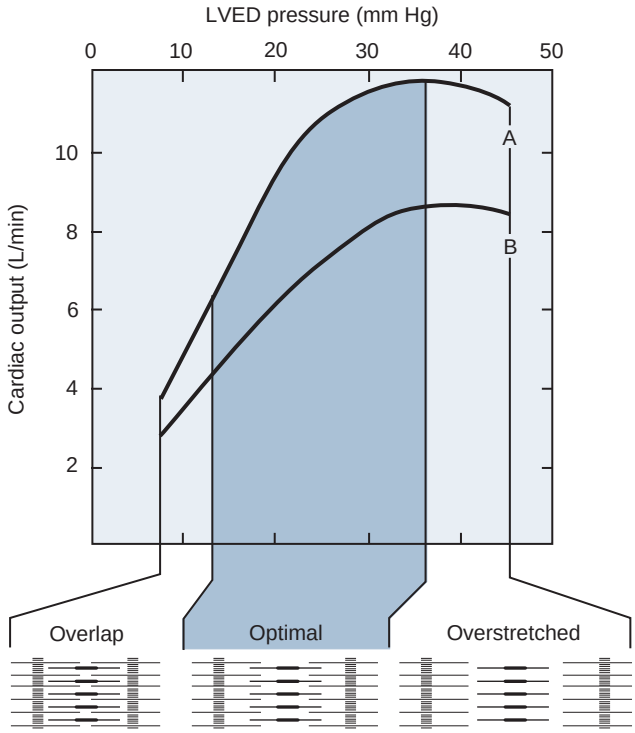


FIGURE 16.10 ▲ **Top:** Starling ventricular function curve in normal heart. An increase in left ventricular end-diastolic (LVED) pressure produced an increase in cardiac output (CO) *(curve B)* by means of the Frank-Starling mechanism. The maximal force of contraction and increased stroke volume are achieved when diastolic filling causes the muscle fibers to be stretched about 2-1/2 times their resting length. In *curve A,* an increase in cardiac contractility produces an increase in CO without a change in LVED volume and pressure. **Bottom:** Stretching of the actin and myosin filaments at the different LVED filling pressures. (From Porth CM: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 464.)

An example of rapid and normal adjustments to changes in preload occurs during the Valsalva maneuver. The first part of the Valsalva occurs when one holds one's breath and bears down, such as during defecation or heavy lifting. Bearing down increases intra-abdominal and intrathoracic pressures, decreasing venous return to the right atrium and ventricle. Right heart preload decreases. Bearing down also stimulates the vagus nerve, and the HR slows.

On exhalation, during the second part of the Valsalva maneuver, intrathoracic pressures decrease rapidly, allowing a sudden increase in venous return. Right atrial and ventricular preloads increase dramatically, stretch increases, and the right ventricular SV increases. Atrial stretch receptors also signal the medulla and lead to sympathetic nervous discharge. HR increases.

Afterload

Afterload is the force or pressure against which a cardiac chamber must eject blood during systole. The most critical factor determining afterload is vascular resistance, in the systemic or pulmonic vessels. Afterload often is equated with systemic vascular resistance or pulmonary vascular resistance.

Afterload affects SV by increasing or decreasing the ease of emptying a ventricle during systole. A decrease in systemic vascular resistance, through vasodilation, presents the left ventricle with relatively large, open, relaxed arteries into which it can pump. Because it is easier to pump, the left ventricle empties easily, which increases SV.

If systemic vascular resistance increases, for example through catecholamine-induced constriction of arteries, it takes a great deal more force for the left ventricle to pump into such a tightened vasculature. SV decreases.

Contractility

Inotropic capabilities and cardiac workload refer to contractile forces. Cardiac muscle forces change in response to neural stimuli and circulating levels of catecholamines. It is thought that through cyclic adenosine monophosphate mechanisms, cardiac cells change intracellular levels of calcium and ATP. These changes lead to increased inotropic actions, although the mechanisms remain unknown.

However, increased inotropic action increases the oxygen consumption of heart cells. This increased consumption also is called *increased workload* and *increased oxygen demand*.

CO depends on HR and SV. Regardless of the initial cause of increased SV (increased preload, increased afterload, or increased inotropic force), an increase in SV increases workload. Similarly, an increased HR, no matter what the cause, increases oxygen demand.

▲ Coronary Circulation

Blood supply to the myocardium is derived from the two main coronary arteries, the left and the right (Fig. 16-11). These arteries originate from the aorta, immediately above the aortic valve. The left main coronary artery has two major branches known as the left anterior descending (LAD) and the left circumflex artery (LCA). The LAD passes down the anterior wall of the left ventricle toward the apex of the myocardium. The LAD supplies blood flow to the anterior two thirds of the ventricular septum, the anterior left ventricle, the apex, and most of the bundle branches (Table 16-2).

The LCA, the other branch of the left main coronary artery, sits in the groove between the left atrium and the left ventricle and wraps around the posterior wall of the heart. The LCA supplies blood flow to the left atrium, the lateral wall of the left ventricle, and the posterior wall of the left ventricle. In about 10% of the population, the LCA is the source of blood flow to the posterior descending coronary artery; when this pattern of flow occurs, the patient is referred to as left dominant. Branches of the LCA provide blood flow to the SA node in about 45% of people and to the AV node in about 10% of people.

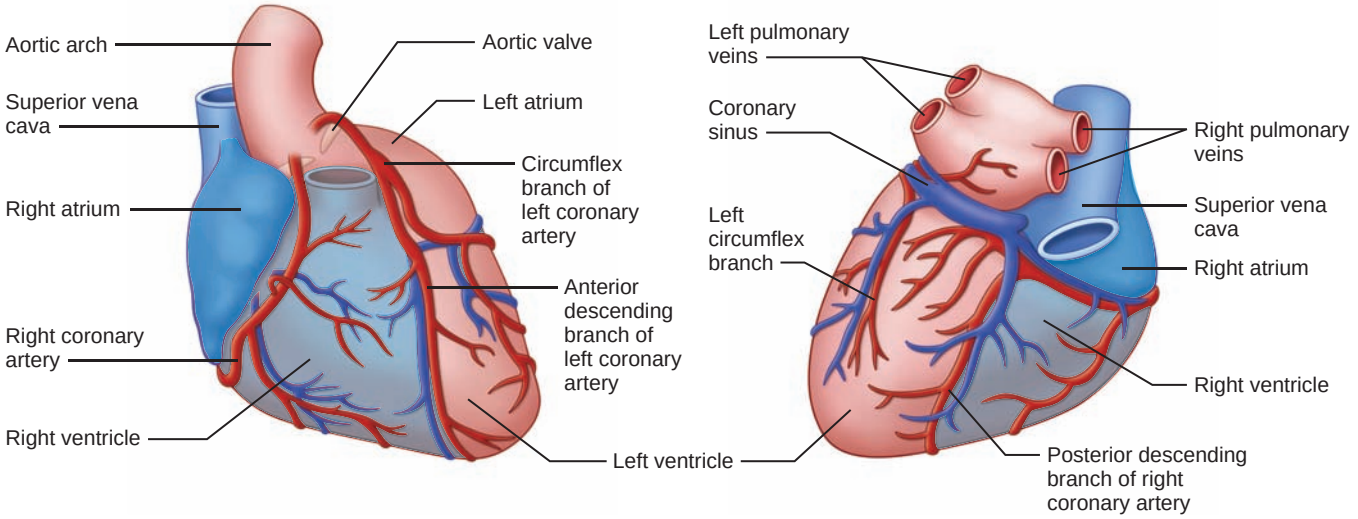


FIGURE 16-11 ▲ Coronary arteries and some of the coronary sinus veins. (Adapted from Porth CM: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 547.)

Table 16-2 Coronary Artery Blood Supply for Cardiac Muscle and Conducting System

Coronary Artery	Cardiac Muscle Supplied	Conducting Tissue Supplied
Left Main Coronary Artery		
Left anterior descending	Anterior ventricular septum Anterior left ventricle The apex	Bundle branches
Left circumflex	Left atrium Left ventricular lateral wall Left ventricular posterior wall	SA node in 45% of hearts AV node in 10% of hearts
Right Coronary Artery		
	Right atrium Right ventricle Posterior ventricular septum Inferior wall of left ventricle	SA node in 55% of hearts AV node in 90% of hearts

The right coronary artery (RCA) also comes off the aorta and branches toward the right atrium; the anterior, lateral, and posterior regions of the right ventricle; and the posterior ventricular septum. The RCA provides blood flow to the right atrium, the right ventricle, and the inferior wall of the left ventricle. In about 90% of the population, the RCA is the source of blood flow to the posterior descending coronary artery, a pattern of flow known as right dominant. The RCA supplies oxygenated blood to the SA node in about 55% of people and to the AV node in about 90% of people.

The coronary arteries initially supply the epicardial layer of the heart and then pass deeper into the heart muscle to provide blood flow to the endocardium. As a result of this flow pattern, poor coronary blood flow initially deprives the subendocardial area of oxygenated blood. If the interruption to flow continues, the effects of decreased oxygenation expand throughout the thickness of the wall of the heart to the subepicardial surface.

Because the coronary arteries derive from the aorta (above the aortic valve) and lie between myocardial fibers, blood flow through the coronary arteries occurs when the aortic valve is closed during ventricular diastole, not systole. Therefore, anything that decreases the diastolic time (eg, tachycardia) decreases coronary perfusion.

▲ Peripheral Circulation

The biological significance of the cardiovascular system is tissue perfusion. Such perfusion supplies the body's cells with oxygen and nutrients while carrying away metabolic wastes, including carbon dioxide. Tissue perfusion is directly proportional to the rate of blood flow, which depends on several factors. One factor is the difference between the mean arterial blood pressure and right atrial pressure (usually represented by the central venous pressure [CVP]). The greater this difference, the faster the flow rate (all else being unchanged). Conversely, if arterial pressure falls or venous pressure rises, flow rate, and thus tissue perfusion, will be decreased.

Another factor affecting flow rate is vascular resistance. The relationship between vascular resistance and blood flow has two general applications. The first describes the flow rate through vessels of differing diameters (eg, arteries, capillaries). The second concerns the ongoing regulation of blood flow by means of adjustments in arteriole diameters (ie, constriction, dilation). Arteriole constriction reduces the radius, thereby increasing resistance and decreasing the flow rate. Conversely, arteriole dilation increases the flow rate.

The other two factors that can affect the flow rate normally are held constant. They are the sum of all vessel lengths and blood viscosity. Because these factors do not normally change significantly, they usually are omitted from flow rate considerations. However, their relationships are obvious. The greater the length of a vessel, the more the resistance and thus the slower the flow rate. Also, the more viscous the blood, the slower the rate of its flow. Blood viscosity is determined by the proportion of solvent (water) to solute and other particles, including blood cells and platelets. The less water and more particles that exist, the more viscous is the blood. The complete equation that describes all four factors is as follows:

$$\text{Flow rate} = \frac{\text{mean arterial pressure} - \text{central venous pressure}}{(\text{resistance} \times \text{viscosity} \times \text{vessel length})}$$

Because blood volume and pressure have such an important influence on tissue perfusion, the factors that alter and regulate them are examined.

Blood Volume

Urinary output and fluid input are the major normal mechanisms for regulating volume. If output is greater or fluid input is less, the volume is less—if all else is held constant. Factors that alter the volume of urine excreted every 24 hours include those that alter the glomerular filtration rate and the tubular reabsorption of water, with or without electrolytes. (For a more detailed explanation of these factors see Chapter 42, specifically the discussion of normal endocrine physiology that considers the antidiuretic hormone.) Pathological conditions that promote any type of fluid loss (eg, burns, severe diarrhea, osmotic diuresis) or a shift of water from the vascular to the interstitial compartment have the potential to reduce blood volume.

Blood Pressure

Because the difference between arterial and venous pressures is the driving force for blood circulation and tissue perfusion, factors that influence CVP are examined first, followed by the factors that regulate arterial blood pressure. CVP is, strictly speaking, the pressure of blood in the venae cavae just before its entry into the right atrium. CVP can be increased by an increase in blood volume (eg, intravenous fluid overload) or a decrease in the pumping ability of the heart (eg, cardiac failure). Because the pulsatile effects of the cardiac cycle are removed by capillary networks, venous pressure is recorded as an average, or mean, and reported in millimeters of mercury (mm Hg).

Arterial blood pressure is the pressure of blood in the arteries and arterioles. It is a pulsatile pressure due to the cardiac cycle, and systolic (peak) and diastolic (trough) numbers are reported in millimeters of mercury. Average or mean arterial blood pressure can be clinically useful as an indicator of average perfusion pressures.

Arterial blood pressure is regulated by the vasomotor tone of the arteries and arterioles, the amount of blood entering the arteries per systole (ie, CO), and blood volume itself. The greater the volume or output, the greater the blood pressure, and vice versa, if vasomotor tone were held constant. The normal regulation of vasomotor tone involves neural and hormonal mechanisms.

Neural regulation is mediated by the vasomotor center of the medulla oblongata. This center consists of vasopressor and depressor subdivisions. The vasomotor center receives neural input from baroreceptors in the carotid sinuses and aorta, atrial diastolic stretch receptors, the limbic system and hypothalamus, the midbrain, and pulmonary stretch receptors. In addition, the center is directly responsive to local hypoxia or hypercapnia. Neural outputs from the vasopressor center result in increased sympathetic stimulation to arterial smooth muscle cells. This increase in sympathetic stimulation results in arterial constriction and an increase in arterial blood pressure. Stimulation of the depressor area decreases such sympathetic stimulation.

Rapid adjustments in arterial blood pressure are effected primarily by the baroreceptor reflexes. An increase in the pressure on these receptors (directly by elevated blood pressure or manual compression and indirectly by increased blood volume) reflexively stimulates the depressor area. This stimulation of the depressor area results in decreased sympathetic stimulation to major arteries and the aorta, which causes a decrease in arterial blood pressure. The decreased baroreceptor stimulation caused by a fall in arterial blood pressure reflexively stimulates the pressor area and results in increased sympathetic stimulation to arterial muscles, causing a rise in arterial blood pressure. Thus, homeostasis of arterial pressure is maintained.

In orthostatic hypotension, the baroreceptor reflex is sluggish. Because arterial pressure is not elevated rapidly enough, the postural change results in a temporary decrease in brain perfusion that leads, in extreme cases, to syncope.

Other factors may alter arterial blood pressure reflexively by their influences on the vasomotor center. Nerve fibers from the limbic system and hypothalamus are believed to mediate emotionally produced alterations in blood pressure. An example of this is fainting, caused by neurally mediated vasodilation in response to the sight of blood or very bad (or good) news. Neural inputs from the midbrain and possibly from ascending spinothalamic fibers in the medulla result in the elevation in arterial pressure that initially accompanies severe pain and in the later decrease in arterial pressure that occurs when severe pain is prolonged. Lung inflation stimulates pulmonary stretch receptors. Their input to the vasomotor center reflexively decreases arterial pressure. Hypercapnia and, to a lesser extent, hypoxia of vasomotor neurons stimulate the pressor area, reflexively causing an increase in arterial pressure. Such stimuli obviously are not part of a normal daily regulatory mechanism but can operate as a normal compensatory mechanism in certain pathological situations. Elevated intracranial pressure can promote medullary hypercapnia and hypoxia. The increase in arterial pressure reflexively produced by these stimuli (Cushing's reflex) increases medullary perfusion, which can ameliorate the medullary hypoxia, hypercapnia, or both. Hormonal regulation of arterial blood pressure is effected by adrenal medullary catecholamines and the renin–angiotensin system. In the former, adrenal medullary catecholamines mimic the action of sympathetic fibers innervating the muscle layer of arteries (tunica media), causing arterial constriction and elevating arterial pressure. The renin–angiotensin system is discussed in Chapter 28. Briefly, a decreased glomerular filtration rate, which can result, for example, from a decrease in blood volume or renal perfusion, stimulates the secretion of renin from the juxtaglomerular apparatus. This stimulation of renin leads to the production of angiotensin II, which acts directly on the tunica media to promote vasoconstriction. Thus, renin elevates arterial pressure, which increases renal perfusion and glomerular filtration.

Finally, arterial blood pressure can be influenced by alterations in the level of unbound calcium in tunica media cells. Such levels are influenced by factors that open or close calcium channels in the membranes of these muscle cells. Drugs that block calcium channels ("calcium blockers") inhibit the entry of calcium into cells. Such decreased calcium influx can lower intracellular calcium levels sufficiently to decrease muscle contractility, including contractility of the heart, thereby promoting a degree of vasodilation and lowering the arterial pressure.

▲ Clinical Applicability Challenges

SHORT ANSWER QUESTIONS

1. Mr. S. was diagnosed with failure of his SA node to fire. How will his heart be electrically excited if the SA node is not functioning?

2. Mrs. J. has been diagnosed with 90% occlusion of her left anterior descending (LAD) coronary artery. Describe which anatomical walls of the heart are affected. Explain which parts of her cardiac conducting system may be affected by the occlusion.

3. Ms. M. has a long history of systemic hypertension. Which of the factors that regulate SV are most affected by her history of hypertension?

References

1. Porth CM: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009

2. Bickley LS: Bates' Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

17 Patient Assessment: Cardiovascular System

Patricia Gonce Morton, Kim Reck, Jillian Hamel, Amanda S. Walther, Kathryn T. Von Rueden, and Jan M. Headley

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Explain the components of the cardiovascular history.
2. Describe the steps of the cardiovascular physical examination.
3. Discuss the mechanisms responsible for the production of the first, second, third, and fourth heart sounds and their timing in the cardiac cycle.
4. Explain the attributes of heart murmurs.
5. Describe components of hematologic studies, coagulation studies, blood chemistries, and serum lipid studies.
6. Compare and contrast the usefulness of serum enzymes and myocardial proteins in diagnosing an acute myocardial infarction.
7. Describe current techniques used for diagnostic purposes in cardiology.
8. Discuss the nursing care and patient education before and after cardiac diagnostic studies.
9. Describe potential complications of cardiac diagnostic procedures.
10. Explain the major features of an electrocardiogram (ECG) monitoring system and steps to troubleshoot the system.
11. Describe the components of the ECG tracing and their meaning.
12. Explain the steps used to interpret a rhythm strip.
13. Describe the causes, clinical significance, and management for each of the dysrhythmias discussed.
14. Describe the parameters of a normal 12-lead ECG.
15. Define electrical axis, and determine the direction of the axis for a 12-lead ECG.
16. Explain the causes, clinical significance, and treatment of bundle branch blocks, atrial enlargement, and ventricular enlargement.
17. Describe the ECG changes associated with serum potassium and calcium abnormalities.
18. Describe the system components required to monitor hemodynamic pressures.
19. Analyze the characteristics of normal systemic arterial, right atrial, right ventricular, pulmonary artery, and pulmonary artery occlusion pressure waveforms.
20. State nursing interventions that ensure accuracy of pressure readings.
21. Discuss the major complications that can occur with an indwelling arterial, central venous, and pulmonary artery catheter.
22. Describe the thermodilution method of measuring cardiac output.
23. Describe alternative minimally invasive and noninvasive methods of obtaining hemodynamic data.
24. Evaluate the factors influencing oxygen delivery and consumption.
25. Use SvO_2 or $ScvO_2$ monitoring to assess oxygen delivery and consumption.

The application of complex technology to the assessment and management of cardiovascular and cardiopulmonary conditions has increased greatly in the past several decades. Use of advanced and complex technologies is an integral part of the care of critically ill patients. Nevertheless, the value of a comprehensive cardiovascular assessment should never be underestimated. The chapter begins with a discussion of the components of cardiovascular assessment that includes the cardiac history, physical examination, and laboratory and diagnostic studies. Among the studies in particular are electrocardiographic monitoring and hemodynamic monitoring.

CARDIAC HISTORY AND PHYSICAL EXAMINATION

The cardiovascular nursing assessment and health history provide physiological and psychosocial information that guides the physical assessment, the selection of diagnostic tests, and the choice of treatment options. During the history, the nurse asks about the patient's chief complaint and the history of the present illness, including a complete analysis of each sign and symptom. Next, the nurse asks about the patient's past health history, family history, and personal and social history. The history concludes with a review of systems that provides additional clues to the patient's health status. The information gathered during the history gives the nurse insight into risk factors and behaviors that promote or jeopardize cardiovascular health. The nurse uses this information to guide health teaching. During the process of taking a thorough history and performing a physical examination, the nurse has an opportunity to establish rapport with the patient and to evaluate the patient's general physical and emotional status.

▲ History

Chief Complaint and History of Present Illness

The nurse begins the history by investigating the patient's chief complaint, asking the patient to describe the problem or reason for seeking health care in his or her own words. The nurse then asks for more information about the present illness, using the NOPQRST format and the questions presented in Box 17-1. Answers to these questions are essential to understanding the patient's perception of the problem. To gain a better understanding of the current illness, the nurse also asks the patient about any associated symptoms, including chest pain, nausea or vomiting, dyspnea, edema of feet or ankles, palpitations, syncope or dizziness, cough and hemoptysis, nocturia, cyanosis, and extremity pain or paresthesias.

Chest Pain

Chest pain is one of the most common symptoms of patients with cardiovascular disease (CVD). Therefore, it is an essential component of the assessment interview. Chest pain is often a disturbing or even frightening experience for a patient, so the patient may be hesitant to initiate a discussion of chest pain. The questions listed in Box 17-1 are particularly useful when assessing chest pain because the answers help determine whether the pain is cardiac in origin.

BOX 17-1 Assessment Parameters: Questions to Ask in a Symptom Assessment

N Normal: Describe your normal baseline. What was it like before this symptom developed?
O Onset: When did the symptom start? What day? What time? Did it start suddenly or gradually?
P Precipitating and palliative factors: What brought on the symptom? What seems to trigger it—factors such as stress, position change, or exertion? What were you doing when you first noticed the symptom? What makes the symptom worse? What measures have helped relieve the symptom? What have you tried so far? What measures did not relieve the symptom?
Q Quality and quantity: How does it feel? How would you describe it? How much are you experiencing now? Is it more or less than you experienced at any other time?
R Region and radiation: Where does the symptom occur? Can you show me? In the case of pain, does it travel anywhere such as down your arm or in your back?
S Severity: On a scale of 0 to 10, with 0 being the absence of pain and 10 being the worst ever experienced, rate your symptom. How bad is the symptom at its worst? Does it force you to stop your activity and sit down, lie down, or slow down? Is the symptom getting better or worse, or staying about the same?
T Time: How long does the symptom last? How often do you get the symptom? Does it occur in association with anything, such as before, during, or after meals?

Because cardiac pain (angina pectoris) is the result of an imbalance between oxygen supply and oxygen demand, it usually develops over time. Typically, anginal pain does not start at maximal intensity. Not all chest pain is cardiac in origin, and careful reporting of the characteristics of the pain and the behaviors (or lack thereof) that precede the onset of pain is required. The nurse asks the patient about his or her normal baseline status before the symptoms developed. It is also important to ask about the onset of the symptoms to determine the date and time of the start of symptoms and whether the onset was sudden or gradual. Symptoms that may accompany chest pain caused by heart disease include nausea and vomiting.

Chest pain caused by coronary artery disease (CAD) is often precipitated by physical or emotional exertion, a meal, or being out in the cold. Palliative measures to relieve anginal pain may include rest or sublingual nitrates; these measures usually do not relieve the pain of a myocardial infarction (MI). The quality of cardiac chest pain is often described as heaviness, tightness, squeezing, or a choking sensation. If the pain is reported as superficial, knifelike, or throbbing, it is not likely to be anginal. Cardiac chest pain is usually located in the substernal region and often radiates to the neck, left arm, back, or jaw. Although the pain is often referred to other areas, anginal pain is visceral in origin, and most complaints include a reference to a "deep, inside" pain. When the patient is asked to point to the painful area, the painful area is about the size of a hand or clenched fist. It is unusual for true anginal pain to be localized to an area smaller than a fingertip. Using a scale of 0 to 10, with 10 being the worst pain the patient has ever experienced and 0 being the absence of pain, the patient is asked to rate the severity of the pain. When asked about time, the patient with cardiac chest pain reports the pain lasting anywhere from 30 seconds to hours.

Pain may be secondary to cardiovascular problems that are unrelated to a primary coronary insufficiency. Therefore, when obtaining the patient's history, the nurse must consider other causes. For example, if the patient reports the pain is made worse by lying down, moving, or deep breathing, it may be caused by pericarditis. If the pain is retrosternal and accompanied by sudden shortness of breath and peripheral cyanosis, it may be caused by a pulmonary embolism.

Dyspnea

Dyspnea occurs in patients with both pulmonary and cardiac abnormalities. In patients with cardiac disease, it is the result of inefficient pumping of the left ventricle, which causes a congestion of blood flow in the lungs. During history taking, dyspnea is differentiated from the usual breathlessness that follows a sudden burst of physical activity (eg, running up four flights of stairs, sprinting across a parking lot). Dyspnea is a subjective complaint of true difficulty in breathing, not just shortness of breath. The nurse determines whether the breathing difficulty occurs only with exertion or also at rest. If dyspnea is present when the patient lies flat but is relieved by sitting or standing, it is orthopnea. If dyspnea is characterized by breathing difficulties starting after approximately 1 to 2 hours of sleep and relieved by sitting upright or getting out of bed, it is paroxysmal nocturnal dyspnea.

Edema of the Feet and Ankles

Although many other problems can leave a patient with swollen feet or ankles, heart failure may also be responsible because the heart is unable to mobilize fluid appropriately. Because gravity promotes the movement of fluids from intravascular to extravascular spaces, the edema becomes worse as the day progresses and usually improves at night after lying down to sleep. Patients or families may report that shoes do not fit anymore, socks that used to be loose are now too tight, and the indentations from sock bands take more time than usual to disappear. The nurse inquires about the timing of edema development (eg, immediately after lowering the extremities, only at the end of the day, only after a significant salt intake) and duration (eg, relieved with temporary elevation of the legs or with constant elevation).

Palpitations and Syncope or Dizziness

Palpitations refer to the awareness of irregular or rapid heart beats. Patients may report the "skipping" of beats, a rushing of the heart, or a loud "thudding." The nurse asks about onset and duration of the palpitations, associated symptoms, and any precipitating events that the patient or family can remember. Because a cardiac dysrhythmia may compromise blood flow to the brain, the nurse asks about symptoms of dizziness, fainting, or syncope that accompany the palpitations.

Cough and Hemoptysis

Abnormalities such as heart failure, pulmonary embolus, or mitral stenosis may cause a cough or hemoptysis. Side effects of medications such as angiotensin-converting enzyme (ACE) inhibitors may also include a cough. The nurse asks the patient about the presence of a cough and inquires about the quality (wet or dry) and frequency of the cough (chronic or occasional, only when lying down or after exercise). If the cough produces expectorant, the nurse inquires about its color, odor, consistency, and amount perceived by the patient. If the patient reports spitting up blood (hemoptysis), the nurse asks if the substance spit up was streaked with blood, frothy bloody sputum, or frank blood (bright or dark).

Nocturia

Kidneys that are inadequately perfused by an unhealthy heart during the day may finally receive sufficient flow during rest at night to increase their output. The nurse asks about the number of times the patient urinates during the night. If the patient takes a diuretic, the nurse also evaluates frequency of urination in relation to the time of day the diuretic is taken.

Cyanosis

Cyanosis reflects the oxygenation and circulatory status of the patient. Central cyanosis is generally distributed and best found by examining the mucous membranes for discoloration and duskiness, and reflects reduced oxygen concentration. Peripheral cyanosis is localized in the extremities and protrusions (hands, feet, nose, ears, and lips) and reflects impaired circulation.

Extremity Pain or Paresthesias

Extremity pain results when the blood supply to exercising muscles is inadequate, and this type of pain is known as claudication. Usually, the cause of claudication is significant atherosclerotic obstruction to the lower extremities. The limb is asymptomatic at rest unless the obstruction is severe. Blood supply to the legs is inadequate to meet metabolic demands during exercise, and ischemic pain results. The patient describes a cramping, "charley horse" ache, or weakness in the foot, calf, thigh, or buttocks that improves with rest. The patient is asked to describe the severity of the pain and how much exertion is required to produce the pain.

Past Health History

The history includes information about the patient's past health. When assessing the patient's past health history, the nurse inquires about childhood illnesses and other previous illnesses, as well as past surgeries, previous diagnostic tests and interventions, medication use, allergies, and transfusions (Box 17-2). The nurse also asks about risk factors (Box 17-3, p. 210).[1,2]

Family History

The nurse asks about the age and health, or age and cause of death, of immediate family members, including parents, grandparents, siblings, children, and grandchildren. The nurse inquires about cardiovascular problems, such as CAD, hypertension, diabetes mellitus, sudden cardiac death, stroke, peripheral vascular disease, and lipid disorders (see Box 17-2).

Personal and Social History

Although the physical symptoms provide many clues regarding the origin and extent of cardiac disease, the personal and social history also adds to the patient's health status. An

BOX 17-2 HEALTH HISTORY for Cardiovascular Assessment

Chief Complaint
- Patient's description of the problem

History of the Present Illness
- Complete analysis of the following signs and symptoms (using the NOPQRST format; see Box 17-1, p. 207)
- Chest pain
- Nausea and/or vomiting
- Dyspnea
- Edema
- Palpitations
- Syncope/dizziness
- Cough and hemoptysis
- Nocturia
- Cyanosis
- Extremity pain or paresthesias

Past Health History
- Relevant childhood illnesses and immunizations: rheumatic fever, murmurs, congenital anomalies, streptococcal infections
- Past acute and chronic medical problems including treatments and hospitalizations: heart failure, hypertension, coronary artery disease (CAD), myocardial infarction (MI), hyperlipidemia, valve disease, cardiac dysrhythmias, diabetes mellitus, endocarditis, thrombophlebitis, deep venous thrombosis, peripheral vascular disease, chest injury, pneumonia, pulmonary embolism, thyroid disease, tuberculosis
- Risk factors: age, heredity, gender, race, tobacco use, elevated cholesterol, hypertension, physical inactivity, obesity, diabetes mellitus (see Box 17-3, p. 210)
- Past surgeries: coronary artery bypass grafting, valvular surgery, peripheral vascular surgeries
- Past diagnostic tests and interventions: electrocardiogram (ECG), echocardiogram, stress test, electrophysiology studies, myocardial imaging studies, thrombolytic therapy, cardiac catheterization, percutaneous transluminal cardiac angioplasty, stent placement, atherectomy, pacemaker or implantable cardioverter defibrillator implantation, valvuloplasty
- Medications, including prescription drugs, over-the-counter drugs, vitamins, herbs, and supplements: angiotensin-converting enzyme (ACE) inhibitors, anticoagulants, antihypertensives, antiplatelets, antiarrhythmics, angiotensin II receptor blockers (ARBs), β-blockers, calcium channel blockers, antihyperlipidemics, diuretics, electrolyte replacements, nitrates, inotropes, hormone replacement therapies, oral contraceptives
- Allergies and reactions to medications, foods, contrast dye, latex or other materials
- Transfusions, including type and date

Family History
- Health status or cause of death of parents and siblings: CAD, hypertension, diabetes mellitus, sudden cardiac death, stroke, peripheral vascular disease, lipid disorders

Personal and Social History
- Tobacco, alcohol, and substance use
- Family composition
- Occupation and work environment
- Living environment
- Diet: restrictions, supplements, caffeine intake
- Sleep patterns: number of pillows used
- Exercise
- Cultural beliefs
- Spiritual or religious beliefs
- Coping patterns and social support systems
- Leisure activities
- Sexual activity: use of agents for erectile dysfunction
- Recent travel

Review of Other Systems
- HEENT: retinal problems, visual changes, headaches, carotid artery disease
- Respiratory: shortness of breath, dyspnea, cough, lung disease, recurrent infections, pneumonia, tuberculosis
- Gastrointestinal: nausea, vomiting, weight loss, change in bowel habits
- Genitourinary: incontinence, erectile dysfunction
- Musculoskeletal: pain, weakness, varicose veins, change in sensation, peripheral edema
- Neurological: transient ischemic attacks, stroke, change in level of consciousness, changes in sensations
- Endocrine: thyroid disease, diabetes mellitus

understanding of the topics listed in Box 17-2 contributes to the nurse's knowledge of the patient as a person and guides interaction with the patient and family as well as patient education.

Review of Other Systems

The health history concludes with a review of relevant systems. This information gives the nurse a better understanding of the patient's total health status, and it also helps the nurse determine the impact of CVD on the functioning of other body systems (see Box 17-2).

▲ Physical Examination

Cardiac assessment requires examination of all aspects of the individual, using the standard steps of inspection, palpation, percussion, and auscultation. A thorough and careful examination helps the nurse detect subtle abnormalities as well as obvious ones.

Inspection

General Appearance

Inspection begins as soon as the patient and nurse interact. General appearance and presentation of the patient are key elements of the initial inspection. Critical examination reveals a first impression of age, nutritional status, self-care ability, alertness, and overall physical health. It is necessary to note the ability of the patient to move and speak with or without distress. Consider the patient's posture, gait, and musculoskeletal coordination.

Jugular Venous Distention

Pressure in the jugular veins reflects right atrial pressure (RAP) and provides the nurse with an indication of heart hemodynamics and cardiac function. The height of the level of blood in the right internal jugular vein is an indication of RAP because there are no valves or obstructions between the vein and the right atrium.

BOX 17-3 Risk Factors for Cardiovascular Disease

Major Uncontrollable Risk Factors

- **Age:** There is an increased incidence of all types of atherosclerotic disease with aging. More than 83% of people who die from CAD are age 65 or older. Women at older ages who have a MI are twice as likely as men to die of it within a few weeks.
- **Heredity (including race):** The tendency for development of atherosclerosis seems to run in families. The risk is thought to be a combination of environmental and genetic influences. Even when other risk factors are controlled, the chance for development of CAD increases when there is a familial tendency. African Americans have more severe hypertension than Caucasians and a higher risk of heart disease. The risk of heart disease is higher in Mexican Americans, American Indians, native Hawaiians, and some Asian Americans.
- **Gender:** Men have a greater risk for development of CAD than women at earlier ages. After menopause, women's death rate from MI increases but is not as great as men's.

Major Risk Factors That Can Be Modified, Treated, or Controlled

- **Tobacco smoking:** A smoker's risk for developing heart disease is two to four times that of nonsmokers. For smokers with coronary heart disease, cigarette smoking is a powerful independent risk factor for sudden cardiac death. Cigarette smoking, combined with other risk factors, greatly increases the risk of coronary heart disease. Exposure to smoke from others increases the risk of coronary heart disease for nonsmokers.
- **High blood cholesterol levels:** The risk for coronary heart disease increases as the blood cholesterol level rises. When other risk factors are present, this risk increases even more.
- **Hypertension:** Known as the "silent killer," hypertension is a risk factor with no specific symptoms and no early warning signs. Men have a greater risk for hypertension than women until the age of 55 years. The risk for development of hypertension is about the same for men and women between the ages of 55 and 75 years. After the age of 75 years, hypertension is more likely to develop in women than in men. African Americans are more likely to have hypertension than whites. Hypertension increases the risk for stroke, MI, kidney failure, and heart failure.
- **Physical inactivity:** A lack of physical exercise is a risk factor for CAD. Moderate to vigorous regular exercise plays a significant role in preventing heart disease and blood vessel disease. Even moderate-intensity exercise is beneficial if performed regularly and long-term. Physical activity also plays a role in controlling cholesterol, diabetes, obesity, and hypertension.
- **Obesity:** People who have excess body fat, especially at the waist, are more likely to develop heart disease and stroke even if they have no other risk factors. Excess weight raises blood pressure, blood cholesterol, and blood triglyceride levels. Excess weight lowers high-density lipids and makes diabetes more likely to develop.
- **Diabetes mellitus:** Even when blood glucose levels are under control, diabetes greatly increases the risk for heart disease and stroke. If blood glucose is not well controlled, the risk is even greater. Most people with diabetes die of some form of heart or blood vessel disease. Many people with diabetes also have high blood pressure, increasing their risk even more.

Other Contributing Factors

- **Stress:** A person's response to stress may be a contributing factor to heart disease. Stress in a person's life, his or her health behaviors, and socioeconomic status may all contribute to established risk factors. For example, individuals under stress may overeat, smoke, and not exercise.
- **Excessive alcohol intake:** Drinking too much alcohol can raise blood pressure, cause heart failure, lead to stroke, contribute to high triglycerides and obesity, and produce dysrhythmias. The risk for heart disease in individuals who drink moderate amounts of alcohol (an average of one drink for women and two drinks for men per day) is lower than in those who do not drink alcohol.

Adapted from American Heart Association: Risk Factors and Coronary Heart Disease and American Heart Association: Heart and Stroke Facts 2011 (see http://www. AHA.org).

The internal jugular veins are not directly visible because they lie deep to the sternomastoid muscles in the neck (Fig. 17-1). The goals of the examination are to determine the highest point of visible pulsation in the internal jugular veins, to note the level of head elevation, and to measure this point of visible pulsation as the vertical distance above the sternal angle. The patient is positioned supine in the bed with the head of the bed elevated 30, 45, 60, and 90 degrees. The patient is examined at each elevation with the head slightly turned away from the examiner. The nurse uses tangential light to observe for the highest point of visible pulsation.[3,4]

Next, the angle of Louis is located by palpating where the clavicle joins the sternum (suprasternal notch). The examining finger is slid down the sternum until a bony prominence is felt. This prominence is known as the angle of Louis. A vertical ruler is placed on the angle of Louis. Another ruler is placed horizontally at the level of the pulsation. The intersection of the horizontal ruler with the vertical ruler is noted, and the intersection point on the vertical ruler is read.

Normal jugular venous pulsation should not exceed 3 cm above the angle of Louis. See Figure 17-2 for an illustration of the procedure for assessment of jugular venous pressure. A level more than 3 cm above the angle of Louis indicates an abnormally high volume in the venous system. Possible causes include right-sided heart failure, obstruction of the superior vena cava, pericardial effusion, and other cardiac or thoracic diseases. An increase in the jugular venous pressure of more than 1 cm while pressure is applied to the abdomen for 60 seconds (hepatojugular or abdominojugular test) indicates the inability of the heart to accommodate the increased venous return.

Chest

The chest is inspected for signs of trauma or injury, symmetry, chest contour, and any visible pulsations. The inspection may reveal the location of the point of maximal impulse (PMI). In most patients, the apical pulse is the PMI; however, in some pathological conditions, these may be two distinct areas on the chest.[3] Thrusts (abnormally strong precordial pulsations) are noted. Any depression (sternum excavatum) or bulging of the precordium is recorded.

Extremities

A close inspection of the patient's extremities can also provide clues about cardiovascular health. The extremities

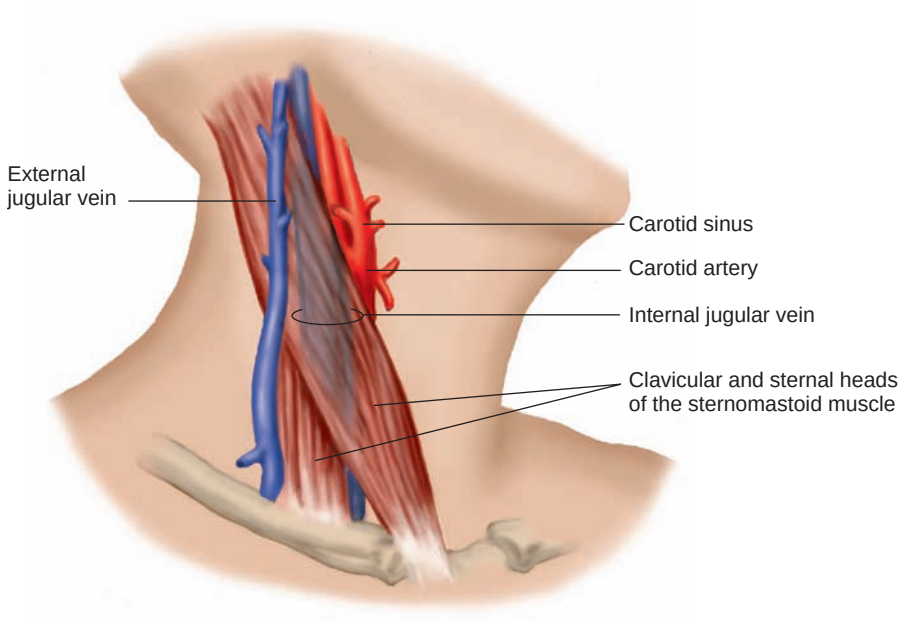


FIGURE 17-1 ▲ Internal jugular veins. (From Bickley L: Bates' Guide to Physical Examination and Health History, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 237.)

are examined for lesions, ulcerations, unhealed sores, and varicose veins. Distribution of hair on the extremities also is noted. A lack of normal hair distribution on the extremities may indicate diminished arterial blood flow to the area.

Skin

Skin is evaluated for moistness or dryness, color, elasticity, edema, thickness, lesions, ulcerations, and vascular changes. Nail beds are examined for cyanosis and clubbing, which may indicate chronic cardiac or pulmonary abnormalities. (For a further discussion of nail assessment, see Chapter 51). General differences in color and temperature between body parts may provide perfusion clues.

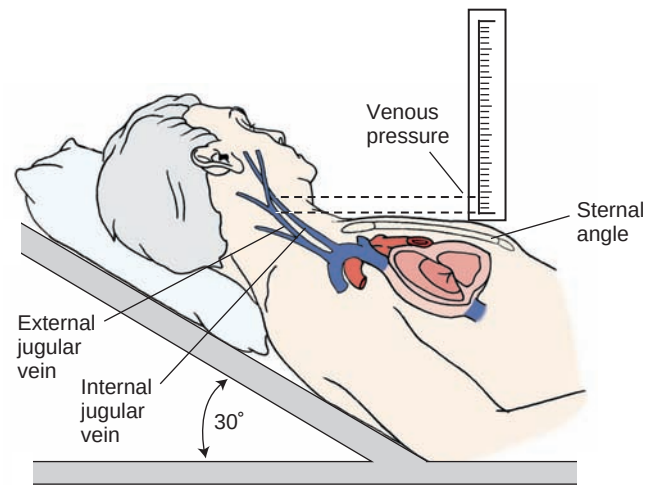


FIGURE 17-2 ▲ Assessment of jugular venous pressure. Place the patient supine in bed and gradually raise the head of the bed to 30, 45, 60, and 90 degrees. Using tangential lighting, note the highest level of venous pulsation. Measure the vertical distance between this point and the sternal angle. Record this distance in centimeters and the angle of the head of the bed.

Palpation

Pulses

Cardiovascular assessment continues with palpation and involves the use of the pads of the finger and balls of the hand. Using the pads of the fingers, the carotid, brachial, radial, femoral, popliteal, posterior tibial, and dorsalis pedis pulses are palpated (Fig. 17-3). The peripheral pulses are compared bilaterally to determine rate, rhythm, strength, and symmetry. The 0-to-4 scale described in Box 17-4 is used to rate the strength of the pulse. The carotid pulses should never be assessed simultaneously because this can obstruct flow to the brain.

Pulses can also be described according to their characteristics. For example, pulsus alternans is a pulse that alternates in strength with every other beat; it is often found in patients with left ventricular failure. Pulsus paradoxus is a pulse that disappears during inspiration but returns during expiration. To determine whether the condition is pathological, the sphygmomanometer is deflated until the pulse is heard only during expiration and the corresponding pressure noted. As the cuff continues to deflate, the point at which the pulse is heard throughout the inspiratory and expiratory cycle is noted. The second systolic pressure reading is subtracted from the first; if the difference is greater than 10 mm Hg during normal respirations, it is considered pathological. During the assessment of pulses, the nurse compares the warmth and size of the palpated areas to monitor perfusion.

Precordium

The chest wall is palpated to assess for the PMI, thrills, and abnormal pulsations. Palpation starts with locating the PMI. In most patients, the PMI represents the point at which the apical pulse is most readily felt. Using light pressure, the nurse first uses the palmar surface of the hand to feel for pulsations and then uses the pads of the finger to palpate the

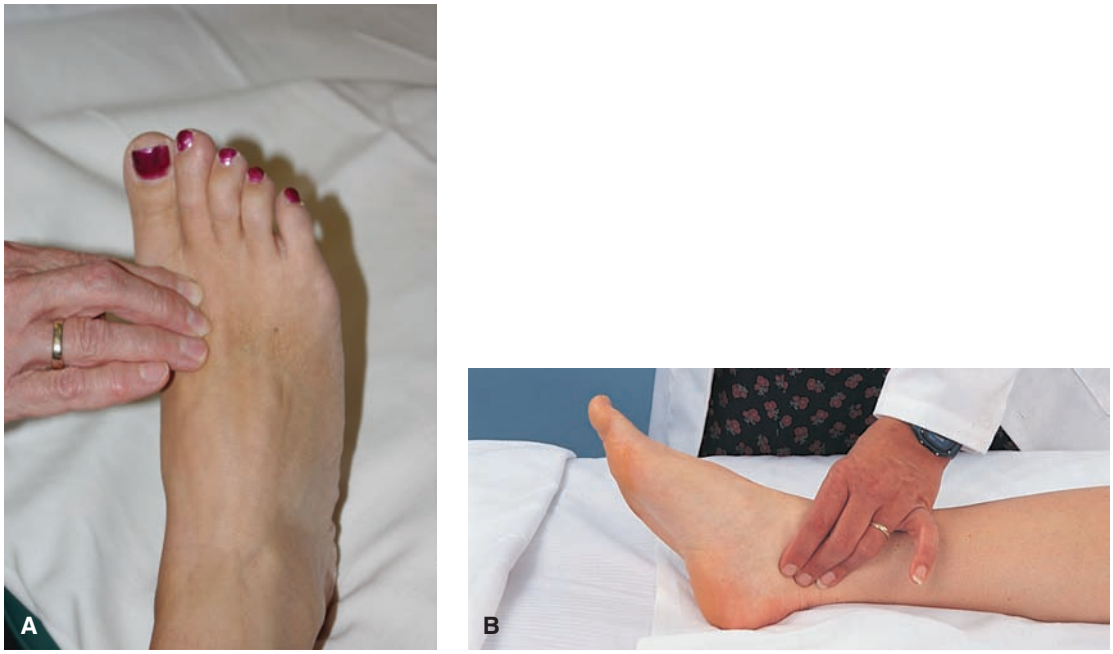


FIGURE 17-3 ▲ **A:** Palpating the dorsalis pedis pulse. **B:** Palpating the posterior tibial pulse. (From Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 401.)

apical pulse (Fig. 17-4). The PMI is palpated, noting its location, diameter, amplitude, and duration. Usually, the PMI is located in the midclavicular line at about the fourth or fifth intercostal space. If the pulse is difficult to palpate, it may be necessary to ask the patient to turn on the left side (left lateral decubitus position).

Next, the nurse palpates the lower left sternal border area, the upper left sternal border area, the sternoclavicular area, the right upper sternal border area, the lower right sternal border area, and finally the epigastric area. During palpation of these areas, the nurse feels for a thrill, which is a palpable vibration. A thrill usually represents a disruption in blood flow related to a defect in one of the semilunar valves.

Percussion

With the advent of radiological means of evaluating cardiac size, percussion is not a significant contributor to cardiac assessment. However, a gross determination of heart size can be made by percussing for the dullness that reflects the cardiac borders.

BOX 17-4 Rating Scale Used for Assessing Strength of Pulses

0 Absent
1 Palpable but thready and weak, easily obliterated
2 Normal, not easily obliterated
3 Increased
4 Bounding, cannot obliterate

Auscultation

Data obtained by careful and thorough auscultation of the heart are essential in planning and evaluating care of the critically ill patient. In this section, the following topics are discussed: the basic principles underlying cardiac auscultation; the factors responsible for the production of normal heart sounds; and the pathophysiological conditions responsible for the production of extra sounds, murmurs, and friction rubs.

To facilitate accurate auscultation, the patient should be relaxed and comfortable in a quiet, warm environment with adequate lighting. The patient is placed in a recumbent position with the trunk elevated 30 to 45 degrees. To help hear abnormal sounds, the patient may be asked to roll partly onto the left side (left lateral decubitus position). This position helps bring the left ventricle closer to the chest wall. The patient also may be asked to sit up, lean forward, and exhale. In this position, it may be easier to hear murmurs caused by aortic regurgitation (Fig. 17-5).

A good-quality stethoscope is essential. The earpieces should fit the ears snugly and comfortably and follow the natural angle of the ear canals. Sound waves that travel a shorter distance are more intense and less distorted, therefore, the tubing of the stethoscope should be approximately 12 inches long and somewhat rigid. It is best to have two tubes leading from the head of the stethoscope, one to each ear. The head of the stethoscope should be equipped with both a diaphragm and a bell on a valve system that allows the clinician to switch easily between the two components. The diaphragm is used to hear high-frequency sounds, such as the first and second heart sounds (S_1, S_2), friction rubs, systolic murmurs, and diastolic insufficiency murmurs. The diaphragm should be placed firmly on the chest wall to create a tight seal. Low-frequency sounds, such as the third and

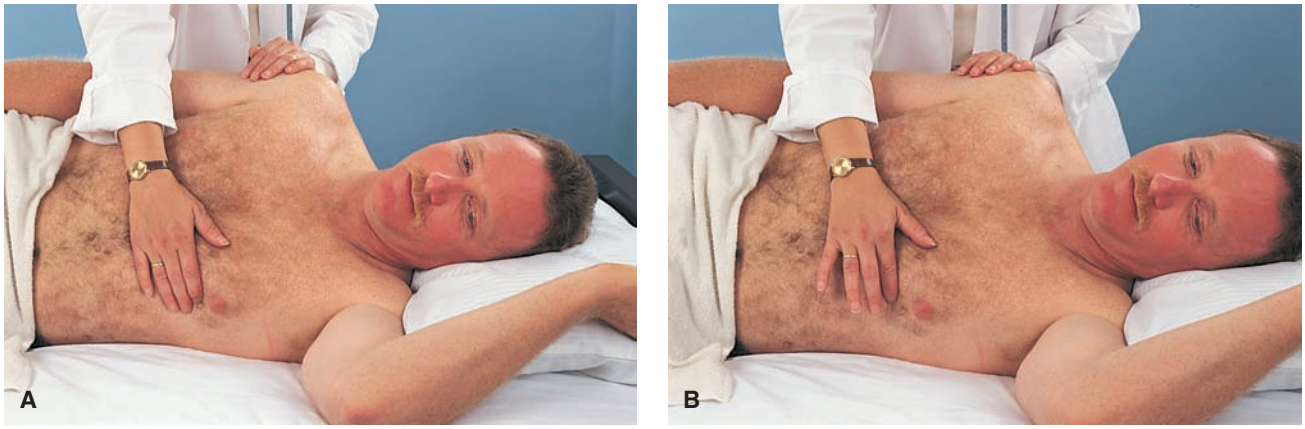
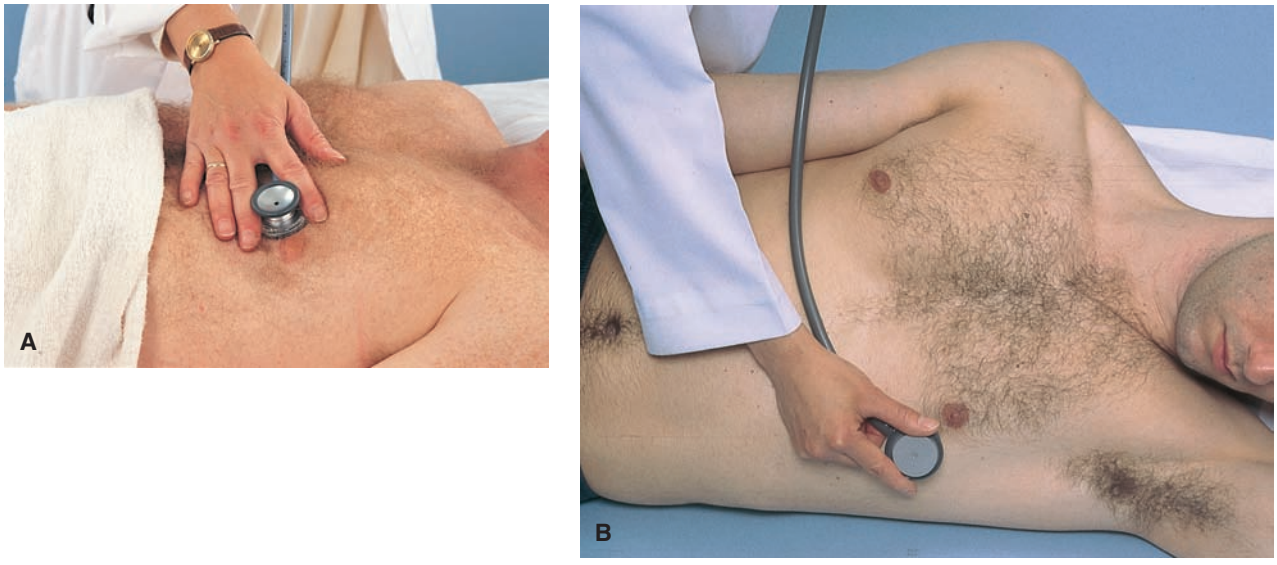


FIGURE 17-4 ▲ Locate the apical impulse with the palmar surface (**A**), then palpate the apical pulse with the fingerpad (**B**). (From Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 367.)



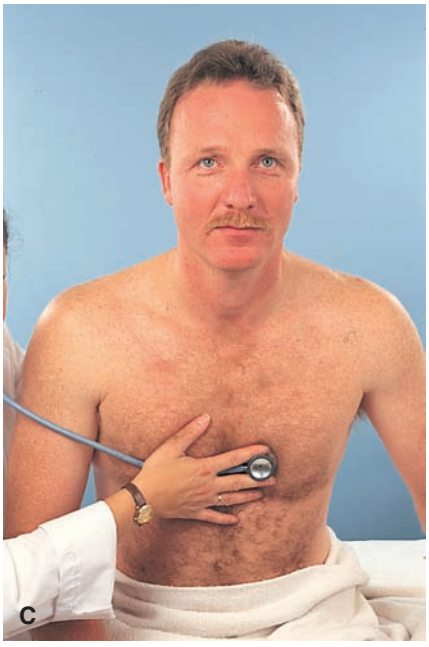


FIGURE 17-5 ▲ **A:** Auscultating the heart with the patient in a recumbent position. **B:** Auscultating the heart with the patient in a left lateral decubitus position. **C:** Auscultating the heart with the patient sitting up, leaning forward, and exhaling. (**A** and **C,** From Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 368–369. **B,** From Bickley L: Bates' Guide to Physical Examination and Health History, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 363.)

fourth heart sounds (S_3, S_4) and the diastolic murmurs of mitral and tricuspid stenosis, are best heard with the stethoscope bell, which should be placed lightly on the chest wall only to seal the edges.

The precordium is auscultated systematically (see Fig. 17-6). Some authorities suggest the use of anatomical names for the auscultation areas (eg, aortic and pulmonic), whereas others discourage the use of such labels because murmurs of more than one origin can be heard in a given area.[3,4] Instead, some suggest the use of anatomical landmarks such as intercostal spaces and relationship to the sternal border.[3,4]

The nurse begins the examination by listening with the stethoscope diaphragm in the right second intercostal space along the sternum. This area is sometimes called the aortic area and is the place where S_2 is loudest. Next, the nurse places the stethoscope in the left second intercostal space along the sternum, which is known as the pulmonic listening area, and from there moves the stethoscope down the left sternal border between the second and fifth spaces, one intercostal space at a time. The lower left sternal border area is sometimes referred to as the tricuspid area. Finally, the nurse moves the stethoscope to the mitral area or apex of the heart, where S_1 is the loudest. This pattern is then repeated with the stethoscope bell.

In each area auscultated, the nurse identifies S_1, noting the intensity of the sound, respiratory variation, and splitting. S_2 should then be identified and the same characteristics assessed. After S_1 and S_2 are identified, the presence of extra sounds is noted—first in systole, then in diastole. Finally, each area is auscultated for murmurs and friction rubs.

First Heart Sound

S_1 is timed with the closure of the mitral and tricuspid valves at the beginning of ventricular systole (Fig. 17-7A). Because mitral valve closure is responsible for most of the sound produced, S_1 is heard best in the mitral or apical area. The upstroke of the carotid pulse correlates with S_1 and can be used to help distinguish S_1 from S_2.

The intensity (loudness) of S_1 varies with the position of the atrioventricular (AV) valve leaflets at the beginning of ventricular systole and the structure of the leaflets (thickened or normal). A loud S_1 is produced when the valve leaflets are wide open at the onset of ventricular systole and corresponds to a short PR interval on the surface electrocardiogram (ECG) tracing. A lengthening of the PR interval produces a soft S_1 because the leaflets have had time to float partially closed before ventricular systole. Mitral stenosis also increases the intensity of S_1 due to a thickening of the valvular structures.

In general, S_1 is heard as a single sound. However, if right ventricular systole is delayed, S_1 may be split into its two component sounds. The most common cause of this splitting is delay in the conduction of impulses through the right bundle branch; the splitting correlates with a right bundle branch block (RBBB) pattern on the ECG. Splitting of S_1 is heard best over the tricuspid area.

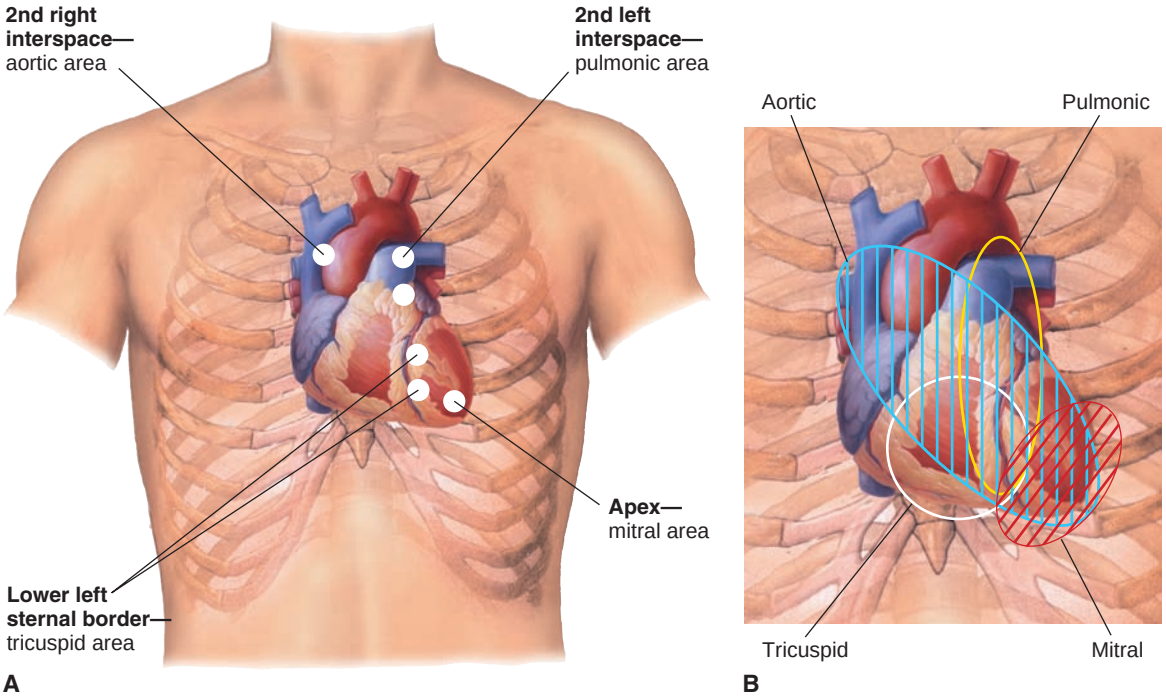
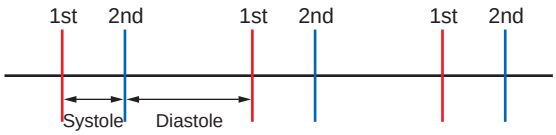
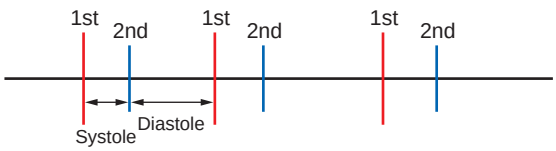


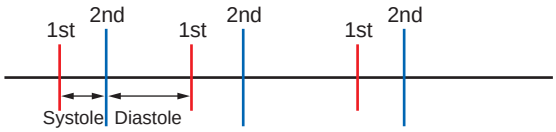
FIGURE 17-6 ▲ **A:** Areas of auscultation. Aortic area (second intercostal space to the right of the sternum). Pulmonic area (second intercostal space to the left of the sternum). Tricuspid area (fifth intercostal space to the left of the sternum). Mitral or apical area (fifth intercostal space, midclavicular line). **B:** Heart sounds and murmurs that originate in the four valves range widely. Use anatomical location rather than valve area to describe where murmurs and sounds are best heard. (**A** From Bickley L: Bates' Guide to Physical Examination and Health History, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 362. **B** Redrawn from Leatham: Introduction to the Examination of the Cardiovascular System, 2nd ed. Oxford, Oxford University Press, 1979.)



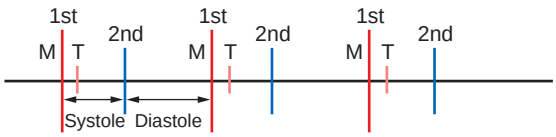
Normal: S_1 is produced by the closure of the AV valves and correlates with the beginning of ventricular systole. It is heard best in the apical or mitral area.



Loud First Sound: The intensity of the first heart sound may be incerased when the PR interval is shortened, as in tachycardia, or when the valve leaflets are thickened as in mital stenosis.

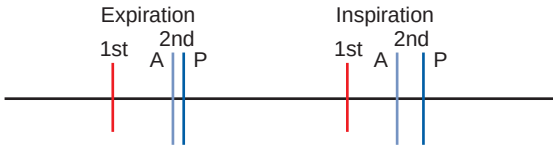


Soft First Sound: A soft S_1 is heard when the PR interval is prolonged.

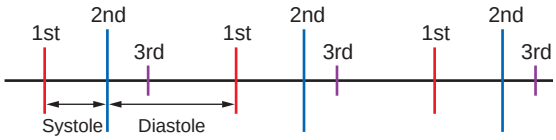


Split First Sound: A split S_1 is heard when right ventricular emptying is delayed. The mitral valve closes before the tricuspid valve and "splits" the sound into its two components.

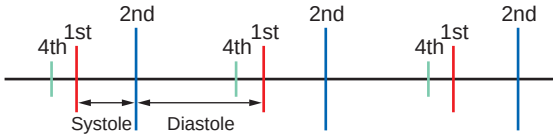
A. First heart sound (S_1)



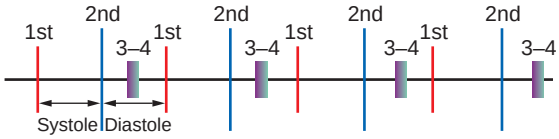
B. Second heart sound (S_2)



C. Third heart sound (S_3)



D. Fourth heart sound (S_4)



E. Summation gallop

FIGURE 17-7 ▲ A: First heart sound. **B:** Second heart sound. The second heart sound is produced by the closure of the semilunar valves (aortic and pulmonary). During inspiration, there is an increase in venous return to the right side of the heart, which causes a delay in the emptying of the right ventricle and the closure of the pulmonic valve. This allows the two components of the second heart sound to separate or split during inspiration. **C:** Third heart sound. An S_3 or ventricular gallop is heard in early diastole, shortly after the second heart sound. The presence of a pathological S_3 may be indicative of heart failure. **D:** Fourth heart sound. An S_4 is a late diastolic sound that occurs just before S_1. It is a low-frequency sound heard best with the bell of the stethoscope. **E:** Summation gallop. With rapid heart rates, S_3 and S_4 may become audible as a single, very loud sound that occurs in middiastole. This sound is a summation gallop.

Second Heart Sound

S_2 is produced by the vibrations initiated by the closure of the aortic and pulmonic semilunar valves and is heard best at the base of the heart (The term "base of the heart" is a clinical term that refers to the superior aspect of the heart at right and left second intercostal spaces next to the sternum.) (Fig. 17-7B). This sound represents the beginning of ventricular diastole.

Like S_1, S_2 consists of two separate components. The first component of S_2 is aortic valve closure; the second component is pulmonic valve closure. With inspiration, systole of the right ventricle is slightly prolonged because of increased filling of the right ventricle. This causes the pulmonic valve to close later than the aortic valve and S_2 to become "split" into its two components. This normal finding is termed physiological splitting and is heard best on inspiration with the stethoscope placed in the second intercostal space to the left of the sternum.

The intensity of S_2 may be increased in the presence of aortic or pulmonic valvular stenosis or with an increase in the diastolic pressure forcing the semilunar valves to close, as occurs in pulmonary or systemic hypertension.

Third Heart Sound

An S_3 may be physiological or pathological (Fig. 17-7C). A physiological S_3 is a normal finding in children and healthy young adults; it usually disappears after 25 to 35 years of age. An S_3 in an older adult with heart disease signifies ventricular failure.

An S_3 is a low-frequency sound that occurs during the early, rapid-filling phase of ventricular diastole. A noncompliant or failing ventricle cannot distend to accept this rapid inflow of blood. This causes turbulent flow, resulting in the vibration of

the AV valvular structures or the ventricles themselves, producing a low-frequency sound. An S_3 associated with left ventricular failure is heard best at the apex with the stethoscope bell. The sound may be accentuated by turning the patient slightly to the left side. A right ventricular S_3 is heard best at the xiphoid or lower left sternal border and varies in intensity with respiration, becoming louder on inspiration.

Fourth Heart Sound

An S_4 or atrial gallop is a low-frequency sound heard late in diastole just before S_1. It is rarely heard in healthy patients (Fig. 17-7D). The sound is produced by atrial contraction forcing blood into a noncompliant ventricle that, by virtue of its noncompliance, has an increased resistance to filling. Systemic hypertension, MI, angina, cardiomyopathy, and aortic stenosis all may produce a decrease in left ventricular compliance and an S_4. A left ventricular S_4 is auscultated at the apex with the bell of the stethoscope. Conditions affecting right ventricular compliance, such as pulmonary hypertension or pulmonic stenosis, may produce a right ventricular S_4 heard best at the lower left sternal border; it increases in intensity during inspiration.

Summation Gallop

With rapid heart rates, as ventricular diastole shortens, if S_3 and S_4 are both present, they may fuse together and become audible as a single diastolic sound. This is called a summation gallop (Fig. 17-7E). This sound is loudest at the apex and is heard best with the stethoscope bell while the patient lies turned slightly to the left side.

Heart Murmurs

Murmurs are sounds produced either by the forward flow of blood through a narrowed or constricted valve into a dilated vessel or chamber or by the backward flow of blood through an incompetent valve or septal defect. Murmur classification is based on several attributes (Box 17-5). Timing in the cardiac cycle is an important attribute that refers to the presence of the murmur during either systole or diastole. Systolic murmurs occur between S_1 and S_2. Diastolic murmurs occur after S_2 and before the onset of the following S_1. Location of maximum intensity refers to the anatomical location on the anterior chest where the sound of the murmur is heard the loudest. Anatomical landmarks are used to describe the radiation of the sound. The pitch of the sound helps further differentiate the type of murmur. The shape of the murmur refers to any changes in intensity over time. The intensity or loudness of a murmur is described using a grading system from 1 to 6. The quality of the sound produced is described as blowing, harsh, rumbling, vibratory, or musical. The effect of ventilation or a change in body position on the murmur is another important attribute.

Systolic Murmurs

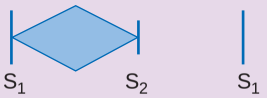
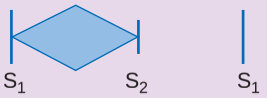
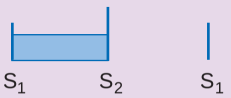
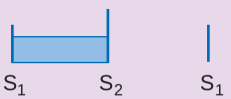
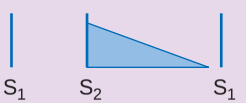
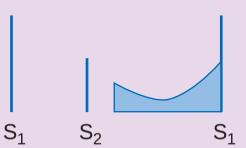
As previously described, S_1 is produced by mitral and tricuspid valve closure and signifies the onset of ventricular systole. Murmurs occurring after S_1 and before S_2 are therefore classified as systolic murmurs.

During ventricular systole, the aortic and pulmonic valves are open. If either of these valves is stenotic or narrowed, a sound classified as a midsystolic ejection murmur is heard. Because the AV valves close before blood is ejected through the aortic and pulmonic valves, there is a delay between S_1 and the beginning of the murmur. The murmurs associated with aortic stenosis and pulmonic stenosis are described as crescendo–decrescendo or diamond shaped (Table 17-1), meaning that the sound increases and then decreases in intensity. The quality of these murmurs is harsh, and they are of medium pitch. The murmur caused by aortic stenosis is heard best in the aortic area and may radiate into the neck. The murmur of pulmonic stenosis is heard best over the pulmonic area. In severe pulmonic stenosis, S_2 may be widely split.

BOX 17-5 Attributes of Heart Murmurs

Timing: A systolic murmur is heard between S_1 and S_2. A diastolic murmur is heard between S_2 and S_1.
Systolic murmurs are classified into three groups:
Midsystolic murmur begins after S_1 and stops before S_2.
Pansystolic (holosystolic) murmur starts with S_1 and stops with S_2 without a gap between the murmur and the heart sound.
Late systolic murmur starts in mid to late systole and continues up to S_2.
Diastolic murmurs also are divided into three categories:
Early diastolic murmur starts right after S_2 and fades before the next S_1.
Middiastolic murmur starts a short time after S_2 and fades away or merges into a late diastolic murmur.
Late diastolic murmur starts late in diastole and continues up to S_1.
Location of maximal intensity: The anatomical location where the murmur is heard best. The location is identified based on intercostal space and its relation to the sternum, the apex, the mid-clavicular line, or one of the axillary lines.
Radiation or transmission from the point of maximal intensity: The nurse notes the site farthest from the location of the greatest intensity at which the sound is still heard. The farthest site is identified using anatomical landmarks as described previously.
Pitch: The terms *high, medium,* and *low* are used to describe the pitch of the murmur.
Shape: The shape of a murmur is determined by its intensity over time. A *crescendo murmur* grows louder. A *decrescendo murmur* grows softer. A *crescendo-decrescendo murmur* first rises in intensity, then falls. A *plateau murmur* has the same intensity throughout.
Intensity: A grading system is used to describe the intensity of the murmur.
Grade 1: barely audible in a quiet room, very faint; may not be heard in all positions.
Grade 2: quiet, but clearly audible.
Grade 3: moderately loud.
Grade 4: loud with palpable thrill.
Grade 5: very loud with an easily palpable thrill; may be heard when the stethoscope is partly off the chest.
Grade 6: very loud with an easily palpable thrill; may be heard with stethoscope entirely off the chest.
Quality: The terms such as *harsh, rumbling, vibratory, blowing,* and *musical* are used to describe the quality of the sound.
Ventilation and position: Note if the murmur is affected by inspiration, expiration, or a change in body position.

Table 17-1 Common Systolic and Diastolic Murmurs

Type	Possible Causes	Where to Auscultate	Radiation	Pitch	Shape	Quality	Ventilation and Position
Systolic Murmurs							
Aortic stenosis	Calcification, rheumatic fever, congenital malformation of valve cusps, degenerative process	Aortic area, right second intercostal space	Neck, upper back, right carotid, down the left sternal border to the apex	Medium	Crescendo–decrescendo May be diminished 	Harsh, may be musical at the apex	Heard best with the patient sitting and leaning forward, loudest during expiration
Pulmonic stenosis	Congenital malformation	Pulmonic area, second and third left intercostal spaces	Left side of neck, toward left shoulder	Medium	Crescendo–decrescendo 	Often harsh	Loudest during inspiration
Mitral regurgitation	Chronic rheumatic fever, acute bacterial endocarditis, myocardial ischemia or infarction, calcification, dilation of valvular apparatus secondary to dilated left ventricle (eg, heart failure), mitral valve prolapse	Mitral area, apex	Left axilla, less often to the left sternal border	Medium to high	Plateau Diminished 	Blowing, harsh	Heard best with patient in the left lateral decubitus position, does not become louder with inspiration
Tricuspid regurgitation	Right ventricular failure, dilation of valvular apparatus secondary to dilated right ventricle, bacterial endocarditis (rare)	Tricuspid area, lower left sternal border	Right sternal border, to the xiphoid area, and perhaps to the left midclavicular line, but not to the axilla	Medium	Plateau Diminished 	Blowing, harsh	May increase slightly with inspiration
Diastolic Murmurs							
Aortic regurgitation	Bacterial endocarditis, trauma, rheumatic fever, congenital malformation	Aortic area, right second intercostal space	Sternal border, apex	High	Decrescendo 	Blowing	Heard best with the patient sitting, leaning forward, with breath held after exhalation
Mitral stenosis	Rheumatic fever, congenital malformation (rare)	Mitral area, apex	Usually none	Low	Decrescendo–crescendo Loud 	Rumbling	Best heard with the patient in a left lateral position Mild exercise and listening during exhalation also make the murmur easier to hear

Systolic regurgitant murmurs are caused by the backward flow of blood from an area of higher pressure to an area of lower pressure. Mitral or tricuspid valvular insufficiency or a defect in the ventricular septum produces systolic regurgitant murmurs, which are harsh and blowing in quality. The sound is described as holosystolic, meaning that the murmur begins immediately after S_1 and continues throughout systole up to S_2 (see Table 17-1).

Mitral insufficiency produces a murmur, heard most easily in the apical area with radiation to the left axilla. The type of murmur associated with tricuspid regurgitation is heard loudest at the left sternal border and increases in intensity during inspiration (see Table 17-1). Both types of regurgitant murmurs are often accompanied by a diminished S_1.

Diastolic Murmurs

Diastolic murmurs occur after S_2 and before the next S_1. During diastole, the aortic and pulmonic valves are closed while the mitral and tricuspid valves are open to allow filling of the ventricles.

Aortic or pulmonic valvular insufficiency produces a blowing diastolic murmur that begins immediately after S_2 and decreases in intensity as regurgitant flow decreases through diastole. These murmurs are described as early diastolic decrescendo murmurs (see Table 17-1).

The murmur associated with aortic regurgitation is heard best in the aortic area and may radiate along the sternal border to the apex. Pulmonic valve regurgitation produces a murmur that is loudest in the pulmonic area.

Stenosis or narrowing of the mitral or tricuspid valve also produces a diastolic murmur. The AV valves open in middiastole shortly after the aortic and pulmonic valves close, causing a delay between S_2 and the start of the murmur of mitral and tricuspid stenosis. This murmur decreases in intensity from its onset and then increases again as ventricular filling increases because of atrial contraction; this is termed decrescendo–crescendo (see Table 17-1).

The murmur associated with mitral stenosis is heard best at the apex with the patient turned slightly to the left side. Tricuspid stenosis produces a murmur that increases in intensity with inspiration and is loudest in the fifth intercostal space along the left sternal border.

Friction Rubs

A pericardial friction rub can be heard when the pericardial surfaces are inflamed. This high-pitched, scratchy sound is produced by these inflamed layers rubbing together. A rub may be heard anywhere over the pericardium with the diaphragm of the stethoscope. The rub may be accentuated by having the patient lean forward and exhale. A pericardial friction rub, unlike a pleural friction rub, does not vary in intensity with respiration.

CARDIAC LABORATORY STUDIES

Knowledge of the purpose and significance of laboratory values in relation to the diagnosis and prognosis of CVD can enhance the quality of nursing care available to patients. Laboratory studies include both routine serum analysis and special studies, such as serum and cardiac enzymes. Nurses who have a basic understanding of laboratory studies can exercise judgment in interpreting results relative to other information about the patient. The ability to use this kind of judgment may well affect the patient's clinical course or prognosis.

▲ Routine Laboratory Studies

Appropriate assessment of normal and compromised cardiac function is essential to ensure accurate evaluation and correct diagnosis of the patient experiencing symptoms consistent with a cardiovascular disorder or CAD. Nurses can more appropriately plan the care of the patient and initiate interventions if they have an understanding of these laboratory tests and recognize their implications. Valuable information may be obtained by assessing levels of hematological components, coagulation factors, electrolytes, and phospholipids. Determination of these laboratory studies may vary with institutional techniques and equipment used. Normal and abnormal assay ranges have been universally established, and a brief listing of frequently ordered laboratory studies with their normal values can be found in Table 17-2. A more extensive explanation of the effects of abnormal laboratory determinations is provided in other parts of this text and is not addressed here.

Hematological Studies

Accurate assessment of the patient with a possible cardiac disorder merits review of hematological function. It is important for the critical care nurse to understand the role of blood cells in cardiac function and their contribution to the maintenance of healthy tissue. Blood is the transport medium for nutrients, such as oxygen and glucose, as well as electrolytes, plasma proteins, hormones, and medications. It is also the vehicle for removal of the products of metabolism. Changes in blood cell integrity and total cell count may reflect specific disorders of the cardiac system and should be considered an integral part of the laboratory assessment.

Knowledge of normal blood values is vital to understand deviations from normal that can be seen with various cardiac disruptions. It is necessary to review both the red blood cell count, which assesses cellular nutrition, and the white blood cell (WBC) count, which assesses defensive capability against infections, when diagnosing specific insults. Table 17-2 lists the components of these helpful hematological studies.

Coagulation Studies

Coagulation studies are also warranted in the laboratory assessment of patients with cardiac disease. Establishment of a baseline for coagulation function provides important information about the patient's ability to form, maintain, and dissolve blood clots. Such information may prove

Table 17-2 **Normal Reference Ranges for Laboratory Blood Tests**

Blood Test	Reference Range
Hematological Studies	
Red blood cell count	
Men	4.6–6.2 × 10^6
Women	4.2–5.4 × 10^6
Hematocrit	
Men	40%–50%
Women	38%–47%
Hemoglobin	
Men	13.5–18.0 g/100 mL
Women	12.0–16.0 g/100 mL
Corpuscle indices	
Mean corpuscular volume	82–98 FL
Mean corpuscular hemoglobin	27–31 pg/cell
Mean corpuscular hemoglobin concentration	32%–36%
White blood cell count	
Total	4,500–11,000/mm^3
Differential (in number of cells/mm^3 blood)	
Total leukocytes	5,000–10,000 (100%)
Total neutrophils	3,000–7,000 (60%–70%)
Lymphocytes	1,500–3,000 (20%–30%)
Monocytes	375–500 (2%–6%)
Eosinophils	50–400 (1%–4%)
Basophils	0–50 (0.1%)
Sedimentation rate	0–30 mm/h
Coagulation Studies*	
Platelet count	250,000–500,000/mm^3
Prothrombin time	12–15 s
Partial thromboplastin time	60–70 s
Activated partial thromboplastin time	35–45 s
Activated clotting time	75–105 s
Fibrinogen level	160–300 mg/dL
Thrombin time	11.3–18.5 s
Blood Chemistries	
Serum electrolytes	
Sodium	135–145 mEq/L
Potassium	3.3–4.9 mEq/L
Chloride	97–110 mEq/L
Carbon dioxide	22–31 mEq/L

Blood Test	Reference Range
Blood Chemistries (cont.)	
Blood gases	
pH	7.35–7.45
PaO_2	80–105 mm Hg
$PaCO_2$	35–45 mm Hg
Bicarbonate	22–29 mEq/L
Base excess, deficit	0 ± 2.3 mEq/L
SaO_2	98%
$Sv–CO_2$	75%
Bilirubin	
Total	0.2–1.3 mg/dL
Direct	0–20 mg/dL
Calcium	
Total	8.9–10.3 mg/dL
Free (ionized)	4.6–5.1 mg/dL
Creatinine	
Men	0.9–1.4 mg/dL
Women	0.8–1.3 mg/dL
Glucose (fasting)	65–110 mg/dL
Magnesium	1.3–2.2 mEq/L
Phosphorus	2.5–4.5 mg/dL
Phosphatase, alkaline	35–148 units
Protein (total)	6.5–8.5 g/dL
Urea nitrogen	8–26 mg/dL
Uric acid	65–110 mg/dL
Men	4.0–8.5 mg/dL
Women	2.8–7.5 mg/dL
Serum Enzymes*	
CK-MM	95%–100%
CK-MB	0%–5%
CK-BB	0%
Aspartate aminotransferase	<50 units/L
Myocardial Proteins	
Troponin-I	<0.1 ng/mL
Troponin-T	<0.1 mcg/mL
Myoglobin	
Men	20–90 ng/mL
Women	10–75 ng/mL

*Examples; regional laboratory techniques and methods may result in variations.

instrumental in patient care decisions. This is especially true in relation to the administration of anticoagulation agents, whether for long-term management, such as warfarin for the management of atrial fibrillation, or for emergency interventions, such as the use of fibrinolytic therapy during an acute MI. Coagulation studies are listed in Table 17-2.

Blood Chemistries

Mechanisms that ensure homeostatic function at the cellular and tissue level depend on the appropriate production and modulation of intracellular and extracellular electrolytes. It is important that the nurse understand normal electrolyte functions and the unique, perhaps life-threatening, situations that may occur when they are significantly abnormal. A thorough analysis of basic electrolyte chemistries is always appropriate in screening of the patient with cardiac disease, whether in the inpatient or outpatient setting. These studies are almost universally obtained during the initial clinical examination. The blood chemistries most commonly assessed are sodium, potassium, chloride, carbon dioxide, calcium, glucose, magnesium, and phosphorus. Table 17-2 provides the normal assay values for common electrolytes.

Common Electrolytes

Sodium is the most abundant cation in the body. It is essential in the maintenance of acid–base balance and osmolality of extracellular fluids as well as in the transmission of nerve impulses. It plays a pivotal role in fluid balance, and its concentration is primarily regulated by the kidneys. Significant alterations of cellular function are evident when sodium levels are lower than normal (hyponatremia) or greater than normal (hypernatremia).

Potassium is the major intracellular cation. Its role in the evaluation of cardiac patients is important because it is released when cells are damaged. It is essential for maintenance of oncotic pressure, intracellular osmolality, and acid–base balance, as well as for its role in cellular reactions. In addition, potassium is vital to the normal functioning of skeletal, smooth, and cardiac muscle. It is particularly important in the regulation of cardiac rate and force of contraction.

Chloride is another major extracellular cation. Like sodium and potassium, it plays a role in acid–base balance and is an important component in the evaluation of acid–base balance.

The carbon dioxide electrolyte is a reflection of carbon dioxide content (mainly bicarbonate), not carbon dioxide gas. In some settings, carbon dioxide is reported as bicarbonate (HCO_3^-).

Other Blood Chemistries

Calcium, like potassium, is important for cardiac function. It plays a significant role in the initiation and propagation of electrical impulses and in myocardial contractility. It is also important for blood clotting, teeth and bone formation, and intracellular energy production. Ionized calcium (free calcium) is responsible for cardiac and neuromuscular excitability. Calcium is reported as total and free (ionized) values.

Glucose levels are important to monitor with baseline laboratory studies because they reflect the nutritive status of the cell. Alterations in glucose, such as in diabetes mellitus, can provide the clinician with both diagnostic as well as prognostic information.

Magnesium is the second major intracellular cation after potassium. It is important in many metabolic processes and is necessary for the normal functioning of the neuromuscular system. It facilitates enzyme activities that help maintain protein synthesis and metabolism, carbohydrate and lipid metabolism, and nucleic acid synthesis. Alterations in normal magnesium levels are reflected in disruptions in neuromuscular activity, such as in the patient with dysrhythmia.

Phosphorus reflects levels of serum phosphate. It is controlled by the parathyroid gland and regulated in the kidneys. Phosphate is important for normal cellular function and for oxygen delivery. It is reciprocal to calcium. Abnormalities can be seen with alterations in heart rate, alterations in neuromuscular function, and reciprocal changes in serum calcium.

Serum Lipid Studies

A review of serum lipid levels is a critical part of the information needed by the nurse to assess cardiovascular risk in any given patient. Patients without a history of CAD need lipid analysis for primary prevention (ie, prevention of the development of CAD). Patients with a new or past history of CAD need lipid analysis for secondary prevention to prevent progression of known disease after a cardiac event. Standard elements of a lipid profile are total cholesterol, low-density lipoprotein (LDL) cholesterol, very–low-density lipoprotein (VLDL) cholesterol, high-density lipoprotein (HDL) cholesterol, and triglycerides. All treatment of lipids for primary and secondary prevention is based on LDL cholesterol.

In 2004, the American Heart Association (AHA) and American College of Cardiology (ACC) updated their guidelines for care of patients with a history of coronary and other atherosclerotic diseases.[5,6] Their guidelines, along with the National Cholesterol Education Program (NCEP) Adult Treatment Panel (ATP) III, set standards for identification and treatment of elevated cholesterol in patients with and without known CAD.[7] Patients who present with a suspected or confirmed acute cardiovascular or coronary event should have a lipid panel sent to a laboratory within 24 hours of presentation. The nurse is ideally positioned to advocate for this element of care.

Cholesterol, a pearly, fatlike substance, is a precursor of bile acids and steroid hormones. Most of the body's cholesterol is synthesized in the liver, but some is absorbed from the diet. The NCEP III recommends total cholesterol less than 200 mg/dL to lower the probability of CAD in patients with no history of cardiac disease, and to slow the progression of disease in patients with known CAD. Patients with known CAD should have a blood cholesterol less than 160 mg/dL.[7]

LDLs constitute 60% to 70% of the total cholesterol in the bloodstream. Based on numerous large-scale studies, LDL cholesterol is known to be directly correlated with the development of CAD and subsequent CVD in susceptible individuals. The target LDL cholesterol is less than 130 mg/dL as primary prevention and less than 100 mg/dL as secondary prevention; values less than 70 mg/dL are considered reasonable.[7]

Triglycerides originate from VLDLs. Although VLDL cholesterol is not considered to be atherogenic, elevated levels can be a marker for a genetic form of cholesterol disorder. The NCEP ATP III recommends treatment of triglycerides above 500 mg/dL because of the association between hypertriglyceridemia and pancreatitis.[7]

Standard cholesterol tests measure only the percentages of the lipid elements mentioned previously. Particles of LDL cholesterol and HDL cholesterol come in various sizes, and accumulating data suggest that particle size affects atherogenicity. Particle sizes are measured by a test called subfraction analysis, which is used most frequently as an investigative tool and a guide for therapy in patients with known CAD. This test has become increasingly available. It costs about $100 and may not be covered by insurance unless the patient has a diagnosis of CAD.

Table 17-3 summarizes the various serum cholesterol levels, and Table 17-4 describes lipid abnormalities and associated mechanisms.

▲ Enzyme Studies

Enzymes are found in all living cells and act as catalysts in biochemical reactions. They are present in low amounts

Table 17-3 Serum Cholesterol Levels

Cholesterol Level (mg/dL)	Description
Low-Density Lipoprotein	
<100 (<70)	Optimal (optional)
100–129	Near or above optimal
130–159	Borderline high
160–189	High
190 or more	Very high
Total Cholesterol	
<200	Desirable
200–239	Borderline high
240 mg/dL or more	High
High-Density Lipoprotein	
<40	Low
60 or more	High
Serum Triglycerides	
<150	Normal
150–199	Borderline high
240–499	High
500 or more	Very high

From Grundy SM, Cleeman JI, Merz NB, et al: Implications of Recent Clinical Trials for the National Cholesterol Education Program Adult Treatment Panel III Guidelines. Circulation 110:227–239, 2004

Table 17-4 Lipid Abnormalities and Associated Mechanisms

Lipid Abnormality	Mechanisms
Elevated total cholesterol	High dietary intake of saturated fat and cholesterol LDL receptor deficiency or downregulation
Elevated LDL cholesterol	LDL receptor deficiency Apoprotein B-100 genetic defect High dietary intake of saturated fat and cholesterol
Elevated triglycerides	Deficiency in lipoprotein lipase Obesity, physical inactivity, insulin resistance, glucose intolerance Excessive alcohol intake
Low HDL	Apoprotein A-1 deficiency Reduced VLDL clearance Cigarette smoking, physical inactivity Insulin resistance Elevated triglycerides Overweight and obesity Very high CHO intake (more than 60% total calories), certain drugs (beta-blockers, anabolic steroids, progestational agents)
Increased lipoprotein remnants	
(VLDL is a surrogate marker for lipoprotein remnants when Tg is more than 200 mg/dL)	Defective apolipoprotein E, seen in familial combined hyperlipidemia
Lipoprotein(a)	Level is genetically determined
Small LDL particles	Particle size is determined by level of Tg; LDL particle is denser and more atherogenic at higher levels of Tg
HDL subspecies	Low levels of HDL 2 and 3 may increase CVD risk, genetically determined vs. lifestyle and other lipid levels
Apolipoprotein B	May be potential marker for all atherogenic lipoprotein
Apolipoprotein A-1	Increased CVD risk when Apo A-1 is low
Combined dyslipidemias small, dense LDL, high triglycerides, low HDL, elevated LDL and triglycerides	Defects in VLDL and LDL receptor activities coexisting with environmental influences such as obesity, physical inactivity, diet high in saturated fat, and cigarette smoking

HDL, high-density lipoprotein; LDL, low-density lipoprotein; Tg, triglycerides; VLDL, very-low-density lipoprotein; CHO, cholesterol; CVD, cardiovascular disease

From Woods SL, Froelicher ESS, Motzer SU, et al: Cardiac Nursing, 6th ed. Philadelphia, PA: Lippincott, Williams & Wilkins, 2009.

in the serum of healthy people. However, when cells are injured, enzymes leak from damaged cells, resulting in serum enzyme concentrations greater than the usual low levels. No single enzyme is specific to the cells of a single organ. Each organ contains a variety of enzymes, and there is considerable overlap among organs in the enzymes they contain. However, the distribution of enzymes in the cells of organs is relatively organ specific. When organ damage occurs, the presence of abnormally high levels of enzymes in the serum, their distribution, and the timing of their appearance and disappearance make the clinical use of serum enzyme studies relevant.

Cardiac enzymes are enzymes found in cardiac tissue. When cardiac injury occurs, as in acute MI, these enzymes are released into the serum, and their concentrations can be measured (Fig. 17-8). Cardiac tissue enzymes are present in other organs as well, so elevation of one or more of these enzymes is not a specific indicator of cardiac injury. However, because cardiac damage does result in above-normal serum concentrations of these enzymes, the quantification of cardiac enzyme levels, along with other diagnostic tests and the clinical presentation of the patient, is routinely used for diagnosing cardiac disease, particularly acute MI.

The challenge is to identify an enzyme or "marker" that correctly identifies cardiac cell death. In essence, a cardiac marker is a surrogate for thrombus formation in the coronary arteries. An ideal marker for cardiac injury should have several important characteristics. It should be easy to measure; be inexpensive; be cardiac specific, with a direct proportional relationship between the extent of myocardial injury and the measured level of the marker (zero blood concentration in the absence of cardiac injury); have rapid serum levels after the onset of injury; and stay in the serum long enough to be measured in patients who delay seeking

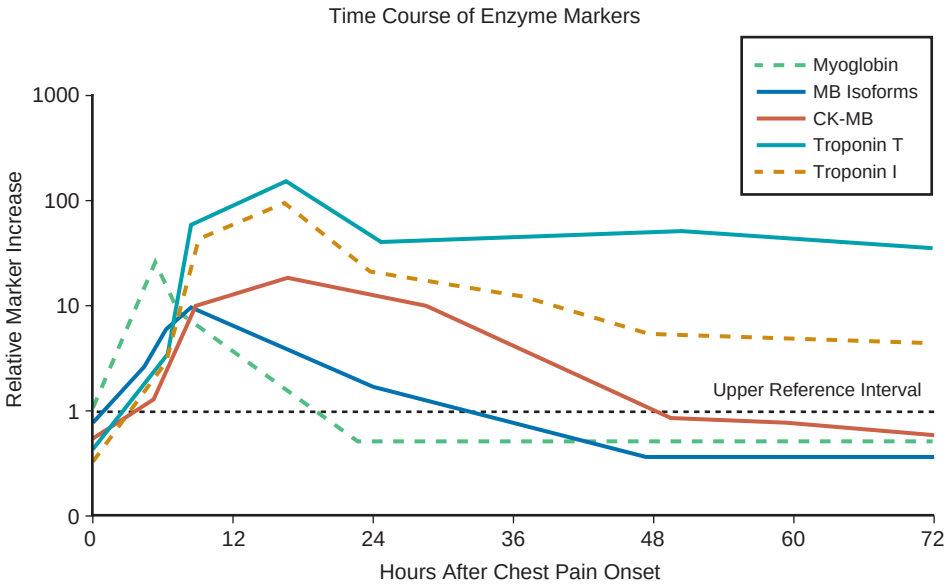


FIGURE 17-8 ▲ Peak elevation and duration of serum enzymes after acute myocardial infarction. (Data from Antman EM: Acute myocardial infarction. In Braunwald E [ed]: Heart Disease: A Textbook of Cardiovascular Medicine, 5th ed. Philadelphia, PA: WB Saunders, 1997, pp 1184–1228.)

treatment. No available biomarkers fit these criteria, but troponins, discussed later in this text, have several important features, making them the most useful of the biomarkers at present.

Creatine Kinase

Creatine kinase (CK) is an enzyme that is found in heart muscle, skeletal muscle, and brain tissue. CK values start rising 4 to 6 hours after the onset of myocardial necrosis, typically peak at 18 to 24 hours, and return to baseline at 36 to 40 hours. To rule out an MI, blood samples may be drawn every 8 hours for 24 hours. Given the wide tissue distribution of CK, the currently published guidelines of the ACC/AHA do not recommend serial measurements of total CK to diagnose MI.[8] However, total CK remains a relevant test because it provides the basis for calculation of the mass of the more specific CK enzymes.

The three CK isoenzymers routinely reported are CK-MM, CK-MB, and CK-BB, which are found in skeletal muscle, heart muscle and brain, respectively. CK-MB in amounts greater than 5% of total CK generally is considered diagnostic for myocardial damage in the presence of chest pain or other symptoms believed to represent myocardial ischemia. Negative values 10 to 12 hours after the onset of pain indicate the absence of myocardial necrosis. Patients who present more than 24 hours after the onset of symptoms may not benefit from measurement of CK isoenzymes because the levels already may have returned to normal (see Table 17-5). Cardiac disorders other than MI, and some noncardiac disorders can elevate CK and CK-MB (see Box 17-6).

▲ Biochemical Markers: Myocardial Proteins

Troponins are cardiac proteins that are released into the circulation after necrosis and rupture of myocardial cells.

Table 17-5 Molecular Biomarkers for the Evaluation of Patients With ST-Elevation Myocardial Infarction

Biomarker	Range of Times to Initial Elevation	Mean Time to Peak Elevations (Nonreperfused)	Time to Return to Normal Range
Frequently used in clinical practice			
CK-MB	3–12 h	24 h	48–72 h
cTnI	3–12 h	24 h	5–10 d
cTnT	3–12 h	12 h to 2 d	5–14 d

CK-MB, MB isoenzyme of creatine kinase; cTnI, cardiac troponin I; cTnT, cardiac troponin T.

Sources: Kushner FG, Hand M, Smith SC, et al: 2009 Focused Updates: ACC/AHA Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction (Updating the 2004 Guideline and 2007 Focused Update) and ACC/AHA/SCAI Guidelines on Percutaneous Coronary Intervention (Updating the 2005 Guideline and 2007 Focused Update): A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 54:2205–2241, 2009.

BOX 17-6 Other Causes of CK-MB Elevation

- Pericarditis
- Myocarditis
- Cardioversion
- Defibrillation
- Prolonged supraventricular tachycardia
- Hypothyroidism
- Myocardial contusion
- Collagen disorders
- Alcoholism
- Cardiac surgery

Three subforms have been identified: cardiac troponin-I (cTnI), troponin-T (cTnT), and troponin-C (cTnC). Both cTnI and cTnT are highly specific to cardiac tissue. Assays for both isoforms have become increasingly sensitive, and very minute elevations of the troponins can be detected. Both cTnI and cTnT have equal sensitivity and specificity in detecting myocardial necrosis. Some clinicians believe that very low troponin values in the absence of CK-MB elevations indicate minor myocardial damage, but may be markers for other conditions that can cause troponin elevations (see Box 17-7).[8] In critically ill patients, elevated troponins signify a worse overall outcome.

▲ Neurohumoral Hormones: Brain-Type Natriuretic Peptide

Brain-type natriuretic peptides (BNP) and pro-BNP are neurohormonal hormones released by the heart in response to decompensation. BNP and pro-BNP are sometimes used in evaluating patients with heart failure. BNP and pro-BNP are discussed more thoroughly in Chapter 20.

▲ Newer Diagnostic Markers

C-reactive protein, a newer marker of inflammation and necrosis, has been implicated as a factor that can cause disruption of fibrous cap lesions underlying acute coronary events. An acute-phase protein and marker of systemic inflammation, C-reactive protein has been shown to be elevated in patients with acute coronary syndromes. Normal values are 0 to 2 mg/dL. Serum values greater than 3 mg/dL in patients with acute coronary syndrome or greater than 5 mg/dL in patients after a coronary interventional procedure may indicate a higher risk and merit closer monitoring or more thorough evaluation.[9,10]

D-dimer is another physiological marker that may be useful in predicting the risk for cardiac events. It represents the end product of thrombus formation and dissolution that occurs at the site of active plaques in acute coronary syndromes; this process precedes myocardial cell damage and release of protein contents.[11] This marker has also been extensively studied for the diagnosis of deep venous thrombosis and pulmonary embolism. Although D-dimer can be elevated in MI and congestive heart failure, it is being used more commonly to detect other organ thromboembolic events.

BOX 17-7 Other Conditions Causing Troponin Elevations

- Sepsis/Systemic Inflammatory Response Syndrome
- Hypovolemia
- Supraventricular tachycardia
- Stroke
- Heart failure
- Pulmonary embolism
- Myocarditis
- Pulmonary hypertension
- Myocardial contusion
- Renal failure
- Left ventricular hypertrophy
- Chronic obstructive pulmonary disease

CARDIAC DIAGNOSTIC STUDIES

Cardiovascular diagnostic techniques have expanded dramatically in the past few years, especially in the area of noninvasive testing. The critical care nurse often cares for patients who undergo one or more of these procedures. Understanding the principles on which the procedures are based enables the nurse to answer questions, incorporate diagnostic findings into the patient's plan of care, and provide high-level nursing care. The critical care nurse also can decrease the anxiety of patients and their families by providing an explanation of the procedure.

▲ Standard 12-Lead Electrocardiogram

The standard ECG records electrical impulses as they travel through the heart. In patients with normal conduction, the first electrical impulse for each cardiac cycle originates in the sinus node and is spread to the rest of the heart through the specialized conduction system—the intra-atrial tracts, AV node, bundle of His, and right and left bundles. As the impulse traverses the conduction system, it penetrates the surrounding myocardium and provides the electrical stimuli for atrial and ventricular contraction. The change in electrical potential in cells of the specialized conduction system as the impulse proceeds is very small and cannot be measured from electrodes outside the body. However, the change in electrical potential of myocardial cells produces an electrical signal that can be recorded from the surface of the body, as is done with an ECG.

Impulses that originate in sites other than the sinus node or impulses that are prevented from traversing the conduction system because of disease or drugs interrupt the normal order of electrical sequences in the myocardium. An ECG

may be used to record these abnormal patterns of impulse formation or conduction. A clinician then has a visual record of the abnormal pattern from which to identify the dysrhythmia.

In addition, an abnormal ECG tracing may result from diseased myocardial cells. For example, in patients with left ventricular enlargement, impulses traversing the enlarged muscle mass of the left ventricle produce a larger electrical signal than normal. In contrast, impulses are unable to traverse myocardial cells that are irreversibly damaged, such as in MI, and no electrical signal is present in the infarcted cells of the left ventricle.

Procedure

The standard 12-lead ECG is so named because the usual electrode placement and recording device permit the electrical signal to be registered from 12 different views. The four limb and six precordial lead wires are attached to the patient as shown in Figure 17-9. For the limb leads, the recording device alternates the combination of electrodes that are active during recording of electrical signals from the heart (Fig. 17-10). This results in six standard views or leads (I, II, III, augmented voltage of the right arm [aVR], augmented voltage of the left arm [aVL], and augmented voltage of the left foot [aVF]) that are recorded in the heart's frontal plane. The six precordial leads (V_1, V_2, V_3, V_4, V_5, and V_6) are arranged across the chest to record electrical activity in the heart's horizontal plane (see Fig. 17-9).

Used routinely in intensive care unit (ICU) patients, ECGs assess dysrhythmias and myocardial ischemia or MI. An ECG is performed easily at the bedside, with the patient ideally placed in the supine position and the electrodes arranged as previously described. In some patients, chest bandages may preclude placement of the precordial leads. It is important that the patient remain still during the ECG recording so that skeletal muscle movement does not result in extraneous noise or artifact in the electrical signal. Additional horizontal plane leads may be recorded by placing electrodes on the right side of the chest to view right ventricular activity or the back of the chest to view left ventricular posterior wall activity (see Fig. 17-9).

In the clinical setting, it is important that the nurse remember where the positive electrode is located in each of the 12 leads of the ECG. The positive electrode is like a camera and provides a view of the heart from that

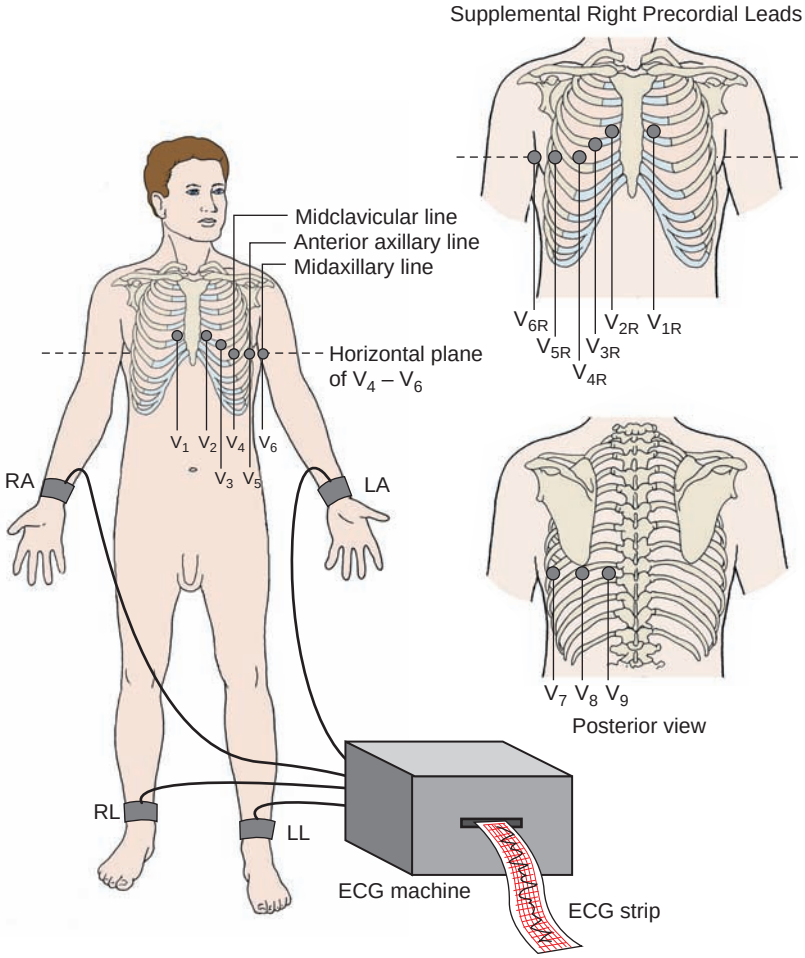


FIGURE 17-9 ▲ Electrocardiogram (ECG) electrode placement. The standard left precordial leads are V_1, fourth intercostal space, right sternal border; V_2, fourth intercostal space, left sternal border; V_3, diagonally between V_2 and V_4; V_4, fifth intercostal space, left mid-clavicular line; V_5, same horizontal line as V_4, anterior axillary line; V_6, same horizontal line as V_4 and V_5, mid-axillary line. The right precordial leads, placed across the right side of the chest, are the mirror opposite of the left leads. For the posterior leads, V_7 is placed at the left posterior axillary line, V_8 is placed at the left mid-scapular line, and V_9 is placed at the left border of the spine. All are placed on the same horizontal line as V_6.

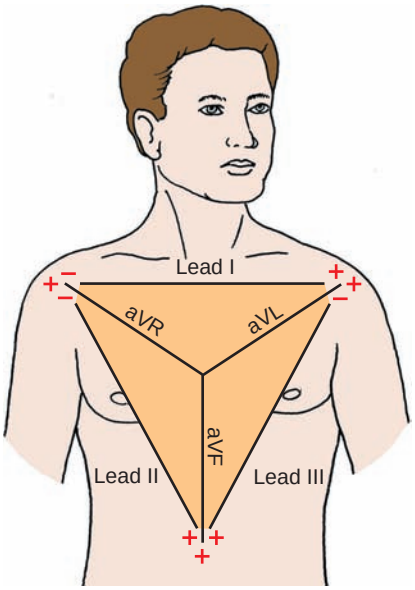


FIGURE 17-10 ▲ Frontal plane leads: standard limb leads, I, II, III, plus augmented leads aVR, aVL, and aVF. This allows an examination of electrical conduction across a variety of planes (eg, left arm to leg, right arm to left arm).

perspective. In lead I, the positive electrode is on the patient's left arm, giving a left lateral view of the heart. In leads II and III, the positive electrode is on the patient's left leg, resulting in an inferior view of the heart. For the augmented leads, the name of the lead corresponds with the placement of the positive electrode. In lead aVR, the view of the heart is poor because the positive electrode is far from the heart on the right arm. In lead aVL, the positive electrode is on the left arm, providing a left lateral view of the heart. In lead aVF, the positive electrode is placed on the patient's left leg, resulting in an inferior view of the heart. Each of the electrodes placed on the patient's chest is a positive electrode. Therefore, V_1 through V_4 provide a view of the anteroseptal wall of the heart, and V_5 and V_6 provide a view of the left lateral wall of the heart. Figure 17-11A and B shows the six limb leads in the frontal plane and the six chest leads in the horizontal plane, and the location of the positive electrode in each. The right-sided chest leads V_4R through V_6R offer the best view of the right ventricle. Leads V_7 through V_9 give the best view of the posterior wall of the heart (see Fig. 17-9). Table 17-6 summarizes the electrocardiographic leads and the corresponding views of the heart.

Nursing Assessment and Management

Critical care nurses often record an ECG in the event of a change in patient status. This change in status includes the development of dysrhythmias. Evaluation of a rhythm strip in relationship to dysrhythmias is discussed later in this chapter. Often, an ECG is obtained during episodes of chest pain before and after the administration of sublingual nitroglycerin. The ECG provides documentation of ST-segment changes associated with the pain.

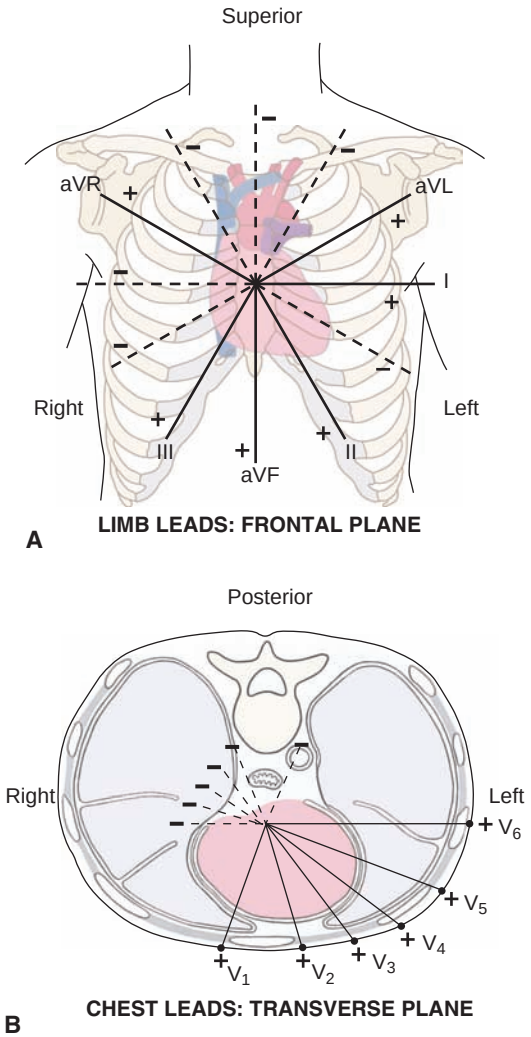


FIGURE 17-11 ▲ **A:** Positioning of the positive and negative electrodes for the limb leads in the frontal plane. **B:** Positioning of the positive electrodes on the chest wall giving a horizontal plane view. (From Bickley L: Bates' Guide to Physical Examination and Health History, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 331.)

Some patients fear being shocked by the ECG recorder. Preparatory instruction for patients should include an explanation of the manner in which the electrical impulses of the heart are recorded and that the patient will feel no sensation during the recording of the ECG.

Table 17-6 Electrocardiographic Leads and Corresponding Views of the Heart

Lead	View of the Heart
II, III, aVF	Inferior
I, aVL, V_5, and V_6	Left lateral
V_1 through V_4	Anteroseptal
Right-sided V_4 through V_6	Right ventricle
V_7 through V_9	Posterior

▲ Electrophysiologic Studies

Holter or 24-Hour Monitoring

Holter monitoring involves the use of ECG monitoring to quantify the frequency and complexity of cardiac ectopic activity that occurs during a patient's usual activities. It is a noninvasive method of assessing for dysrhythmias, response to antiarrhythmic therapy, and development of ECG changes suggestive of ischemia. Holter monitoring is indicated for patients with syncope, near syncope, dizziness, or palpitations. Patients with infrequent symptoms may not benefit from this method of gathering data because Holter monitoring is most useful in patients who are experiencing their dysrhythmias multiple times in a day. Holter monitoring is rarely used in the inpatient setting where telemetry monitoring is also available.

Holter monitoring involves placement of anterior chest electrodes that are then connected to a portable recording device, or Holter monitor. The Holter monitor is a small recording device that may be carried in a shirt pocket. Commonly, two leads are recorded continuously on tape through four or five electrodes placed on the patient's anterior chest; the electrodes are arranged so that one lead reflects the anterior wall and the other reflects the inferior wall of the heart. Data are collected from two leads simultaneously to minimize the effect of artifact on the final analysis. Continuous recording of the ECG leads is usually performed for 24 to 48 hours, and the electrodes must remain in place for the entire session. At the completion of the test, the recorder is returned for analysis.

Optimal skin preparation and electrode placement are crucial to obtaining high-quality ECG readings. Patients must bathe before placement of the electrodes. Bathing is prohibited while the electrodes are in place because contact with water can cause them to loosen. Fishnet placed over the electrodes helps keep them in place. It is necessary to caution patients against removal of the electrodes because loss of electrical contact can mimic dysrhythmias.

Holter monitoring requires significant patient participation. Patients receive instructions about keeping a diary to record medications, activities, and symptoms during the monitoring period. Usually, patients should maintain normal activities while wearing the monitor and record an entry in the diary at least every 2 hours. An understanding of the physical and emotional stressors, as well as the patient's symptoms, enhances analysis of the recordings. Compliance with record keeping is necessary. Hospitalized patients may require the assistance of nursing staff to maintain their diaries.

Event (Continuous Loop) Monitoring

In event (continuous loop) monitoring, the patient wears electrodes and a recording device, but the device does not record continuously. Instead, the patient is required to activate the recorder when a symptom occurs, and recording can go on for the duration of symptoms. The ECG is recorded on a continuous loop tape so that information before, during, and after the event is recorded. Results can be communicated to a monitoring agency by telephone, allowing for rapid analysis and feedback to patient and caregiver. Cardiac event recorders can be worn for up to 1 month.

Cardiac event recorders are most useful for patients who have relatively infrequent episodes of dysrhythmias, who are aware and able to respond to symptoms, and who are willing to wear electrodes and carry the recorder, possibly for as long as a month. As with Holter monitoring, electrode placement on clean, intact skin is crucial. Electrodes must be kept in place for the duration of the study, making immersion bathing impractical. The patient diary, a source of detailed information, requires significant participation on the part of the patient.

Implantable Loop Monitoring

The implantable loop monitor (ILR) is a device that is implanted subcutaneously and provides continuous ECG monitoring for up to 14 months. ILRs were developed to provide long-term monitoring for patients with presyncope and syncope. The major limitations of the ILR are its requirement for subcutaneous implantation and cost. The use of the device requires familiarity with implantation techniques and programming. The ILR is used for patients in whom less expensive tests, such as Holter monitoring, have failed to provide a diagnosis.

Implantation of the ILR is a surgical procedure that exposes the patient to the risk of infection and bleeding. Patients undergoing ILR implantation must understand the potential risks of the procedure. They need instruction regarding postoperative site care and use of the device.

Signal-Averaged Electrocardiography

Signal-averaged electrocardiography (SAECG) is performed in the same manner as a resting ECG, except that the heart's electrical activity is monitored for 15 to 20 minutes. The purpose of SAECG is to filter out random noise and to allow very low levels of electrical activity, called late potentials, to be recorded. This electrical activity, which is not detected by standard ECG, is thought to be coming from the cardiac substrate. Late potentials have been shown to be useful in identifying patients at risk for lethal heart rhythms.

SAECG must be performed by specially trained personnel in a very quiet room, with no extraneous electrical or electromagnetic signal-emitting equipment present. The patient must be cooperative and restful for the duration of the study.

Diagnostic Electrophysiology Study

The diagnostic electrophysiology study is a type of heart catheterization during which access to the heart is obtained through the femoral veins, or, for some more complex studies, the upper extremity (brachial, external jugular, or subclavian) veins. Multiple catheters are usually placed in one or more vessels. An arterial line is typically placed to provide continuous blood pressure monitoring during the case.

Diagnostic electrophysiology studies are performed to evaluate a broad spectrum of cardiac dysrhythmias. They can help assess the function of the sinoatrial (SA) node, the AV node, and the His-Purkinje system; determine the characteristics of reentrant dysrhythmias; map the location

of dysrhythmogenic foci for potential ablation; and assess the efficacy of antiarrhythmic drugs and devices. The basic electrophysiology protocol involves measurement of baseline conduction intervals; atrial pacing to assess SA node and AV node properties; assessment of the His-Purkinje system conductivity; ventricular pacing to evaluate for retrograde conduction and ventricular dysrhythmia potentials; and drug testing.[12]

Diagnostic electrophysiology studies are very safe. The risks associated with the procedure are similar to those encountered with cardiac catheterization and include hemorrhage, thromboembolism, phlebitis, and infection. Because most diagnostic electrophysiology studies do not require arterial puncture, the risk for serious vascular damage is very low. The risk for death due to the induction of lethal dysrhythmias is close to zero, in part because the procedural setting is uniquely equipped to terminate hemodynamically unstable dysrhythmias.

The following preprocedure preparations are necessary for patients undergoing diagnostic electrophysiology studies:

- The physician or nurse reviews the procedure so that the patient clearly understands its purpose and nature. Appropriate personnel obtain informed consent.
- Because sedation is used, the patient must be NPO for 8 hours before the procedure.
- The ordering physician or nurse practitioner must review patient medications to be sure they are to be administered on the day of the procedure. Antiarrhythmic drugs are typically withdrawn before the procedure.
- Excessive anxiety can increase catecholamine release and affect sympathetic tone, so the nurse should alert the ordering physician or nurse practitioner to signs of anxiety.

Postprocedure, the following nursing care applies:

- The nurse checks the patient's blood pressure and heart and respiratory rates frequently according to the institution's protocols.
- If the diagnostic electrophysiology study failed to induce dysrhythmias, the patient may not require telemetry monitoring. If dysrhythmias were induced, the patient does need continuous telemetry monitoring.
- The nurse monitors venous and arterial access sites for bleeding. This monitoring may include serial complete blood counts to ensure stable hemoglobin and hematocrit counts.

Tilt Table Testing for Syncope

Tilt table testing, or upright tilt table testing, refers to maintaining the patient in a head-up position for a brief period to provoke syncope, bradycardia, or hypotension. In tilt table testing, the patient is positioned on a tilt table in the supine position and tilted upright to a maximum of 60 to 80 degrees for 20 to 45 minutes. Isoproterenol may be given to provoke syncope in patients who do not become symptomatic within the testing period.

Tilt table testing is performed on patients who are suspected of having vasodepressor or vasovagal syncope. Upright posture is associated with gravitational pooling of blood, which results in a decline in central venous pressure (CVP), stroke volume (SV), and blood pressure. These effects normally lead to activation of arterial and cardiopulmonary baroreceptor reflexes that maintain blood pressure. In people susceptible to vasovagal syncope, these reflexes are reversed, resulting in bradycardia and hypotension that leads to syncope.

The patient experience during tilt table testing can be unpleasant, particularly if the patient has syncope during the study. Patients undergoing tilt table testing need to be NPO for 8 hours before the study. They need intravenous (IV) access and should be informed that they may receive a vasoactive agent such as isoproterenol.

▲ Chest Radiography

Chest radiography is a routine diagnostic test used to assess critically ill patients with cardiac disease. The test can be performed easily at the bedside in patients too ill to be transported to the radiology department. The image obtained on a radiograph that allows visualization of vascular and cardiac shapes is based on the premise that thoracic structures vary in density and permit different amounts of radiation to reach the film.

Chest radiography may be used for the evaluation of cardiac size, pulmonary congestion, pleural or pericardial effusions, and position of intracardiac lines, such as transvenous pacemaker electrodes or pulmonary artery (PA) catheters. Figure 17-12 shows the structures that can be seen on a normal posteroanterior chest radiograph.

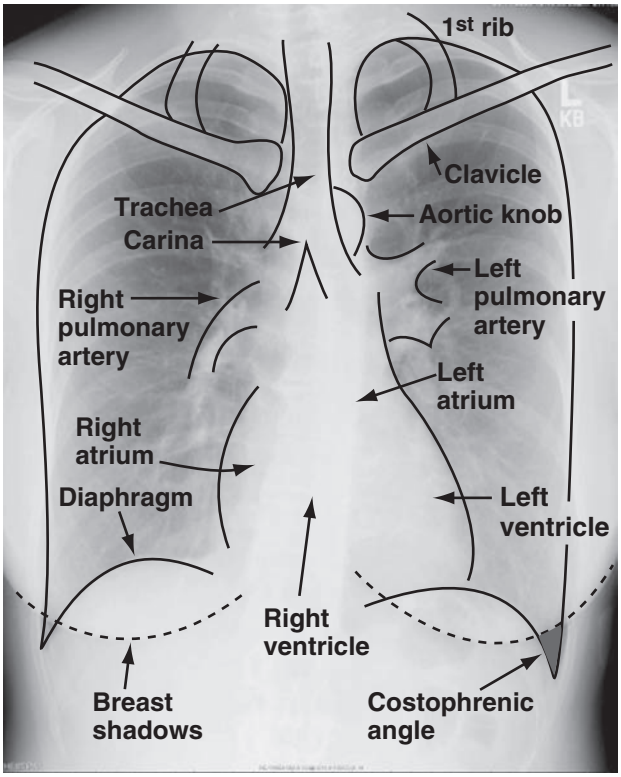


FIGURE 17-12 ▲ Outline of structures visible on normal posteroanterior chest radiograph. (Adapted from Woods SL, Froelicher ESS, Motzer SU, Bridges EJ: Cardiac Nursing, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 268.)

Procedure

Cardiac size is evaluated best in the radiology department, where the procedure can be standardized with the patient standing and the radiograph taken from posterior and lateral views at a distance of 6 feet. Portable bedside chest radiographs usually are taken only from an anterior view with the patient lying supine or sitting erect and are not standardized.

Patients undergoing radiography of the chest are instructed not to move while the radiograph is being taken. Proper positioning of the radiographic plate behind the patient is important to ensure that thoracic structures are aligned on the film. Care is taken to remove all metal objects, including fasteners on clothing, from the field of view because metal blocks the x-ray beam. Patients usually are asked to take a deep breath and hold it when the radiograph is taken to displace the diaphragm downward; this may be uncomfortable for patients who have undergone recent thoracic surgery.

Nursing Assessment and Management

The critical care nurse's role in obtaining diagnostic thoracic radiographic films often is limited to the ICU, where portable radiographs are made. With unstable patients, the nurse must decide when the film can be taken. It is important that IV lines not become tangled or loosened while one is trying to place the radiographic plate in the proper position.

Female patients of childbearing potential should have a lead drape placed over the abdomen to protect the ovaries from any radiation scatter. For the same reason, caregivers and family members should leave the patient's room when the radiograph is taken. When caregivers cannot leave the patient's bedside, a lead apron should be worn.

▲ Echocardiography

Echocardiography refers to a group of tests that use ultrasound technology to provide information about cardiac structures. Specifically, a transducer emits ultrasound waves and receives a signal from the reflected sound waves; it alternates periods of sound transmission and reception. As the sound waves are emitted and travel through tissues with a homogeneous density, such as when the sound waves move through the left ventricular wall, the signal travels in a straight line. When the density of the structures changes, such as when the waves move from the ventricular wall into the blood-filled left ventricle, the direction of the sound waves changes, and this difference is recorded by the receiver. These density changes are called interfaces, and they form the basis for distinguishing one structure from another. Ultrasound waves do not travel well through bone; thus, bony paths are avoided during the examination.

Echocardiography is most often used to assess ejection fraction, wall motion and thickness, systolic and diastolic ventricular volumes, valvular function and disease, vegetations, intracardiac masses or thrombi, and pericardial fluid. It is a helpful diagnostic tool in the presence of sudden clinical deterioration in acute MI, in which significant complications may be observed or suspected. In addition, it also may be used in evaluating function of all four cardiac valves, including calculation of gradients and orifice size, intracardiac tumors, and aortic dissection. Contrast echocardiography is a technique for improving resolution during echocardiographic studies. Agitated saline solution, injected intravenously, is used to identify intracardiac shunts. In addition, several phospholipid IV contrast agents have been developed to improve visualization of the endocardial border. These agents would be used when visualization of the endocardial border is hindered by body habitus or artifact.

The quality and usefulness of echocardiographic studies depend on the relative age of the technology being used, the skill of the technologist performing the study, the habitus of the patient, and the skill of the study's interpreter. Accuracy may decrease up to 20% in patients who are obese or who have chronic obstructive pulmonary disease or chest wall deformities. These physical features increase the distance the ultrasound waves must travel and thus increase the likelihood of artifact. Transthoracic echocardiography is of limited usefulness in investigating the left atrium and left atrial appendage because these structures are at the back of the heart.

Echocardiography is performed in a specifically designed laboratory with dim lighting and minimal sound distraction. It can also be performed at the bedside with lighting optimized to enhance the quality of the study. Patients should be able to tolerate lying flat or nearly flat. The technician asks them periodically to change position, and they should be able to turn onto their left side for several minutes at a time. In addition, they should be able to breathe in deeply or hold their breath. They do not need to be fasting.

M-Mode Echocardiography

Motion mode, or M-mode, echocardiography allows recording of amplitude and of the rate of motion of moving objects with great accuracy. It is often referred to as an "ice pick" view because it uses a single beam of sound that allows a small region of the heart to be visualized at any point in time. The four positions of the transducer depicted in Figure 17-13 are the typical views used during an M-mode echocardiogram. It provides rapid assessment of valvular motion and chamber wall thickness. The transmitter is placed on the anterior chest in an intercostal space or subcostal position to avoid bony structures.

Two-Dimensional Echocardiography

Two-dimensional (2D) images of cardiac structures can be obtained by using multiple crystals to generate a cross-sectional imaging plane. The ultrasound beam is pie shaped, resulting in a "plane" of reflected echoes. Visually, 2D echocardiography creates a cross-sectional slice of the heart from parasternal, subcostal, apical, and suprasternal positions. This approach is useful for evaluating the thickness of the left ventricular wall, left ventricular wall mass, and wall motion abnormalities.

Three-Dimensional Echocardiography

Three-dimensional (3D) echocardiography allows for the imaging and analysis of cardiac structures as they move in time and space. 3D echocardiography uses the principles of

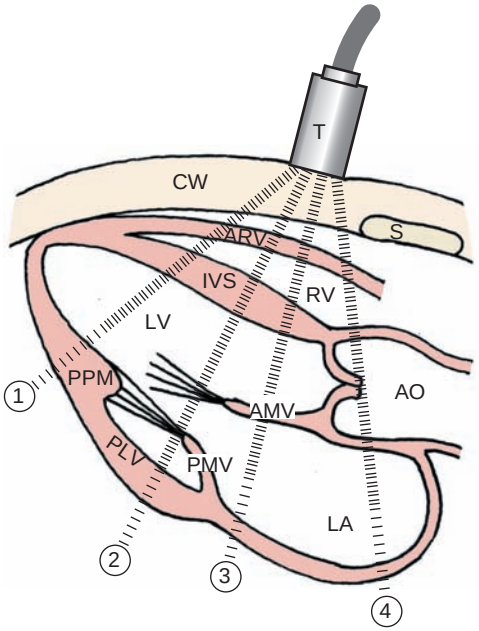


FIGURE 17-13 ▲ Echocardiographic views of the heart. A cross-section of the heart shows the structures through which the ultrasonic beam passes as it is directed from the apex (*1*) toward the base (*4*) of the heart. AMV, anterior mitral valve; AO, aorta; ARV, anterior right ventricular wall; CW, chest wall; IVS, interventricular septum; LA, left atrium; LV, left ventricle; PLV, posterior left ventricular wall; PMV, posterior mitral valve; PPM, posterior papillary muscle; RV, right ventricular cavity; S, sternum; T, transducer.

ultrasound imaging with advanced real-time reconstruction capabilities to generate a more authentic representation of the heart. Early in the evolution of 3D echocardiography, the generated images required lengthy postprocedure processing, which meant that immediate results were not available. Current technology allows for real-time images, making this modality a valuable diagnostic and treatment tool.

Doppler Echocardiography

Doppler echocardiography superimposes Doppler techniques on either M-mode or 2D images. The direction of blood flow can be assessed by measuring echoes reflected from red blood cells as they move away from or toward the transducer. This type of study is particularly useful in patients with valvular disease. Stenotic valves cause turbulence in the forward flow of blood through the heart, and regurgitant valves cause turbulence in blood flowing retrograde through the cardiac chambers. When the direction of flow is color encoded, the study is known as a color Doppler echocardiogram. Audio signals usually are recorded during Doppler studies. Contrast material may also be used in conjunction with M-mode or 2D echocardiography. Although many agents have been used as contrast material, almost any liquid injected intravenously contains microbubbles. As the microbubbles travel through the heart, they produce multiple echoes. This technique is especially useful in identifying right-to-left intracardiac shunts because of the early appearance of the microbubble echoes on the left atrium or ventricle.

Transesophageal Echocardiography

Transesophageal Echocardiography (TEE) involves the placement of a 2D transducer at the end of a flexible endoscope to obtain high-quality images of cardiac structures. Because the heart rests directly on the esophagus, the ultrasound signal travels millimeters to reach the heart. This reduces the amount of signal artifact and attenuation, yielding a clearer image. TEE is useful in patients with emphysema, obesity, and chest wall deformities. In addition, TEE provides a posterior approach to the heart; thus, it also allows for better imaging of the aorta, PA, valves of the heart, both atria, atrial septum, left atrial appendage, and coronary arteries.

Patients undergoing TEE should be NPO for 6 hours before the study. IV sedation is given, and several staff members, including technicians, nurses, and a physician, are required throughout the study. TEE takes substantially longer than a transthoracic echocardiogram and can be uncomfortable for patients. There is also a risk for esophageal perforation (1 in 10,000) associated with the procedure. Table 17-7 summarizes nursing considerations for caring for the patient undergoing TEE.

Bedside Vascular Access Testing

Obtaining vascular access in a critically ill patient frequently represents a challenge to clinicians. Portable devices have been designed for use at the bedside to locate vascular anatomy in real time, allowing placement of IV lines with great accuracy.

Intravascular Ultrasound

Intravascular ultrasound (IVUS) uses ultrasound technology to visualize the lumen and wall structure of the coronary arteries. It is an adjunct to cardiac catheterization and is discussed more fully in that section (see Cardiac Catheterization, Coronary Angiography, and Coronary Intervention).

▲ Stress Testing

Stress testing is an important tool in the evaluation of the patient with suspected ischemic CVD. Stress testing is used to assess prognosis and to determine functional capacity. Stress testing involves monitoring physiological parameters, such as blood pressure and ECG, when the heart is in a resting state and again when the heart has been stressed either by exercise or by a pharmacological agent chosen to simulate exercise. A great deal more information can be obtained by also looking at images of the heart at rest and with activity. These images can be obtained by a variety of methods, including radioactive tracers and echocardiography. Boxes 17-8 and 17-9 present indications and contraindications for stress testing, respectively. These contraindications largely relate to advanced disease states; stress testing in patients with these conditions could precipitate a catastrophic event.

Table 17-7 **Nursing Interventions for the Patient Undergoing Transesophageal Echocardiography (TEE)**

Nursing Action	Rationale
Preprocedure	
1. Evaluate patient for contraindications.	Patients with history of dysphagia or esophageal disease are not candidates for TEE.
2. Instruct patient and family about procedure.	Some discomfort may occur; patient will receive moderate sedation but will be closely monitored.
3. Ensure that documented patient history is adequate and informed consent has been signed.	Medication allergies should be noted; procedure requires consent signature.
4. Ensure that patient has been NPO for 6 h before procedure.	Aspiration precaution is vital.
5. Prepare patient for procedure.	Remove oral prosthetics, as indicated; have patient void.
6. Insert peripheral intravenous (IV) catheter.	IV access is required for routine medication administration; emergency vascular access line should be available.
7. Place patient on cardiac monitor with blood pressure and pulse oximetry.	Patient must be continually monitored during procedure.
8. Ensure that emergency resuscitation equipment is nearby, including medications, defibrillator, and suction apparatus.	Cardiac arrest precaution.
During Procedure	
1. Monitor cardiac rhythm, blood pressure, pulse oximetry, and airway patency per institutional policy.	Continuous observation is required after moderate sedation.
2. Assist physician with patient positioning and endoscope placement.	Allaying fears enhances patient cooperation.
3. Monitor for complications.	Vagal stimulation may occur with a resultant vasovagal reaction; transient tachycardia/ bradycardia and blood pressure alterations may appear; patient may experience hypoxia or laryngospasm.
4. Reassure patient throughout procedure.	Allaying fears enhances patient cooperation.
5. Document patient response to procedures.	Per institutional protocol.
Postprocedure	
1. Assess vital signs at conclusion of procedure; document per institutional policy.	Comparison with baseline is necessary to monitor sedation recovery.
2. Assist patient to position of comfort or on one side.	Position provides comfort and patent airway support.
3. Keep patient NPO until gag reflex is assessed.	Prudent, given aspiration risk.
4. If gag reflex is present, encourage patient to cough; offer lozenges or ice to soothe sore throat; keep NPO per physician order.	Interventions provide patient opportunity to clear residual secretions and obtain comfort.
5. If outpatient, instruct patient not to drive for at least 12 h.	If patient was sedated during procedure, it is best if family member or another drives patient home.
6. Instruct patient to seek care or contact physician in event of dyspnea, hemoptysis, or severe pain.	If symptoms of complications occur, patient should be reevaluated.

Fundamentally, stress testing provides information about the heart's response to activity. The heart extracts 70% of the oxygen carried by each unit of blood perfusing the myocardium. Cardiac metabolism is nearly entirely aerobic, meaning that the heart is unable to create energy in anaerobic conditions or when the oxygen supply is insufficient. Therefore, an increase in oxygen demand on the heart requires additional coronary artery blood flow to meet the new metabolic requirements. Narrowing of the coronary arteries can limit the amount of blood delivered to a portion of the myocardium, resulting in ischemia.

BOX 17-8 Indications for Stress Testing

1. Differential diagnosis of chest pain (ie, evaluation of patients with suspected ischemic heart disease)
2. Assessment of the level of exercise at which ischemic manifestations occur in a patient with known ischemic heart disease
3. Evaluation of therapy for dysrhythmias and angina
4. Evaluation of functional disability secondary to organic heart disease (eg, valvular heart disease)
5. Risk stratification of the asymptomatic patient with multiple risk factors for ischemic heart disease

BOX 17-9 Contraindications for Stress Testing

1. Recent MI (4 to 6 weeks), except when submaximal protocols are used (65% of maximum predicted heart rate or symptom-limited exercise stress testing before hospital discharge)
2. Unstable angina or angina at rest
3. Rapid ventricular or atrial dysrhythmias
4. Advanced atrioventricular block, unless chronic
5. Uncompensated congestive heart failure
6. Acute noncardiac illnesses
7. Severe aortic stenosis
8. Blood pressure greater than 170/100 mm Hg before the onset of exercise

Exercise Stress Testing

Patients must be ambulatory to participate in exercise stress testing. Functional limitations due to orthopedic, neurological, pulmonary, or peripheral vascular issues can affect the patient's ability to complete the stress test protocol.

Procedure

In exercise stress testing, the heart rate is monitored continuously while exercise is performed on a treadmill or bicycle. The patient is exercised to a target heart rate that is 85% of the maximum predicted for that individual. By convention, the maximum predicted heart rate is calculated as 220 beats/min for men (210 beats/min for women) minus the patient's age in years. Attainment of maximum heart rate is a good prognostic sign.

The blood pressure, heart rate and rhythm, ECG, presence or absence of symptoms, and workload performed are monitored. Workload is determined by metabolic equivalents (METs) or by the double product (blood pressure × heart rate). METs are defined as the resting respiratory oxygen uptake for a 70-kg, 40-year-old man, and 1 MET is equivalent to 3.5 mg/min/kg of body weight. Work activities are calculated in terms of METs. Stair climbing, for example, is approximately equivalent to 4 METs. A reasonable workload for most active adults is 10 METs. Although the double product correlates well with the degree of CVD, it is less often used as a measure of workload.

Initial ECG readings are performed before exercise to document a baseline, with continuous 12-lead monitoring used throughout the study. The lead system is the same as used for the standard 12-lead ECG. However, it is necessary to move the limb leads to the torso to prevent arm or leg movement during exercise from interfering with ECG recording. Skin preparation and electrode attachment require careful attention to permit interpretable recordings during maximal exercise. It may be necessary to wrap material or fishnet over the electrodes and cables on the patient's torso to reduce movement artifact. Treadmill stress testing without a concomitant imaging modality is less reliable in females; therefore, exercise stress testing in females is typically paired with radionuclide or echocardiographic imaging. Baseline ECG abnormalities, such as left bundle branch block (LBBB), make analysis of exercise-induced ECG changes more complex.

The treadmill protocol chosen should reflect the patient's physical capacity and the purpose of the test. All treadmill protocols are multistaged, using increments in time, speed, and elevation of the treadmill platform. The protocol is selected based on the condition of the patient and the purpose of the study. For example, the Ellestad protocol uses small increments in workloads of shorter duration, whereas the Bruce protocol uses larger workload increments of longer duration. The Ellestad protocol may be better suited to a patient with less exercise tolerance. The Bruce protocol is among the most popular in use for reasonably functional people; a large body of diagnostic and prognostic data supports its use.

Patients who have not previously undergone exercise testing should be allowed briefly to practice walking on the treadmill or riding the bicycle. Before starting the test, a resting baseline ECG and blood pressure are obtained with the patient in sitting and standing positions. The ECG and heart rate are monitored continuously throughout the test, and blood pressure is monitored every few minutes. The monitoring continues for at least 6 to 10 minutes into recovery, or until symptoms or blood pressure and ECG changes have resolved to document the patient's return to baseline values.

It is mandatory that emergency personnel and equipment be available in areas where exercise testing is performed. Indications of myocardial ischemia during exercise testing are the development of ST-segment depressions, chest pain or the anginal equivalent, or failure to increase blood pressure to 120 mm Hg or the sustained decrease of 10 mm Hg with progressive stages of exercise. The test is terminated for any of the following reasons:

1. The target heart rate is reached.
2. The patient is unable to continue exercising because of shortness of breath, fatigue, claudication, or severe chest pain.
3. There is ECG evidence of complete AV block, ventricular Tachycardia (VT), or premature ventricular contractions (PVCs).
4. There is ECG evidence of ST-segment changes consistent with ischemia or infarction.
5. The patient's systolic blood pressure is greater than 220 mm Hg or diastolic blood pressure is greater than 120 mm Hg during exercise, or the patient's blood pressure drops below baseline at any time during the exercise protocol.

In the absence of ECG evidence of ischemia or the development of life-threatening dysrhythmias, every effort should be made to reach the maximum predicted heart rate to improve the diagnostic accuracy of the test. When the maximum predicted heart rate is not reached, the diagnostic reliability of the study is low. Exercise stress test results are considered reliable only if patients reach the maximum predicted heart rate value (85% of their maximal predicted effort).

Nursing Assessment and Management

Adequately preparing patients for the stress test maximizes the information obtained from the study. Patients should be NPO for 4 to 6 hours before the test to minimize blood diversion to the gastrointestinal tract, which decreases available coronary blood supply. In particular, they should not drink caffeine-containing beverages because of the effect of caffeine on the heart rate. Beta blockers blunt the heart rate response to exercise and may prevent achievement of the maximum predicted heart rate, so they should be withheld on the day of the test. Digitalis may also be withheld because of its negative chronotropic effects. Badly deconditioned patients, or those with comorbidities that could affect their ability to ambulate, may not be able to complete the required exercise. Appropriate attire, including comfortable walking shoes, is necessary to maximize patient comfort and performance.

The critical care nurse may be responsible for explaining the general format of the exercise test to the patient and family. It is important that patients understand why the test is indicated and what will be expected of them. The nurse reassures patients that someone will observe them closely

throughout the test and encourages them to express any concerns before, during, and after the procedure. Patients should also understand that they may have to continue exercising after angina develops but will not be expected to exercise more than is safe.

Pharmacological Stress Testing

Pharmacological stress testing is performed in patients who are unable to bike or walk on a treadmill. Patients referred for stress testing may have limitations that prevent them from performing adequate physical exercise. This has led to the development of alternative methods of simulating the effect of exercise on the heart using adrenergic agents, such as dobutamine, or vasodilators, such as adenosine or dipyridamole. These studies require no activity on the part of the patient. There is continuous monitoring of the ECG, along with frequent blood pressure measurements. In addition, an imaging modality, such as echocardiography or nuclear imaging, is always used.

Pharmacological agents used include several drugs. Dobutamine increases myocardial oxygen demand by increasing contractility, heart rate, and systemic blood pressure. Because dobutamine has some heart rate–blunting characteristics, supplemental atropine may be required to achieve target heart rate values. With the infusion of dobutamine, coronary blood flow increases up to twofold in normal coronary arteries but less so in arteries with flow-limiting lesions. Vasodilators, such as adenosine and dipyridamole, also cause an increase in coronary blood flow. They simulate the effects of exercise on the heart by producing arteriolar and coronary artery vasodilation. Regadenoson is a selective adenosine agonist that incites coronary vasodilation, producing maximal effects quickly, and maintaining hyperemia for the duration of perfusion imaging.[13] Although information on functional capacity is not obtained during pharmacological stress testing, it is expected to provide a reasonable equivalent to that gathered during physical exercise.

ECG changes, secondary to the infusion of dobutamine, adenosine, and dipyridamole, have a very low sensitivity for the detection of significant CAD. Therefore, pharmacological stress testing is always accompanied by a nuclear or echocardiographic imaging modality to increase the sensitivity of the study.

Nuclear Imaging with Stress Testing

Noninvasive, rapid, and accurate imaging of cardiac structure and function using radiotracers is a routine part of inpatient assessment of patients with known or suspected CVD. Broadly speaking, this is known as radionuclide cardiac imaging. Single-photon emission computed tomography (SPECT) and positron emission tomography (PET) are both widely available types of radionuclide imaging. SPECT and PET cameras capture the photons emitted by infused radiotracers and provide information on the magnitude and location of the uptake. The images are ECG gated, or collected in synchrony with ongoing ECG monitoring, so that the final data interpretation can be presented in the context of the full cardiac cycle of contraction and relaxation. In SPECT imaging, the final result is also referred to as myocardial perfusion imaging (MPI). PET is discussed at greater length later in this chapter (see Positron Emission Tomography).

Nuclear imaging is combined with exercise or the infusion of a pharmacological agent in a variety of protocols. Protocols may involve the injection of several radiotracers over several hours, with imaging performed 24 hours later. The purpose of the protocols is to obtain information on the heart at rest and with stress. Protocols vary widely because of patient comorbidities, patient size, and available personnel and equipment.

Myocardial Perfusion Imaging

MPI uses SPECT technology to look at coronary blood flow, giving information about the location, quantity, and severity of cardiac disease. MPI uses radiopharmaceutical agents that, once injected into the venous bloodstream, accumulate in viable myocardium in proportion to the blood flow to a particular area. After injection of the tracer, the SPECT camera is used to record an image of radioactive counts from the entire myocardium.

During MPI studies, images are obtained of the heart at rest and with stress. Typically, at rest in the normal heart, the radiotracer is spread uniformly throughout the myocardium, and the camera reads counts equally from throughout the myocardium. During exercise, a similar scan is obtained in patients without significant coronary artery stenosis because blood flow increases uniformly to meet myocardial oxygen demands.

However, in patients with significant CAD, the image obtained during exercise is altered. The amount of coronary blood flow is limited in stenotic arteries, and the quantity of tracer in myocardial segments supplied by stenotic arteries is diminished or absent compared with segments supplied by nonstenotic arteries. An area of decreased tracer uptake during exercise compared with at rest is known as a reversible perfusion defect. Reversible perfusion defects are suggestive of impaired blood flow, or ischemia, in a region. In patients with previous infarction, decreased uptake may be present on both the rest and exercise scans in the infarcted segments; this pattern is known as a fixed perfusion defect and usually signifies nonviable myocardium. It is possible for patients to have fixed perfusion defects in some myocardial segments, reversible defects in others, and normal perfusion in the remaining segments.

Because of the many patients who are physically unable to exercise, pharmacological agents may be used to mimic the heart's response to exercise. Vasodilating agents, such as dipyridamole, adenosine, and dobutamine, administered IV mimic exercise conditions in the heart by dilating nonstenotic coronary arteries. Coronary blood flow is increased preferentially through normal, nonstenosed arteries; this results in relative hypoperfusion in myocardial segments supplied by stenosed coronary arteries. A radiotracer injected during the peak action of the pharmacological agent produces images similar to those seen with exercise. As of this writing, only dipyridamole is approved by the U.S. Food and Drug Administration (FDA) for use in perfusion imaging.

PROTOCOLS. Three radioactive tracers, thallium-201, technetium (Tc)-99m, and sestamibi are approved for perfusion imaging. Characteristics of the three agents differ and are responsible for the varying imaging protocols used.

Thallium Protocol. The cardiac half-life of thallium is approximately 7.5 hours, meaning that 50% of the tracer still is present in myocardial cells 7.5 hours after it is administered. It also redistributes readily, so thallium in normally perfused areas moves to previously underperfused areas after the myocardial blood flow demands in that territory have decreased. The standard protocol for thallium perfusion studies begins first with the exercise portion; thallium is injected at the peak of exercise, and imaging starts within 5 minutes of injection. The rest portion is obtained 2 to 4 hours later. Because of redistribution, no additional thallium is required. However, in some patients with perfusion defects on both the rest and exercise scans, significant redistribution may not occur, and it is recommended that an additional dose of thallium be administered.

Sestamibi Protocol. Perfusion imaging with sestamibi typically begins with the rest scan. Because significant uptake also occurs in the liver, imaging is delayed for approximately 60 minutes. This delay allows sestamibi to be cleared from the liver but not the heart. In addition, a glass of milk or small fatty meal is taken shortly after radiotracer injection to enhance hepatic clearance. A second dose of sestamibi is administered during peak exercise, and the exercise scan is obtained 60 minutes after injection, again allowing time for hepatic clearance. Because sestamibi redistributes very slowly, the image obtained 60 minutes after peak exercise reflects the perfusion conditions at the time of injection. Initially, perfusion studies with sestamibi were performed on two different days, but it now is customary to complete both portions of the study in one day. It has been shown that exercise sestamibi myocardial perfusion SPECT can provide incremental prognostic information in patients who have not suffered a previous MI or undergone cardiac catheterization and who are determined to be at low risk.

Nursing Assessment and Management

All the directions and precautions that pertain to exercise ECG also apply to exercise radionuclide imaging. When pharmacological agents are used in place of exercise, minor side effects, such as flushing, headache, and nausea, may occur. Serious side effects due to the radiotracer are extremely rare. Medications to counteract serious side effects should be readily available. Some patients who receive sestamibi report a metallic taste several minutes after injection. Patients often are anxious about the radiation involved and the appearance of the equipment. It is important for the nurse to allay these anxieties.

Table 17-8 outlines some of the tests that are used to detect the presence of myocardial ischemia.

Radionuclide Angiocardiography

A radionuclide ventriculogram or multigated acquisition (MUGA) is a precise means of calculating both right and left ventricular ejection fraction. MUGA scan has, for many years, been the gold standard for measuring ejection fraction, but other technologies, such as cardiac magnetic resonance imaging (MRI), echocardiography, and angiography, can now provide equally useful information. MUGA is performed by labeling the patient's red blood cell pool with a radioactive tracer, usually technetium 99m, and measuring

Table 17-8 Diagnostic Tests Used to Detect Myocardial Ischemia

Procedure	Abnormal Findings	Special Considerations
Standard 12-lead electrocardiogram (ECG)	Transient ST-segment and T-wave changes in patients with chest pain at rest or of prolonged duration	
Holter monitoring	Transient ST-segment and T-wave changes occurring at rest or with activity	Only two ECG leads monitored
Stress echocardiogram	Segmental wall motion abnormality associated with echocardiogram obtained during exercise	May be used in patients with ventricular conduction defects Pharmacological agents may be used in patients who cannot exercise
Exercise ECG	Transient ST-segment and T-wave changes occurring with exercise	Cannot be used in patients who are unable to exercise or who have left bundle branch block (LBBB) or paced rhythm Does not provide good information on the location of the coronary artery disease
Radionuclide perfusion stress study	"Cold spot" image or perfusion defect associated with scan obtained during exercise	May be used in patients with ventricular conduction defects Pharmacological agents may be used in patients who cannot exercise
Online ischemia analysis	Myocardial ischemia dynamic analysis (MIDA)* analyzing eight leads to detect ST-segment levels indicating ischemia and QRS complex changes corresponding to infarct evolution	Noninvasive Hastens clinical decision making Graphic trends monitored online Reocclusion readily identified Helps differentiate chest pain related to ischemia from nonischemic symptoms

*MIDA CoroNet, Hewlett-Packard, Andover, MA, Product Literature.

radioactivity with a gamma camera positioned over the chest. The number of counts recorded per unit of time is proportional to the blood volume moving through the cardiac chambers. Information is collected on cardiac wall motion, dilation, and wall thickness.

A *first pass MUGA* is performed specifically when information is required regarding right ventricular function. In this study, the gamma camera is positioned to begin counting before the radioactive tracer is injected. That way, when the venous blood makes its first pass through the right side of the heart, information can be obtained on right ventricular function. Once the tracer completes a circulatory cycle, leading to labeled blood in both the right and left ventricles, right ventricular function is obscured by the left ventricle.

Nurses caring for patients who have undergone radionuclide imaging should be aware of precautions; this information is available through the radiation safety department of their institution. The length of time that any precautions may be necessary is related to the half-life of the radiotracer used. In general, nurses who are pregnant should avoid caring for patients for 24 to 48 hours after the study, and all nurses should wear gloves when handling body fluids during the 24- to 48-hour period.

Stress Echocardiography

There are several important advantages to the use of echocardiography as the imaging modality with stress testing. The echocardiogram identifies regional wall motion abnormalities, which are the final result of myocardium that is poorly perfused, secondary to CAD. Echocardiograms can be read immediately, do not involve ionizing radiation, and are more cost effective than nuclear imaging. The disadvantages of stress echocardiography include the difficulty in obtaining quality images secondary to the experience of the technologist and because of the patient's body habitus. Images must be obtained both at rest and at peak exercise for comparison.

Stress echocardiography can be used with exercise stress testing or pharmacological stress testing. A baseline 2D echocardiogram is performed before exercise is performed or the drug is infused. (For pharmacological stress testing, dobutamine or dipyridamole is used as the provocative agent.) The imaging is continued through and for 10 minutes after stopping the exercise or drug infusion. The echocardiogram is examined for wall motion abnormalities indicative of poor regional myocardial perfusion. A study is considered positive if, after exercise or drug infusion, new wall motion abnormalities are detected.

▲ Computed Tomography

Computed tomography (CT) scanning is a noninvasive technique used to evaluate the heart and its surrounding structures. CT scanning involves passing x-ray beams through a patient's body while a detector gathers and records images generated by the beams. Computers reconstruct the images into 2D or 3D images that provide strikingly detailed views of the anatomy. See Figure 17-14. Cardiac CT scanning is used to detect structural diseases of the heart, including congenital anomalies and aneurysms.

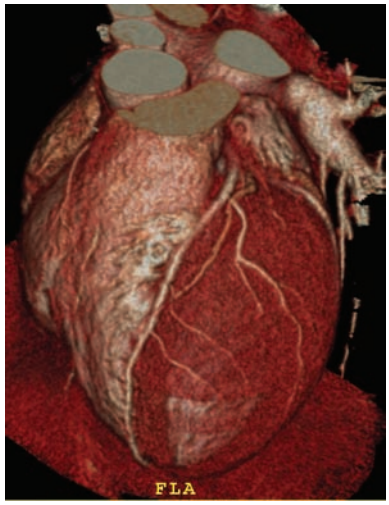


FIGURE 17-14 ▲ Multislice CT image of the heart, a 64-slice cardiac computed tomography scan of the heart, frontal plane.

Coronary artery calcium (CAC) is an indicator of atherosclerosis that can be assessed by CT. Atherosclerotic plaque evolves though stages where instability and rupture may be followed by calcification. Although arterial calcification indicates a later stage of vascular disease, its absence does not exclude the presence of noncalcified plaque that is vulnerable to rupture.[14] A CAC score (CACS) of zero, consistent with no detectable CAC, may be falsely reassuring as this measurement does not identify plaque that has yet to be calcified. There is no direct association between the CACS and degree of stenosis of atherosclerotic lesions. In other words, it is possible to have a very high CACS with no flow limiting intravascular lesions, or a very low CACS with significant flow limiting intravascular lesions. CACS is most useful in determining a patient's CVD event risk by virtue of its strong association with total coronary atherosclerotic disease burden.[9] The Agatston Score is used to measure the amount of calcium detected during a coronary CT. Scores of less than 10, 11 to 99, 100 to 400, and greater than 400 are used to categorize individuals into groups having minimal, moderate, increased, or extensive amounts of calcification, respectively. Although it has not replaced coronary angiograms as the gold standard of detection and quantification of CAD, the CACS adds independent prognostic information of all-cause mortality when used in conjunction with risk assessment algorithms (such as the Framingham risk score).[15]

Coronary computed tomography angiogram (CCTA) is a noninvasive method to visualize the actual lumen of the coronary arteries. CCTA can identify narrowings and stenoses of coronary arteries, whether or not calcium deposits are present. Patients undergoing ECG-gated CCTA may need heart rate modulation. Optimal heart rates are 55 to 60 beats/min and can be safely achieved in most patients with medications, particularly β-blockers. Patients require placement of an IV line for contrast administration. Monitoring for contrast-induced allergic reactions and renal toxicity is also necessary.

▲ Magnetic Resonance Imaging

MRI allows high-resolution assessment of cardiovascular anatomy, function, blood flow, metabolism, and perfusion. It permits assessment of cardiac structure and function at rest or during exercise or pharmacological stress testing. MRI is used in the diagnosis of CAD, coronary artery bypass graft disease, cardiomyopathy, valvulopathy, congenital heart disease, cardiac masses, intracardiac thrombi, and diseases of the pericardium. Myocardial viability studies can be performed using MRI; thus, viable or ischemic tissue can be distinguished from scarred or infarcted tissue. Centers performing electrophysiology procedures use MRI to map pulmonary veins before an atrial fibrillation pulmonary vein ablation procedure. Atrial septal defects can be characterized before the percutaneous placement of an occlusion device.

Cardiovascular MRI has other advantages. It is useful in patients unable to tolerate iodine-based contrast material because of allergy or renal insufficiency. Gadolinium, the MRI contrast medium, can cause both allergic reactions and nephrotoxicity, but the frequency of both complications appears to be less than that associated with iodinated contrast material. In addition, cardiovascular MRI with a stress testing protocol is being used to provide a comprehensive evaluation of cardiac structure, wall function, valvular function, myocardial perfusion, angiography, and viability. The provocative agent used for MRI stress testing can be adenosine, dobutamine, or dipyridamole.

Coronary MRI remains challenging because of several unique issues, including the small size of coronary arteries and their nearly constant motion during respiration and cardiac cycles. MRI requires expensive equipment that may not be available at all centers.

Obesity may be a contraindication for MRI (or magnetic resonance angiography) because the patient is required to fit into a fixed-sized tunnel within the scanner. The patient must be able to lie flat and remain composed despite confinement and loud noises generated by the scanner. Non–MRI-compatible aneurysm clips, implanted devices (including implanted cardiac defibrillators and pacemakers), and other body metal are all contraindications to MRI scanning. Patients with a history of metalworking may have metal shards in their eyes, making them unsuitable for MRI scanning. Contraindications to MRI are listed in Box 17-10. Although tattoo dye may contain metallic oxides that heat up during MRI, tattoos are not contraindicated in MRI scanning. Breath holding is required to avoid breathing artifacts at intervals during MRI, so cardiac MRI scanning may not be appropriate for patients who are unable to hold their breath. An IV line is required for instilling the contrast medium. Central catheters are not used because of the high pounds per square inch injection pressure used.

BOX 17-10 Contraindications to Magnetic Resonance Imaging

Absolute	Relative (Individual Assessment Required)
Cardiac pacemaker	Prosthetic joints
Aneurysm clips	Certain foreign objects in body (eg, dental braces)
Epicardial pacing wires	Nonmetallic prosthetic heart valves
Metal prosthetic heart valves	Surgical staples
Implanted cardioverter–defibrillator	Coronary stents (if recently deployed)
Implanted infusion pumps	
Cochlear implants	
Metal intrauterine devices	
Metal debris (eg, bullets, shrapnel)	

▲ Positron Emission Tomography

PET has significantly contributed to the knowledge of cardiac physiology and metabolism. It detects coronary stenoses (perfusion) and assesses myocardial viability (metabolism). PET scanning is the gold standard for testing myocardial viability.

Rubidium-82 and nitrogen-13–labeled ammonia are tracers used in evaluating regional myocardial blood flow. Fluorodeoxygenase (FDG) and carbon-11–labeled acetate are used for evaluating glucose and fatty acid metabolism, respectively. If perfusion testing with rubidium-82 and nitrogen-13 ammonia demonstrates decreased blood flow, and metabolic testing with FDG and carbon-11 acetate demonstrates absent metabolic activity, then that region of myocardium is considered nonviable. That is, there is a matched decrease in flow and metabolism. If, on the other hand, the flow appears reduced by the perfusion tracers but the metabolic activity is preserved, that region of myocardium is considered ischemic and viable. This would indicate a mismatch of flow and viability, and it would direct the patient's treatment to an intervention that would restore flow to a viable area of tissue.

The patient should be NPO for 6 hours before the study. Caffeinated beverages should be restricted for 24 hours before the procedure.

▲ Cardiac Catheterization, Coronary Angiography, and Coronary Intervention

During cardiac catheterization and related procedures, radiographic contrast is injected into the chambers of the heart and the coronary arteries under fluoroscopic guidance. These studies are commonly performed and are well established as the gold standard for evaluating the coronary artery lumen. Intracoronary lesions may be targets for a variety of interventions, including angioplasty and stenting, or coronary artery bypass. Coronary anomalies and other disease states, including aneurysms and myocardial bridging, can also be seen. Coronary interventions are performed based on the information gained during the diagnostic cardiac catheterization.

The major limitations related to cardiac catheterization involve cost, experience of the operator, level of risk afforded, and ability to determine whether an identified lesion can cause ischemia. The cost of cardiac catheterization, which is in the thousands of dollars, is much greater

than for noninvasive modalities. Extensive data indicate that the physician operator must perform at least 75 procedures annually to maintain the skills necessary to perform a safe and interpretable procedure. Although this procedure can locate blockages in the coronary arteries, more information may be required to understand the ischemic potential of a given lesion before an angioplasty is performed.

Patients undergoing cardiac catheterization require careful preprocedure evaluation, including a recent history and physical examination to identify a history of contrast media allergy as well as a recent set of laboratory studies, including a complete blood count, prothrombin and partial thromboplastin time, International Normalized Ratio, and chemistry panel (serum potassium, creatinine, and blood urea nitrogen levels). Women who are premenopausal and could be pregnant must have pregnancy tests performed within 48 hours of the procedure. The patient must also be NPO for at least 8 hours before the procedure; appropriate medications may be taken with a sip of water on the morning of the procedure. Patients need IV line placement, and consideration may be given to insertion of a Foley's catheter if the patient may have difficulty urinating postprocedure. Patients must be able to lie still and almost flat on a procedure table for the duration of the examination. For nursing considerations for the patient undergoing cardiac catheterization, see Box 17-11. After the procedure, the patient requires careful monitoring of vital signs (blood pressure, heart rate, and respirations with pulse oximetry). The percutaneous entry site of the procedure needs close monitoring for signs of bleeding. Any bleeding or hematoma formation must be managed to prevent serious vascular complications, including retroperitoneal bleeding. IV fluids after the procedure promote elimination of the renal-toxic contrast media and protect the patient from hypotension due to dehydration or increased vagal tone during potentially painful portions of the recovery. Bed rest for several hours after an arteriotomy is mandatory to allow the site to stabilize and further protect the patient from vascular bleeding complications. A summary of patient teaching for patients undergoing cardiac catheterization can be found in Box 17-12.

IVUS is an adjunctive technique performed in patients undergoing cardiac catheterization. IVUS uses ultrasound technology to obtain information regarding the lumen and wall structure of the coronary artery. It permits detailed cross-sectional imaging of coronary arteries and allows for a risk assessment of individual lesions. It is frequently performed in conjunction with coronary angiograms to determine lumen measurements and characteristics, including plaque morphology and burden. The information obtained from IVUS can be used to determine the need for coronary angioplasty or stenting. It is also used to evaluate the final outcome from coronary angioplasties with and without stenting.

The standard method for determining when to use angioplasty to treat an intracoronary lesion is angiography alone. However, determining which lesions cause ischemia can be difficult, and coronary angiography may under- or overestimate a lesion's functional severity. Fractional flow reserve (FFR) is a measurement that is obtained to help determine the ischemic potential of coronary stenoses. FFR is performed in the cardiac catheterization laboratory in conjunction with angiography and is defined as the ratio of maximal blood flow in a stenotic blood vessel compared with normal maximal blood flow. A pressure-sensor guidewire is threaded beyond the lesion in question, and the pressure gradient across the blockage is measured at peak hyperemia, usually induced by

BOX 17-11 NURSING INTERVENTIONS

For the Patient Undergoing Cardiac Catheterization

Preprocedure

- Explain procedure to patient and family.
- Verify that the patient has taken nothing by mouth for at least 6 hours before the procedure except prescribed medications as advised by the physician.
- Ensure that ordered preoperative laboratory studies have been completed and results are available.
- Verify patient, identify allergy information; alert physician if patient is allergic to radiographic dye, medications, or specific foods.
- Ensure that informed consent has been obtained.
- Establish intravenous (IV) access per institutional protocol or physician order.
- Place patient on cardiac monitoring system with blood pressure and pulse oximetry monitoring.
- Provide supplemental oxygen as ordered/indicated.
- Premedicate patient per physician order.
- Obtain vital signs before transfer to catheterization laboratory.

During Procedure

- Continually assess patient vital signs, oxygenation, level of consciousness, and cardiac rhythm per institutional protocol.
- Alert attending physician to significant changes in vital signs, oxygenation, and presence of malignant cardiac dysrhythmias (eg, premature ventricular contractions, ventricular tachycardia, ventricular fibrillation).
- Be prepared to initiate cardiac resuscitation with emergency equipment and medications.

Postprocedure

- Ensure that patient vital signs are stable before transfer.
- Check catheterization site dressing for bleeding and integrity.
- Check distal pulse below catheterization site; if femoral site was used, check distal pulse, extremity color, capillary refill, and neurosensory status.
- Keep extremity straight and instruct patient not to bend leg or arm.
- Maintain IV infusion per physician order or institutional protocol.
- Maintain supplemental oxygenation support as ordered or indicated.
- Encourage oral fluids as ordered.
- Check patient's coagulation status per institutional protocol before sheath removal.
- When catheter is removed:
  - Apply direct pressure over invasive site for 20 to 30 minutes to prevent bleeding or apply commercial hemostatic compression device per institutional protocol.
  - Check distal extremity for pulse, color, capillary refill, and sensorium.
  - Remind patient to lie flat for 4 to 6 hours per institutional protocol.
  - Check site dressing every 4 to 6 hours for bleeding and integrity.

BOX 17–12 TEACHING GUIDE Cardiac Catheterization

Preprocedure

- Instruct the patient not to take anything by mouth for at least 6 hours before the procedure, except prescription medications as advised by the physician to reduce the chance of nausea and vomiting during the procedure.
- Tell the patient that an IV line will be placed to allow fluid and medication administration before, during, and after the procedure.
- Tell the patient that preoperative medication will be given before transport to the catheterization laboratory.
- Inform the patient that only a patient gown will be worn during the procedure.
- Advise the patient that the catheterization laboratory is usually cool, and the procedure table is firm and may be uncomfortable after a prolonged time.
- Explain that the patient may be asked to turn the head, hold the breath, or cough during the procedure.
- Advise that some discomfort may be experienced during the procedure but that local anesthesia will be administered to minimize pain.
- Inform the patient that a cardiac monitor will be used for the duration of the procedure and for a few hours after the procedure.

During Procedure

- Instruct the patient to inform the physician and team of any chest pain experienced.
- Remind the patient to lie still.
- Reassure the patient and allay anxiety.
- Encourage and answer the patient's questions.

Postprocedure

- Remind the patient to lie still and keep the extremity straight.
- Instruct the patient to verbalize any chest pain or shortness of breath if present.
- Tell the patient when the catheter sheath is due for removal.
- Encourage the patient to take oral fluids as ordered.
- Advise the patient that the physician will review and explain the catheterization findings.
- Tell the patient that lying flat for several hours after the procedure will minimize the chance of bleeding from the catheter site.
- Inform the patient that oral fluids should be consumed as tolerated after the procedure to assist in eliminating the radiographic contrast material.
- Encourage the patient and family to ask questions.

intracoronary adenosine infusion. It is calculated as the mean distal coronary pressure divided by the mean aortic pressure during maximum blood flow. FFR in a normal coronary artery is a value of 1. An abnormal value of less than 0.75 indicates a flow-limiting lesion and is associated with ischemia. While not mandatory to perform, FFR-guided interventions have been associated with a reduced rate of primary composite end point of death, MI, and repeat vascularization at 1 year, when compared with standard percutaneous coronary interventions guided by angiography alone.[16]

Coronary interventions that may be necessary include percutaneous transluminal coronary angioplasty (PTCA), which involves the displacement of intracoronary plaque or thrombus for intracoronary blockages of 70% or greater. Intracoronary stents are intraluminal scaffolds placed after PTCA to decrease the reclosure rate of angioplasty sites. In directional coronary atherectomy (DCA), the plaque is removed rather than displaced. DCA is a specialized procedure, used far less frequently than PTCA in most centers. Extraction atherectomy is performed using a transluminal extraction catheter and suction to remove thrombi. A more detailed discussion of coronary interventions can be found in Chapter 18.

Left Heart Catheterization

Left heart catheterization provides information about the lumen of the aorta, coronary arteries, aortic and mitral valvular competencies, and wall motion of the left ventricle. Many studies also include pressure measurements in the left atrium and left ventricle, with pressure gradients measured across the aortic and mitral valves, as well as over the left ventricular outflow tract.

A diagnostic left heart catheterization is typically performed percutaneously from either the brachial or femoral artery. This procedure delineates baseline coronary anatomy, and it can identify abnormalities of the coronary arteries, great vessels, and cardiac chambers. Injection of the coronary vessels with radiographic contrast shows the actual lumen of the vessel and defines plaque, thrombus, and dissections that cause obstruction to blood flow. The left ventricular filling pressure is obtained as an indicator of the fluid status of the patient. A left ventriculogram, which involves rapid filling of the left ventricle with contrast media, provides the left ventricular ejection fraction as well as information regarding wall motion abnormalities and size of the left ventricle. Patients with valvular disease can have additional studies to measure valvular gradients and chamber pressures to allow for mathematical calculations of valve area and flow dynamics.

The risk profile for left heart catheterization is significant because the procedure involves cannulation of an artery and use of contrast media. The risks include bleeding at the percutaneous entry site, dissection of any of the vessels traversed during the procedure, perforation of peripheral or coronary arteries, mechanical irritation of the cardiac tissue, plaque embolization leading to MI or cerebrovascular accident, allergic reactions to the contrast media or any other drug given during the procedure, and renal compromise because of the renal toxic effects of the contrast media.

Right Heart Catheterization

Right heart catheterization aids in the differentiation of left ventricular failure versus pulmonary disease as a cause of dyspnea. It is performed in patients with a history of dyspnea, valvular heart disease, and intracardiac shunts.

Diagnostic right heart cardiac catheterizations can be performed from the right or left external jugular or the femoral veins. Right heart chamber pressures and information about the pulmonic valve and PA pressures are obtained. More commonly, the procedure may be performed through the inferior jugular vein to the superior vena cava. The goal is to

sample oxygen saturations and pressures in the right atrium, right ventricle, pulmonary capillary bed, and PA.

The most common problem during right heart catheterization is dysrhythmia resulting from stimulation of the myocardium. The dysrhythmias are self-limiting and usually do not require treatment. Postprocedure restrictions are minimal because a vein is accessed, and the risk for bleeding is low.

ELECTROCARDIOGRAPHIC MONITORING

Cardiac monitoring is used in a variety of settings. Traditionally used in ICUs and operating rooms, cardiac monitors are commonly found in many inpatient units where it is necessary to monitor continuously a patient's heart rate and rhythm or the effects of a therapy. In addition, cardiac monitors are used outside the hospital in settings such as paramedic ambulances, surgical centers, outpatient rehabilitation programs, and transtelephonic monitoring clinics.

Although the type of monitor may differ in each of these settings, all monitoring systems have three basic components: a display system, a monitoring cable, and electrodes. Electrodes are placed on the patient's chest to receive the electrical current from the cardiac muscle tissue. The electrical signal is then carried by the monitoring cable to a screen, where it is magnified and displayed. The display can be obtained both at the patient's bedside and at a central station, along with displays from other patients' monitors.

The capabilities of cardiac monitors have greatly expanded since they were first introduced nearly 50 years ago. Early monitoring systems were used merely for assessing the patient's heart rate and rhythm. Today's monitors have expanded capabilities that include the diagnosis of complex dysrhythmias, the detection of myocardial ischemia, and the identification of prolonged QT intervals. These expanded features are made possible through development of computerized dysrhythmia detection algorithms, ST-segment monitoring software, noise reduction features, multilead monitoring systems, and derived 12-lead ECGs with a minimum number of electrodes.

▲ Equipment Features

Two types of cardiac monitoring equipment are in use: continuous hard-wire monitoring systems and telemetry monitoring systems.

Hard-Wire Monitoring Systems

Hard-wire monitors, which are commonly used in ICU settings, require the patient to be linked directly to the cardiac monitor through the ECG cable. Information is displayed and recorded at the bedside along with simultaneous display and recording at a central station. Because this type of cardiac monitoring limits patient mobility, patients using this system usually are confined to bed rest or are allowed to be up at the bedside only. Hard-wire monitors operate on electricity but are well isolated so that water, blood, and other fluids do not pose an electrical hazard as long as the machine is maintained properly.

Telemetry Monitoring Systems

In telemetry monitoring, no direct wire connection is needed between the patient and the ECG display device. Electrodes are connected by a short monitoring cable to a small battery-operated transmitter. The ECG is then sent by radiofrequency signals to a receiver that picks up and displays the signal on an oscilloscope, either at the bedside or at a distant central recording station. Antennas are built into the receiver and may be mounted in the vicinity of the receiver to widen the range of signal pickup. Batteries are the power source for the transmitter and make it possible to avoid electrical hazards by isolating the monitoring system from potential current leakage and accidental shock. Telemetry systems are used primarily for dysrhythmia monitoring in areas where the patient is fairly mobile, such as a dysrhythmia surveillance or progressive care unit. Because the patient is mobile, stable ECG tracings often are more difficult to obtain. Some hard-wire systems have built-in telemetry capability so that patients may be switched easily from one system to another as monitoring needs change.

Display Systems

Modern electronic technology continues to make sophisticated advances in monitoring equipment, and current display systems incorporate features such as the following:

- Computerized storage capability that permits retrieval of dysrhythmia data
- Automatic chart documentation, in which the ECG recorder is activated by alarms or at preset intervals
- Expanded alarm systems for a variety of parameters
- Multilead or 12-lead ECG display, which facilitates interpretation of complex dysrhythmias
- ST-segment analysis for monitoring ischemic events
- Computer systems that store, analyze, and trend monitored data, allowing the information to be retrieved at any time to aid in diagnosis and to note trends in the patient's status
- Wireless communication devices carried by the nurse that provide data and alarms
- QT-interval monitoring

Monitoring Lead Systems

All cardiac monitors use lead systems to record the electrical activity generated by cardiac tissue. Each lead system is composed of a positive or recording electrode, a negative electrode, and a third electrode used as a ground. As the heart depolarizes, the waves of electrical activity move inferiorly because the normal route of depolarization moves from the SA node and atria, downward through the AV node, His-Purkinje system, and ventricles, and to the left because the

muscle mass in the left side of the heart is greater than the muscle mass of the right side of the heart. Each lead system views these waves of depolarization from a different location on the chest wall and thus produces P waves and QRS complexes of varying configuration.

The terminology used to describe lead systems can be confusing. The wires attached to the patient's chest are called leads, and the pictures produced by these wires are also called leads. A standard ECG uses 10 lead wires with electrodes at the ends (4 placed on the limbs, and 6 placed on the chest) and produces 12 electrical views of the heart, known as 12 leads.

Cardiac monitoring systems currently on the market vary from a simple three-electrode device to the more common five-electrode system. Other systems less commonly used aim to reduce the number of electrodes while monitoring all 12 leads. The discussion of monitoring in this chapter will focus on the three- and five-lead systems.

The three-electrode system produces limited selections of leads I, II, or III with only a single lead viewed on the screen at one time (single-channel recording). Five-electrode systems allow the possibility of viewing any of the 12 ECG leads and permit the nurse to view two or more leads on the monitor screen simultaneously (multichannel recording).

Three-Electrode Systems

Monitors that require three electrodes use positive, negative, and ground electrodes that are placed in the right arm (RA), left arm (LA), and left leg (LL) positions on the chest as designated by markings on the monitor cable. When the electrodes are placed appropriately, the standard leads (leads I, II, III) may be obtained by moving the lead selector on the bedside monitor to the lead I, II, or III position (Fig. 17-15). The lead selector automatically adjusts which electrode is positive, which electrode is negative, and which electrode is ground to obtain an appropriate tracing. When lead I is selected, the LA is positive, the RA is negative, and the LL is ground. For a lead II configuration, the LL is positive, the RA is negative, and the LA is ground. To obtain a lead III, the LL is positive, the LA is negative, and the RA is ground. The configuration of leads I, II, and III, known as Einthoven's triangle, is illustrated in Figure 17-16.

To obtain a chest lead on the monitor that replicates the chest lead from the 12-lead ECG, a five-wire system is needed. (See Fig. 17-9 on page 224 for a review of chest lead placement.) When only three wires are available, a modified version of any of the six chest leads may be obtained. To configure a modified chest lead (MCL), the goal is to position the positive electrode in the designated chest position. For example, an MCL_1 would require the positive electrode to be placed in a V_1 position (fourth intercostal space, right sternal border). The negative electrode is always positioned under the left clavicle. The ground electrode can be positioned anywhere.

To obtain an MCL_1 lead, the monitor is set to lead I (Box 17-13). (By setting the monitor to lead I, the LA electrode is positive, the RA electrode is negative, and the leg wire is ground [Einthoven's triangle].) The positive electrode (LA) is placed in a V_1 position (fourth intercostal space, right sternal border), and the negative electrode (RA) is positioned under the left clavicle. The ground electrode (LL) can be positioned anywhere, but if it is placed in a V_6 position, it is helpful when switching to an MCL_6 lead.

To obtain an MCL_6 lead, the goal is to place a positive electrode in a V_6 position, a negative electrode under the left

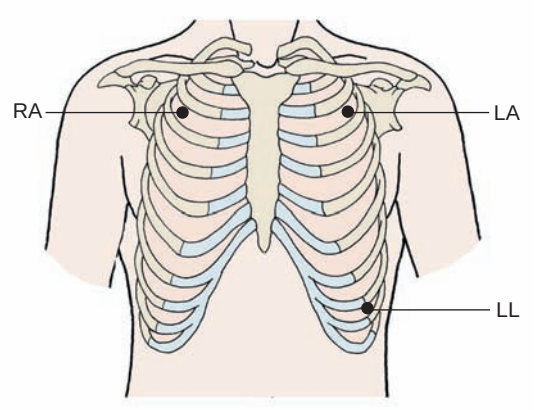


FIGURE 17-15 ▲ Three-electrode monitoring system. Leads placed in this position allow the nurse to monitor leads I, II, and III. The left leg electrode must be placed below the level of the heart. LA, left arm; LL, left leg; RA, right arm.

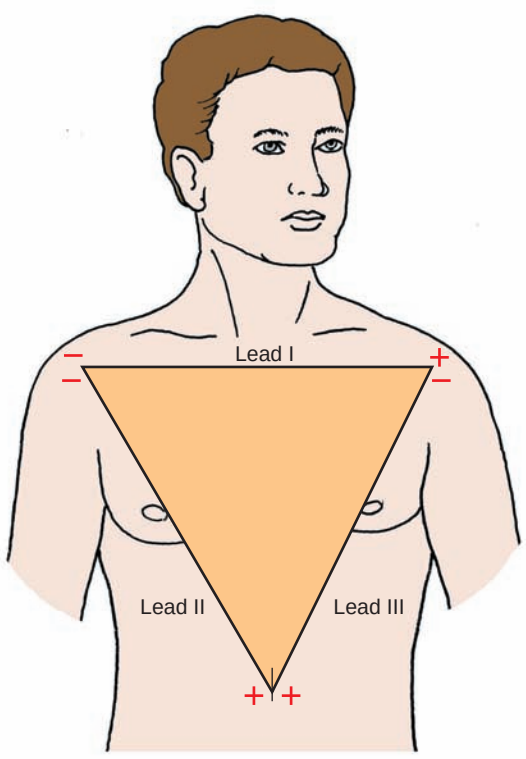


FIGURE 17-16 ▲ Einthoven's triangle. Leads I, II, and III are known as the standard leads. When placed together over the chest, they form what is known as Einthoven's triangle.

Lead I: LA is positive, and RA is negative.

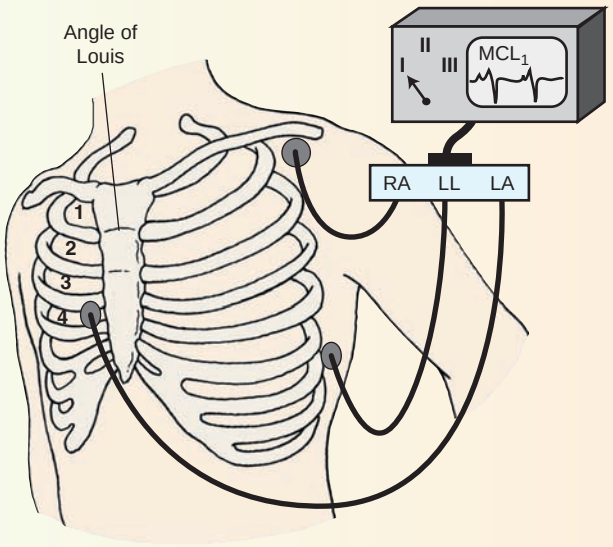
Lead II: LL is positive, and RA is negative.

Lead III: LL is positive, and LA is negative.

BOX 17-13 Three-Electrode System

To monitor modified chest lead $(MCL)_1$ using a three-electrode monitor:

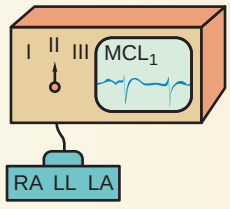
1. Select lead I on the monitor.
2. Refer to Einthoven's triangle to remember that LA is positive, RA is negative, and LL is ground for lead I.
3. Place the positive electrode (LA) in a V_1 position (fourth intercostal space, right sternal border).
4. Place the negative electrode (RA) under the left clavicle.
5. Place the ground wire (LL) in the V_6 position (fifth intercostal space, left midaxillary line).



To monitor MCL_6 using a three-electrode monitor:

1. Select lead II on the monitor.
2. Refer to Einthoven's triangle to remember that LL is positive, RA is negative, and LA is ground for lead II.
3. Place the positive electrode (LL) in the V_6 position (fifth intercostal space, left midaxillary line).
4. Place the negative electrode (RA) under the left clavicle.
5. Place the ground wire (LA) in a V_1 position (fourth intercostal space, right sternal border).

Note: The electrodes are in the same position on the chest for the MCL_1 lead and the MCL_6 lead. To view the two leads, the nurse merely switches the monitor from lead I to lead II.



clavicle, and a ground wire anywhere. By setting the monitor to lead II, the LL electrode is positive, the RA electrode is negative, and the LA electrode is ground (Einthoven's triangle). The positive electrode (LL) is placed in the V_6 position (midaxillary line, same horizontal level as V_4), and the negative electrode (RA) is placed under the left clavicle. The ground wire can be placed anywhere, but if it is placed in a V_1 position, it will be helpful when switching to an MCL_1 lead.

By arranging the electrodes as described, the nurse can monitor both MCL_1 and MCL_6 merely by switching the monitor from a lead I to a lead II without changing the electrode placement on the patient's chest. MCL_1 and MCL_6 are ideal leads for detecting bundle branch block (BBB) rhythms and for differentiating supraventricular wide-QRS tachycardias from VT.

Five-Electrode Systems

The five-electrode system that increases the monitor's capability beyond the three-electrode system is preferred over the three-electrode system. (The four-electrode monitor requires a right leg electrode that is the ground for all leads described in the three-electrode system.) The five-electrode monitor adds an exploring "chest" electrode that allows one to obtain any one of the six chest leads and the six limb leads. In essence, a five-wire monitor system provides all the capabilities of the 12-lead ECG machine. The only difference is that the five-wire monitor has only one chest electrode, whereas the 12-lead ECG machine has six chest electrodes. Newer cardiac monitors now have all six chest electrodes and allow the nurse to view all 12 leads of the ECG simultaneously on the monitor screen.

To monitor a patient with a five-wire system, the four limb electrodes are positioned on the body according to their designations. The fifth chest electrode is placed on the chest in the designated precordial position. For example, if the nurse wants to monitor V_1, the chest electrode is placed in the fourth intercostal space, right sternal border (Fig. 17-17). If the nurse wants to switch to a different chest lead for monitoring, the electrode must be repositioned on the patient's chest. A five-electrode monitor offers the additional advantage of allowing the nurse to view two or more different leads simultaneously on the monitor screen.

Lead Selection

No single monitoring lead is ideal for every patient. Table 17-9 summarizes the use of various leads and the reasons for their use. Lead II is used commonly because it records clear upright P waves and QRS complexes that are helpful in determining the underlying rhythm. In addition to lead II, leads III, aVF, and V_1 or MCL_1 show well-formed P waves and therefore are helpful in identifying atrial dysrhythmias. V_1 or MCL_1 is useful in recognizing RBBB and in differentiating ventricular ectopy from supraventricular rhythms with aberrancy. V_6 or MCL_6 is helpful in identifying LBBB and also is useful in differentiating ventricular ectopy from supraventricular rhythms with aberrancy. Lead I may be tried in the patient with respiratory disease who has much artifact on the tracing because there is less movement of the positive electrode in this lead than in a lead II or a V_1.

As mentioned, there is no one ideal monitoring lead for every patient, and in several situations, multilead recording is desirable. Multilead ECG systems offer multiple views of the heart because they reflect a tracing from each of the major heart surfaces. One of the major uses of multilead monitoring is in the interpretation of complex cardiac dysrhythmias, especially when differentiating aberrancy from ventricular ectopy

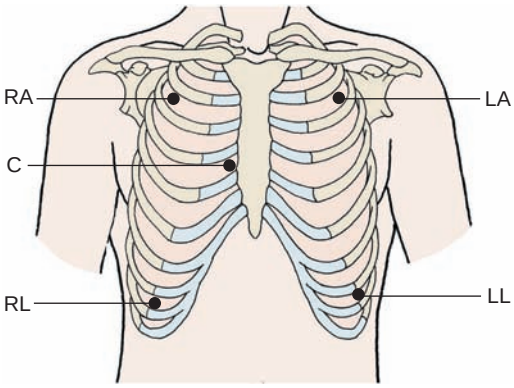


FIGURE 17-17 ▲ Five-electrode monitoring system. Using a five-electrode system allows the nurse to monitor any of the 12 leads of the ECG. The chest electrode must be moved to the appropriate chest location when monitoring the precordial leads.

and in identifying complex atrial dysrhythmias, uncharacteristic-looking ventricular premature beats, and fascicular blocks. Another use of multilead monitoring is in assessment of myocardial ischemia, injury, and infarction. By continuously viewing one lead from each area of the heart, episodes of anginal pain or silent ischemia can be documented. As soon as possible, these changes should be confirmed by a full 12-lead ECG.

Table 17-9 Suggested Monitoring Lead Selection

Lead	Rationale for Use
II	Produces large, upright visible P waves and QRS complexes for determining underlying rhythm
V_1 or modified chest lead $(MCL)_1$	Helpful for detecting right bundle branch block and to differentiate ventricular ectopy from supraventricular rhythm aberrantly conducted in the ventricles
V_6 or MCL_6	Helpful lead for detecting LBBB and to differentiate ventricular ectopy from supraventricular rhythm aberrantly conducted in the ventricles
III, aVF, V_1	Produce visible P waves; useful in detecting atrial dysrhythmias
I	Useful in patients with respiratory distress Left arm and right arm electrodes involved and placements less affected by chest motion compared with other leads
II, III, aVF	Helpful in detecting ischemia, injury, and infarction in the inferior wall Ischemia related to the right coronary artery is best seen in lead III
I, aVL, V_5, V_6	Helpful in detecting ischemia, injury, and infarction in the lateral wall
V_1 through V_4	Helpful in detecting ischemia, injury, and infarction in the anterior wall Ischemia related to the left anterior descending coronary artery or the left circumflex coronary artery is best seen in lead V_3

▲ Procedure

Electrode Application

Proper skin preparation and application of electrodes are imperative for good ECG monitoring. An adequate tracing should reflect (1) a narrow, stable baseline; (2) absence of distortion or "noise"; (3) sufficient amplitude of the QRS complex to activate the rate meters and alarm systems properly; and (4) identification of P waves.

The type of electrode currently used for ECG monitoring is a disposable silver- or nickel-plated electrode centered in a circle of adhesive paper or foam rubber. Most electrodes are pregelled by the manufacturer. They may have disposable wires attached to the electrodes or nondisposable wires that snap onto the electrodes. Electrodes should be comfortable for the patient. If not properly applied, undue artifact and false alarms may result.

When applying electrodes, the following procedure should be followed:

1. Select a stable site. Avoid bony protuberances, joints, and folds in the skin. Areas in which muscle attaches to bone have the least motion artifact.
2. Shave excessive body hair from the site.
3. Rub the site briskly with a dry gauze pad to remove oils and cellular debris. Skin preparation with alcohol may be necessary if the skin is greasy; allow the alcohol to dry completely before applying the electrode. Follow the electrode manufacturer's directions because the chemical reaction between alcohol or other skin-preparation materials and the adhesives used in some electrodes may cause skin irritation or nonadhesion to the skin.
4. Remove the paper backing and apply each electrode firmly to the skin by smoothing with the finger in a circular motion. Attach each electrode to its corresponding ECG cable wire. Sometimes it is necessary to tape over the cable wire connection or make a stress loop with the cable wire for extra stability.
5. Change electrodes every 2 to 3 days, and monitor for skin irritation.

While applying the electrodes, explain the purpose of the procedure to the patient. Reassure the patient that monitor alarm sounds do not necessarily indicate a problem with the patient's heart beat; alarms often occur when an electrode becomes loose or disconnected. See Evidence-Based Practice Highlight 17-1.

Monitor Observation

Cardiac monitors are useful only if the information they provide is "observed," either by computers with alarms for programmed parameters or by the human eye, and appropriately acted on by competent, responsible people. Some critical care units use monitor technicians whose main responsibilities are to observe monitors, obtain chart samples, and give appropriate information to the nurse about each patient's ECG status. Those observing the monitor should know the acceptable dysrhythmia parameters for each patient and should be notified of any interruptions in monitoring, such as those caused by changing electrodes or by changing the patient to

EVIDENCE-BASED PRACTICE HIGHLIGHT 17-1

Dysrhythmia Monitoring

△ Expected Practice

- Select the best monitoring leads for dysrhythmia identification (display two leads when possible).
  - Lead V_1 to diagnose wide QRS complex.
  - Lead II to diagnose atrial activity and measure heart rate.
- Proper electrode placement is required for accurate diagnosis.
- Prepare the patient's skin before attaching ECG electrodes.
- Measure QT interval and calculate QTc using a consistent lead if high risk for Torsades de Pointes.

△ Supporting Evidence

- V_1 is the lead of choice to diagnose wide QRS complexes (ventricular tachycardia vs. supraventricular tachycardia with aberrant conduction; left vs. right bundle branch block). A 5-lead monitoring system is required to monitor V leads. MCL_1 may differ in QRS morphology as compared to V_1 and should be used only when a 5-lead monitoring system is unavailable.[6–10] (Level V)
- When V_1 electrode placement is not possible, V_6 may be used. [7–11] (Level IV)
- Electrode site preparation includes clipping excessive hair and cleansing oily skin with alcohol.[3–4] (Level IV)
- QTc of more than 0.50 seconds (500 ms) is dangerously prolonged and associated with risk for Torsades de Pointes. The QT interval should be corrected for heart rate (QTc) and monitored with any of the following: [9,10,12–15] (Level IV)
  - Antidysrhythmic, antibiotic, antipsychotic, and other drugs that prolong QTc
  - Severe bradycardia
  - Hypokalemia or hypomagnesemia
  - Any drug overdose
- Perform an atrial electrogram (AEG) in cardiac surgical patients with atrial epicardial wires to assist in identifying atrial activity.[16–17] (Level V)

AACN Evidence Leveling System

Level A Meta-analysis of quantitative studies or metasynthesis of qualitative studies with results that consistently support a specific action, intervention or treatment.
Level B Well-designed, controlled studies with results that consistently support a specific action, intervention, or treatment.
Level C Qualitative studies, descriptive or correlational studies, integrative review, systematic reviews, or randomized controlled trials with inconsistent results.
Level D Peer-reviewed professional organizational standards with clinical studies to support recommendations.
Level E Multiple case reports, theory-based evidence from expert opinions, or peer-reviewed professional organizational standards without clinical studies to support recommendations.
Level M Manufacturer's recommendations only.

Excerpted from American Association of Critical-Care Nurses Practice Alert. Available online at http://aacn.org. All references cited in this alert are available with the associated resources related to this chapter. Visit: http://thepoint.lww.com

a portable monitor. The observer also should be aware of the presence of artifact from chest physical therapy or hiccups so that it may be considered in dysrhythmia diagnosis.

Regardless of the system used for monitor observation, certain practices always should be followed. If the monitor alarm sounds, the nurse evaluates the clinical status of the patient before doing anything else to see if the problem is an actual dysrhythmia or a malfunction of the monitoring system. Asystole should not be mistaken for an unattached ECG wire, nor should a patient inadvertently tapping on an electrode be misread as VT. In addition, monitor alarms always should be in the functioning mode. Only when direct physical care is being given to the patient can the alarm system safely be put on "standby." This ensures that no life-threatening dysrhythmia goes unnoticed. If the change on the monitor is not caused by an artifact or a disconnected wire, a full 12-lead ECG should be recorded to evaluate the rhythm change further.

▲ Troubleshooting Electrocardiogram Monitor Problems

Several problems may occur in monitoring the ECG, including baseline but no ECG trace, intermittent traces, wandering or irregular baseline, low-amplitude complexes, 60-cycle interference, excessive triggering of heart rate alarms, and skin irritation. Box 17-14 outlines the steps to follow when such problems occur.

DYSRHYTHMIAS AND THE 12-LEAD ELECTROCARDIOGRAM

Dysrhythmias and abnormalities of the 12-lead ECG commonly encountered in monitored patients can be recognized with a little practice. The types that occur most frequently are discussed in this chapter. Before presenting the individual dysrhythmias and 12-lead ECG abnormalities, the method for evaluating a rhythm strip is addressed.

To understand the causes, clinical significance, and treatment of dysrhythmias, knowledge of the conduction system is essential. Chapter 16 provides a review of the essential elements of the cardiac conducting system.

▲ Evaluation of a Rhythm Strip

Electrocardiogram Paper

An ECG tracing is a graphic recording of the heart's electrical activity. The paper consists of horizontal and vertical lines, each 1 mm apart. The horizontal lines denote time measurements. When the paper is run at a sweep speed of

BOX 17-14 Troubleshooting: ECG Monitor Problem Solving

Excessive Triggering of Heart Rate Alarms
- Is the high–low alarm set too close to the patient's heart rate?
- Is the monitor sensitivity level set too high or too low?
- Is the patient cable securely inserted into the monitor receptacle?
- Are the lead wires or connections damaged?
- Has the monitoring lead been properly selected?
- Were the electrodes applied properly?
- Are the R and T waves the same height, causing both waveforms to be sensed?
- Is the baseline unstable, or is there excessive cable or lead wire movement?

Baseline but No ECG Trace
- Is the size (gain or sensitivity) control properly adjusted?
- Is an appropriate lead selector being used on the monitor?
- Is the patient cable fully inserted into the ECG receptacle?
- Are the electrode wires fully inserted into the patient cable?
- Are the electrode wires firmly attached to the electrodes?
- Are the electrode wires damaged?
- Is the patient cable damaged?
- Call for service if the trace is still absent.
- Is the battery dead (for telemetry system)?

Intermittent Trace
- Is the patient cable fully inserted into the monitor receptacle?
- Are the electrode wires fully inserted into the patient cable?
- Are the electrode wires firmly attached to the electrodes?
- Are the electrode wire connectors loose or worn?
- Have the electrodes been applied properly?
- Are the electrodes properly located and in firm skin contact?
- Is the patient cable damaged?

Wandering or Irregular Baseline
- Is there excessive cable movement? This can be reduced by clipping to the patient's clothing.
- Is the power cord on or near the monitor cable?
- Is there excessive movement by the patient? Are there muscle tremors from anxiety or shivering?
- Is site selection correct?
- Were proper skin preparation and application procedures followed?
- Are the electrodes still moist?

Low-Amplitude Complexes
- Is size control adjusted properly?
- Were the electrodes applied properly?
- Is there dried gel on the electrodes?
- Change electrode sites. Check 12-lead ECG for lead with highest amplitude, and attempt to simulate that lead.
- If none of the preceding steps remedies the problem, the weak signal may be the patient's normal complex.

Sixty-Cycle Interference
- Is the monitor size control set too high?
- Are there nearby electrical devices in use, especially poorly grounded ones?
- Were the electrodes applied properly?
- Is there dried gel on the electrodes?
- Are lead wires or connections damaged?

25 mm/s, each small square measured horizontally is equal to 0.04 second, and a large square (five small squares) equals 0.20 second. Height or voltage is measured by counting the lines vertically. Each small square measured vertically is 1 mm, and the large square is 5 mm (Fig. 17-18). Some ECG paper also is marked by vertical slash marks across the top or bottom. The distance between two vertical markings represents 3 seconds. The distance between 6 seconds is used for rate calculation.

Waveforms and Intervals

During the cardiac cycle, the following waveforms and intervals are produced on the ECG surface tracing (see Fig. 17-18):

- P wave: The P wave is a small, usually upright and rounded deflection representing depolarization of the atria. It normally is seen before the QRS complex at a consistent interval.
- PR interval: The PR interval represents the time from the onset of atrial depolarization until the onset of ventricular depolarization. Included in the interval is the brief delay of the electrical signal at the AV node that allows time for the blood to move from the atria to the ventricles before the ventricles are depolarized. The interval is measured from the beginning of the P wave to the beginning of the QRS complex. A normal PR interval is 0.12 to 0.20 second.
- QRS complex: The QRS complex is a large waveform representing ventricular depolarization. Each component of the waveform has a specific connotation. The initial negative deflection is a Q wave, the initial positive deflection is an R wave, and the negative deflection after the R wave is an S wave. Not all QRS complexes have all three components, even though the complex is commonly called the QRS complex. A normal QRS complex is 0.06 to 0.11 second in width. Figure 17-19 illustrates different kinds of QRS complexes.
- ST segment: The ST segment is the portion of the tracing from the end of the QRS complex to the beginning of the T wave. It represents the time from the end of ventricular depolarization to the beginning of ventricular repolarization. Normally, it is isoelectric. An isoelectric ST segment means the ST segment joins the QRS complex at the baseline. ST segments may be elevated or depressed in a variety of conditions. Elevated ST segments could indicate acute myocardial injury. Depressed ST segments may signify acute myocardial injury or myocardial ischemia. For a more detailed discussion of ST-segment abnormalities, see Chapter 21.
- T wave: The T wave is the deflection representing ventricular repolarization or recovery. The T wave appears after the QRS complex. The atria also have a repolarization phase. However, there is no visible wave on the ECG to represent atrial repolarization because it occurs at the same time as the QRS complex.
- U wave: A U wave is a rarely seen, small, usually positive deflection after the T wave. Its significance is uncertain, but it typically is seen with hypokalemia.
- QT interval: The QT interval is the period from the beginning of ventricular depolarization to the end of ventricular repolarization. The QT interval is measured from the beginning of the QRS complex to the end of the T wave. Because the QT interval varies with heart rate, it is necessary to use a table in which QT intervals for various heart rates are listed. Tables are available for this

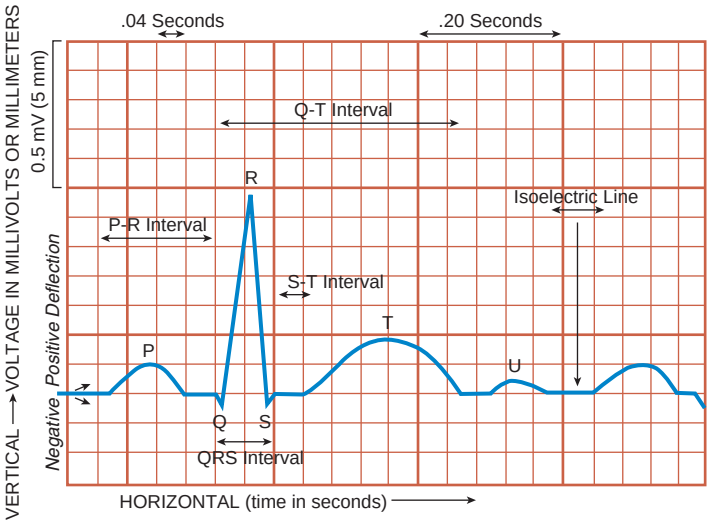


FIGURE 17-18 ▲ Waveforms of the ECG. Schematic representation of the electrical impulse as it traverses the conduction system, resulting in depolarization and repolarization of the myocardium.

purpose in most texts about dysrhythmias (Table 17-10). If such a table is not available, a corrected QT interval (QTc) can be calculated for comparison with normal values. Normal QTc usually does not exceed 0.42 second for men and 0.43 second for women. A quick method for obtaining a QTc is to use half of the preceding RR interval (described later).

Calculation of Heart Rate

Although cardiac monitors and ECG strips can be used to calculate heart rate, the calculated rate is merely an estimate of the number of times per minute the heart has been electrically excited. In the normal heart, each excitation should be followed by cardiac contraction. However, in some situations, electrical activity can occur without contraction, resulting in a lack of perfusion. Therefore, the heart rate obtained from the cardiac monitor or ECG strip should never be substituted for the determination of heart rate by palpating the pulse.

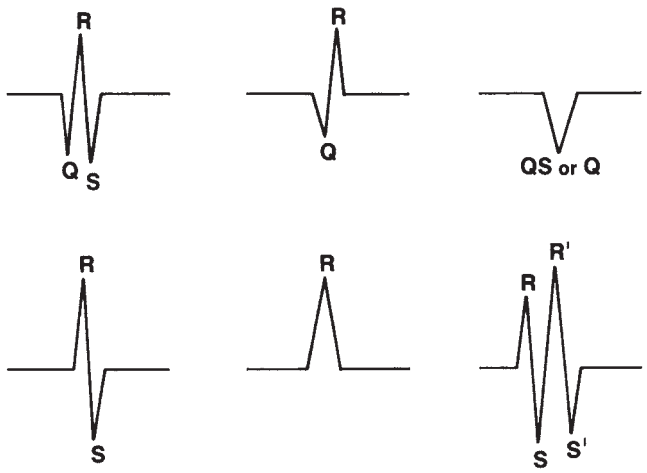


FIGURE 17-19 ▲ Configurations of the QRS complex. A Q wave is a negative deflection before an R wave, an R wave is a positive deflection, and an S wave is a negative deflection after an R wave.

Both the atrial and the ventricular rates can be estimated by examining the ECG. To determine the ventricular rate, count the number of QRS complexes in a 6-second strip and multiply by 10. To estimate the atrial rate, count the number of P waves in a 6-second strip and multiply by 10. In the normal patient, the atrial and the ventricular rates should be the same. This method of rate calculation provides an estimate of heart rate for regular and irregular rhythms.

Another method of rate calculation can be used if the rhythm is regular. The ventricular heart rate is estimated by dividing 300 by the number of large boxes on the ECG paper between two R waves (the RR interval).

Table 17-10 Approximate Normal Limits for QT Intervals in Seconds

Heart Rate per Minute	Men and Children	Women
40	0.45–0.49	0.46–0.50
46	0.43–0.47	0.44–0.48
50	0.41–0.45	0.43–0.46
55	0.40–0.44	0.41–0.45
60	0.39–0.42	0.40–0.43
67	0.37–0.40	0.38–0.41
71	0.36–0.40	0.37–0.41
75	0.35–0.38	0.36–0.39
80	0.34–0.37	0.35–0.38
86	0.33–0.36	0.34–0.37
93	0.32–0.35	0.33–0.36
100	0.31–0.34	0.32–0.35
109	0.30–0.33	0.31–0.33
120	0.28–0.31	0.29–0.32
133	0.27–0.29	0.28–0.30
150	0.25–0.28	0.26–0.28
172	0.23–0.26	0.24–0.26

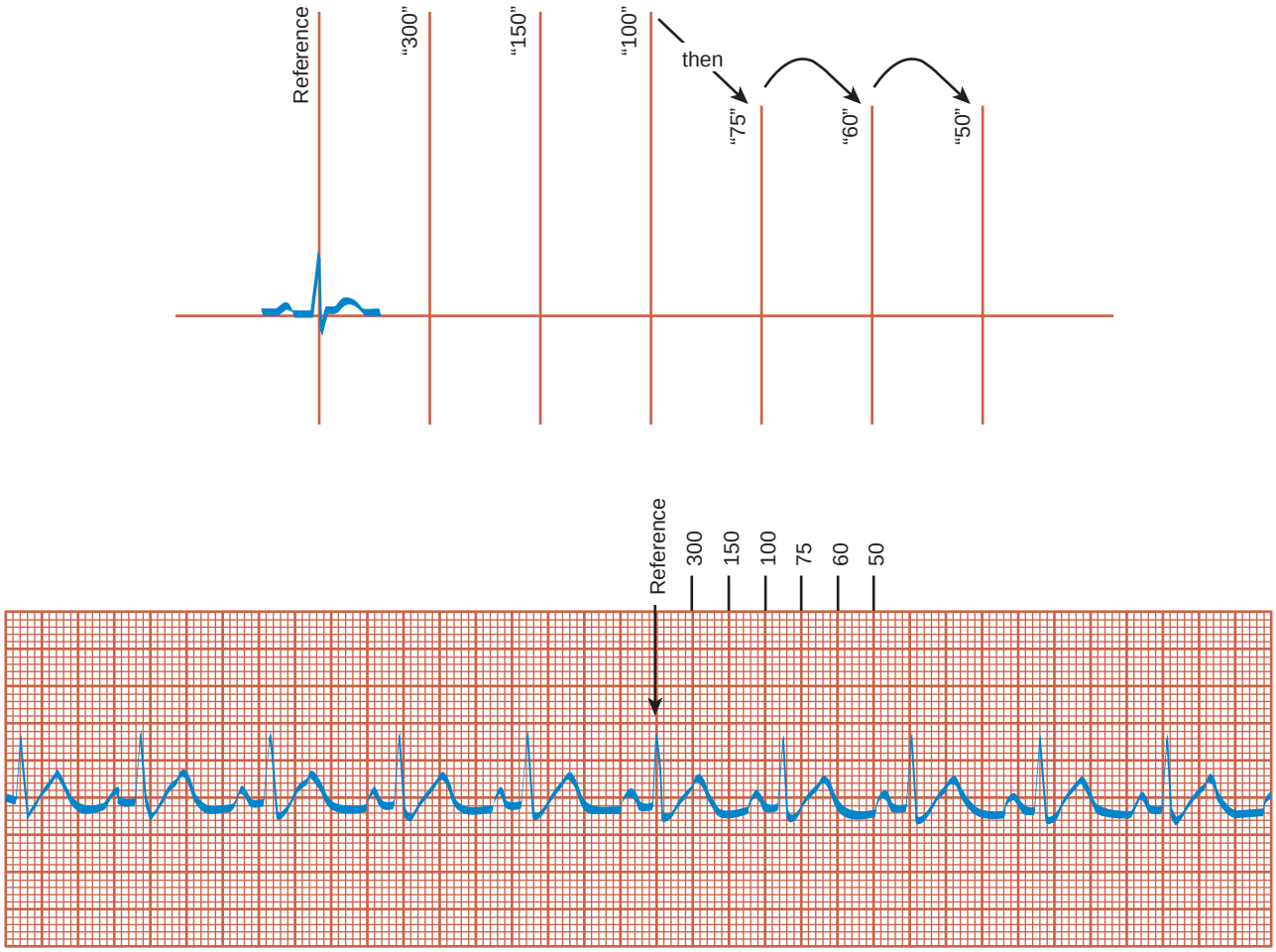


FIGURE 17-20 ▲ Method for estimating heart rate. Using this method, the heart rate is approximately 85 beats/min.

The atrial rate is calculated by dividing 300 by the number of large boxes on ECG paper between two P waves (the PP interval).

Another quick method for estimating rate involves the use of a series of numbers. To use this method for estimating ventricular rate, the nurse first finds a QRS complex that falls directly on a dark line of the ECG paper. This dark line is the reference point. The next six dark lines of the paper are labeled 300, 150, 100, 75, 60, and 50 (Fig. 17-20). Then, the nurse finds the next QRS complex immediately after the reference point and estimates the ventricular rate using the sequence of numbers. The same method can be used for estimating atrial rate by using the P waves.

Steps in Assessing a Rhythm Strip

The following analysis represents a systematic approach to assessment of a cardiac rhythm strip. Whether or not this method is used, it is important to take the time to complete each step because many dysrhythmias are not as they first appear.

1. Determine the atrial and ventricular heart rates.
   - Are they within normal limits?
   - If not, is there a relationship between the two (ie, one a multiple of the other)?
2. Examine the rhythm to see if it is regular.
   - Is there an equal amount of time between each QRS complex (RR interval)?
   - Is there an equal amount of time between each P wave (PP interval)?
   - Are the PP and RR intervals the same?
3. Look for the P waves.
   - Are they present?
   - Is there one or more P waves for each QRS complex?
   - Do all P waves have the same configuration?
4. Measure the PR interval.
   - Is it normal?
   - Is it the same throughout the strip, or does it vary?
   - If it varies, is there a pattern to the variation?
5. Evaluate the QRS complex.
   - Is it normal in width, or is it wide?
   - Are all complexes of the same configuration?
6. Examine the ST segment.
   - Is it isoelectric, elevated, or depressed?
7. Identify the rhythm and determine its clinical significance.
   - Is the patient symptomatic? (Check skin, neurological status, renal function, coronary circulation, and hemodynamic status or blood pressure.)
   - Is the dysrhythmia life-threatening?
   - What is the clinical context?
   - Is the dysrhythmia new or chronic?

▲ Normal Sinus Rhythm

Normal sinus rhythm (Fig. 17-21A) is the normal rhythm of the heart. The impulse is initiated at the sinus node in a regular rhythm at a rate of 60 to 100 beats/min. A P wave appears before each QRS complex. The PR interval is within normal limits and of equal duration (0.12 to 0.20 second), and the QRS is narrow (<0.12 second) unless an intraventricular conduction defect is present.

▲ Dysrhythmias Originating at the Sinus Node

Table 17-11 summarizes and compares ECG characteristics of sinus rhythms.

Sinus Tachycardia

In sinus tachycardia, the sinus node accelerates and initiates an impulse at a rate of 100 times per minute or more (see Fig. 17-21B). The upper limits of sinus tachycardia extend to 160 to 180 beats/min. All other ECG characteristics, except for heart rate, are the same as in normal sinus rhythm.

Sinus tachycardia usually is caused by factors relating to an increase in sympathetic tone. Stress, exercise, and stimulants such as caffeine and nicotine can produce this dysrhythmia. Sinus tachycardia also is associated with such clinical problems as fever, anemia, hyperthyroidism, hypoxemia, heart failure, and shock. Drugs, such as atropine, which blocks vagal tone, and the catecholamines (eg, epinephrine, dopamine) also can produce this rhythm.

The cause of the sinus tachycardia and the underlying state of the myocardium determine the prognosis. Sinus tachycardia alone is not a lethal dysrhythmia but often signals an underlying problem that should be pursued.

In addition, the rapid rate of sinus tachycardia increases oxygen demands on the myocardium and decreases the filling time of the ventricles. In people who already have depleted cardiac reserve, ischemia, or heart failure, the persistence of a fast rate may worsen the underlying condition.

Treatment of sinus tachycardia usually is directed at eliminating the underlying cause. Specific measures may include sedation, oxygen administration, digitalis, and diuretics if heart failure is present, or beta-blockers if the tachycardia is caused by thyrotoxicosis.

Sinus Bradycardia

Sinus bradycardia is defined as a rhythm with impulses originating at the sinus node at a rate of less than 60 beats/min (see Fig. 17-21C). The rhythm (RR interval) is regular, and all other parameters are normal.

Sinus bradycardia is common among people of all ages and may be normal in highly trained athletes. It is present in both healthy and diseased hearts. It may be associated with sleep, severe pain, inferior wall MI, acute spinal cord injury, and certain drugs (eg, digitalis, beta-blockers, verapamil, diltiazem). In people with healthy hearts, slow heart rates are tolerated well. However, in those with severe heart disease, the heart may not be able to compensate for a slow rate by increasing the volume of blood ejected per beat. In this situation, sinus bradycardia leads to a low cardiac output (CO).

No treatment is indicated unless symptoms are present. If the pulse is very slow and the patient is symptomatic, appropriate measures include atropine (to block the vagal effect) or cardiac pacing.

Sinus Dysrhythmia

Sinus dysrhythmia (formerly sinus arrhythmia) is a disorder of rhythm (see Fig. 17-21D) that is said to be present if the RR intervals on the ECG, from the shortest RR interval to

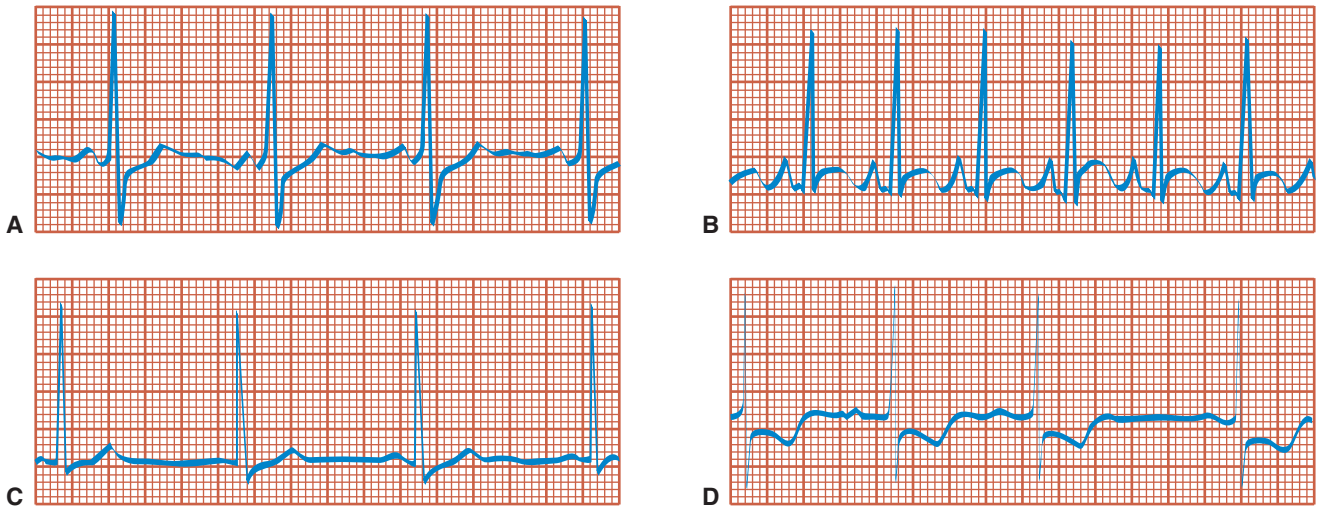


FIGURE 17-21 ▲ Sinus rhythms. **A:** Normal sinus rhythm. (Heart rate = 60 to 100 beats/min.) **B:** Sinus tachycardia. (Heart rate = 100 to 180 beats/min.) **C:** Sinus bradycardia. (Heart rate < 60 beats/min.) **D:** Sinus dysrhythmia. (Difference between shortest and longest RR interval.)

Table 17-11 A Comparison of the Electrocardiographic Characteristics of Sinus Rhythms

	Normal Sinus Rhythm	Sinus Tachycardia	Sinus Bradycardia	Sinus Dysrhythmia
Rate	60–100 beats/min	More than 100 beats/min	<60 beats/min	60–100 beats/min
Rhythm	Regular	Regular	Regular	Irregular
P waves	Present, one per QRS	Present, one per QRS	Present, one per QRS	Present, one per QRS
PR interval	<0.20 s, equal	<0.20 s, equal	<0.20 s, equal	<0.20 s, equal
QRS complex	<0.12 s	<0.12 s	<0.12 s	<0.12 s

the longest, vary by more than 0.12 second. This dysrhythmia is caused by an irregularity in sinus node discharge, often in association with phases of the respiratory cycle. The sinus node rate gradually increases with inspiration and gradually decreases with expiration.

Sinus dysrhythmia is a normal phenomenon, seen especially in young people in the setting of lower heart rates. It also occurs after enhancement of vagal tone (eg, with digitalis or morphine). Because it is a normal finding, sinus dysrhythmia does not imply the presence of underlying disease. Symptoms are uncommon unless there are long pauses between heart beats, and usually no treatment is required.

Sinus Arrest and Sinoatrial Block

Sinus arrest is a disorder of impulse formation. The sinus node fails to form a discharge, producing pauses of varying lengths because of the absence of atrial depolarization. The P wave is absent, and the resulting PP interval is not a multiple of the basic PP interval. The pause ends either when an escape pacemaker from the junction or ventricles takes over or when sinus node function returns.

An SA block often is difficult to differentiate from sinus arrest on a surface ECG tracing. In SA block, the sinus node fires, but the impulse is delayed or blocked from exiting the sinus node. If the block is complete, the duration of the pause is a multiple of the basic PP interval (Fig. 17-22).

Both dysrhythmias may result from disruption of the sinus node by infarction, degenerative fibrotic changes, drugs (digitalis, beta-blockers, calcium channel blockers), or excessive vagal stimulation. These rhythms usually are transient and insignificant unless a lower pacemaker fails to take over to pace the ventricles. Treatment is indicated if the patient is symptomatic. The goal is to increase the ventricular rate, which may require the use of atropine or, in the presence of serious hemodynamic compromise, a pacemaker.

Sick Sinus Syndrome

Sick sinus syndrome refers to a chronic form of sinus node disease (Fig. 17-23). Patients exhibit severe degrees of sinus node depression, including marked sinus bradycardia, SA block, or sinus arrest. Often, rapid atrial dysrhythmias, such as atrial flutter or fibrillation ("tachycardia–bradycardia syndrome"), coexist and alternate with periods of sinus node depression.

Management of sick sinus syndrome requires control of the rapid atrial dysrhythmias with drug therapy and, in selected cases, control of very slow heart rates, often requiring implantation of a permanent pacemaker.

▲ Atrial Dysrhythmias

Premature Atrial Contraction

A premature atrial contraction (PAC) occurs when an ectopic atrial impulse discharges prematurely and, in most cases, is conducted in a normal fashion through the AV conducting system to the ventricles (Fig. 17-24A). On the ECG tracing, the P wave is premature and may even be buried in the preceding T wave; it often differs in configuration from the sinus P wave. The QRS complex usually is of normal configuration. However, because of timing, the QRS complex may appear wide and bizarre if conducted with some

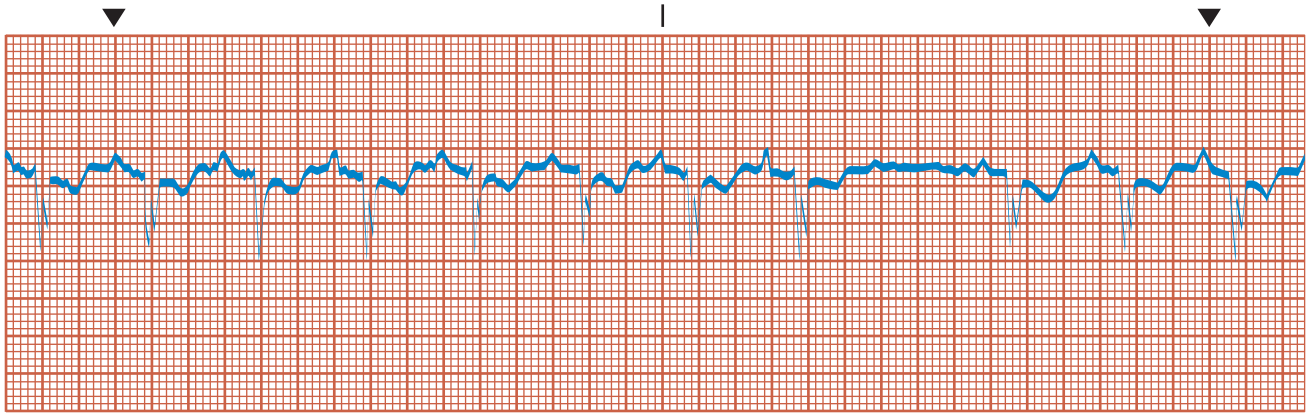


FIGURE 17-22 ▲ Sinoatrial block. The pause is a multiple of the basic PP interval.

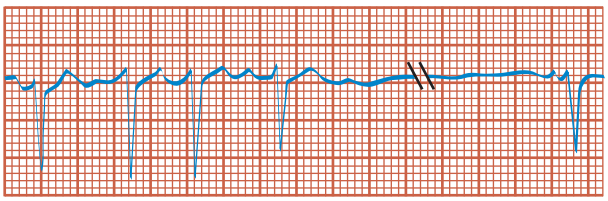


FIGURE 17-23 ▲ Sick sinus syndrome. Atrial fibrillation is followed by atrial standstill. A sinus escape beat is seen at the end of the strip.

degree of delay (aberrant PAC) or may not appear at all if the atrial impulse is blocked from being conducted to the ventricles (blocked PAC). A short pause, usually less than "compensatory," is present (see later definition of PVC on pages 250–253).

People of all ages experience PACs. PACs may occur in healthy people as a result of various stimuli, such as emotions, tobacco, alcohol, and caffeine. PACs also may be associated with rheumatic heart disease, ischemic heart disease, mitral stenosis, heart failure, hypokalemia, hypomagnesemia, medications, and hyperthyroidism.

Alternatively, PACs may be a precursor to an atrial tachycardia, atrial fibrillation, or atrial flutter, indicating an increasing atrial irritability. They also may indicate an underlying condition (eg, heart failure). Patients may have the sensation of a "pause" or "skip" in rhythm when PACs are present.

No treatment is necessary in many cases. The patient should be monitored and frequency of premature beats documented. In addition, the patient should be assessed for underlying conditions and treated.

Paroxysmal Supraventricular Tachycardia

Paroxysmal supraventricular tachycardia (PSVT) describes a rapid atrial rhythm occurring at a rate of 150 to 250 beats/min (Fig. 17-24B). The tachycardia begins abruptly, in most instances with a PAC, and it ends abruptly. P waves may precede the QRS complex but also may be hidden in the QRS complex or precede the T wave at faster rates. (If some of the P waves are not followed by a QRS complex, this is referred to as PSVT with block and usually is caused by digitalis toxicity.) The P waves may be negative in leads II, III, and aVF because of retrograde conduction from the AV node to the atria. The QRS complex usually is normal unless there is an underlying intraventricular conduction problem. The rhythm is regular, and the paroxysms may last from a few seconds to several hours or even days.

The term PSVT is used to identify rhythms previously called paroxysmal atrial tachycardia and paroxysmal nodal or junctional tachycardia, rhythms similar in all respects except in their sites of origin. PSVT also is known as AV nodal reentrant tachycardia because the mechanism most commonly responsible for this dysrhythmia is a reentrant circuit or chaotic movement at the level of the AV node.

PSVT must be differentiated from other narrow QRS complex (supraventricular) tachycardias. Table 17-12 is a guide to the differential diagnosis. The following points favor the diagnosis of PSVT versus a sinus tachycardia:

- An atrial premature beat often initiates the rhythm.
- The tachycardia begins and terminates abruptly.
- The rate often is faster than a sinus tachycardia and tends to be more regular from minute to minute.
- In response to a vagal maneuver, such as carotid sinus massage, the ectopic tachycardia either is unaffected or reverts to a normal sinus rhythm; however, sinus tachycardia slows slightly in response to increased vagal tone.

Like PACs, PSVTs often occur in adults with normal hearts for the same reasons (eg, emotions, tobacco, alcohol,

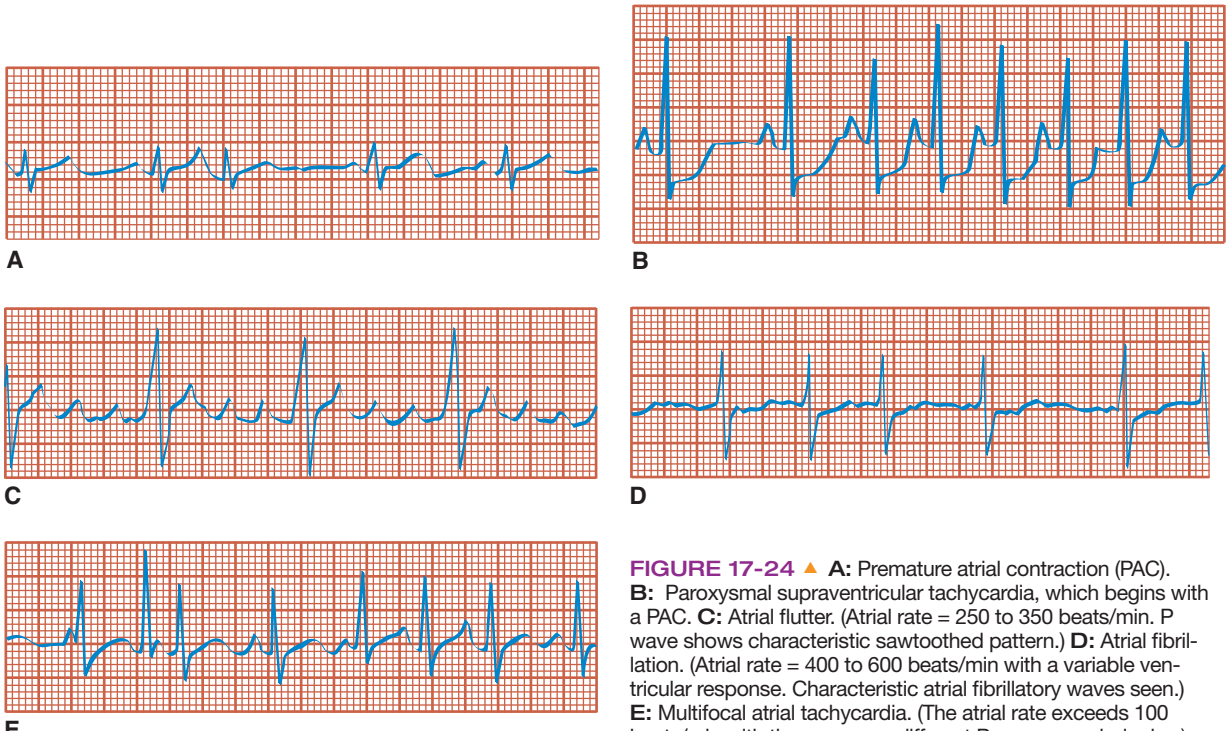


FIGURE 17-24 ▲ **A:** Premature atrial contraction (PAC). **B:** Paroxysmal supraventricular tachycardia, which begins with a PAC. **C:** Atrial flutter. (Atrial rate = 250 to 350 beats/min. P wave shows characteristic sawtoothed pattern.) **D:** Atrial fibrillation. (Atrial rate = 400 to 600 beats/min with a variable ventricular response. Characteristic atrial fibrillatory waves seen.) **E:** Multifocal atrial tachycardia. (The atrial rate exceeds 100 beats/min with three or more different P-wave morphologies.)

Table 17-12 Differential Diagnosis of Narrow QRS Tachycardia

Type of Supraventricular Tachycardia (SVT)	Onset	Atrial Rate	Ventricular Rate	RR Interval	Response to Carotid Massage
Sinus tachycardia	Gradual	100–180 beats/min	Same as sinus rate	Regular	Gradual slowing
Paroxysmal supraventricular tachycardia	Abrupt	150–250 beats/min	Usually same as atrial; block seen with digitalis toxicity and atrioventricular (AV) node disease	Regular, except at onset and termination	May convert to normal sinus rhythm
Atrial flutter	Abrupt	250–350 beats/min	Occurs with 2:1, 3:1, 4:1, or varied ventricular response	Regular or regularly irregular	Abrupt slowing of ventricular response; flutter waves remain
Atrial fibrillation	Abrupt	400–650 beats/min	Depends on ability of AV node to conduct atrial impulse; decreased with drug therapy	Irregularly irregular	Abrupt slowing of ventricular response; fibrillation waves remain

caffeine). When heart disease is present, such abnormalities as rheumatic heart disease, acute MI, and digitalis toxicity may serve as the background for a PSVT.

Often the patient has no underlying heart disease and may experience only palpitations and some lightheadedness, depending on the rate and duration of the PSVT. If the patient has underlying heart disease, dyspnea, angina pectoris, and heart failure may occur as ventricular filling time, and thus CO, is decreased.

Vagal stimulation often terminates the PSVT, either through carotid massage or the Valsalva maneuver. If vagal stimulation is unsuccessful, IV adenosine may be given. Cardioversion or overdrive pacing may be required if drug therapy is unsuccessful. Long-term prophylactic therapy may be indicated.

Atrial Flutter

Atrial flutter is a rapid atrial ectopic rhythm in which the atria fire at rates of 250 to 350 beats/min (Fig. 17-24C). The AV node functions as a "gatekeeper," preventing too many impulses from reaching the ventricle. If the ventricles are stimulated 250 to 350 times per minute, they are unable to respond with effective contractions, and CO is insufficient to sustain life. The AV node may allow only every second, third, or fourth atrial stimulus to proceed to the ventricles, resulting in what is known as a 2:1, 3:1, or 4:1 flutter block.

The rapid and regular atrial rate produces "sawtooth" or "picket-fence" P waves on the ECG. It is usual for a flutter wave to be partially concealed in the QRS complex or T wave. The QRS complex exhibits a normal configuration except when aberrant conduction is present.

When the ventricular rate is rapid, the diagnosis of atrial flutter may be difficult. Vagal maneuvers, such as carotid sinus massage or the administration of adenosine, increase the degree of AV block and allow recognition of flutter waves. Atrial flutter often is seen in the presence of underlying cardiac disease, including CAD, cor pulmonale, and rheumatic heart disease. If atrial flutter occurs in conjunction with a rapid ventricular rate, the ventricular chambers cannot fill adequately, resulting in varying degrees of hemodynamic compromise. Likewise, if atrial flutter is accompanied by a very slow ventricular rate, CO is diminished. The loss of "atrial kick," because atrial contraction is not occurring, is also a concern. The lack of atrial kick can compromise CO. Finally, without atrial contractions, thrombi can form on the walls of the atria. If these thrombi break loose, the result could be pulmonary embolus, cerebral embolus, or MI.

Treatment goals for atrial flutter are to reestablish sinus rhythm or to achieve ventricular rate control. When the ventricular rate is rapid, prompt treatment to control the rate or revert the rhythm to a sinus mechanism is indicated. Drugs may be selected to slow the conduction of the impulses through the AV node or to achieve pharmacological conversion of the rhythm. If pharmacological conversion is not successful, electrical cardioversion can be used. Synchronized cardioversion is especially useful in the prompt treatment of atrial flutter. The patient should be NPO before the procedure and receive sedation. (For a more detailed discussion of cardioversion, see Chapter 18.) If the patient has been experiencing atrial flutter for more than about 72 hours, anticoagulation may be needed before pharmacological or electrical conversion of the rhythm is attempted. Other modes of therapy may be indicated for the long-term management of atrial flutter, such as ablation, pacing, and implantable devices.

Atrial Fibrillation

Atrial fibrillation, which is a rapid atrial ectopic rhythm, occurring with atrial rates of 350 to 500 beats/min (Fig. 17-24D), is characterized by chaotic atrial activity with the absence of definable P waves. Instead, the P waves appear as small, quivering fibrillatory waves. Like atrial flutter, the ventricular rate and rhythm depend on the ability of the AV junction to function as a gatekeeper. If too many atrial stimuli pass through the AV junction, the ventricular response is rapid. If too few atrial stimuli pass through the AV junction, the ventricular response is slow. The ventricular rhythm is characteristically irregular.

Although atrial fibrillation may occur as a transient dysrhythmia in healthy young people, the presence of chronic atrial fibrillation is usually associated with underlying heart disease. One or both of the following are present in patients with chronic atrial fibrillation: atrial muscle disease or atrial distention together with disease of the sinus node. This rhythm commonly occurs in the setting of heart failure, ischemic or rheumatic heart disease, or pulmonary disease, and after open heart surgery. Atrial fibrillation also is seen in congenital heart disease.

The immediate clinical concern in patients with atrial fibrillation is the rate of the ventricular response. If the ventricular rate is too fast, end-diastolic filling time is decreased, and CO is compromised. If the ventricular rate is too slow, CO may again be decreased. As in atrial flutter, patients with atrial fibrillation have lost AV synchrony and atrial kick, resulting in a compromised CO. Patients also are at risk for the formation of mural thrombi and embolic events, such as stroke, MI, and pulmonary embolus.

The treatment principles for atrial fibrillation are the same as those for atrial flutter. The goal of therapy is to achieve rate control or to convert the rhythm to sinus.[17] If a patient has chronic atrial fibrillation, anticoagulant therapy is added to the drug regimen to prevent an embolic event. Cardioversion is indicated for rhythm control when drug therapy fails or in the setting of hemodynamic compromise. Ablation, pacing, and implantable devices are among the therapeutic options.[18]

Multifocal Atrial Tachycardia

Multifocal atrial tachycardia is a rapid atrial rhythm with varying P-wave morphology, resulting from the firing of three or more atrial foci (Fig. 17-24E). The atrial rate exceeds 100 beats/min, and the rhythm usually is irregular. The P waves vary in shape because of the multiple foci. The PR intervals may vary also, depending on the proximity of the focus to the AV node. The QRS complexes are normal unless an impulse is conducted with aberrancy.

This rhythm characteristically occurs in patients with severe pulmonary disease. Such patients often exhibit hypoxemia, hypokalemia, alterations in serum pH, or pulmonary hypertension. They usually manifest symptoms associated with the underlying disease rather than with the dysrhythmia itself. Treatment is directed at controlling the underlying pulmonary disease and slowing the ventricular rate if necessary.

▲ Junctional Dysrhythmias

Junctional Rhythm

A junctional rhythm, also known as a nodal rhythm, is a rhythm originating in the AV node. When the SA node fails to fire, the AV node usually takes control, but the rate is slower. The rate of a junctional rhythm ranges between 50 and 70 beats/min. The P wave in the dysrhythmia can have one of three possible configurations.

1. The AV node fires, and the wave of depolarization travels backward (retrograde conduction) into the atria. The impulse from the AV node then moves forward into the ventricle. When this sequence occurs, the P wave appears as an inverted wave before a normal QRS complex (Fig. 17-25A).
2. The retrograde conduction into the atria occurs at the same time as the forward conduction into the ventricles. The resulting rhythm strip shows an absent P wave with a normal QRS complex. In reality, the P wave is not absent. Instead, it is buried inside the QRS complex (see Fig. 17-25B).
3. Forward conduction of the ventricles precedes retrograde conduction of the atria. When this sequence occurs, a normal QRS complex is followed by an inverted P wave (see Fig. 17-25C).

A junctional rhythm may be the result of hypoxia, hyperkalemia, MI, heart failure, valvular disease, drugs (digoxin, beta-blockers, calcium channel blockers), or any cause of SA node dysfunction. Patients with a junctional rhythm may become symptomatic as a result of the slower rate. Hypotension, decreased CO, and decreased perfusion may occur. The benefit of AV synchrony and atrial kick may be lost when the atria are stimulated with or after ventricular depolarization.

Treatment should be directed at the underlying cause. Symptomatic patients may require immediate treatment. The heart rate can be increased through the use of atropine or cardiac pacing. Interventions are also directed toward improving CO.

Premature Junctional Contractions

A premature junctional contraction (PJC) is an ectopic impulse from a focus in the AV junction, occurring prematurely, before the next sinus impulse (Fig. 17-26). As in all rhythms originating in the AV junction, the QRS complex is narrow (<0.12 second), reflecting normal ventricular conduction. On rare occasions, the QRS complex may be wide if the impulse is conducted aberrantly. The atria are depolarized in a retrograde fashion before, during, or after ventricular excitation, producing inverted P waves that may occur before, during, or after the QRS complex. As with PACs, PJCs may occur in healthy people or in those with underlying heart disease. Ischemia or infarction may activate an ectopic focus in the AV junction, as may stimulants, such as nicotine or caffeine, or pharmacological agents (eg, digitalis).

Frequent PJCs may indicate increasing irritability and may be a precursor of a junctional rhythm. Although usually asymptomatic, patients may experience a "skipped beat." Treatment for PJCs is not necessary.

▲ Ventricular Dysrhythmias

Premature Ventricular Contractions

A PVC is an ectopic beat originating prematurely at the level of the ventricles (Fig. 17-27A). The beat is ventricular in origin and results in no electrical activity in the atria. As a result, no P waves appear. The ventricular depolarization does not travel through the normal rapid ventricular conduction system. Instead, ventricular conduction spreads more slowly through the Purkinje system, resulting in a wide

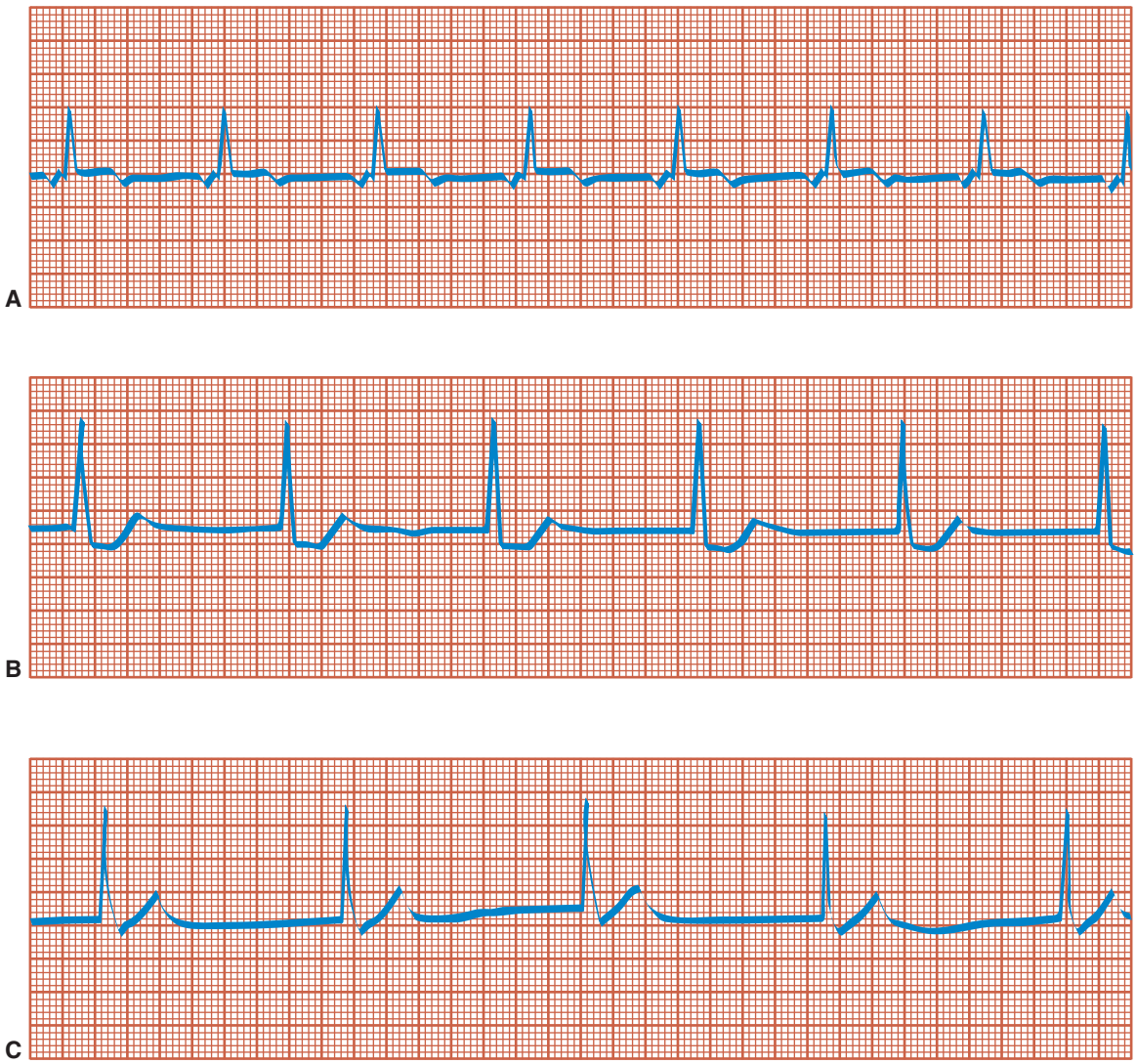


FIGURE 17-25 ▲ Junctional rhythm. **A:** A junctional rhythm in which the inverted P wave appears before a normal QRS complex. **B:** A junctional rhythm in which the inverted P wave is buried inside the QRS complex. **C:** A junctional rhythm in which the inverted P wave follows the QRS complex.

QRS complex with a T wave that is opposite in direction to the QRS complex. A compensatory pause often follows the premature beat as the heart awaits the next stimulus from the sinus node. The pause is considered fully compensatory if the cycles of the normal and premature beats equal the time of two normal heart cycles.

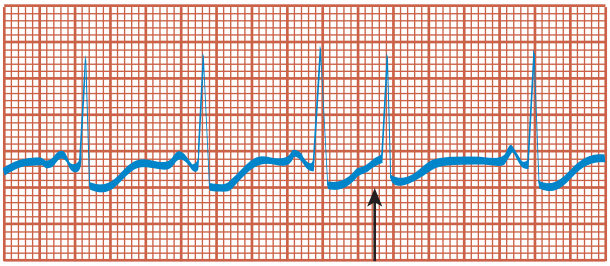


FIGURE 17-26 ▲ Premature junctional contraction.

Ventricular premature beats can be described by their frequency and pattern. They can be rare, occasional, or frequent; optimally, they are described as number of PVCs per minute. If PVCs occur after each sinus beat, ventricular bigeminy is present (see Fig. 17-27B). Ventricular trigeminy is a PVC occurring after two consecutive sinus beats. When PVCs appear in only one form (from one ventricular site), they are referred to as uniformed, as opposed to multiformed, when two or more forms (from more than one ventricular site) of the QRS complex are apparent (see Fig. 17-27C). Two PVCs in a row are a couplet (see Fig. 17-27D), whereas three in a row are a triplet, which is a short run of VT (see Fig. 17-27E).

The most common of all ectopic beats, PVCs can occur with or without heart disease in any age group. They are especially common in people with myocardial disease (ischemia or infarction) or with myocardial irritability (hypokalemia, increased levels of catecholamines, or mechanical irritation with a wire or catheter). The presence

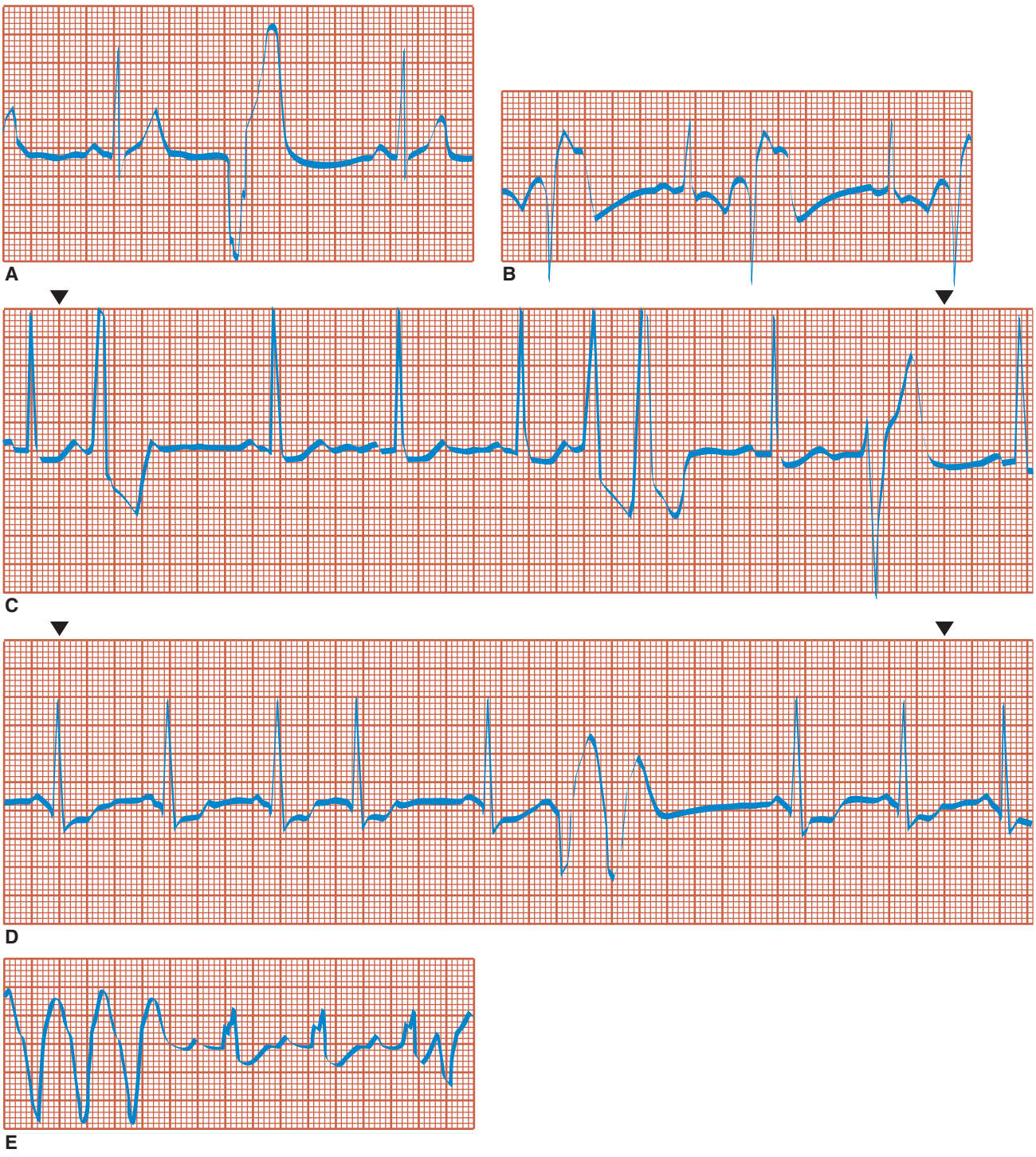


FIGURE 17-27 ▲ Ventricular dysrhythmias. **A:** Premature ventricular contractions (PVCs). **B:** Ventricular bigeminy. (Every other beat is a PVC.) **C:** Multiformed PVCs. **D:** Couplet (two PVCs in a row). **E:** Triplet. (Short run of ventricular tachycardia; the first three beats are VT with the rhythm converting to sinus rhythm with first-degree heart block.)

of PVCs is a sign of ventricular myocardial irritability and, in some patients, may lead to VT or ventricular fibrillation (VF). The nature of the patient's underlying heart disease, rather than the presence of PVCs as such, determines the treatment and prognosis. Numerous and multiformed PVCs in the presence of serious heart disease worsen the prognosis. PVCs approaching the apex of the preceding T wave (R-on-T phenomenon) are of clinical concern. The T wave represents ventricular repolarization, when the heart should not be stimulated. If stimulation occurs during this vulnerable period, VF and sudden death may result (Fig. 17-28).

Infrequent, isolated PVCs require no treatment. Multiple or consecutive PVCs may be managed with antiarrhythmic agents. In the emergency setting, amiodarone and lidocaine

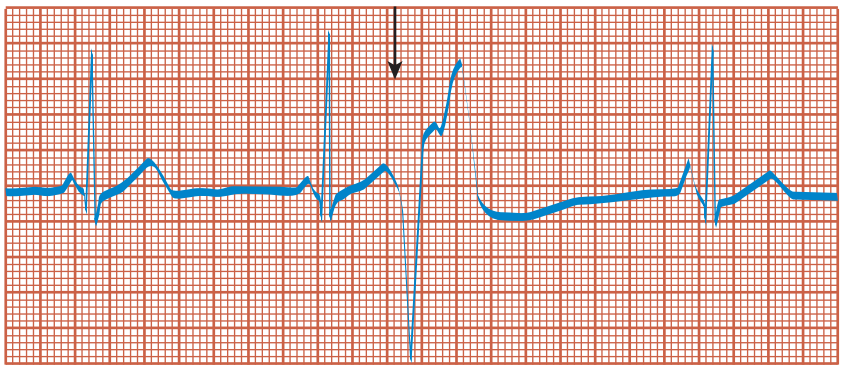


FIGURE 17-28 ▲ R-on-T premature ventricular contraction. (From Huff J: ECG Workout, 4th ed. Philadelphia, PA: Lippincott, Williams & Wilkins, 2002, p 195.)

are the drugs of choice. Many antiarrhythmic agents are available for chronic therapy. If serum potassium is low, potassium replacement may correct the dysrhythmia. If the dysrhythmia is caused by digitalis toxicity, withdrawal of the digitalis may correct it.

Ventricular Tachycardia

In the previous section, VT was defined as three or more PVCs in a row. VT is recognized by wide, bizarre QRS complexes occurring in a fairly regular rhythm at a rate greater than 100 beats/min (Fig. 17-29A). P waves usually are not seen and, if seen, are not related to the QRS complex. VT may be a short, nonsustained rhythm or longer and sustained.

In adults with normal hearts, VT is rare but is a common complication of MI. Other causes are the same as those described for PVCs. VT is a precursor of VF, and signs and symptoms of hemodynamic compromise (eg, ischemic chest pain, hypotension, pulmonary edema, and unconsciousness) may be seen if the rate is fast and the tachycardia is sustained. Serious dysrhythmia progression depends on the underlying heart disease.

If the patient is hemodynamically stable with the dysrhythmia, lidocaine may be administered intravenously. If the patient becomes unstable, synchronized cardioversion

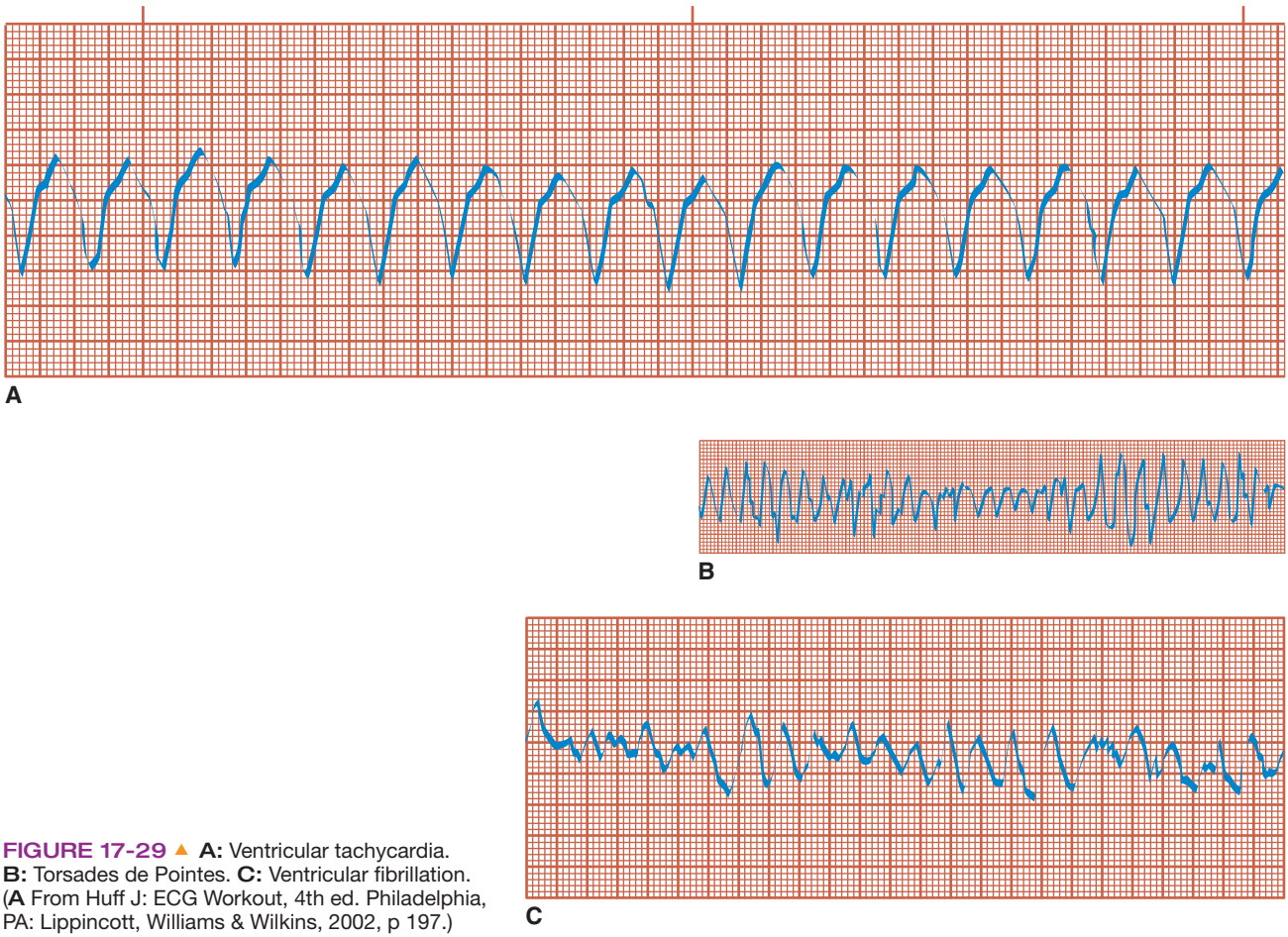


FIGURE 17-29 ▲ **A:** Ventricular tachycardia. **B:** Torsades de Pointes. **C:** Ventricular fibrillation. (**A** From Huff J: ECG Workout, 4th ed. Philadelphia, PA: Lippincott, Williams & Wilkins, 2002, p 197.)

(or in emergency situations, unsynchronized defibrillation) is indicated. Long-term treatment for this dysrhythmia may involve the use of an implantable cardioverter–defibrillator (ICD). See Chapter 18 for a more detailed discussion of ICDs.

Torsades De Pointes

Torsades de pointes ("twisting of the points") is a specific type of VT (Fig. 17-29B). The term refers to the polarity of the QRS complex, which swings from positive to negative and vice versa. The QRS complex morphology is characterized by large, bizarre, polymorphous, or multiformed QRS complexes of varying amplitude and direction, frequently varying from beat to beat and resembling torsion around an isoelectric line. The rate of the tachycardia is 100 to 180 beats/min but can be as fast as 200 to 300 beats/min. The rhythm is highly unstable; it may terminate in VF or revert to sinus rhythm. This form of VT is most likely to develop in myocardial disease when the underlying QT interval has been prolonged.

Torsades de pointes is favored by conditions that prolong the QT interval. Examples include severe bradycardia; drug therapy, especially with type IA antiarrhythmic agents; and electrolyte disturbances, such as hypokalemia and hypocalcemia. Other factors that can precipitate this dysrhythmia include intrinsic cardiac disease, familial QT-interval prolongation, drug-induced prolongation of the QT interval, hypokalemia, hypomagnesemia, and hypocalcemia.[19] Torsades de pointes may terminate spontaneously and may repeat itself after several seconds or minutes, or it may transform into VF.

Treatment for torsades de pointes consists of shortening the refractory period (and thus the QT interval) of the underlying rhythm. IV magnesium sulfate, magnesium chloride, or isoproterenol is effective in suppression of the dysrhythmia. Overdrive pacing also can be used. Treatment is directed at correcting the underlying problem and may necessitate stopping the offending pharmacological agent or correcting the electrolyte imbalance. Emergency cardioversion or defibrillation is indicated if the torsades does not revert spontaneously to sinus rhythm.

Ventricular Fibrillation

VF is defined as rapid, irregular, and ineffectual depolarizations of the ventricle (Fig. 17-29C). No distinct QRS complexes are seen. Only irregular oscillations of the baseline are apparent; these may be either coarse or fine in appearance.

VF may occur in the following circumstances: myocardial ischemia and infarction, catheter manipulation in the ventricles, electrocution, prolonged QT interval, or as a terminal rhythm in patients with circulatory failure. As in asystole, loss of consciousness occurs within seconds in VF. There is no pulse and no CO. VF is the most common cause of sudden cardiac death and is fatal if resuscitation is not instituted immediately.

If VF occurs, rapid defibrillation is the management of choice (see the discussion of cardiopulmonary resuscitation in Chapter 18). The patient should be supported with cardiopulmonary resuscitation and drugs if there is no response to defibrillation. An ICD may be indicated for long-term management of VF (see Chapter 18 for a discussion of ICDs).

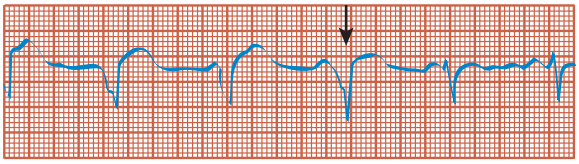


FIGURE 17-30 ▲ Accelerated idioventricular rhythm. The first three beats are of ventricular origin. The fourth beat (*arrow*) represents a fusion beat. The subsequent two beats are of sinus origin.

Accelerated Idioventricular Rhythm

Accelerated idioventricular rhythm (AIVR) is produced by a "speeding up" of ventricular pacemaker cells, which normally have an intrinsic rate of 20 to 40 beats/min (Fig. 17-30). When the idioventricular rate accelerates above the sinus rate, the ventricular pacemaker becomes the primary pacemaker for the heart. AIVR is characterized by wide QRS complexes occurring regularly at a rate of 50 to 100 beats/min. AIVR may last for a few beats or may be sustained.

Typically, AIVR is seen with acute MI, often in the setting of coronary artery reperfusion after thrombolytic therapy. It may occur less commonly as a result of ischemia or digitalis intoxication. Patients usually are not symptomatic. Adequate CO can be maintained, and degeneration into a rapid VT is rare.

In most cases, treatment is not necessary. If a patient is hemodynamically compromised, the sinus rate is increased with atropine or atrial pacing to suppress the AIVR.

▲ Atrioventricular Blocks

A disturbance in some portion of the AV conduction system causes an AV block. The sinus-initiated beat is delayed or completely blocked from activating the ventricles. The block may occur at the level of the AV node, bundle of His, or the bundle branches because the AV conduction system contains all of these structures. In first- and second-degree AV block, the block is incomplete; some or all of the impulses eventually are conducted to the ventricles. In third-degree or complete heart block, none of the sinus-initiated impulses is conducted. Table 17-13 summarizes and compares heart block rhythms.

First-Degree Atrioventricular Block

In first-degree block, AV conduction is prolonged and equal in time. All impulses eventually are conducted to the ventricles (Fig. 17-31A). P waves are present and precede each QRS complex in a 1:1 relationship. The PR interval is constant but exceeds the upper limit of 0.20 second in duration.

First-degree heart block occurs in people of all ages and in healthy and diseased hearts. PR prolongation may be

Table 17-13 A Comparison of the Electrocardiographic Characteristics of Heart Block Rhythms

	First-Degree Heart Block	Second-Degree Heart Block—Mobitz Type I (Wenckebach)	Second-Degree Heart Block—Mobitz Type II	Third-Degree Heart Block
Rate	Usually 60–100 beats/min	Usually 60–100 beats/min	May be slow depending on number of blocked P waves	Rate determined by ventricular focus, usually very slow
Rhythm	Regular	Irregular due to dropped QRS	Often regular but depends on pattern of block	May be regular or irregular ventricular focus
P waves	Present, one per QRS	Present, one per QRS until QRS is missed	Present, more than one P wave per QRS	Present, more than one P wave per QRS; P waves no relationship to QRS complexes
PR interval	>0.20 s, equal throughout	Progressively gets longer until QRS is missed; pattern repeats	May be normal or prolonged, equal throughout	May be normal or prolonged, unequal throughout
QRS complex	<0.12 s	<0.12 s	Usually more than 0.12 s	More than 0.12 s

caused by drugs, such as digitalis, β-blockers, or calcium channel blockers; CAD; a variety of infectious diseases; and congenital lesions. First-degree block is of no hemodynamic consequence but should be seen as an indicator of a potential AV conduction system disturbance. First-degree block may progress to second- or third-degree AV block.

No treatment is indicated for first-degree heart block. The PR interval should be monitored closely, watching for further block. The possibility of a drug effect also should be evaluated.

Second-Degree Atrioventricular Block—Mobitz I (Wenckebach)

Mobitz type I (Wenckebach) block occurs when AV conduction is delayed progressively with each sinus impulse until eventually the impulse is completely blocked from reaching the ventricles. The cycle then repeats itself (see Fig. 17-31B). Of the two types of second-degree block, Mobitz I (Wenckebach) and Mobitz II, Mobitz I occurs more commonly.

On the ECG tracing, P waves are present and related to the QRS complex in a cyclical pattern. The PR interval progressively lengthens with each beat until a QRS complex is not conducted. The QRS complex has the same configuration throughout the underlying rhythm.

A Mobitz type I block usually is associated with block above the bundle of His. Therefore, any drug or disease process that affects the AV node, such as digitalis, myocarditis, or an inferior wall MI, may produce this type of second-degree block.

Patients with Mobitz type I second-degree AV block rarely are symptomatic because the ventricular rate usually is adequate. Wenckebach block often is temporary, and if it progresses to third-degree block, a junctional pacemaker at a rate of 40 to 60 beats/min usually takes over to pace the ventricles. No treatment is required for this rhythm except to discontinue a drug if it is the offending agent. The patient should be monitored for further progression of block.

Second-Degree Atrioventricular Block—Mobitz II

Mobitz type II block is described as an intermittent block in the AV conduction, usually in or below the bundle of His. Mobitz type II block is characterized by a fixed PR interval when AV conduction is present and a nonconducted P wave when the block occurs (see Fig. 17-31C). This block in conduction can occur occasionally or be repetitive with a 2:1, 3:1, or even 4:1 conduction pattern. Because there is no disturbance in the sinus node, the PP interval is regular. Often there is accompanying BBB, so the QRS complex may be wide.

A Mobitz type II pattern is seen in the setting of an anterior wall MI and various diseases of the conducting tissue, such as fibrotic disease. A Mobitz type II block is potentially more dangerous than a Mobitz type I block. Mobitz type II block often is permanent, and it may deteriorate rapidly to third-degree heart block with a slow ventricular response of 20 to 40 beats/min.

Constant monitoring and observation for progression to third-degree heart block are required. Medications, such as atropine, or cardiac pacing may be required if a patient becomes symptomatic or if the block occurs in the setting of an acute anterior wall MI. Permanent pacing often is indicated for long-term management.

Third-Degree (Complete) Atrioventricular Block

In third-degree or complete heart block, the sinus node continues to fire normally, but the impulses do not reach the ventricles (see Fig. 17-31D). The ventricles are stimulated

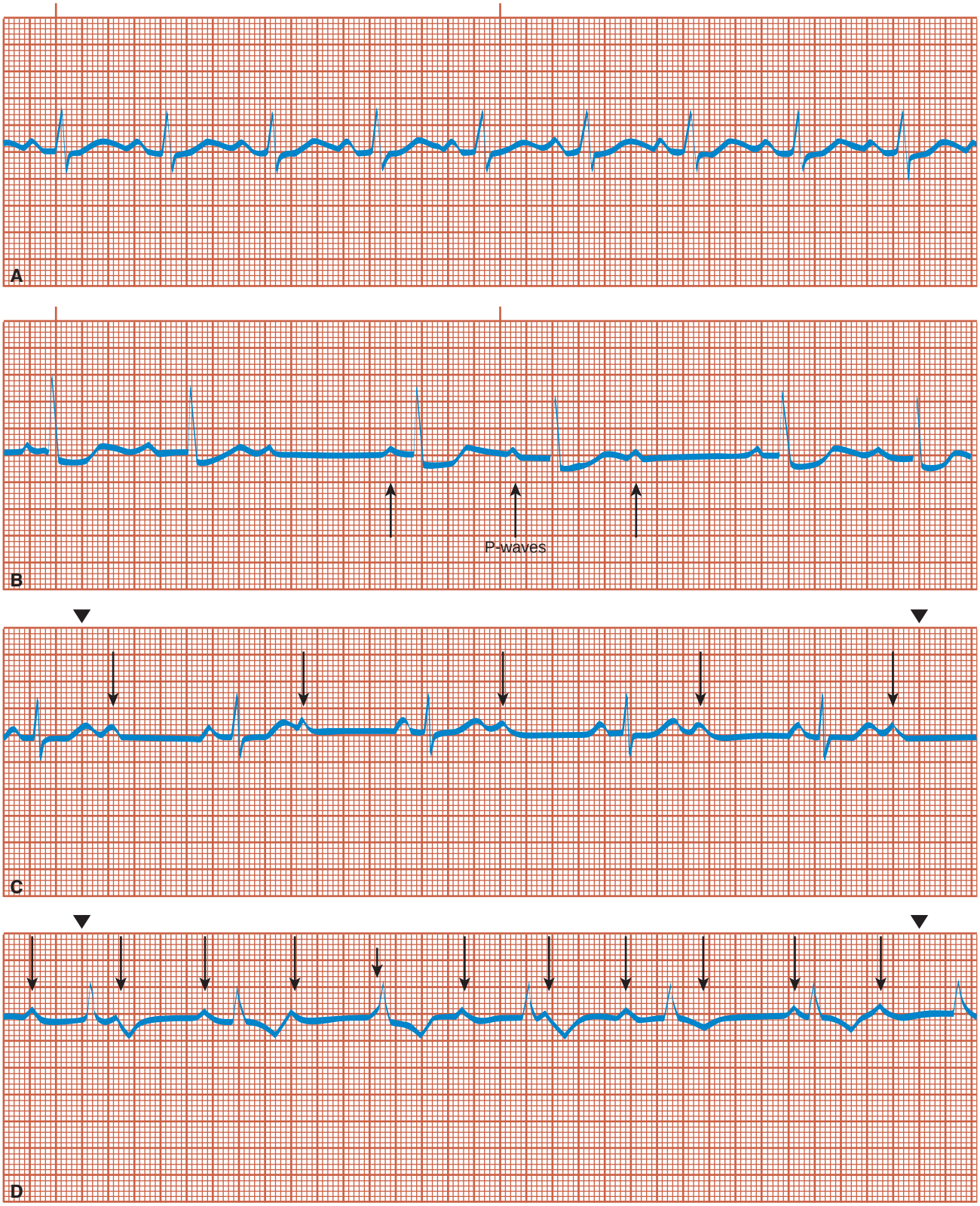


FIGURE 17-31 ▲ Heart block rhythms. **A:** First-degree heart block. **B:** Second-degree heart block: Mobitz type I. **C:** Second-degree heart block: Mobitz type II. *Arrows* denote blocked P wave (2:1 block). **D:** Third-degree heart block (complete atrioventricular block). *Arrows* denote P waves. Note the lack of relationship between the atria (P wave) and ventricles (QRS). (**A** and **B,** From Huff J: ECG Workout, 4th ed. Philadelphia, PA: Lippincott, Williams & Wilkins, 2002, pp 150, 156.)

from escape pacemaker cells either in the junction (at a rate of 40 to 60 beats/min) or in the ventricles (at a rate of 20 to 40 beats/min), depending on the level of the AV block.

On the ECG tracing, P waves and QRS complexes are both present, but there is no relationship between the two. Therefore, complete heart block is considered one form of AV dissociation. The PP and RR intervals are each regular, but the PR interval is variable. If a junctional pacemaker paces the ventricles, the QRS complex is narrow. A pacemaker site lower in the ventricles produces a wide QRS complex.

The causes of complete heart block are the same as for lesser degrees of AV block. Complete heart block is often poorly tolerated. The rate and dependability of the ventricular pacemaker depend on its location. If the escape rhythm is ventricular in origin, the rate is slow, and the pacemaker site is unreliable. The patient may be symptomatic because of a low CO. A pacemaker site high in the bundle of His may provide an adequate rate and is more dependable. The patient may remain asymptomatic if the escape rhythm supports a normal CO.

A temporary pacing wire is usually inserted immediately, and when the patient is stabilized, a permanent pacemaker is implanted.

▲ The 12-Lead Electrocardiogram

The Normal 12-Lead Electrocardiogram

As previously described, the ECG provides 12 electrical views of the heart. The first three electrical views are provided by the standard leads I, II, and III. The next three electrical views are provided by the augmented leads, aVR, aVL, and aVF. The standard and augmented leads are referred to as the limb leads and provide a view from a vertical plane. The remaining six electrical views of the heart, the precordial leads, chest leads, or V leads, V_1 through V_6, provide a horizontal plane view of the heart (Fig. 17-32).

In the normal 12-lead ECG, the P wave representing atrial depolarization is usually upright and rounded. Each component of the QRS complex (ventricular depolarization) is analyzed separately. The Q wave, the initial downward deflection of the QRS complex, should be absent or small. The R component is the tallest upright portion of the QRS complex in the limb leads except aVR. In the precordial leads, the R wave begins as a small wave in V_1 and gradually progresses to a tall wave by V_6. The S wave, the downward stroke after the R wave, is small or absent in the limb leads. The S wave begins as a deep wave in V_1 and gradually disappears by V_6 in the precordial leads. The ST segment is isoelectric but may be slightly elevated in V_1 through V_3. The T wave, representing ventricular repolarization, is usually upright, although a variety of configurations can be normal. Table 17-14 summarizes the normal 12-lead ECG.

The 12-lead ECG may be useful in determining the electrical axis of the heart and detecting abnormalities that require more than one electrical view. These abnormalities include BBB; atrial or ventricular enlargement; and patterns of ischemia, injury, or infarction.

Electrical Axis

Electrical axis refers to the general direction of the wave of excitation as it moves through the heart. In the normal heart, the flow of electrical forces originates in the SA node, spreads throughout atrial tissue, passes through the AV node, and moves throughout the ventricles. This flow of forces is normally downward and to the left, a pattern known as normal axis.

The ventricles make up the largest muscle mass of the heart and therefore make the most significant contribution to the determination of the direction of the flow of forces in the heart. For this reason, the QRS complex is examined when deciding the electrical axis.

A quick way to estimate the axis of the heart is to examine the direction of the QRS complex in leads I and aVF (Fig. 17-33). A QRS complex that is mainly upright in both leads represents a normal axis. A QRS complex that is upright in lead I and downward in lead aVF represents left

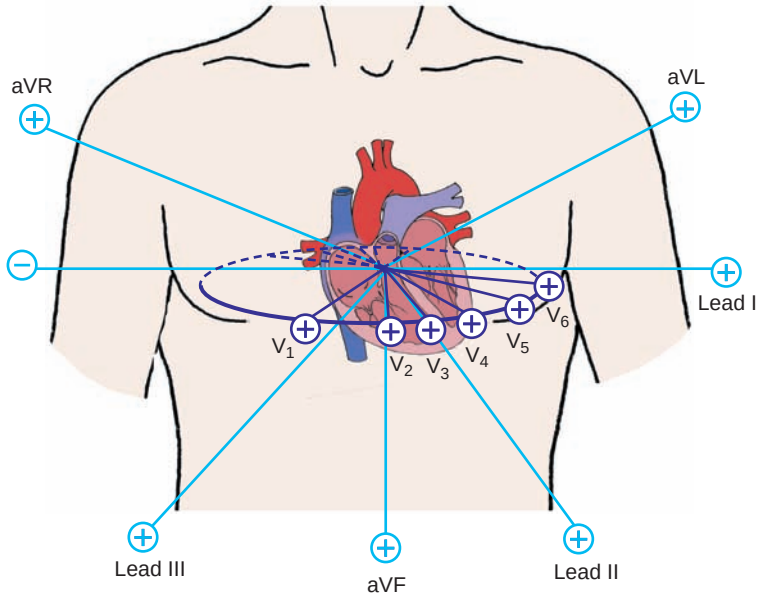


FIGURE 17-32 ▲ Electrocardiographic views of the heart.

Table 17-14 The Normal 12-Lead Electrocardiogram

Lead	P	Q	R	S	S-T	T
I	Upright	Small, 0.04 s, or none	Dominant	Less than R or none	Isoelectric +1 to –0.5 mm	Upright
II	Upright	Small or none	Dominant	Less than R or none	+1 to –0.5 mm	Upright
III	Upright Flat Diphasic Inverted	Small or none	None to dominant	None to dominant	+1 to –0.5 mm	Upright Flat Diphasic Inverted
aVR	Inverted	Small, or large	Small or none	Dominant	+1 to –0.5 mm	Inverted
aVL	Upright Flat Diphasic Inverted	Small, none, or large	Small, none, or dominant	Small, none, or dominant	+1 to –0.5 mm	Upright Flat Diphasic Inverted
aVF	Upright Flat Diphasic Inverted	Small or none	Small, none, or dominant	None to dominant	+1 to –0.5 mm	Upright
V_1	Upright Flat Diphasic	None May be QS	Small	Deep	0 to +3 mm	Inverted Flat Upright Diphasic
V_2	Upright	None			0 to +3 mm	Upright Diphasic Inverted
V_3	Upright	Small or none			0 to +3 mm	Upright
V_4	Upright	Small or none			+1 to –0.5 mm	Upright
V_5	Upright	Small			+1 to –0.5 mm	Upright
V_6	Upright	Small	Tall	Small or none	+1 to –0.5 mm	Upright

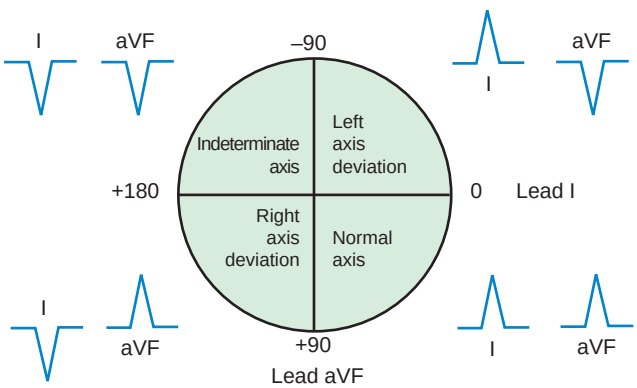


FIGURE 17-33 ▲ Determining electrical axis. To determine the axis of the heart, examine the direction of the QRS complex in leads I and aVF.

Lead I	*Lead aVF*	*Axis*
Negative	Negative	Indeterminate axis
Negative	Positive	Right axis deviation
Positive	Negative	Left axis deviation
Positive	Positive	Normal axis

axis deviation. A QRS complex that is downward in lead I and upright in lead aVF represents right axis deviation. A QRS complex that is downward in leads I and aVF is uncommon and represents indeterminate axis.

The direction of the flow of forces in the heart can change as a result of an anatomical shift of the heart in the chest wall. An anatomical shift may occur in very obese patients or in patients with large abdominal tumors or abdominal ascites. Left axis deviation can be caused by LBBB, left ventricular enlargement, or inferior wall MI. Right axis deviation can be caused by RBBB, right ventricular enlargement, or an anterior wall MI.

Patients with an axis shift are asymptomatic. The only way an axis shift can be detected is through a 12-lead ECG. The axis shift usually represents some underlying abnormality, and treatment is directed at the underlying cause.

Bundle Branch Block

A BBB develops when there is either a functional or pathological block in one of the major branches of the intraventricular conduction system. As conduction through one

bundle is blocked, the impulse travels along the unaffected bundle and activates one ventricle normally. The impulse is delayed in reaching the other ventricle because it travels outside of the normal conducting fibers. The right and left ventricles are thus depolarized sequentially instead of simultaneously. The abnormal activation produces a wide QRS complex, representing the increased time it takes for ventricular depolarization (Fig. 17-34). The broad QRS complex has two peaks (RSR′), indicating that depolarization of the two ventricles was not simultaneous.

An RBBB and LBBB are diagnosed on the 12-lead ECG but can also be identified on the bedside monitor using a V_1 or MCL_1 tracing and a V_6 or MCL_6 tracing (see section on Electrocardiographic Monitoring for description of lead selection). To identify the presence of a BBB, the QRS complex duration must be prolonged to 0.12 second or longer, representing the delay in conduction through the ventricles. An RBBB alters the configuration of the QRS complex in the right-sided chest leads, V_1 and V_2. Normally, these leads have a small, single-peaked R-wave and deep S-wave configuration. With an RBBB, depolarization of the right ventricle is delayed, and the ECG pattern changes. An RBBB is evidenced by an RSR′ configuration in V_1. If the initial peak of the QRS complex is smaller than the second peak, the pattern would be described as rSR′. An "r" is used to describe the first, smaller peak, and an "R" is used to describe the second, taller peak. Likewise, if the initial peak of the QRS complex is taller than the second peak, the pattern is described as an RSr'. Whenever ventricular depolarization is abnormal, so is ventricular repolarization. As a result, ST-segment and T-wave abnormalities may be seen in leads V_1 and V_2 for patients with an RBBB.

An LBBB changes the QRS complex pattern in the left-sided chest leads, V_5 and V_6. Normally, these leads have a tall, single-peaked R wave and a small or absent S wave. Instead, the double-peaked RSR′ pattern is noted. In addition, V_1 shows a small R wave with a widened S wave, indicating delayed conduction through the ventricles. Like RBBB, the ST segments and T waves may be abnormal in the left-sided chest leads V_5 and V_6 when the patient has an LBBB (see Fig. 17-34).

The most common causes of BBB are MI, hypertension, heart failure, and cardiomyopathy. RBBB may be found in healthy people with no clinical evidence of heart disease.

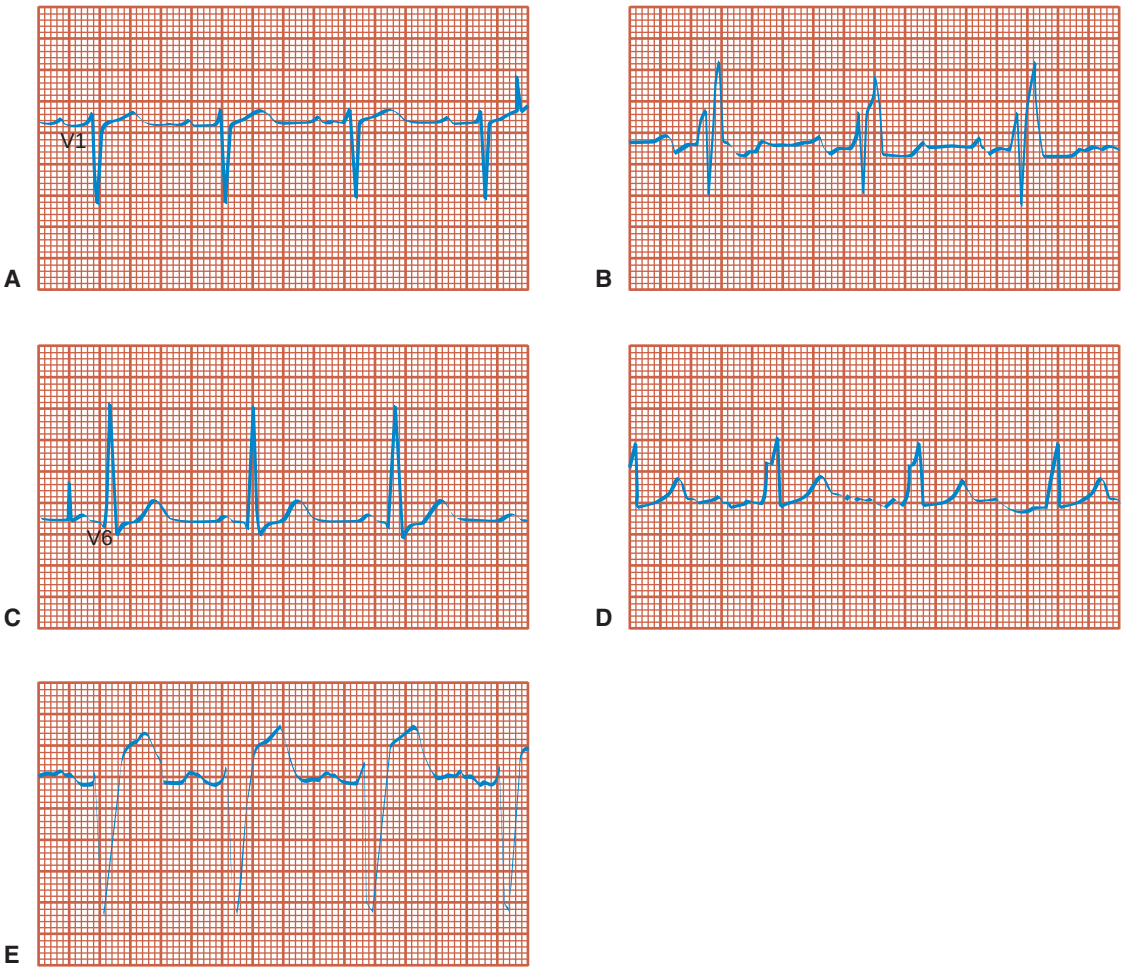


FIGURE 17-34 ▲ Comparison of right versus left bundle branch block (LBBB). **A:** A normal V_1 tracing. Note the small narrow R and deep narrow S wave. **B:** V_1 tracing showing the wide QRS complex and double-peaked R wave, indicating a right bundle branch block. **C:** A normal V_6 tracing. Note the tall narrow R wave and absent S wave. **D:** A V_6 tracing showing the wide QRS complex and double-peaked R wave, indicating a LBBB. **E:** A V_1 tracing. Note the small narrow R and deep wide S wave, indicating a LBBB.

Congenital lesions involving the septum and right ventricular hypertrophy (RVH) are other causes of RBBB. LBBB is usually associated with some type of underlying heart disease. Long-term CVD in the older patient is a common cause of LBBB.

BBB signifies underlying disease of the intraventricular conduction system. Patients should be monitored for involvement of the other bundles or fascicles or for progression to complete heart block. Progression of block may be very slow or rapid, depending on the underlying cause. A new-onset LBBB in conjunction with an acute MI is associated with a higher mortality rate.

The underlying heart disease determines treatment and prognosis. Patients with an MI and new-onset BBB are closely monitored for progression to a type of complete heart block. A temporary pacemaker may be inserted.

Enlargement Patterns

Enlargement of a cardiac chamber may involve hypertrophy of the muscle or dilation of the chamber. The most common causes include pumping for a prolonged period against high pressures or pumping for a prolonged period to move blood through narrowed valves. Electrocardiography is not an ideal diagnostic tool for determining the cause of the enlargement. Echocardiography is more helpful in determining if the enlargement is the result of hypertrophy or dilation. The terminology used to describe enlargement patterns on the ECG can be confusing. The term ventricular hypertrophy is commonly used because hypertrophy is the most frequent cause of the enlargement pattern in the ventricles. The general terms atrial abnormality and atrial enlargement are often used rather than the specific terms atrial hypertrophy or atrial dilation because atrial changes on the ECG may result from a variety of causes, including atrial dilation, hypertrophy, or other conditions. (See Fig. 17-35A for comparison.)

Right Atrial Enlargement

When the atria enlarge, changes are seen in the P wave because the P wave represents atrial depolarization. Right atrial (RA) enlargement is noted on the ECG by the presence of tall, pointed P waves in leads II, III, and aVF. The P wave in V_1 may show a diphasic wave with an initial upstroke that is larger than the downstroke (Fig. 17-35B).

The right atrium is more likely to enlarge as a result of pressures created by pulmonary causes, such as pulmonary hypertension and chronic obstructive pulmonary disease. For this reason, RA enlargement is often referred to as P pulmonale. RA enlargement is often associated with RVH.

Treatment is directed at the underlying cause. Often, however, the underlying cause may be a chronic condition that cannot be cured.

Left Atrial Enlargement

Left atrial enlargement is noted on the ECG by the presence of broad, notched P waves in leads I, II, and aVL. The P wave in V_1 may show a diphasic wave with a terminal downstroke that is larger than the initial upstroke (see Fig. 17-35C).

The left atrium is more likely to enlarge because of increased pressures created by trying to pump blood through a stenotic mitral valve. For this reason, left atrial enlargement is often referred to as P mitrale. When a left atrial enlargement pattern is noted on the ECG, the patient should be evaluated for the presence of mitral stenosis. An echocardiogram is a helpful diagnostic tool in addition to cardiac auscultation. Treatment is directed at the underlying cause. A valve replacement may be necessary.

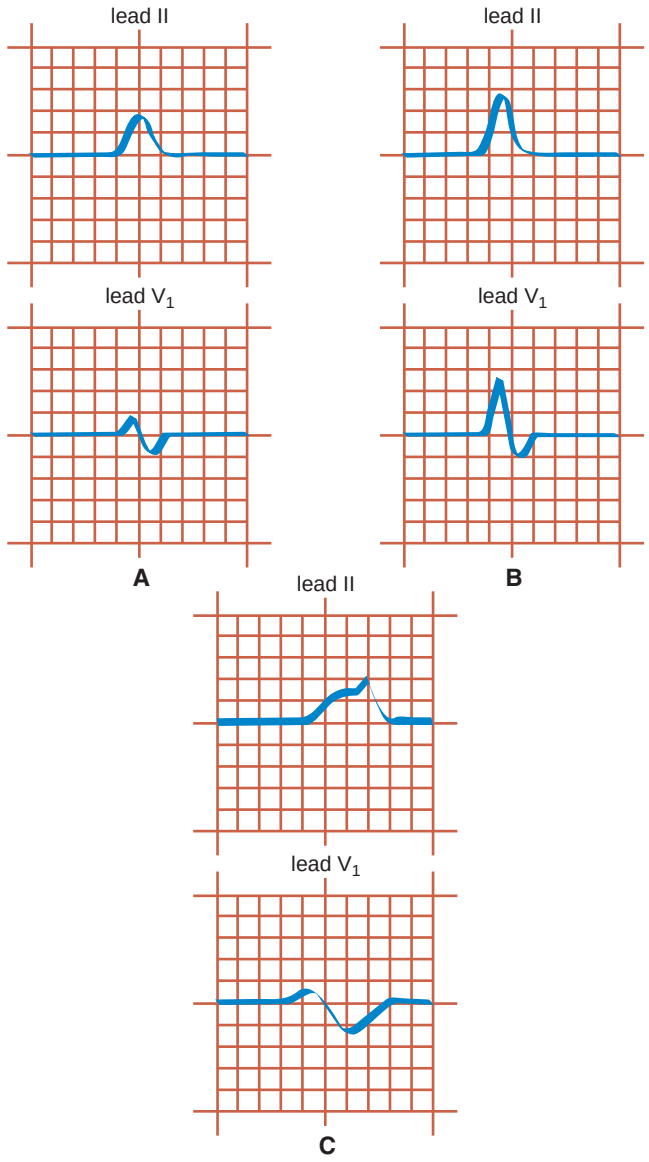


FIGURE 17-35 ▲ Right versus left atrial enlargement. **A:** The normal P wave in leads II and V_1. **B:** Right atrial (RA) enlargement. Note the increased amplitude of the early, RA component of the P wave in V_1 and the tall, pointed P wave in lead II. **C:** Left atrial enlargement. Note the increased terminal downstroke and duration of the P wave in V_1 and the broad, notched P wave in lead II.

Right Ventricular Hypertrophy

RVH may exist without clear evidence on the ECG because the left ventricle normally is larger than the right and can mask changes in the size of the right ventricle. ECG evidence suggestive of RVH includes RA enlargement and right axis deviation. In addition, the normal QRS complex pattern across the precordial leads is reversed. Normally, R waves are small in V_1 and gradually

grow tall by V_6. With RVH, the R wave is tall in V_1 and progresses to small by V_6. Precordial S waves persist rather than gradually disappear.

The presence of RVH is most likely an indicator of a chronic pulmonary condition, most likely chronic obstructive pulmonary disease, pulmonary hypertension, or pulmonic stenosis. RA enlargement is usually seen with an accompanying RVH. Treatment is directed at the underlying pulmonary disease.

Left Ventricular Hypertrophy

Numerous criteria exist for the detection of left ventricular hypertrophy (LVH) on the ECG. The simplest criterion involves remembering the number "35." LVH is determined by adding the deepest S wave in either V_1 or V_2 to the tallest R wave in either V_5 or V_6. If the sum is 35 mm or more and the patient is older than 35 years of age, LVH is suspected. In addition, the T waves in V_5 and V_6 may be asymmetrically inverted, and a left axis shift is likely.

Usually, LVH is the result of chronic systemic hypertension, a chronic cardiovascular problem, or aortic stenosis. LVH may result in a displacement of the PMI when palpating the apical pulse. Treatment of LVH is directed at the underlying condition.

Ischemia, Injury, and Infarction Patterns

The 12-lead ECG can be very useful in detecting evidence of myocardial ischemia, injury, or infarction. Ischemia is seen on the ECG by ST-segment depressions and T-wave inversions. Acute patterns of injury are noted by ST-segment elevations. The presence of significant Q waves indicates an MI. For a more detailed discussion of patterns of ischemia, injury, and infarction, see Chapter 21.

EFFECTS OF SERUM ELECTROLYTE ABNORMALITIES ON THE ELECTROCARDIOGRAM

Maintenance of adequate fluid and electrolyte balance assumes high priority in the care of patients in any medical, surgical, or coronary ICU. Patients being treated for renal or cardiovascular diseases are especially vulnerable to electrolyte imbalances. The cure may well be worse than the disease if electrolyte abnormalities go undetected or ignored because they frequently are caused by the treatment rather than by the disease itself.

Diuresis can very quickly cause major shifts in electrolytes. Certainly, the often insidious drop of serum potassium levels in the patient with cardiac disease, who has been taking digitalis and then starts diuretics, is well known. Diuretics also are used frequently as part of the medical regimen for the control of hypertension. Any addition, deletion, or change in diuretic therapy warrants close monitoring of serum electrolytes. A history of any of these problems should alert the nurse to check the patient's serum electrolytes on an ongoing basis.

Potassium and calcium are probably the two most important electrolytes involved in the proper function of the heart. Because of their effects on the electrical impulse in the heart, an excess or insufficiency of either electrolyte frequently causes changes in the ECG (Table 17-15). The nurse who is aware of and able to recognize these changes may well suspect electrolyte abnormalities before laboratory findings or clinical symptoms appear and hazardous dysrhythmias occur.

However, it is necessary to remember that just as a patient who sustains MI may not have chest pain, the patient with electrolyte abnormalities may not exhibit any of the ECG changes described in the following sections. The ECG manifestations are valuable primarily in arousing suspicion of electrolyte abnormalities. Not one of them even approaches being diagnostic.

▲ Potassium

Potassium is the primary intracellular cation found in the body. Inside the cardiac cell, potassium is important for repolarization and for maintaining a stable, polarized state.

Hyperkalemia

The earliest sign of hyperkalemia on the ECG is a change in the T wave. It usually is described as tall, narrow, and "peaked" or "tented" in appearance (Fig. 17-36). As the serum potassium level increases, the P-wave amplitude decreases and the PR interval is prolonged. Atrial asystole occurs, along with a widening of the QRS complex. At high, near-lethal potassium levels, the widened QRS complex merges with the T wave and starts to resemble a sine wave. Various dysrhythmias may occur during this time, with progression to VF and asystole. Clinically, the described changes in T waves begin to appear at serum levels of 6 to 7 mEq/L, and QRS complex widening is seen at serum levels of 8 to 9 mEq/L. Vigorous treatment must be instituted to reverse the condition at this point because sudden death may occur at any time after these levels are reached.

The ECG changes in hyperkalemia also may be associated with other conditions. Tall, peaked T waves may be a normal finding or may occur in the early stages of MI. QRS

Table 17-15 Electrocardiographic Changes Associated With Electrolyte Imbalances

Hyperkalemia	Tall, narrow, peaked T waves; flat, wide P waves; widening QRS complex	Sinus bradycardia; sinoatrial block; junctional rhythm; idioventricular rhythm; ventricular tachycardia (VT); ventricular fibrillation (VF)
Hypokalemia	Prominent U waves; ST segment depression; T-wave flattening or inversion	Premature ventricular beats; SVT; VT; VF
Hypercalcemia	Shortened QT interval	Premature ventricular contractions
Hypocalcemia	Lengthened QT interval; T-wave flattening or inversion	Ventricular tachycardia

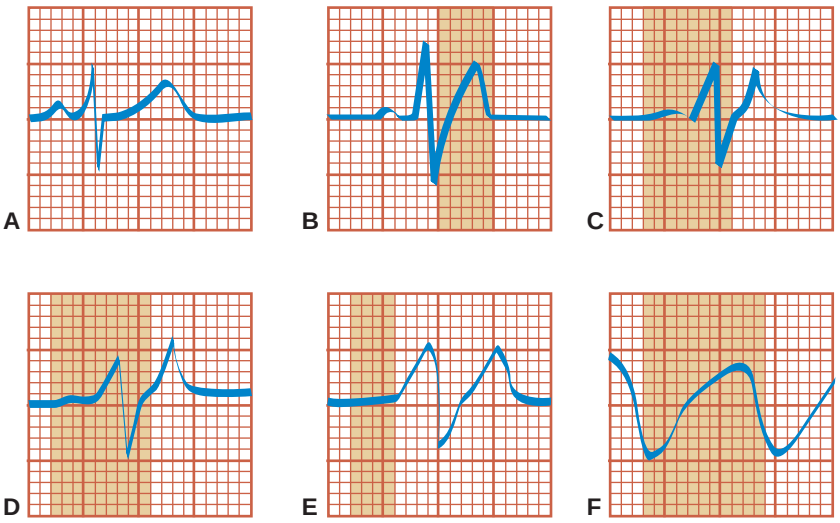


FIGURE 17-36 ▲ The effect of hyperkalemia on an ECG. **A:** This waveform is produced when the serum potassium level falls within the normal range—usually considered to be 3.5 to 5 mEq/L. **B:** When the serum potassium level rises above 5.5 mEq/L, the T wave begins to peak (*see highlighted area*). The P wave and QRS complex are normal. **C:** When the potassium level exceeds 6.5 mEq/L, the P wave grows wider, and its amplitude falls. The QRS complex also widens (*see highlighted area*) as intraventricular conduction velocity diminishes. **D:** When the potassium level reaches 10 mEq/L, the P wave becomes almost indiscernible; the QRS complex is slurred and widened (*see highlighted area*). **E:** When the potassium level ranges from 10 to 12 mEq/L, the P wave is undetectable (*see highlighted area*) because the atria are no longer excitable. **F:** When the potassium level exceeds 12 mEq/L, the QRS complex is no longer identifiable. The waves are known as sine waves (*see highlighted area*). Ventricular fibrillation and cardiac arrest follow. (From Springhouse: ECG Interpretation: Clinical Skillbuilders. Springhouse, PA: Author, 1990, p 113.)

complex widening may be seen with quinidine and procainamide toxicity.

Hypokalemia

Hypokalemia is associated with the appearance of U waves. Although the presence of U waves may be normal in many people, these waves also may be an early sign of hypokalemia (Fig. 17-37). Usually easily recognized (best seen in lead V_3), a U wave may encroach on the preceding T wave and go unnoticed. The T wave may look notched or prolonged when it is hiding the U wave, giving the appearance of a prolonged QT interval. With increased potassium depletion, the U wave may become more prominent as the T wave becomes less so. The T wave becomes flattened and may even invert. The ST segment tends to become depressed, somewhat resembling the effects of digitalis on the ECG. Only at very low serum levels is there reasonable correlation between ECG changes and serum potassium concentrations.

The changes seen in hypokalemia also are observed in other conditions. The U wave may be accentuated in association with digitalis, LVH, and bradycardia.

Untreated hypokalemia enhances instability in the myocardial cell. Ventricular premature beats are the most common manifestation of this imbalance, but supraventricular dysrhythmias, conduction problems, and eventually VT and VF may occur. Hypokalemia also increases the sensitivity of the heart to digitalis and its accompanying dysrhythmias, even at normal

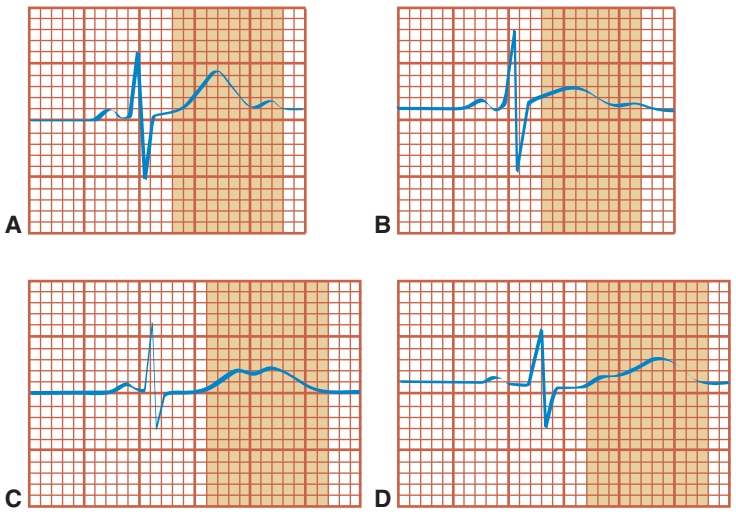


FIGURE 17-37 ▲ The effect of hypokalemia on an ECG. **A:** When the potassium level is normal—usually considered to be 3.5 to 5 mEq/L—the T wave is much higher than the U wave (*see highlighted area*). **B:** When the potassium level falls to 3 mEq/L, the T wave and U wave are almost the same height (*see highlighted area*). **C:** When the potassium level falls to 2 mEq/L, the U wave starts rising above the T wave (*see highlighted area*). **D:** As the potassium level reaches 1 mEq/L, the U wave starts to resemble a T wave (*see highlighted area*). The duration of the QT interval remains the same, but it cannot be measured because the two waves are fusing. (From Springhouse: ECG Interpretation: Clinical Skillbuilders. Springhouse, PA: Author, 1990, p 114.)

serum levels. The severity of the dysrhythmias associated with hypokalemia requires early recognition of this problem.

▲ Calcium

Like potassium, calcium is important in normal cardiac function. It is essential for the initiation and propagation of electrical impulses and for myocardial contractility. Abnormal calcium levels are not commonly seen unless they are associated with an underlying disease, and therefore they are not as common as serum potassium abnormalities.

Hypercalcemia

On an ECG, the major finding associated with hypercalcemia is shortening of the QT interval (Fig. 17-38). Because the QRS complex and T wave usually are unaffected by changes in serum calcium levels, the shortened QT interval is a result of shortening of the ST segment. QT-interval shortening also is seen in patients taking digitalis. In addition, ST-segment depression occasionally occurs, and T-wave inversion may be seen.

Hypocalcemia

On an ECG, low serum calcium levels prolong the QT interval because of a lengthening of the ST segment (see Fig. 17-38). The T wave itself is not prolonged but may be inverted in some cases. The prolongation of the QT interval in hypocalcemia should not be mistaken for a prolonged QTU interval seen in hypokalemia. In patients with chronic renal failure, hypocalcemia may be associated with decreased potassium levels.

QT interval prolongation also may be seen in cerebral vascular disease and after cardiac arrest. Several antiarrhythmic agents produce prolonged QT intervals and always should be considered when evaluating an ECG for hypocalcemic changes.

HEMODYNAMIC MONITORING

Hemodynamic monitoring is a means of evaluating intracardiac and intravascular volume, pressures, and cardiac function. The purposes of hemodynamic monitoring are to aid in the diagnosis of various cardiovascular disorders, guide therapies to optimize cardiac function, and evaluate the patient's response to therapy.

Because a primary goal of management of a critically ill patient is to ensure adequate oxygenation of tissues and organs, indications for hemodynamic monitoring include conditions in which CO is insufficient to deliver oxygen to the cells due to alterations in intravascular volume (preload), alterations in vascular resistance (afterload), or alterations in myocardial contractility. Hemodynamic monitoring may be indicated as a mechanism to assess the balance of oxygen supply (oxygen delivery) and demand as evaluated by measurement of oxygen consumption or venous oxygen saturation. The determinants of CO, oxygen delivery, and oxygen utilization are discussed in detail in the later portion of this section and in Chapters 16, 23, and 54.

Patients who are in cardiogenic shock, severe heart failure, severe sepsis or septic shock, multiple system organ dysfunction, or acute respiratory distress syndrome, or those who have had cardiac surgery, are examples of candidates for invasive and minimally invasive hemodynamic monitoring. In addition, noninvasive hemodynamic technology now affords clinicians the ability to evaluate cardiovascular performance in areas outside of the critical care arena and in the outpatient setting.

To incorporate hemodynamic data into the care of the critically ill, the nurse must understand the following:

- Cardiorespiratory anatomy and physiology
- Monitoring system components to measure cardiac and vascular pressures and CO
- Rationales for interventions directed toward enhancing CO, oxygen delivery, and oxygen consumption
- Potential complications
- Differences between physiological changes and mechanical or monitoring system problems

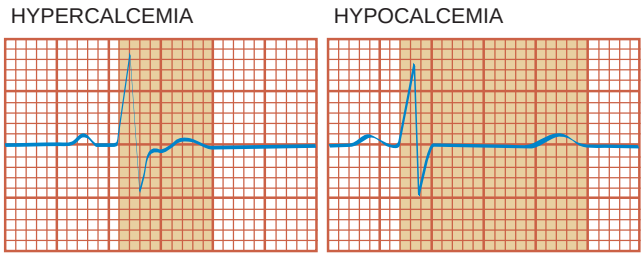


FIGURE 17-38 ▲ The effects of hypercalcemia and hypocalcemia on an ECG. Changes in serum calcium levels are reflected in phase 2 of the action potential. Hypercalcemia shortens the QT interval, whereas hypocalcemia lengthens it (*see highlighted areas*). (From Springhouse: ECG Interpretation: Clinical Skillbuilders. Springhouse, PA: Author, 1990, p 115.)

▲ Pressure Monitoring System

Basic equipment necessary to measure and monitor invasive hemodynamic pressures includes a hollow-tube catheter, a fluid-filled pressure monitoring system comprised of flush solution, IV tubing with drip chamber, noncompliant tubing, stopcocks, a flush device, one or more transducers, and a monitor that amplifies and displays the pressures and waveforms (Fig. 17-39). Pressures from the intravascular space or cardiac chambers are transmitted through the catheter and the fluid-filled noncompliant pressure tubing to the pressure transducer, which then converts the physiological signal from the patient into an electrical one. Transducers are usually disposable and precalibrated, and come packaged with the pressure system. The monitor converts and amplifies the electrical signal generated by the transducer to a pressure tracing and digital value. In general, bedside physiological monitoring systems have the capability to display several pressure digital readings and waveforms simultaneously. The monitors also include mechanisms to label waveform locations, set or adjust alarms and tracing scale size, and zero the system.

Using a continuous flush solution maintains a patent pressure system. The flush solution is typically normal saline or

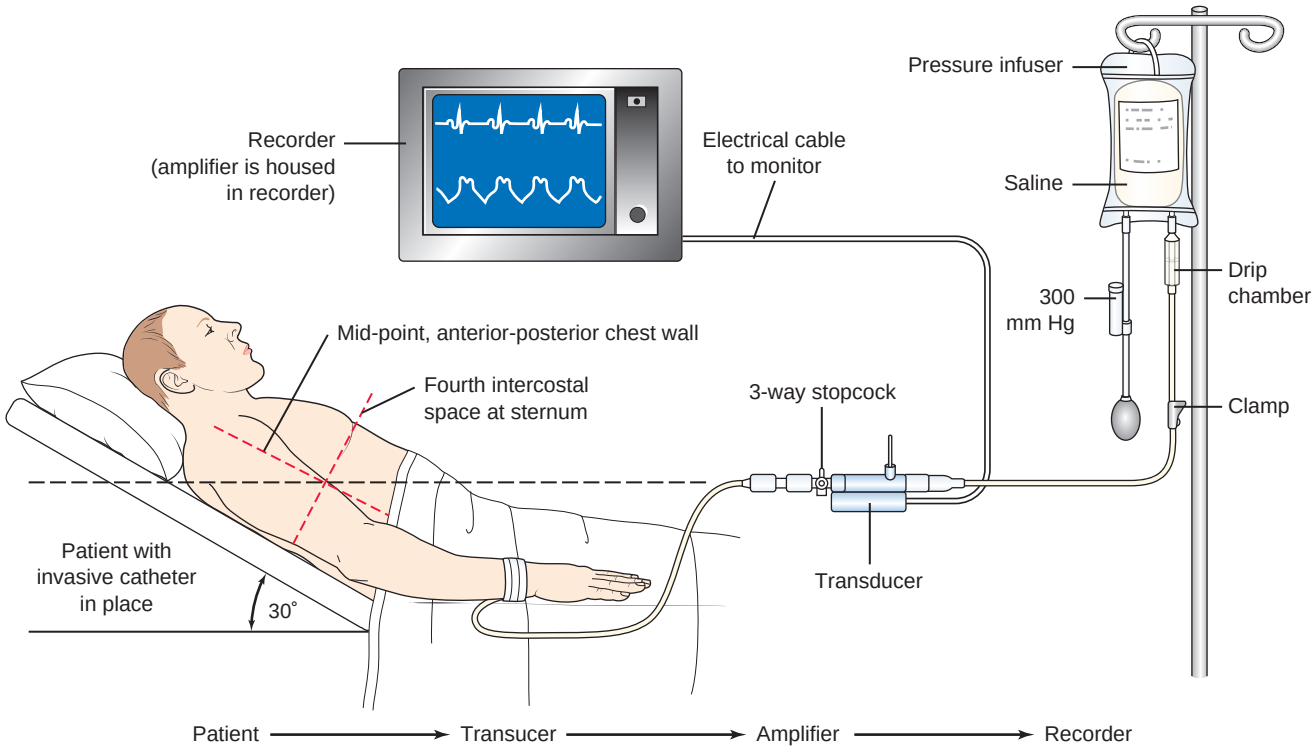


FIGURE 17-39 ▲ Invasive pressure monitoring. An indwelling catheter is attached by pressure tubing to a transducer. The transducer is connected to an amplifier/monitor that visually displays a waveform and systolic, diastolic, and mean pressure values. The system is composed of a flush solution under pressure, a continuous flush device, and a series of stopcocks. Typically, the stopcock closest to the insertion site is used to draw blood samples from the artery, and the stopcock located nearest the transducer is used for zeroing.

dextrose and water (D_5W) and may be heparinized. The bag of solution is placed in a continuous pressure infusion bag or device to exert approximately 300 mm Hg. This maintains a constant pressure through the restrictor in the flush device and system. A continuous flow of approximately 3 to 5 mL/h prevents backflow of blood through the catheter and tubing, thereby maintaining system patency and accurate transmission of pressures. The system is flushed manually by activating the flush device.

Optimizing the Pressure Monitoring System

An optimal pressure monitoring system is one that accurately reproduces the physiological signals transmitted through it. For optimal use of invasive monitoring systems, it is essential to ensure accurate pressure recordings and waveform display. Technical or mechanical factors can produce erroneously high or low pressures and altered waveforms. Before determining whether abnormal pressures are a result of altered physiology or a response to interventions, the nurse assesses the system to determine whether the pressures recorded are accurate. Table 17-16 describes causes of technical factors affecting invasive pressure monitoring and troubleshooting techniques. Any impedance between the patient and transducer, such as air bubbles, blood, or additional stopcocks, can alter the signal and subsequently the pressures and waveforms. Less than 300 mm Hg in the continuous pressure device, soft compliant IV tubing, or additional length of pressure tubing may also distort the signal to the transducer. Stopcocks, used for zeroing the transducer and blood sampling, as well as any other connectors, are kept to a minimum. Luer-Lok–type connections, rather than slip-lock connections, help preserve the integrity of the system.

After assessing the pressure system components to identify any potential mechanical problems, the nurse performs a square-wave test to determine the dynamic response of the system. In addition, the nurse ensures proper leveling of the air–fluid interface and zeroing of the transducer to optimize the system.[20–23]

Square-Wave Test: Dynamic Response Testing

For a rapid bedside assessment of the dynamic response of the system, a simple evaluation of dynamic response can be obtained by performing a square-wave test and observing the resultant oscillations. Checking the dynamic response of the system determines the natural frequency and damping coefficient. Factors affecting the response of the system include the natural frequency of the system itself, the pressure tubing quality, number of stopcocks, and other components, such as blood sampling systems. The steps required to measure the natural frequency and damping coefficient accurately are complex and time consuming. Other references describe the steps for performing this process.

Table 17-16 Troubleshooting Pressure Monitoring Systems and Measurements

Problem	Cause	Prevention	Intervention
1. No waveform	Transducer not open to catheter	Check stopcocks for proper position.	Check and correct stopcock position. Check scale setting and monitor setup. Aspirate blood clot. Do not fast flush or irrigate with syringe.
	Settings on bedside monitor incorrect or off		Check function with cable checking device.
	Catheter clotted	Use correct setting on bedside monitor.	Check function of transducer with mercury, water column, or supplemental pressure device.
	Faulty cable	Maintain continuous flush.	
	Faulty transducer	Use functioning cables.	Change transducer if necessary.
2. Overdamped waveforms	Improper scale selection		Change to proper scale.
	Air bubbles in tubing and near transducer	Flush system by gravity. Remove any air bubbles.	Flush air from system. On initial setup, expel all air from flush solution bag.
	Blood clot partially occluding catheter tip	Maintain continuous flush; use heparinized solution according to hospital protocol.	Aspirate clots with syringe. Use heparinized solution according to institution policy.
	Forward migration of catheter		Reposition patient. Check for kinks in catheters.
	Catheter tip occluded by balloon or vessel wall		Reposition by pulling back catheter while observing waveforms.
	Leak in pressure system	Tighten all connections and stopcock up on set up.	Tighten all connections and stopcocks. Change faulty system components if necessary.
	Pressure bag not inflated at 300 mm Hg	Inflate or apply pressure to device to 300 mm Hg.	Reinflate bag or activate device. Change device if faulty.
3. Underdamped waveforms; whip or ringing	Excessive movement of catheter	Correct catheter placement. Use appropriate catheter size for vessel.	Try different catheter tip position. Eliminate excessive tubing.
	Air bubbles in tubing	Eliminate excessive length of pressure tubing. Check for very rigid pressure tubing.	Change tubing. Eliminate excessive stopcocks.
4. False low readings	Leveling or zero reference (transducer) is too high	Check level periodically. Level air–fluid interface of stopcock nearest the transducer to the phlebostatic axis.	Relevel transducer air–fluid interface to phlebostatic axis.
	Improper zeroing	Check monitor settings. Observe waveforms.	Rezero monitor.
	Overdamped waveforms	Perform square-wave test.	Optimize length of pressure tubing.
5. False high readings	Leveling or zero reference (transducer) is too low	Check level periodically. Level air–fluid interface of stopcock nearest the transducer to the phlebostatic axis.	Relevel transducer air–fluid interface to phlebostatic axis.
	Improper zeroing	Check monitor settings. Observe waveforms.	Rezero monitor.
	Overdamped waveforms	Perform square-wave test.	Remove excessive length of pressure tubing.
6. Inappropriate pressure waveform	Incorrect catheter position		Reposition patient. Obtain chest x-ray. Reposition catheter.*
	Migration of PAC into mechanical wedge position	Establish optimal position carefully during the insertion process, ensuring use of 1.25–1.5 mL air for proper balloon inflation volume for obtaining a PAOP tracing.	Observe waveforms and confirm with initial insertion tracings. If right ventricular tracing is observed from PAC distal tip, slowly inflate balloon to allow PAC to "float" into pulmonary artery. If PAOP tracing is observed with balloon deflated, withdraw catheter slightly while observing waveforms. Stop withdrawing as soon as a pulmonary artery tracing is observed.
7. Bleed back into pressure tubing or transducer	Loose connections	Ensure all connections are tight.	Tighten connections.
	Stopcocks not returned to proper position	Return stopcocks to proper positions.	Ensure stopcocks are in correct position.
	Pressure bag not at 300 mm Hg	Maintain 300 mm Hg of pressure.	Check pressure device.

*Repositioning the PAC usually is done by a physician or advanced practice nurse such as a nurse practitioner and varies with hospital policies. PAC, pulmonary artery catheter; PAOP, pulmonary artery occlusion pressure.

To perform a square-wave test, a flush device that can be activated and released rapidly is required. Activating the flush device opens the internal restrictor and increases the fluid flow through the system. The nurse observes the bedside monitor for the increase in pressure. The waveform sharply rises and "squares off" at the top of the scale. After the flush device is released, the restrictor closes. The nurse observes the waveform as it returns to baseline, counts the number of oscillations, and observes the distance between them.

In an ideal system, also called optimally damped, the square wave has a straight vertical upstroke from the baseline, a straight horizontal component, and more importantly, a straight vertical downstroke back to the baseline with approximately one and one-half to two sharp oscillations. The distance between the oscillations is also close.[20] Figure 17-40 depicts a normal square wave and examples of square waves from nonoptimized hemodynamic monitoring systems. Overdamped systems produce lower than actual systolic pressures and potential loss of dicrotic notch identification. Underdamped systems produce artificially high systolic pressures and low diastolic pressures. By performing a square-wave test, the bedside clinician can quickly assess if the abnormal waveform tracing is a result of the patient's physiology or a less-than-optimal system.[20–23]

Leveling and Zeroing

After a square-wave test is performed, the system is leveled to an external landmark and then zeroed to atmospheric pressure to ensure accurate pressure monitoring. Typically, the stopcock nearest the transducer is used as the air–fluid interface for leveling and zeroing; however, any stopcock port in the system can be used as long as it is leveled to the phlebostatic axis. The phlebostatic axis is best described as the bisection of the fourth intercostal space and the midpoint of the anterior–posterior chest diameter (see Fig. 17-39, p. 264); it is often called the zero reference point. Zeroing the transducer is the action of opening the pressure system to atmospheric air and observing a reading of zero on the bedside monitor. With the stopcock turned off to the patient and opened to air, the influence of hydrostatic pressure is negated from the fluid-filled pressure system. Subsequent pressures recorded on the monitor now reflect those generated by the patient, not external forces. Bedside monitor manufacturers vary; however, most have a function key to ensure that the zeroing process has been successful. Newer disposable transducers come from the manufacturer precalibrated and do not require adjustment to an electronic zero. The term "zeroing" is used, however, when referencing to atmospheric pressure.

Once the zero reference point is established, the patient's chest is marked to ensure consistent leveling when other practitioners obtain subsequent pressure readings. With the patient positioned supine, a carpenter-type level or laser-light level device is used to align the air–fluid interface with the phlebostatic axis. Further pressure measurements are taken with the patient in the supine position.

If the alignment of the air–fluid interface changes after initial leveling and zeroing, an inversely related error of approximately 2 mm Hg for every inch misaligned occurs. For example, if the transducer air–fluid interface is raised from initial leveling, the values displayed will be about 2 mm Hg too low, and if lowered from initial leveling, the values recorded will be erroneously too high.

The head of the bed may be elevated as much as 60 degrees, provided that the air–fluid interface is releveled after any changes in patient position. Lateral or side-lying positions may be used if the external landmark is properly identified. Because some patients respond differently to head of bed elevation and side-lying positions, their hemodynamic values should be compared from supine.[20–23]

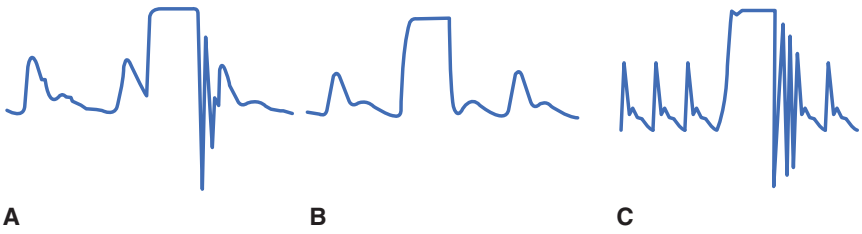


FIGURE 17-40 ▲ Steps to perform a square wave test include:
1. Activate the snap or pull tab of the flush device.
2. Observe the square wave generated on the bedside monitor.
3. Count the oscillations after the square wave.
4. Observe the distance between the oscillations.

A. Optimally damped system: Activation of the fast flush device generates a sharp vertical upstroke, horizontal line, and straight vertical downstroke ending with 1.5 to 2 oscillations close together before returning to baseline.
B. Overdamped system: Activation of the fast flush device generates a slurred upstroke and downstroke with less than 1.5 oscillations above or below the baseline. Causes include system leaks, blood clots, or large air bubbles in the tubing or transducer. Systolic pressures read erroneously low, diastolic pressures occasionally read low.
C. Underdamped system: Activation of the fast flush device generates more than 2 to 3 oscillations above and below the baseline. Causes include small air bubbles in the system, very rigid pressure tubing, and additional length of tubing. Systolic pressures read erroneously high, diastolic pressures read erroneously low.

(Courtesy of Edwards Lifesciences LLC.)

After the level and zero is verified, the only way to determine whether the pressures displayed on the monitor are accurate is to apply a known value to the transducer with a piece of external tubing and water column. Some transducer manufacturers provide a device that applies a known pressure to the transducer for rapid determination of accurate pressure recordings.

▲ Arterial Pressure Monitoring

Invasive arterial pressure monitoring uses an intra-arterial catheter connected to the pressure monitoring system. This allows continuous monitoring of the systemic arterial blood pressure and provides vascular access for obtaining blood samples by withdrawing blood from a stopcock or closed system device in the system. Indications for intra-arterial blood pressure monitoring include monitoring patients with vasoactive IV infusions; cardiovascular instability; and fluctuating, unstable blood pressures.

Arterial Line Insertion

The most common sites for arterial catheter insertions are the radial, brachial, and femoral arteries. Alternative and less frequent sites include the axillary and dorsalis pedis arteries in adults and the temporal and umbilical arteries in neonates. The following factors are considered for selecting the artery for cannulation:

- Size of the artery in relation to the size of the catheter; the artery should be large enough to accommodate the catheter without occluding or significantly impeding flow.
- Accessibility of the site; the chosen site should be easily accessible and free from contamination by body secretions.
- Blood flow to the limb distal to the insertion site; there should be adequate collateral flow in the event that the cannulated artery becomes occluded.

The radial artery, which satisfies these criteria, is the most frequent site for an arterial catheter. It is superficially located and therefore easy to palpate. Cannulation of this artery also usually poses the least limitation on the patient's mobility.

Before a catheter is inserted into the radial artery, the presence of adequate collateral circulation to the hand by the ulnar artery is assessed by performing Allen's test (Fig. 17-41). The Allen's test is performed by having the patient clench their fist several times while the nurse is occluding both the radial and ulnar arteries. The patient then extends their hand with the palm side up to show it is blanched. Pressure on the ulnar artery is released, and the hand is observed for return of color. If the hand remains blanched for longer than about 10 seconds, ulnar circulation is considered inadequate, in which case the radial artery should not be cannulated. Use of ultrasound devices for assessing blood flow in place of the Allen's test is becoming more common.

Regardless of the site chosen for arterial catheter placement, the insertion is performed using sterile technique. The pressure monitoring system is assembled and flushed, and the transducer is leveled and zeroed before the catheter is inserted. Once the catheter is in place, it should be secured and the site dressed according to institutional policy.[20–23]

Arterial Pressure Waveform

The normal arterial waveform should have a rapid upstroke, a clear dicrotic notch, and a definite end-diastole, as shown in Figure 17-42. The mechanical activity of systole and diastole follows the electrical activity of depolarization and repolarization, respectively. The initial sharp upstroke of the waveform results partly from the rapid ejection of blood from the left ventricle into the aorta. On a dual-channel tracing of both the ECG and arterial waveforms, the QRS complex precedes the rapid rise in arterial pressure. The dicrotic notch reflects a slight backflow of blood in the aorta, reflecting closure of the aortic valve or may be a reflective wave from the periphery.

Obtaining Arterial Pressures

The value measured at the peak of the waveform is the systolic pressure. A normal arterial systolic pressure is typically 90 to 140 mm Hg. The dicrotic notch typically indicates the end of ventricular systole and the beginning of

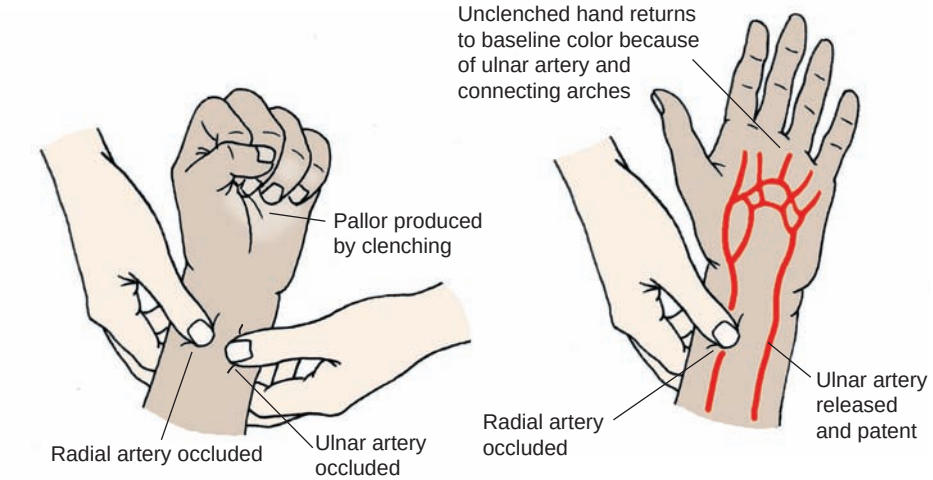


FIGURE 17-41 ▲ Modified Allen's test.

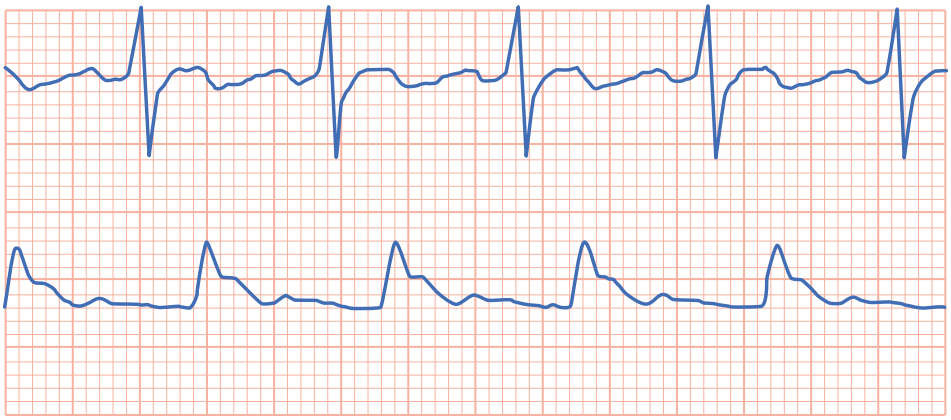


FIGURE 17-42 ▲ Normal relationship of ECG and arterial pressure waveform.

diastole. As blood flows to the periphery, the pressure in the arterial system decreases. The lowest point of the waveform is the diastolic pressure, which is normally between 60 and 90 mm Hg.

Mean arterial pressure (MAP) is used to evaluate perfusion of vital body organs. Normal MAP is 70 to 105 mm Hg. The MAP calculation incorporates the impact of diastolic time being approximately two times longer than systole during a cardiac cycle. Therefore, MAP = diastolic pressure +⅓ pulse pressure, or

$$\frac{\text{Systolic pressure} + (\text{Diastolic pressure} \times 2)}{3}$$

Most bedside monitors automatically calculate and continuously display the MAP. Manufacturer algorithms to determine MAP may vary; however, most incorporate assessment of the area under the full arterial waveform rather than a mathematical model.

The difference between the systolic and diastolic pressure is the pulse pressure (PP). This value more closely reflects the SV from the ventricle. SV is proportional to PP and inversely related to the aortic compliance. Bedside monitors do not automatically display this valuable parameter. Clinicians who want to assess the patient's volume status should include PP in their assessment because it is an indirect reflection of the SV. The range of PP may be as wide as 30 to 100 mm Hg at the far ends of the spectrum. A wide PP occurs typically with elevated systolic pressures resulting from aortic regurgitation and some vascular conditions. A narrow PP may result from hypovolemic states when the diastolic pressure rises.[22]

Complications

Infection

Proper attention to sterile technique during catheter insertion; care of the insertion site; blood sampling; and maintenance of a sterile, closed monitoring system reduce the risk of infection. The following should be performed according to institution policy: assessment of insertion site for signs of infection; use of sterile technique when changing dressings, tubing, and flush solution; and maintenance of the integrity of the system. Opening the pressure system to air for either zeroing or blood sampling provides opportunity for infection. Applying sterile nonvented or "dead-ender" caps to the stopcock ports helps eliminate contamination. Closed systems for blood sampling help reduce the potential for open stopcock infections and assist with managing potential blood loss.

Accidental Blood Loss

Accidental blood loss from an arterial catheter can be catastrophic and often can be prevented. All connections in the system should use a Luer-Lok–type connector. The extremity in which the catheter is placed may be immobilized (eg, placing the wrist on an arm board). If some type of patient self-protective device is used, it should not be placed over the insertion site. Easy access to the insertion site and connections is imperative.

Impaired Circulation to Extremity

Circulation to the extremity in which the arterial line is placed must be monitored frequently. Initial assessment of color, sensation, temperature, and movement of the extremity is made after insertion of the arterial catheter and as frequently as the institution policy states. Any indication of impaired circulation may be an indication for catheter removal and is reported immediately.

Nursing Considerations

Blood pressures obtained by an intra-arterial catheter and with an optimal pressure monitoring system are most accurate. Comparisons between intra-arterial and cuff pressures may be misleading because the methods of measurement reflect different physiological events and are therefore not truly comparable.[23] Direct or invasive monitoring measures pressure, and indirect cuff measurements are based on flow. In normotensive patients, intra-arterial pressures are typically higher by about 5 to 10 mm Hg than the pressures obtained using a cuff. Indirect methods tend to overestimate direct measurements in hypotensive patients and underestimate them in hypertensive patients. Variations as wide as 20 to 60 mm Hg occur, depending on specific patient conditions.[22]

For interventions when accurate values are important for therapeutic decisions, intra-arterial pressures remain the gold standard. Using a trend value from one source is often more helpful than comparing values obtained between different

technologies. Documenting the site of pressure measurements and the type of technique used is key.

Patient safety measures include the proper setting and activation of all alarms on the bedside physiological monitor. Bedside monitor alarms provide warning that a change has occurred either in the system or in the patient's physiological status. Alarms are set either around a patient's specific parameter or according to institution policy. Typically, high and low alarms are set for systolic, diastolic, and mean pressures and within 10 to 20 mm Hg of the patient's blood pressure. The alarms must be visible and audible to the caregiver for the specific patient environment. Troubleshooting steps for an alarm are listed in Table 17-16 on page 265.

General steps to ensure accurate pressures from invasive lines include assessing the patient first, then checking the pressure monitoring system, and then inspecting the monitor itself. Assess the insertion site: Is the catheter kinked? Are there any blood clots? Is there any sign of bleeding? Next, evaluate the pressure system: Are any stopcocks turned the wrong way? Is there sufficient pressure in the pressure bag (ie, does the pressure read 300 mm Hg)? Are there any air bubbles? Is the bedside monitor functioning properly? Are the alarms set correctly?

If catheter patency is in question, blood and fluid are aspirated from the blood-drawing port or stopcock in an attempt to remove a blood clot (if present), and then the system is flushed using the fast flush device. The system should not be flushed with a syringe. No additional IV solution or medication should be administered through the arterial pressure monitoring system at any time.

▲ Central Venous Pressure Monitoring

CVP is typically measured in the superior vena cava near the right atrium via a catheter placed in the jugular or subclavian vein. It therefore reflects the pressure of blood in the right atrium and provides information about intravascular blood volume, right ventricular end-diastolic pressure (RVEDP), and right ventricular function. To a limited degree in persons with normal pulmonary vasculature and left ventricular function, the CVP indirectly reflects left ventricular end-diastolic pressure (LVEDP) and function because the left and right sides of the heart are linked by the pulmonary vascular bed. Alterations in intravascular volume status or ventricular function usually are associated with abnormally high or low CVP measurements.[20–22]

Catheter Insertion

The CVP catheter is long and flexible. It is inserted under maximum sterile conditions with a chlorhexidine site preparation. The physician or nurse practitioner uses a sterile field with a full sterile drape, sterile gloves and gown, and a mask and cap. Those assisting the physician also should wear a cap and mask and sterile gloves if near the catheter or insertion site. The best insertion site to minimize infection risk is the subclavian vein.[24] Catheters also can be inserted into an antecubital, jugular, or femoral vein if necessary. It is threaded into position in the vena cava close to the right atrium. Occasionally, the catheter may advance into the right atrium. In this situation, the catheter is withdrawn several centimeters.

The hemodynamic monitoring system components and preparation for CVP monitoring are identical to those described for arterial pressure monitoring. After insertion of the catheter, the pressure tubing is connected to the catheter hub. The CVP waveform and value appear on the bedside monitor.

Complications

Infection

Infection may occur within the catheter or around the insertion site. Central venous catheter–associated bloodstream infection is diagnosed and verified by blood cultures. Occasionally after catheter removal, the tip is cut off with sterile scissors and sent to the microbiology laboratory. Signs and symptoms of infection include erythema at the insertion site, fever, or elevated WBC count. Primary measures to prevent infection include routine dressing and IV fluid tubing changes, as outlined by the Centers for Disease Control and Prevention[24] and hospital policy, as well as adherence to sterile technique during catheter insertion and dressing changes. When catheters are left in place for an extended period of time, antibiotic-impregnated catheters may be used to reduce the risk for infection. See Evidence-Based Practice Highlight 17-2.

Thrombosis

Thromboses occasionally form and may vary in size from a thin fibrin sheath over the catheter tip to a large thrombus. A small thrombus may be flushed away without causing harm, but a larger thrombus occluding the catheter and vein should not be flushed into the venous circulation. A large thrombus may be detected by loss of hemodynamic waveform and inability to infuse fluid or withdraw blood from the catheter. The patient may have edema of the arm closest to the catheter site, varying degrees of neck pain (that may radiate), and jugular vein distention. A large thrombus is classified as an emergency because it may impair circulation to a limb. A nurse may attempt to aspirate this clot if hospital policy permits. Frequently, hospitals also have protocols to administer small doses of thrombolytic agents to dissolve the clot. At the very least, the nurse is responsible for reporting suspected catheter occlusion to a physician.

Air Embolism

Air embolism occurs as a result of air entering the system and traveling through the vena cava to the right ventricle. Usually, air entry into the catheter is associated with disconnection of the catheter from the IV tubing. Changes in intrathoracic pressure with inspiration and expiration draw air into the catheter and vena cava. Sudden hypotension may be the first indicator of this sometimes lethal problem.

Approximately 10 to 20 mL of air must enter the venous system before the patient becomes symptomatic. Signs of such an emergency may include confusion, lightheadedness, anxiety, and unresponsiveness. The physiological event is the creation of foam in the ventricle with each heart contraction

EVIDENCE-BASED PRACTICE HIGHLIGHT 17-2

Preventing Catheter Related Bloodstream Infection

△ Expected Practice

- Cleanse hands with waterless cleaning solution or, if visibly soiled, with soap and water before and after patient contact.
- Disinfect clean skin utilizing friction with an appropriate antiseptic (preferably 2% chlorhexadine) before catheter insertion and during site care.
- Utilize full barrier precautions when inserting central venous access devices.
- Educate all staff inserting and caring for intravascular catheters, assess competency of same at regular intervals, and advocate adherence to standards of care.
- Replace peripheral IV sites in the adult patient population at least every 96 hours but no more frequently than every 72 hours. Leave peripheral venous catheters in children until IV therapy is completed, unless complications (eg, phlebitis and infiltration) occur.
- Replace IV tubing at least every 96 hours but no more frequently than every 72 hours.
- When adherence to aseptic technique during intravascular catheter insertion cannot be ensured (eg, prehospital, code situation), replace the catheter as soon as possible, but within 48 hours.

△ Supporting Evidence

- A substantial proportion of hospital-acquired infections result from cross-contamination from the hands of health care workers. Alcohol-based hand rub, compared with traditional handwashing with unmedicated soap and water or medicated hand antiseptic agents, may offer better results because it requires less time, acts faster, and is less likely to irritate skin. Thus, the Centers for Disease Control and Prevention recommends the use of alcohol-based hand rubs between patient contacts as an adjunct to traditional handwashing alone.
- Chlorhexidine gluconate solutions utilized for vascular catheter site care reduce catheter related bloodstream infections and catheter colonization more effectively than povidone-iodine solutions. Moreover, 80% of resident and transient skin flora are found in the first five epidermal layers of the skin. There is clinical evidence to support the efficacy of applying antiseptics with sufficient friction to assure that the solution reaches into the cracks and fissures of the skin. There is no evidence that supports use of traditional concentric prepping technique. Although a 2% chlorhexidine-based preparation is preferred, tincture of iodine, an iodophor, or 70% alcohol can be used. Allow any solution used to dry before the catheter is inserted.[1,4,5]
- Compared with peripheral venous catheters, CVCs carry a substantially greater risk for infection; therefore, the level of barrier precautions needed to prevent infection during insertion of CVCs should be more stringent. Maximal sterile barrier precautions (eg, cap, mask, sterile gown, sterile gloves, and full body sterile drapes) during the insertion of CVCs substantially reduce the incidence of CRBSI compared with standard precautions (eg, sterile gloves and small drapes)[1,3,2,10] There are some studies that demonstrate infection rates are lower when the subclavian site is used. Selection of central line insertion site, however, is based on patient risk factors.
- Healthcare workers who insert and care for intravascular devices should receive formalized education and training in indications for intravascular catheterization, proper placement, maintenance, and infection control. Educational programs focusing on central venous catheter insertion and care have lead to a substantial decrease in cost, morbidity, and mortality attributable to central venous catheterization. Ongoing education and reinforcement of appropriate technique serve as a reminder of current best practices, and studies demonstrate that consistent reinforcement of aseptic techniques leads to decreased CRBSI.[1,8,9,10,11]
- Studies of peripheral intravenous (IV) catheters show that there is not a substantial difference in phlebitis rates between catheters left in place 72 hours and those left in place 96 hours. There is no evidence to support that routine replacement of central venous catheters is more effective in decreasing bloodstream infections than replacing central venous catheters as needed.[1,12]
- Studies show that IV tubing containing crystalloids can be replaced every 72 to 96 hours. If monitoring using a transducer system, replace the transducer, tubing, flush device and flush solution every 96 hours.

Excerpted from American Association of Critical-Care Nurses Practice Alert. Available online at http://aacn.org. All references cited in this alert are available with the associated resources related to this chapter. Visit: http://thepoint.lww.com

and loss of SV due to air instead of blood in the ventricle, causing a sudden decrease in CO. Cardiac arrest may occur.

If this problem is suspected, turning the patient on the left side in the Trendelenburg position may allow the air to rise to the wall of the right ventricle and improve blood flow. Oxygen should be started unless contraindicated.

Strategies to prevent disconnections include having Luer-Lok connections on all central line catheters and tubings, careful manipulation of catheter and tubing during dressing changes, and routine monitoring of the connections. There is no substitute for close observation by skilled and educated nursing staff.

Nursing Considerations

Ensuring the integrity of the monitoring system, obtaining and documenting accurate data, and monitoring trends in CVP are critical to the interpretation and utilization of information to assess a patient's cardiovascular function and response to interventions. Evidence-Based Practice Highlight 17-3 summarizes current evidence related to obtaining accurate measurement of CVP pressure.

Normal CVP is less than 8 mm Hg. Low CVP indicates a hypovolemic state often requiring fluid administration. The anticipated response to fluid therapy is an increase in the CVP. Similarly, diuretic therapy reduces intravascular volume, and its administration is expected to be associated with a decrease in the CVP. Vasodilation from sepsis or vasodilating drugs may also lead to a low or decreasing CVP; both create a relative hypovolemia because the intravascular space has become greater relative to the blood volume, which has not changed. Increased CVP may be caused by a number of complex and interrelated factors, each of which requires scrutiny. Two of the more common causes of increased CVP are right ventricular failure and mechanical ventilation. Rarely is intravascular volume overload and hypervolemia alone a cause of increased CVP.

EVIDENCE-BASED PRACTICE HIGHLIGHT 17-3
Pulmonary Artery/Central Venous Pressure Measurement

△ Expected Practice

- Verify the accuracy of the invasive pressure monitoring system by performing a square waveform test at the beginning of each shift and anytime the system if disturbed (eg, blood draw).
- Position the patient in supine position with head of bed (HOB) between 0 and 60 degrees, lateral position 20, 30 or 90 degrees or prone before the pulmonary artery pressure (PAP), pulmonary artery occlusion pressure (PAOP), and central venous pressure (CVP) measurements. HOB elevation can be at any angle from 0 (flat) to 60 degrees if the patient is in the supine position. Allow the patient to stabilize 5 to 15 minutes after a position change.
- Level and reference the transducer air-fluid interface to the phlebostatic axis (supine or prone position), fourth intercostal space (ICS) ½ antero-posterior diameter of the chest or lateral angle specific reference using a laser or carpenters level before PAP/PAOP/CVP measurements.
- Obtain PAP/PAOP/CVP measurements from a graphic (analog) tracing at end-expiration or adjust the measurement point if the patient is receiving airway pressure release ventilation (APRV) or is actively exhaling.
- Use simultaneous ECG tracing to assist with proper PAP/PAOP/CVP waveform identification.
- PA catheters can be safely withdrawn and removed by competent registered nurses.

△ Supporting Evidence

- The square waveform test, or dynamic response test, determines the ability of the transducer system to correctly reflect invasive pressures.[14,15] The dynamic response is affected by system problems, such as air bubbles in the tubing, excessive tubing length, loose connections, and catheter patency. Removal of micro bubbles during system set up result in an "adequate" or "optimal" system in over 95% of cases.[16,17] Any of these problems affect the accuracy of PAP/PAOP/CVP measurements and must be corrected prior to pressure measurement. Perform the square waveform test: on the initial system setup, at least once each shift, after opening the catheter system (eg, for rezeroing, drawing blood, or changing tubing), and whenever the PAP/PAOP/CVP waveform appears to be damped or distorted.[14] (Level A)
- Consider the following changes in PA pressures as clinically significant (ie, not reflective of the normal variability in PA pressures): PAS more than 4 to 7 mm Hg; PAEDP more than 4 to 7 Hg; PAOP more than 4 mm Hg.[18–20] (Level B)
- Studies in a variety of patient populations found that PAP/PAOP/CVP measurements are accurate when the HOB is elevated to any angle between 0 and 60 degrees[21–27] or when the patient is in a 20 degree[28], 30[20,29] or 90 degree[30] lateral position with the HOB flat, as long as the correct angle-specific reference is used. PAP/PAOP/CVP measurements should not be performed when the patient is in Trendelenburg[31] position or if the legs are in a dependent position.[21] There are no studies to support PAP/PAOP/CVP measurements in reverse Trendelenburg. Reliable cardiac output (CO) measurements have been obtained only in the supine position with a backrest at 20[32] or 45 degrees[33] and in the prone position.[34–37] Clinically significant changes in CO may occur in the 20-degree lateral positon[32,38] that may limit concurrent PAP/PAOP/CVP and CO PAP/PAOP/CVP compared to the flat, supine position using a standardized approach.[17,39] (Level A)
- The use of position-specific reference is critical to accurate PAP/PAOP/CVP measurement.[20,40–44] In the supine position the phlebostatic axis (fourth ICS at ½ the anteroposterior diameter of the chest) is the most commonly used reference point.[45] In the lateral position, the following reference points should be used: 30 degrees lateral (half distance from surface of bed to the left sterna border); 90-degree right lateral position (fourth ICS /midsternum); and in the 90-degree left position (fourth intercostal space/Left parasternal border).[41,42,45] A laser or carpenter's level (not the eyeball technique) should be used to correctly reference the system.[45] Once the correct reference location is identified, a mark should be placed on the chest wall. (Level A)
- Before obtaining PAP/PAOP/CVP measurements, patients may require 5 to 15 minutes for stabilization depending on the patient's left ventricular function.[47] No specific recommendations are available for the stabilization period after proning; measurements have been performed 20 to 30 minutes after repositioning from supine to prone for patients with acute lung injury[35–37] and in patients with ARDS, measurements were performed 20 minutes after stabilization of the SvO_2 (60 to 90 minutes after proning).[34] (Level A)
- Changes in intrathoracic pressure during respiration alter intracardiac pressures. PAP/PAOP/CVP measurements are obtained by convention at end-expiration when pleural pressure is minimal.[48] For patients receiving APRV, the PAOP should be measured at the end of the positive pressure plateau, which can be observed on the ventilator and is the point immediately before the release of airway pressure and the initiation of inspiration.[49] With active exhalation (suspect if respiratory-induced fluctuation in PAOP is >10 to 15 mm Hg) read the PAOP at the midpoint between the end-expiratory peak and the end-inspiratory lowpoint.[50] The addition of the airway pressure tracing to the analog strip may further improve measurement accuracy.[51] (Level A)
- A simultaneous ECG should be used to facilitate correct PAP/PAOP/CVP waveform measurement[55–57] and should be read from analog tracings or using the stop cursor method.[52–54] Digital readouts should not be used as they reflect pressures obtained throughout respiration and may be significantly different from and expiratory pressures. (Level A)
- Registered nurses, who demonstrate competency, can safely withdraw and/or remove PA catheters.[58,59] Before incorporation withdrawing and/or removing PA catheters into nursing practice, verify that it is within your state's scope of practice for registered nurses. (Level B)

AACN Evidence Leveling System

Level A Meta-analysis of quantitative studies or metasynthesis of qualitative studies with results that consistently support a specific action, intervention, or treatment.
Level B Well-designed, controlled studies with results that consistently support a specific action, intervention, or treatment.
Level C Qualitative studies, descriptive or correlational studies, integrative reviews, systematic reviews, or randomized controlled trials with inconsistent results.
Level D Peer-reviewed professional organizational standards with clinical studies to support recommendations.
Level E Multiple case reports, theory-based evidence from expert opinions, or peer-reviewed professional organizational standards without clinical studies to support recommendations.
Level M Manufacturer's recommendations only.

Excerpted from American Association of Critical-Care Nurses Practice Alert. Available online at http://aacn.org. All references cited in this alert are available with the associated resources related to this chapter. Visit: http://thepoint.lww.com

Mechanical ventilation increases intrathoracic pressure, which is transmitted to the pulmonary vasculature, heart, and great vessels. This pressure may directly affect CVP, which may increase as well, because intrathoracic pressure compresses the pulmonary vessels, creating resistance to blood flow from the right side to the left side of the heart and causing blood to "back up" in the right ventricle, right atrium, and vena cava. In extreme cases, the increased intrathoracic pressure associated with mechanical ventilation causes significant right ventricular dysfunction, and the CVP is elevated because of reduced forward blood flow into the pulmonary vasculature, resulting in increased volume and pressure of the blood in the right atrium and vena cava.

Increased CVP is associated with right ventricular failure due to CAD or left ventricular failure. The inability of the right ventricle to pump blood through the pulmonary vasculature because of injured or infarcted myocardium results in increased volume and pressure in the right atrium and vena cava. Left ventricular failure may increase CVP as the pressure of blood volume congests the pulmonary vasculature and impairs flow from the right ventricle, causing right ventricular dilation and subsequent failure. Again, the increased pressure is reflected backward to the right atrium and vena cava. In these instances, interventions are directed toward facilitating forward blood flow by improving ventricular contractility and reducing the intravascular blood volume. A decrease in the CVP is an indication of the effectiveness of therapy.

CVP is always interpreted in conjunction with other clinical observations, such as auscultation of breath sounds, heart and respiratory rate, ECG, neck vein distention, and urine output. For example, increased CVP associated with pulmonary basilar crackles and decreased urine output is often indicative of left ventricular failure. Distended neck veins but clear breath sounds and a high CVP might be caused by increased intrathoracic pressure from mechanical ventilator effects. Patients who are septic may have a low CVP that is associated with fever, elevated WBC count, tachycardia, and tachypnea, whereas patients who are taking vasodilating agents may have a low CVP that is associated with an increased heart rate but none of the other aforementioned clinical signs. A CVP value alone is meaningless, but when used in conjunction with other clinical data, it is a valuable aid in managing and predicting the patient's clinical course.[22]

▲ Pulmonary Artery Pressure Monitoring

The PA catheter provides assessment of right ventricular function, pulmonary vascular status, and, indirectly, left ventricular function. RA, right ventricular (RV), and PA pressures, as well as pulmonary artery occlusion pressure (PAOP), are measured using a PA catheter. PA catheters with a thermistor have the capability of determining CO. The pressures and CO obtained allow the clinician to calculate derived parameters and facilitate diagnosis of cardiovascular and cardiopulmonary dysfunction, determine the therapy needed, and evaluate the effectiveness of the interventions.

Pulmonary Artery Catheters

Several types of flow-directed, balloon-tipped PA catheters are available in different sizes. The type of catheter used is determined by the parameters to be monitored and additional requirements governed by the patient's condition. The 7.5- or 8-French (F; a measure of catheter size) thermodilution catheter is the size most commonly used (Fig. 17-43). All PA catheters have several external ports or lumen hubs corresponding to internal lumens and lumen openings into the right side of the heart and PA. A typical PA catheter has four lumens with external hubs or ports: the proximal hub and lumen, distal hub and lumen, balloon inflation valve and lumen, and thermistor connector and lumen.

The proximal or RA lumen opens into the right atrium; in smaller patients, the location might be in the superior or inferior vena cava, depending on insertion location. The lumen is used for infusion of fluids and is often connected to a transducer to provide RAP measurements and display of the RAP waveform. The RA lumen port also is used as the injectate port for measuring COs.

The distal or PA lumen hub is always attached to a transducer and a continuous flush system. The PA waveform is displayed continuously, as are the PA systolic, diastolic, and mean pressures. The PA port is used for the withdrawal of mixed venous blood, which is necessary for venous oxygen saturation, oxygen extraction, oxygen consumption, and intrapulmonary shunt measurements. Use of the PA distal port for fluid or medication administration is not recommended.

The balloon inflation port and lumen enable inflation of the balloon near the catheter tip with a small volume of air to measure the PA occlusion pressure, known as the PAOP. The balloon capacity of most PA catheters is 1.5 mL, and the balloon should not be inflated with more than this amount of air. Fluid is never inserted into the balloon inflation port.

Near the tip of the PA catheter is a thermistor. A cable to the bedside monitor or to a CO computer connects to the external thermistor port. The thermistor permits measurement of the patient's temperature in the PA (core temperature), and it detects the blood temperature change when solution is injected through the RA port to obtain a CO.

Specialty PA catheters include the previously described components and additional features and lumens. Some of the features include additional lumens in the RA, RV, or both for added infusion. A distal lumen containing fiber-optic filaments allows continuous measurement of mixed venous oxygen saturation (SvO_2). The external optic connecting cable is attached to an optics module and then special oximetry monitor. Catheters modified with a thermal filament and combined with a thermal filament connector and special monitor provide and display CO on a continuous basis. Other advanced catheters with advanced monitor algorithms determine RV ejection fraction and additional derived parameters such as end-diastolic volume. Figure 17-44 shows various types of PA catheters.

Specially designed catheters may also be used for temporary pacing. There are PA catheters that house pacing electrodes for both atrial and ventricular pacing as well as PA catheters with lumens in the RA and RV for placing of special probes for pacing.[20]

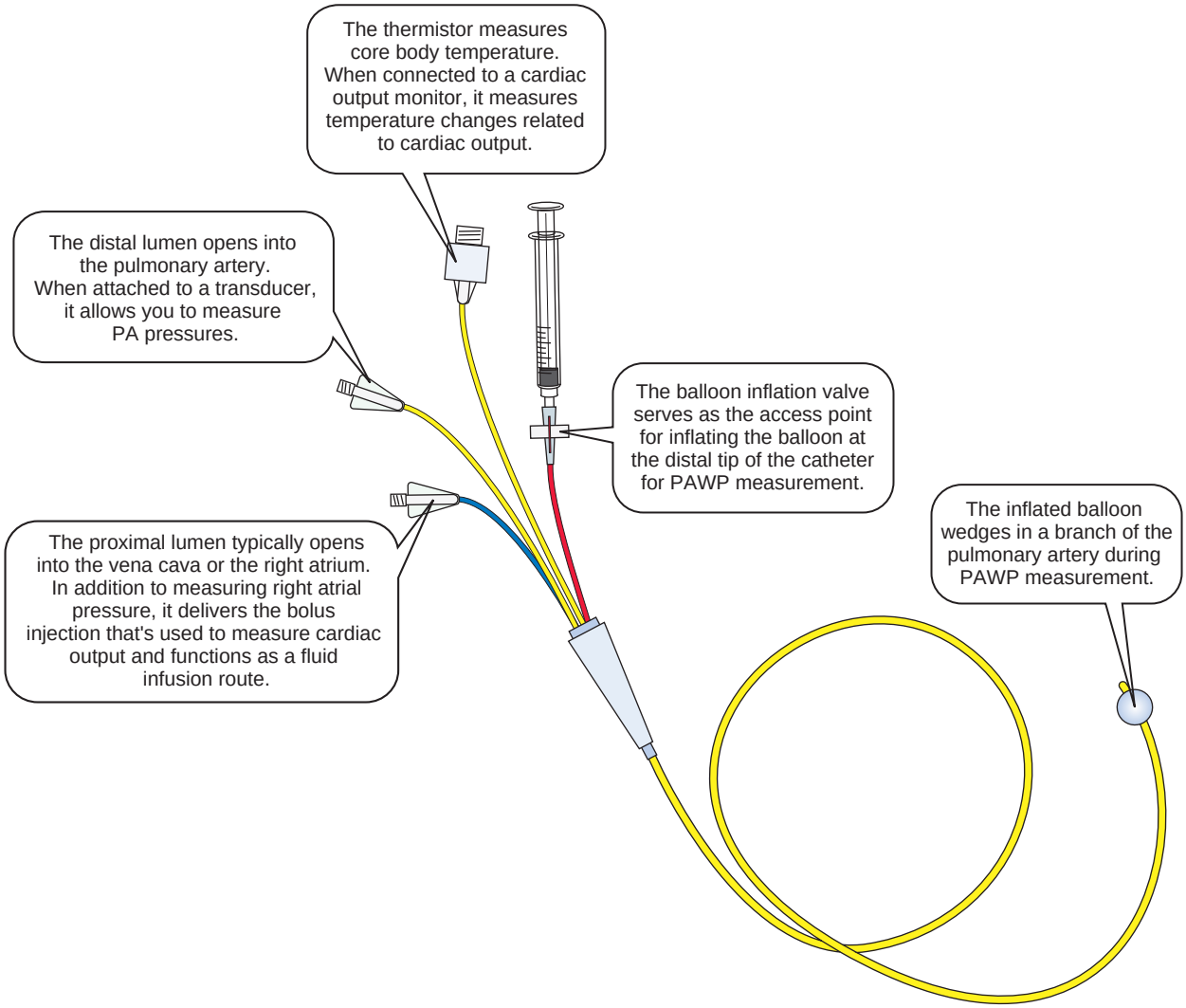


FIGURE 17-43 ▲ Pulmonary artery catheter. PA, pulmonary artery; PAOP, pulmonary artery occlusion pressure. (Courtesy of Edwards Lifesciences, LLC.)

Pulmonary Artery Catheter Insertion

Before the PA catheter is inserted, all necessary equipment should be assembled and prepared according to institution policies. The flushed pressure monitoring system is placed at the zero reference point, leveled, and zeroed. Each lumen of the PA catheter is flushed with sterile solution from the flush system. (Note that fiber-optic SO_2-monitoring catheters must be calibrated in the calibration cup housed in the sterile tray package before flushing the PA lumens.) The balloon lumen is inflated with air to ensure proper inflation and to check for leaks; it is then deflated before insertion. The PA port is then connected to the prepared pressure tubing, and the other lumens are connected to either a pressure monitoring system or an IV solution.

Strict sterile technique, including a full sterile drape, is required for the insertion procedure. The clinician performing the procedure wears a cap, mask, gown, and gloves. The nurse assisting wears a cap and mask and, if manipulating the catheter, gloves. The PA catheter is inserted into a large vein through an introducer catheter, which is usually placed by a percutaneous approach. The most common insertion sites are the right internal jugular, right or left subclavian, and femoral veins. Occasionally, the antecubital vein is used; this requires a venous cutdown.

Determination of the catheter tip location is established by monitoring the waveform and pressures on the bedside monitor as the catheter passes through the heart chambers and vessels. Black catheter markings occur every 10 cm, with a heavier black line at the 50- and 100-cm points. Distances are identified from the distal tip (ie, the proximal lumen exits 30 cm from the distal tip). These marking are also used during the insertion procedure to assist with catheter tip placement. When the catheter tip is approximately 15 cm into the introducer, the tip has typically exited the sheath and lies in the vena cava and RA junction. Waveforms on the monitor show respiratory excursions.

At this time, the balloon is inflated with the recommended balloon inflation volume of 1.5 mL air or CO_2. The clinician gently but rapidly advances the catheter with the balloon inflated. This helps "float" the catheter into the right atrium, through the tricuspid valve into the right ventricle,

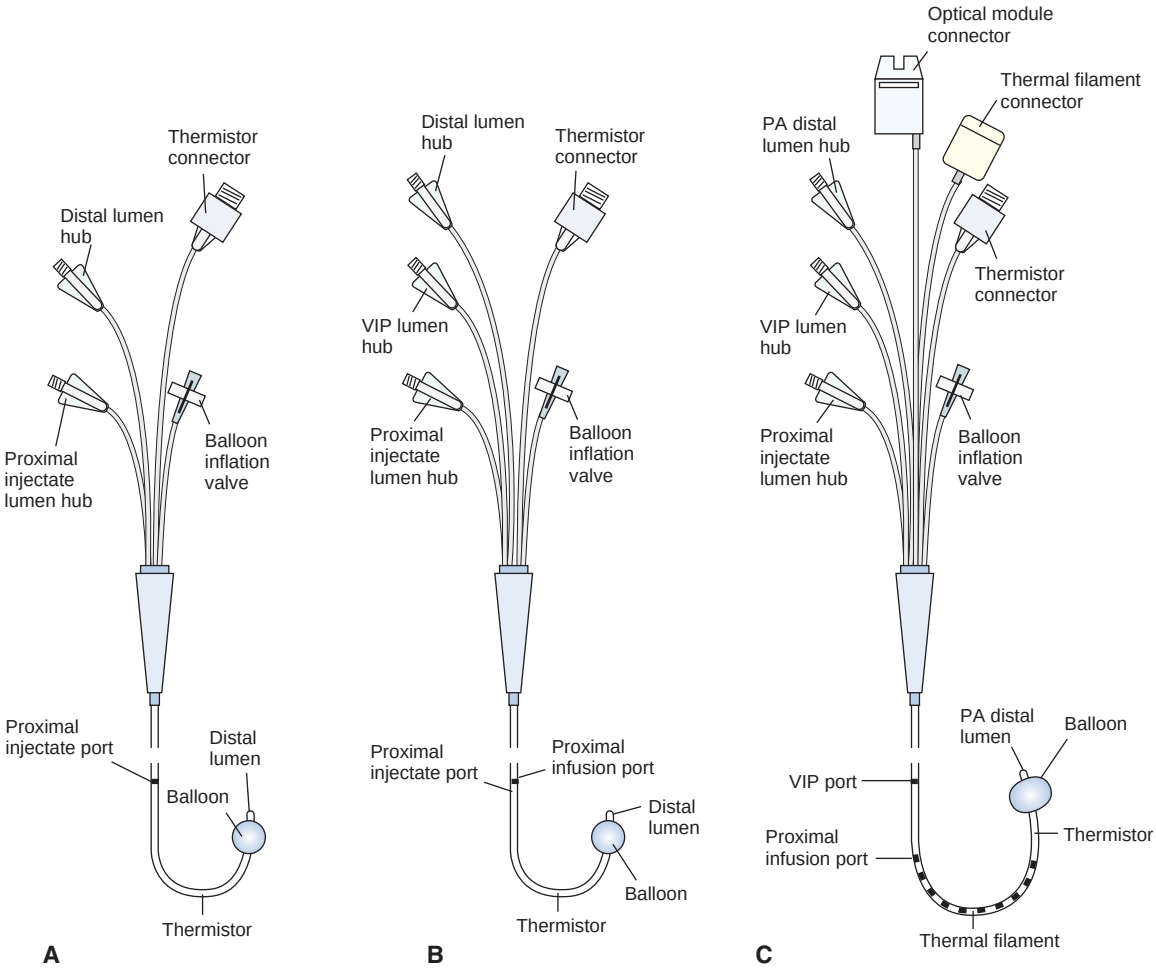


FIGURE 17-44 ▲ Types of PA catheters. **A:** Four-lumen catheter. **B:** Five-lumen catheter that includes an additional venous infusion port (VIP) into the right atrium. **C:** Seven-lumen catheter that includes a VIP port and two additional lumens for continuous cardiac output (CCO) and thermal filament, and continuous mixed venous oxygen saturation (SvO_2) monitoring (optical module connector). An additional option is to combine use of the CCO filament and the thermistor response time to calculate end-diastolic volume monitoring. (Courtesy of Edwards Lifesciences, LLC.)

across the pulmonic valve into the PA, and eventually into the wedged position (Fig. 17-45). The balloon is allowed to deflate passively after the PA wedge waveform is noted on the monitor and return of the PA waveform is confirmed. The PA catheter is slowly pulled back 1 to 2 cm to reduce or remove any redundant length or loop in the right atrium or right ventricle. The balloon is then reinflated to determine the minimum inflation volume required to obtain a wedge tracing. The catheter should be in position where the full or near full inflation volume (1.25 or 1.5 mL for a 7- or 8-F PA catheter) is required to obtain a wedge pressure tracing. Again, the balloon is passively deflated, and return of the PA tracing is observed. The PA catheter is made of a material that softens in vivo. This additional step during insertion helps decrease distal migration of the PA catheter after it has been placed. The PA catheter is then secured, and a sterile dressing is placed over the insertion site. Catheter position is also verified with a chest radiograph after the insertion.

Nursing responsibilities during the insertion procedure include ensuring use of sterile technique, monitoring the changes in hemodynamic waveforms, recording the pressures in each chamber of the heart as the catheter is passed through, and monitoring the patient for complications. Ventricular dysrhythmias are the most common complications during PA catheter insertion (see section on Complications on pages 277–279). Therefore, it is advisable to have a lidocaine bolus and defibrillator available for the insertion procedure.[20]

Waveform Interpretation

All hemodynamic pressures and waveforms are generated by pressure changes in the heart during the various phases of the cardiac cycle. Electrical activity (depolarization and repolarization) precedes mechanical activity of systole and diastole. Therefore, interpretation of the hemodynamic waveforms depends on the correlation of mechanical to electrical activities using the ECG. There are three categories of hemodynamic waveforms: atrial, which includes RA, left atrial, and PA wedge (that indirectly reflects the left atrial waveform); ventricular, which includes left and right ventricular; and arterial, which includes PA and systemic arterial. The waveforms in each category are similar because

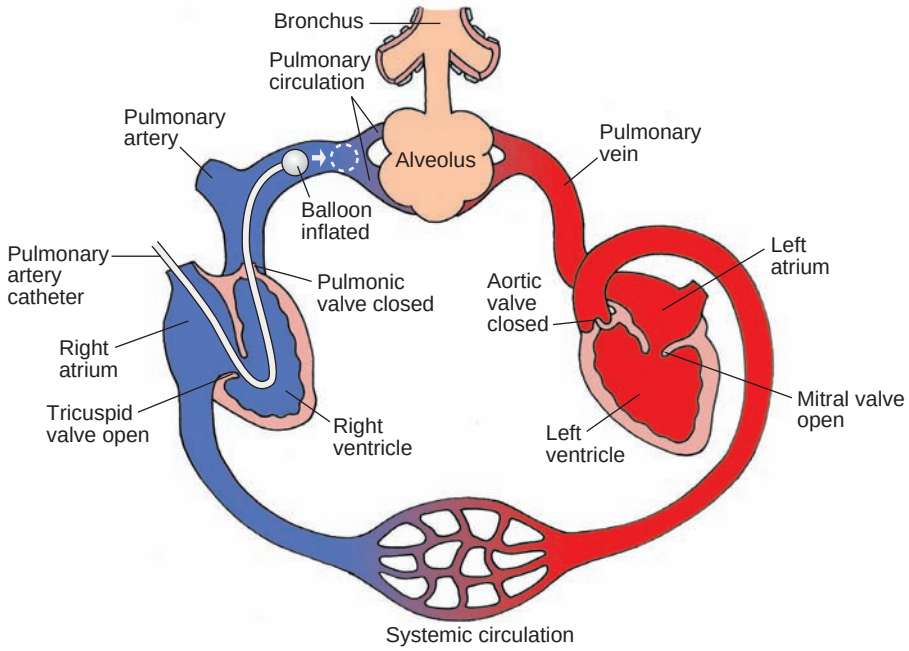


FIGURE 17-45 ▲ Position of the PA. When the balloon is inflated and the catheter is in the wedge position, there is an unrestricted vascular channel between the tip of the catheter and the left ventricle in diastole. PAOP thus reflects left ventricular end-diastolic pressure, an important indicator of left ventricular function. (Courtesy of Philips.)

they result from the same cardiac events. The measurements are different because the pressures generated in the right side compared with the left side of the heart differ.

Right Atrial Pressure

The right atrium is a low-pressure chamber, receiving blood volume passively from the venae cavae. Normal pressure is 2 to 6 mm Hg. Both atrial waveforms have three positive waves: a, c, and v. The a wave reflects the increase in atrial pressure during atrial systole. The c wave results from a small increase in pressure associated with closure of the AV valve and early atrial diastole. The v wave represents atrial diastole and reflects the increase in pressure caused by filling of the atrium with blood. It also occurs during ventricular systole. Figure 17-46 shows the RA waveform.

Accurate identification of the a, c, and v waves requires correlation of the waveform with the ECG. On the ECG, the P wave represents atrial depolarization, which causes RA and then left atrial (LA) contraction. Therefore, the a wave occurs after the P wave and usually in the PR interval. The QRS complex represents ventricular depolarization and causes ventricular contraction. Simultaneously, the atria relax and fill with blood. The v wave generated by these events thus falls after the QRS complex and in the T-to-P interval.

Atrial pressure tracings also have two primary negative waves or descents: x and y. The x descent follows the a or c wave if present and represents a decrease in pressure caused by atrial relaxation at the beginning of atrial diastole. The y descent follows the v wave and represents the initial, passive atrial emptying into the ventricle as the AV valve opens.[20–22]

Right Ventricular Pressure

The RV is a low-pressure chamber. RVEDP is usually 0 to 8 mm Hg. When the tricuspid valve is open, the RAP and the RVEDP are similar. Right ventricular systolic pressure is normally 20 to 30 mm Hg because the RV must generate only enough pressure to open the pulmonic valve and move blood through the low-pressure pulmonary vasculature.

The RV waveform has a distinctive "square root" configuration. Figure 17-46 shows the RV waveform. The initial rapid increase in RV pressure represents isovolumetric contraction, which follows the QRS complex of the ECG. The RV pressure continues to increase as the tricuspid and pulmonic valves are closed until the ventricular force generated exceeds the PA pressure. Rapid ejection occurs when the pulmonic valve opens. After ventricular systole, the pulmonic valve closes, and the right ventricular pressure rapidly decreases, creating a diastolic dip. Next in the cardiac cycle, the tricuspid valve opens and the RV passively fills with blood from the RA. Right ventricular diastole occurs within the period from the T wave to the next Q wave on the ECG. The point on the waveform just before the rapid increase in pressure represents RVEDP.[20–22]

Pulmonary Artery Pressure

The pulmonary vasculature is a relatively compliant low-resistance, low-pressure system in healthy people. Normal PA systolic pressure is 20 to 30 mm Hg. Normal diastolic PA pressure is 8 to 15 mm Hg, with a mean of 10 to 20 mm Hg. Systolic PA pressure and the peak of the PA waveform are generated by RV systolic ejection; therefore, the PA systolic pressure and the RV systolic pressure are the same as long as the pulmonic value is not stenotic. The PA waveform characteristics are similar to the systemic arterial waveform previously described (see Fig. 17-46). The dicrotic notch in the downward slope of the PA waveform corresponds with pulmonic valve closure at the beginning of RV diastole and is the beginning of the PA diastolic phase. PA diastolic pressure reflects the resistance of the pulmonary vascular bed and, to a limited degree, LVEDP. In normal conditions, with no obstructions or resistance from the PA to the left ventricle, PA diastolic pressure theoretically is an indirect measure of

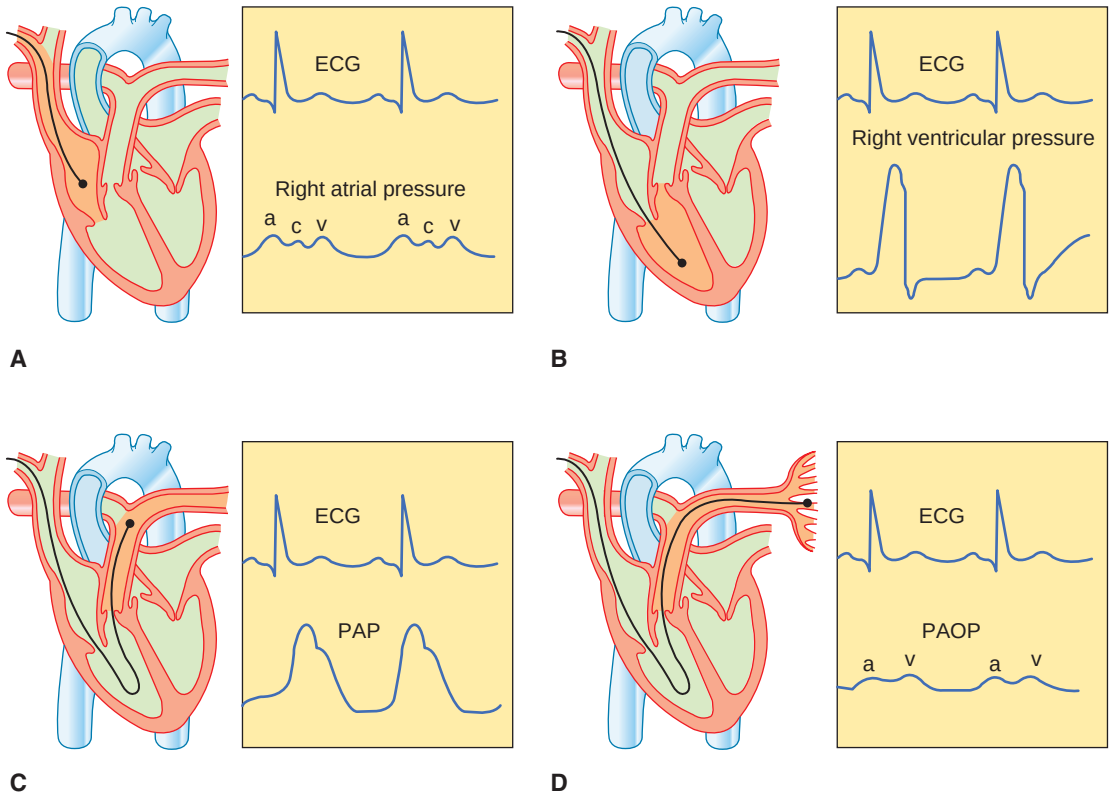


FIGURE 17-46 ▲ Normal PA waveforms. During PA insertion, the waveforms change as the catheter advances through the heart. **A:** When the catheter enters the right atrium (RA), a waveform with three small upright waves appears. The a waves represent the RA systole; the v waves, RA filling. **B:** When the catheter reaches the right ventricle, a waveform with sharp systolic upstrokes and lower diastolic dips appears. **C:** When the catheter "floats" into the PA, a PA pressure (PAP) waveform appears. Note that the upstroke is smoother than on the right ventricle waveform. The dicrotic notch indicates pulmonic valve closure. **D:** When the catheter "floats" into a distal branch of the PA, the balloon wedges where the vessel becomes too narrow for it to pass, and a PAOP waveform, with two small upright waves, appears. The a wave represents left atrial systole; the v wave, left atrial filling. ECG, electrocardiogram. (Courtesy of Edwards Lifesciences, LLC.)

LVEDP because the pulmonary vasculature, left atrium, and open mitral valve allow equalization of pressure from the left ventricle back to the tip of the PA catheter.[20–22]

Pulmonary Artery Occlusion Pressure

When the PA catheter is properly positioned, the PAOP (also called pulmonary artery wedge pressure [PAWP]) is obtained by inflating the balloon at the catheter tip. The balloon occludes forward flow in the branch of the PA, decreasing the influence of pulmonary vascular resistance (PVR) on the pressure reading, and creates a static column of blood from that portion of the PA through the LA, an open mitral valve during diastole, and the LV. In this way, the PAOP reflects LVEDP. Normal PAOP is 8 to 12 mm Hg. The PAOP more closely measures LA pressure and LVEDP than the PA diastolic pressure.

Inflation of the PA balloon causes the PA waveform on the monitor to become a PAOP tracing. No more than 1.5 mL of air is used to inflate the balloon. If less than 1 to 1.25 mL of air generates a PAOP tracing, the PA catheter has migrated distally and with the balloon passively deflated, needs to be withdrawn slightly. Depending on institution policies, a physician or advanced practice clinician performs this procedure.

An LA tracing has a, c, and v waves and x and y descents. The electrical and mechanical events of the heart generating these waves are identical to those of the RA waveform. The a wave corresponds to LA contraction, and the v wave corresponds to LA filling and LV contraction. With a direct LA line, the c wave is typically visible as in an RA tracing. The c wave is rarely visible on the PAOP tracing because the slight increase in pressure from backward bulging of the mitral valve is difficult to observe.

The ECG may be correlated with the PAOP waveform just as with the RA waveform. The primary difference between the PAOP and the RA waveforms is the slight delay of a and v waves in PAOP relative to the ECG because of the distance from the left side of the heart to transmit pressures to the catheter placed in the right side. The a wave now falls more closely in line with the QRS complex, although it may be within the PR interval. The v wave correlates with the T-to-P interval.[20–22]

Figure 17-47 shows the normal values and waveforms during a PA catheter insertion. Note that the RA pressure is equivalent to the RVEDP, the RV systolic pressure is equivalent to the PA systolic pressure, and the PA diastolic pressure is equivalent to the PAOP. Observe the diastolic value during insertion; it increases when the PA catheter "floats" into the PA, and the systolic pressures from the RV to the PA are similar.

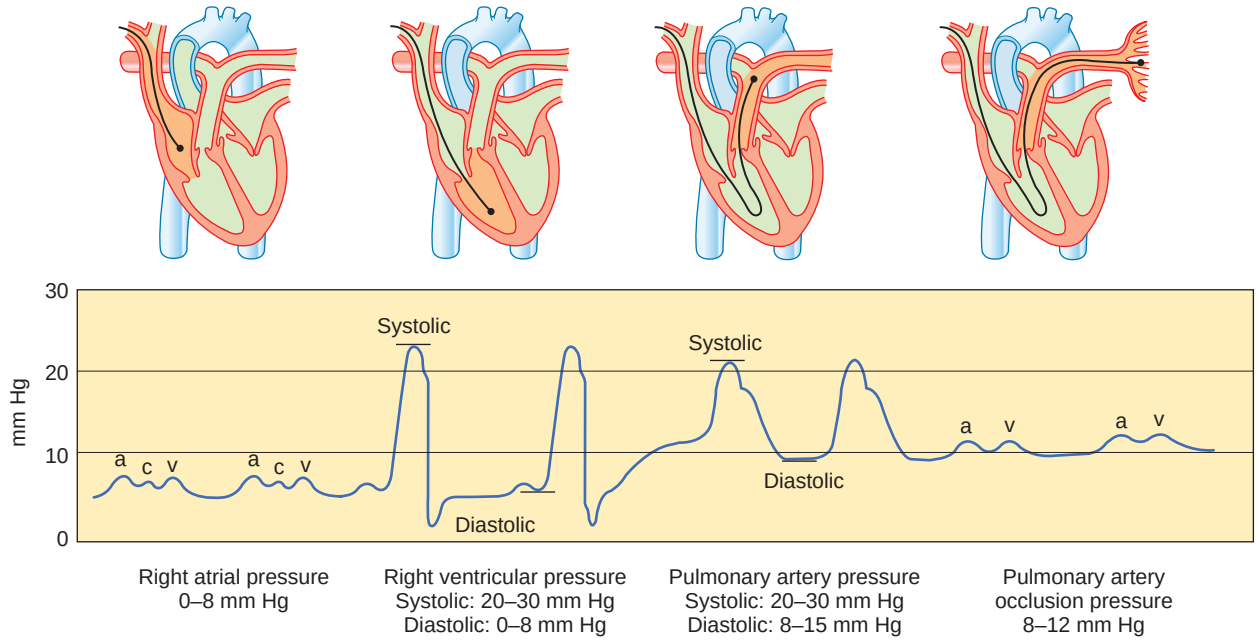


FIGURE 17-47 ▲ Normal values and wave configurations produced by the PA catheter.

Physiological Causes of Abnormal Waveforms

Hemodynamic waveform analysis provides valuable additional information for differential diagnosis. Specific conditions produce abnormalities in the a, c, and v waves, in the x and y descents, or in a combination of both. Clinical assessment, coupled with waveform analysis and interpretation of hemodynamic pressure, enhances the skills of the critical care nurse. Table 17-17 summarizes the causes of abnormal hemodynamic pressures.

Abnormalities of RA waveform include large, elevated a or v waves. Increased resistance to RV filling and impaired atrial emptying cause an elevated a wave. Examples of pathological causes of large a waves are tricuspid stenosis and RV failure. Elevated v waves are related to regurgitant flow from the ventricle back into the atrium during ventricular contraction. Examples of pathological causes of large v waves are tricuspid valve insufficiency and RV failure. Elevations in either the a or v wave cause the mean RA pressure to be higher.

Increased PA pressures may be systolic or diastolic or both. Because PA systolic pressure is a reflection of RV systolic pressure, factors that increase RV pressures such as increased PVR, hypervolemia, LV failure, and mechanical ventilation can produce an increased PA systolic pressure. Left ventricular failure, hypervolemia, and increased PVR cause increased PA diastolic pressure. Increased PVR may result from acute respiratory distress syndrome, primary pulmonary hypertension, or pulmonary embolus.

Left ventricular dysfunction and mitral valve disease occur more frequently than RV or tricuspid valve disorders, and therefore abnormal PAOP waveforms are more common than abnormal RA waveforms. Abnormalities of the PAOP waveform usually are large, elevated a or v waves. Increased resistance to LV filling and impaired atrial emptying cause elevated a waves. Examples of pathological causes of large a waves are mitral stenosis and LV failure. Elevated v waves relate to regurgitation from an incompetent mitral valve, allowing blood to flow from the ventricle back into the atrium during ventricular contraction. In both these valvular diseases, the PAOP does not accurately reflect LVEDP. Left ventricular failure usually causes elevation of both the a and v waves and significantly increases the PAOP because of reduced contractility and forward blood flow. Elevated PAOP frequently is due to LV dysfunction or hypervolemia. In some cases, such as in acute respiratory distress syndrome or with mechanical ventilator settings that generate extremely high intrathoracic pressure, PAOP is elevated because of noncardiogenic causes. In these cases, the normal PA diastolic pressure/PAOP gradient of 1 to 4 mm Hg widens. A widened pressure gradient is a differential diagnostic sign of pulmonary hypertension or increased PVR.[20,22]

Complications

Generally, most complications that occur with use of the PA catheter relate to the process of percutaneous central venous access. The other complications such as infection, thrombus, and air embolus are discussed in the earlier section on CVP.

Pneumothorax

Pneumothorax is a complication from vessel access through the subclavian vein. Anatomical factors can make placement of a PA catheter difficult, particularly if the patient is obese or has torturous subclavian veins. The needle or introducer sheath may pass through the vessel wall and puncture the lung during insertion, causing an apical pneumothorax. Signs and symptoms of a pneumothorax and routine postinsertion chest radiograph are used to diagnose this complication.

Table 17-17 Interpreting Hemodynamic Monitoring Pressures

Pressure and Description	Normal Values	Causes of Increased Pressure	Causes of Decreased Pressure
Central Venous Pressure (CVP) or Right Atrial Pressure (RAP)			
The CVP or RAP reflects right ventricular function and end-diastolic pressure.	Mean pressure: 2–8 mm Hg	• Right-sided heart failure • Volume overload • Tricuspid valve stenosis or insufficiency • Constrictive pericarditis • Cardiac tamponade • Pulmonary hypertension • Right ventricular infarction	Reduced circulating blood volume
Right Ventricular Pressure			
Typically, right ventricular pressure monitored only on initial pulmonary artery catheter insertion. Right ventricular systolic pressure normally equals pulmonary artery systolic pressure; RAP reflects right ventricular end-diastolic pressure.	Systolic pressure: 20–30 mm Hg Diastolic pressure: 0–8 mm Hg	• Mitral stenosis or insufficiency • Pulmonary disease • Hypoxemia • Constrictive pericarditis • Chronic heart failure • Atrial and ventricular septal defects • Patent ductus arteriosus	Reduced circulating blood volume
Pulmonary Artery Systolic Pressure			
Pulmonary artery systolic pressure results from right ventricular systolic pressure and reflects right ventricular function.	Systolic pressure: 20–30 mm Hg Mean pressure: 8–15 mm Hg	• Left-sided heart failure • Increased pulmonary blood flow (left or right shunting, as in atrial or ventricular septal defects) • Any condition causing increased pulmonary arteriolar resistance (such as pulmonary hypertension, volume overload, mitral stenosis, or hypoxia)	Reduced circulating blood volume
Pulmonary Artery Diastolic Pressure			
Pulmonary artery diastolic pressure is an indirect reflection of left ventricular end-diastolic pressure (LVEDP) in a patient without significant pulmonary artery disease.	Diastolic pressure: 8–12 mm Hg	• Any condition causing increased pulmonary arteriolar resistance (such as pulmonary hypertension, volume overload, mitral stenosis, or hypoxia)	Reduced circulating blood volume
Pulmonary Artery Occlusion Pressure (PAOP) or Left Atrial Pressure			
PAOP indirectly reflects left atrial and LVEDPs, unless the patient has obstructions from the tip of the pulmonary artery catheter to the left ventricle. Changes in PAOP reflect changes in left ventricular filling pressure.	Mean pressure: 8–12 mm Hg	• Left-sided heart failure • Mitral stenosis or insufficiency • Pericardial tamponade	Reduced circulating blood volume
Pulse Pressure (PP)			
PP is the difference between systolic and diastolic arterial pressure. PP can be used to assess the patient's stroke volume (SV).	Normal range: 40–60 mm Hg with a wider range of 30–100 mm Hg	• Increased SV • Decreased vascular resistance • Peripheral vascular disease • Aortic insufficiency	Decreased SV Severe vasodilation in conditions such as late sepsis, various shock states

Modified from Springhouse: Critical Care Made Incredibly Easy. Springhouse, PA: Author, 2004, p 170.

Infection

Systemic infection and sepsis are caused by contamination of the PA catheter, insertion site, or pressure monitoring system. Careful attention to sterile technique during pressure tubing assembly, using the maximum sterile barrier for insertion, and dressing changes helps prevent infection.[24] Protocols for changing the PA catheter and monitoring system should be followed carefully. Diagnosis of PA catheter-related sepsis is based on blood cultures, WBC count, and fever in the absence of other sources of infection.

Ventricular Dysrhythmias

Ventricular dysrhythmias may occur during the insertion of a PA catheter. As the catheter passes through the right ventricle, it may irritate the endocardium and cause premature ventricular complexes and occasionally ventricular tachycardia. The dysrhythmias typically resolve when the catheter is advanced into the PA. After the PA catheter is in proper position, it may become dislodged if it is not well secured, and the tip may "fall back" into the right ventricle. The patient may experience dysrhythmias, and the hemodynamic pressures and waveform reflect those of the right ventricle. Usually in this situation, because of potential contamination at the insertion site, the catheter is withdrawn or occasionally by inflating the balloon, the catheter may "refloat" into the PA. It is essential to have ready access to emergency drugs and equipment in case the ventricular dysrhythmias persist. Many introducer kits contain sterile sheaths; when placed over the PA catheter, they provide additional protection from contamination.

Pulmonary Artery Rupture or Perforation

A rare but very serious and potentially fatal complication is rupture or perforation of the PA. Perforation of the PA may occur during insertion, manipulation, or upon subsequent rewedging of the PA catheter. Patients with friable PAs may be at some risk. However, proper advancement of the catheter with the balloon fully inflated with 1.5 mL of air and avoidance of advancing the catheter too far into a small artery minimize the chance of PA perforation. Close observation of the PA waveform as the balloon is inflated and filling the balloon only with the amount of air necessary to obtain a PAOP tracing prevent overdistending a small PA. As previously stated, the catheter should become wedged when inflated with 1.25 to 1.5 mL of air. If less air is required to obtain the PAOP waveform, the catheter has migrated out of proper position.[1]

Nursing Considerations

Nursing care of the patient undergoing PA pressure monitoring is complex. Critical care nurses must be able to interpret waveforms and pressure data as well as be alert to potential complications. It is necessary to ensure accurate readings and minimize operator error. Consistency of leveling and measurement techniques is especially important because small variations in the zero reference point elicit large and erroneous changes in the pressures observed. Table 17-16 outlines problems and troubleshooting strategies associated with hemodynamic pressure monitoring. Evidence-Based Practice Highlight 17-3 on page 271 summarizes current evidence related to obtaining accurate measurement of PA pressures.

Measurement of all hemodynamic pressures is most accurate when obtained at the end of expiration in the respiratory cycle. In the healthy person, intrathoracic pressure at end expiration is about equal to atmospheric pressure. During the end-expiration period, there is minimal airflow and little variation in pleural pressures that influence cardiac pressures. Thus, end expiration provides a standard reference point for obtaining measurements. Spontaneous breathing causes negative intrathoracic pressure during inspiration, which produces a decline in the waveform. The waveform used for measurement is the last clear wave occurring just before the inspiratory dip. Mechanical ventilation causes increased intrathoracic pressure during inspiration, which produces an inspiratory "push" or rise in the waveform. The end-expiratory wave used for measurement is the last clear wave occurring just before the inspiratory rise (Fig. 17-48).

Closely set alarm parameters alert the nurse to potential physiological or technical complications. For example, one indication of a pulmonary embolus is an acute increase in PA pressures. Distal migration of the PA catheter may cause the catheter to wedge spontaneously without balloon inflation, and PA pressures may decrease to that of a PAOP. With properly set alarms, conditions such as these are detected.[20,22]

▲ Determination of Cardiac Output

CO is the volume of blood ejected from the heart per minute, expressed in liters per minute. Normally, CO is 4 to 8 L/min at rest. CO is a function of heart rate and SV. The left ventricle must generate enough pressure in systole to overcome aortic pressure and systemic vascular resistance (SVR) and eject sufficient blood volume to perfuse the organs of the body. The determination of CO and assessment of its determinants are important adjuncts to the care of critically ill patients. Routine evaluation of CO is essential when any CO monitoring technology is used.

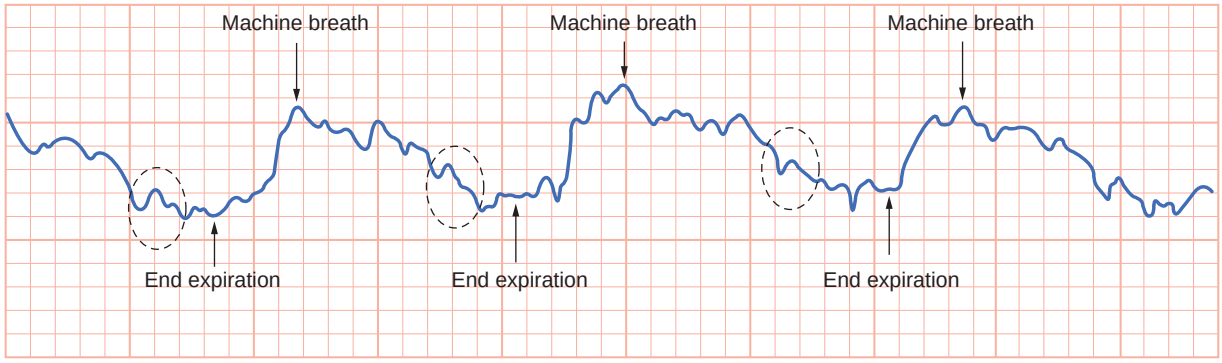


FIGURE 17-48 ▲ PAOP tracing showing respiratory variation from positive pressure mechanical ventilation. Measurement of PAOP is made at the last clear tracing before the inspiratory rise as identified by the open circles.

Cardiac index relates CO to body size. Normally, the cardiac index is 2.5 to 4 L/min/m^2. To obtain it, the CO is divided by the patient's body surface area (BSA). Standard bedside monitors and CO computers automatically calculate the cardiac index when the patient's height and weight are entered. The BSA is also used to index other valuable hemodynamic parameters (Table 17-18).

Factors That Determine Cardiac Output

CO is determined by heart rate and SV as described in Chapter 16. Alterations in CO are caused by changes in heart rate, preload, afterload, and contractility. Analysis of each of these is essential in directing interventions to address the underlying pathophysiological process. One strategy to consider is to evaluate CO, then systematically assess the determinants of heart rate, SV with preload first, afterload second, and finally contractility.

An increased or decreased CO provides global information only and needs to be evaluated in light of the components affecting it. Bradycardia due to conduction defects or medications can cause CO to decrease. An increase in heart rate can produce an increase in CO; however, this may be a compensatory physiological response to emotional or physiologic stress, or to a decreased SV. Tachycardia increases myocardial oxygen demands and may place a compromised patient at risk for myocardial ischemia. Tachycardia also may decrease CO because of shortened diastole and decreased filling time of the ventricles. If the elevated heart rate is due to external stimuli, identify the cause and direct interventions to eliminate or decrease the stimuli. Conditions to assess for are pain, fever, stress, and hypermetabolic states.

Table 17-18 Calculation of Cardiac Hemodynamic Parameters

Parameter	Definition	Formula	Normal Values
Cardiac Output (CO)	The number of liters pumped by the heart per minute.	Heart rate × SV	4–8 L/min
Cardiac Index (CI)	CO indexed to the patient's body surface area (BSA).	CO/BSA	2.5–4 L/min/m^2
Stroke Volume (SV)	The mL of blood ejected from the ventricle with each contraction.	CO/HR × 1,000	60–100 mL/beat
Stroke Volume Index (SVI)	SV indexed to the patient's BSA.	CI/heart rate	33–47 mL/beat/m^2
Mean Arterial Pressure (MAP)	The calculated average arterial pressure over a full cardiac cycle.	[Systolic BP + (diastolic BP × 2)]/3	70–105 mm Hg
Right Atrial Pressure (RAP)	Pressure created by volume of blood in the right heart.	Direct measurement	0–8 mm Hg
Left Atrial Pressure (LAP)	Pressure created by volume of blood in the left heart.	Direct measurement	6–12 mm Hg
Pulmonary Artery Occlusion Pressure (PAOP)	Pressure measured in the pulmonary artery when the pulmonary artery catheter's balloon is inflated.	Direct measurement	8–12 mm Hg
Right Ventricular End-Diastolic Volume Index (RVEDVI)	Amount of volume in the right ventricle at the end of diastole.	SVI/RV ejection fraction	60–100 mL/m^2
Left Ventricular End-Diastolic Volume Index	Amount of volume in the left ventricle at the end of diastole.	SV/LV ejection fraction	40–80 mL/m^2
Systemic Vascular Resistance (SVR)	Refers to the resistance to blood flow offered by the systemic vasculature.	[(MAP – RAP) × 80]/CO	800–1,200 dyne/s/cm^{-5}
Systemic Vascular Resistance Index (SVRI)	SVR indexed to patient's BSA.	[(MAP – RAP) × 80]/CI	1,360–2,200 dyne/s/cm^{-5}
Pulmonary Vascular Resistance (PVR)	Refers to the resistance to blood flow offered by the pulmonic vasculature.	(MPAP – PAOP) × 80/CO	<250 dyne/s/cm^{-5}
Pulmonary Vascular Resistance Index (PVRI)	PVR indexed to patient's BSA.	(MPAP – PAOP) × 80/CI	<425 dyne/s/cm^{-5}
Left Ventricular Stroke Work Index (LVSWI)	A measure of work performed by the left ventricle each beat.	SVI (MAP–PAOP) × 0.0136	40–70 g/m^2/beat
Right Ventricular Stroke Work Index (RVSWI)	A measure of work performed by the right ventricle each beat.	SVI (MPAP – RAP) × 0.0136	5–10 g/m^2/beat
Stroke Volume Variation	Variation in SV over a respiratory cycle.	SV maximum – SV minimum/SV mean	<10%–15%

SV, the volume of blood ejected by each ventricular contraction, is influenced by preload, afterload, and contractility (see Chapter 16 for detailed discussion). Preload is the degree of stretch on the myocardial muscle fibers at end-diastole and is determined by ventricular filling (end-diastolic) volume. Within physiological limits, increases in end-diastolic volume cause stretch of the myofibrils and increase the force of the next ventricular contraction (Frank-Starling law of the heart). Preload is primarily influenced by total blood volume. Because the PA catheter measures pressure, not volume, assumptions are made that volume and pressure can be equated. Many factors alter the pressure–volume relationship; therefore, the use of pressures (eg, CVP or PAOP) to evaluate preload must be considered in light of factors that can affect pressures, such as mechanical ventilation or ventricular compliance. A specialized PA catheter is able to provide right ventricular ejection fraction and volumetric data. Indirect assessment of preload uses the RAP or CVP for the right ventricular preload, and the PA diastolic pressure, left atrial pressure, and PAOP for the left ventricular preload.[20–22]

Decreases in preload can be due to hypovolemia, secondary to blood loss, dehydration, or third spacing of fluids. Preload is also reduced related to massive vasodilation, for example in septic, anaphylactic, or neurogenic shock. Hypovolemia or decreased venous return associated with mechanical ventilation and elevated intrathoracic pressures can cause a decreased preload.

Afterload is often defined as the impedance or resistance to ejection of blood from the ventricles. PVR is a clinical assessment of right ventricular afterload. Left ventricular afterload is clinically evaluated by calculating SVR. PVR and SVR can be indexed to body size using the patient's BSA (see Table 17-18, p. 280). Primary factors affecting afterload are semilunar valve abnormalities and vascular resistance.

Vasoconstriction causes elevated afterload and has several causes. Increased SVR may be a compensatory response to hypovolemia caused by vasoconstriction to maintain peripheral perfusion in this state. An increase in afterload occurs with some medications, hypothermia, and the compensatory vascular response to cardiogenic shock. This increase in afterload may also be accompanied by a decrease in CO and an increase in myocardial oxygen demand and work. A decrease in afterload due to vasodilation reduces resistance to ejection of blood, thus increasing CO. Vasodilating medications, septic states, and allergic and anaphylactic reactions are all causes of vasodilation and thus increased CO.

Contractility, the third determinant of SV, is an inherent property of the heart. It is not affected by preload or afterload and cannot be directly measured. Indices used to assess contractility include determining SV and calculating the stroke work index for both the left and right ventricles. Myocardial oxygen supply and demand balance, electrolytes, and minerals (eg, calcium) influence myocardial contractility.

Reduction in contractility decreases CO. Examples include insufficient oxygen delivery to the myocardium, causing myocardial ischemia and infarction; medications, such as beta-blocking agents; or metabolic imbalances, such as low serum levels of calcium, phosphorus, or magnesium. Positive inotropic agents or correction of impaired myocardial oxygenation or metabolic derangements may cause enhanced contractility, most often resulting in increased CO.[20–22]

Obtaining Cardiac Output Values

Several methods for evaluating CO are available. These include invasive, minimally invasive, and noninvasive technologies. All techniques have certain assumptions and limitations that need to be considered to provide an understanding of the indications and applications of each. This section discusses the more common methods of CO monitoring used in the critical care areas.

Fick's Method for Cardiac Output Determination

The Fick method, originally developed in the 1800s by Adolf Fick, is the historical laboratory gold standard. The Fick method is based on the principle that the uptake or release of a substance by an organ divided by the arterial and venous concentration of that substance is the product of flow or CO. The classic method of determining CO uses oxygen as the substance and the lungs as the organ. In order for this relationship to be valid, simultaneous samples of arterial and venous blood must be obtained and accurately measured. In addition, inspired and expired oxygen concentration must be measured by indirect calorimetry to determine the oxygen consumption. Other technologies use these principles; however, they use carbon dioxide as the measured substance.

Indicator-Dilution Methods for Cardiac Output Determination

Stewart proposed the principles of the indicator-dilution method, and Hamilton further refined them. The Stewart-Hamilton equation is based on use of a known indicator as a signal and determination of the dilution rate of that signal over a given period of time. Three indicators in clinical use are dye, thermal, and small doses of lithium. The indicator is injected into the venous system, and a time–concentration curve is generated from a blood sample obtained from the arterial system. Analysis of the curve allows CO calculation.[20–22]

Thermodilution is the most common method used to measure CO and is considered the clinical gold standard. Cold or room temperature solution is the indicator and is injected into the RA port of the PAC. A thermistor near the end of the catheter continuously measures the temperature of blood flowing past it. A dilution curve is generated by the change in blood temperature after indicator injection. Based on this curve, CO is calculated by the computer.

Determination of CO through thermodilution is obtained either on an intermittent or continuous basis. Intermittent CO requires the injection of a known amount of "cooler than blood" injectate, and a single CO curve is produced. Specialty catheters house thermal filaments and, with a dedicated thermal cable and computer, emit energy as the indicator. The "warmer than blood" signal is measured at the thermistor, and thermodilution curves are produced on a 30- to 60-second frequency for continuous CO assessment.[20]

Procedure for Intermittent Thermodilution Cardiac Output Determination

A computation constant, based on the catheter size, volume, and temperature of the injectate, as well as injection method, is set on the computer or programmed into the bedside CO

module. Five or ten milliliter of sterile D_5W or normal saline solution is used as the injectate solution and volume. The injectate syringe used should be part of a closed system that remains intact and attached to the RA port by a stopcock (Fig. 17-49). Iced (0°C to 4°C) or room-temperature solution may be used. A temperature difference between the patient's blood temperature and the injectate of at least 10°C improves accuracy. In most conditions, room-temperature injectate with a 10-mL volume provides accurate results. With hypothermia or very low CO states, iced solution and a 10-mL injectate volume provide a greater signal and increased accuracy.

Steps for performing a manual CO determination vary according to bedside monitor or CO computer manufacturer. See specific operations manuals for directions for use. General steps include:

- Ensuring the accurate amount of injectate volume in the syringe
- Injecting the volume in a smooth and rapid manner, in less than 4 seconds
- Waiting approximately 1 minute between injections to allow the catheter thermistor to return to baseline

When injected, the solution passes a temperature probe in the closed system and flows through the right atrium and right ventricle, past the thermistor at the tip of the PAC. A curve is produced and used for determining the CO. The average of several CO determinations is required to obtain a final measurement. Serial measurements and averaging are necessary because of the number of physiological variables and the performance of the technical procedure. Three or more consecutive measurements are usually necessary. Measurements included in the averaging process should be within 10% to 15% of the mean, and each one should be associated with a normal CO curve. Abnormal curves are eliminated from the CO averaging process.

Interpretation of Cardiac Output Curves

Many bedside monitors and CO computers are equipped with a means of visualizing the CO curves. Normal CO curves have a smooth upstroke from the rapid injection followed by a gradual decline (Fig. 17-50). The area under the curve is inversely proportional to the CO. Curves associated with a high CO have a small area under the curve, with a steeper upstroke and more rapid return to baseline, and curves associated with a low CO have a greater area under the curve, with a more sloped upstroke and slower return to baseline.

Arterial Pressure- and Waveform-Based Cardiac Output Determinations

Uses of arterial pressures, arterial waveforms, or both are other methods for determining CO and SV. The basic premise relates to the proportionality relationship of PP to SV and the inverse relationship of PP to aortic compliance. As PP widens, SV increases and aortic compliance decreases (becomes more rigid, less elastic). Other factors considered with this relationship are the impact of conditions that change vascular tone: larger vessel compliance and peripheral vessel resistance.[25,26]

General components required for these methods are an existing arterial line, a special sensor, and a specific monitor

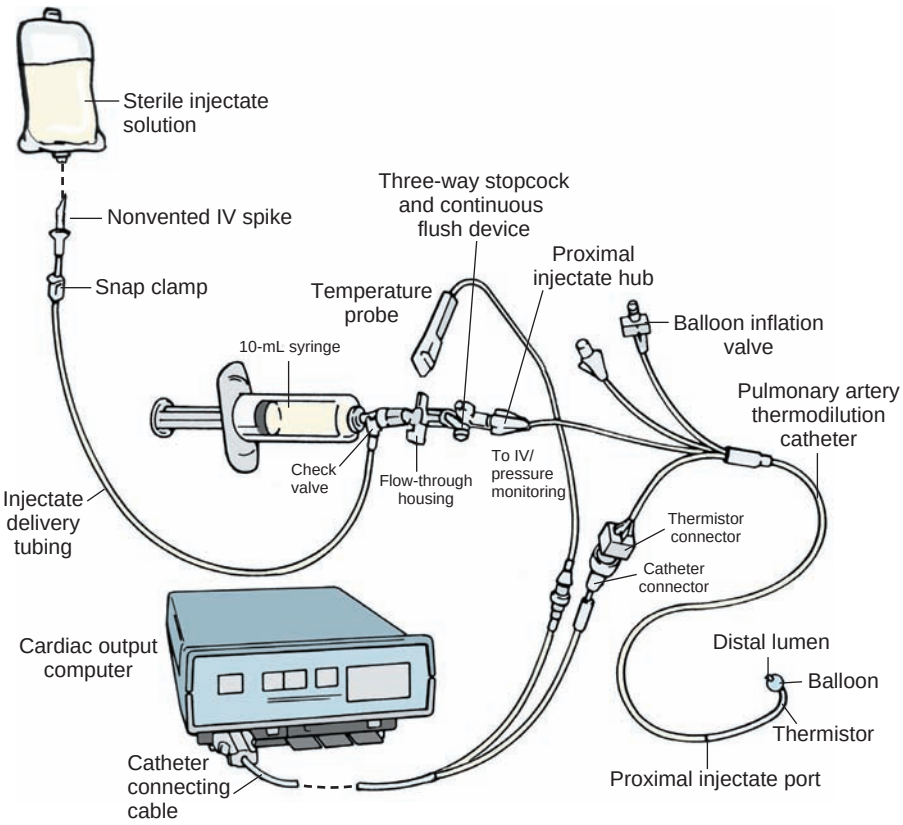


FIGURE 17-49 ▲ A closed room-temperature injectate system for measurement of cardiac output (CO). (Courtesy of Edwards Lifesciences.)

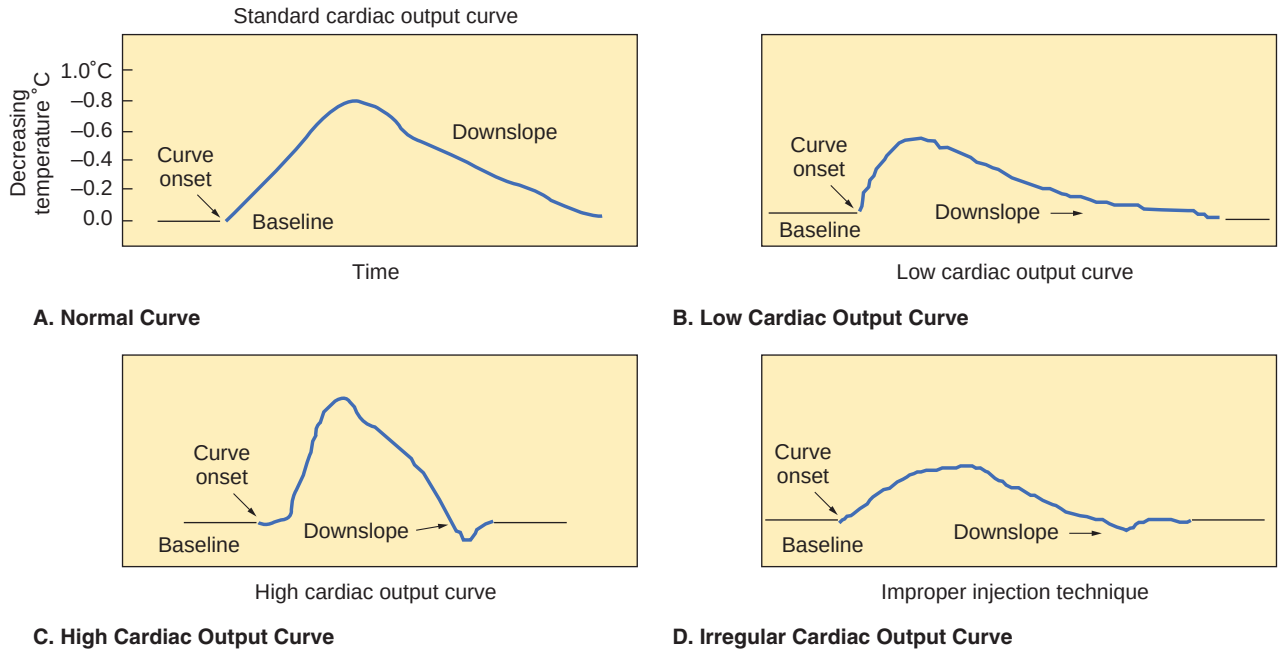


FIGURE 17-50 ▲ Examples of thermodilution curves observed on a bedside monitor or strip chart recorder. **A:** Normal curve with smooth upstroke and gradual decline to baseline. Note that the temperature change is actually lower than patient baseline temperature; however, the graph is shown in an upright orientation. **B:** Low CO produces a greater area under the curve. The upstroke is normal with a more gradual decline. **C:** A high CO has a smaller area under the curve. The upstroke is more rapid with a faster return to baseline. **D:** Irregular curve shows an erroneously low CO probably due to irregular or uneven emptying of the injectate syringe. (Courtesy of Edwards Lifesciences.)

that uses a unique algorithm for the SV and CO determinations. Some systems use the shape of the arterial wave to determine the dicrotic notch location, which signifies the end of systole. This method is referred to as pulse contour.[27] The area under the curve then represents the amount of volume ejected into the arterial vascular bed and reflects SV. Other systems assess the systolic and diastolic pressure to obtain a mean value to arrive at SV. This method is described as pulse power.[28] Another method samples the full waveform for pressures and uses waveform characteristics for CO determinations. This method is termed arterial pressure–based CO.[29] External calibration with either thermodilution or lithium is required for both the pulse contour and pulse power methods. The arterial pressure–based CO method does not require external calibration. Figure 17-51 depicts the various methods for obtaining SV from the arterial pulse wave.

Once the SV is determined, the pulse rate, assessed by the arterial waves, reflects the heart rate; SV × heart rate = CO. All the technologies, regardless of the specific algorithm used to determine the CO value, use the arterial pressure. This requires obtaining of accurate values and ensuring optimal waveforms.

Other parameters obtained with an arterial pressure system include stroke volume variation (SVV) or its surrogate, pulse pressure variation (PPV), and systolic pressure variation (SPV). These parameters evaluate the difference between the maximum and minimum values of systolic pressure, PP, or SV during a respiratory cycle. They are called dynamic parameters and are better predictors of fluid responsiveness than static measures such as CVP/RAP in the critically ill. A natural phenomenon occurs during the respiratory cycle in which the arterial pressure falls during inspiration and rises during expiration. The variation is a result of changes in intrathoracic pressure during respiration; a negative pressure during inspiration results in a fall in systolic pressure, and a relatively higher intrathoracic pressure during expiration causes a rise in systolic pressure. The normal variation during a respiratory cycle is 5 to 10 mm Hg. When the difference is greater, the condition is termed *pulsus paradoxus*. Reverse pulsus paradoxus is the same phenomenon that occurs during

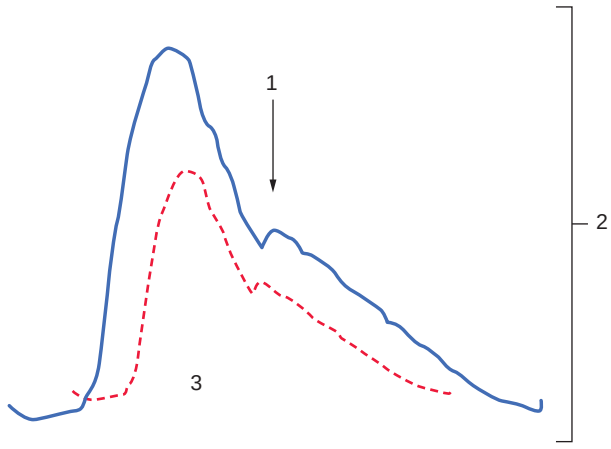


FIGURE 17-51 ▲ Methods to obtain stroke volume using arterial pulse wave. 1. Pulse Contour: Identification of dicrotic notch required. 2. Pulse Power: Mean determined, systolic and diastolic extrapolated. 3. Pulse Pressure/Flow: Full wave measured for pressure and waveform shape assessed.

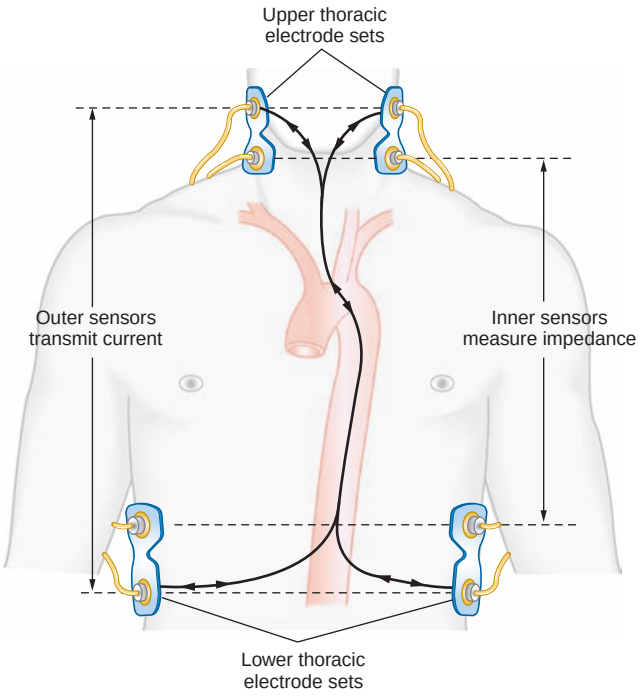


FIGURE 17-52 ▲ Placement of thoracic impedance sensors. (Courtesy of Cardiodynamic International.)

controlled mechanical ventilation. The mechanics are opposite of spontaneous breathing in that the arterial pressure rises during inspiration and falls during expiration.[20,25,29]

SPV, PPV, and SVV can be calculated from the arterial tracing as long as the respiratory cycle is noted. In addition, other technologies to obtain this value include bedside physiological monitors with special software and the pulse oximetry plethysmographic waveform.[30,31]

Nursing Considerations

Patient assessment includes assessing for pulsus paradoxus that occurs during cardiac tamponade, obstructive lung diseases, and hypovolemic states. A SVV greater than 10% to 15% has a high level of sensitivity and specificity for determining the need for fluid and in predicting preload responsiveness. A patient is preload responsive if after a fluid bolus their SV or CO increases by 10% to 15%. Technical considerations for the use of arterial pressure–based technologies include those that affect the accuracy of the arterial waveform. Optimal pressure system maintenance and leveling of the device is required. Limitations to using variation parameters relate to factors that cause changes in intrathoracic pressure and those that affect ventricular filling time. Any cardiac dysrhythmia can affect the overall value because of irregular ventricular responses and therefore the value should be used with caution in those conditions. Intravascular volume resuscitation increases preload, which in turn increases CO.[20,22,25]

Impedance-Based Cardiac Output Determination

Impedance (Z) is resistance to flow of an electrical current. Impedance decreases in the presence of fluid. Two types of impedance-based CO technologies are available for clinical use: bioreactance and impedance cardiography (ICG) (also called thoracic electrical bioimpedance). Both provide noninvasive, continuous, real-time CO and other hemodynamic data using pairs of skin electrode-sensors at the upper and lower thorax.

Bioreactance technology delivers a small alternating current via the upper electrodes and analyzes the changes in frequency of the electrical impulses as they traverse the chest. The frequency changes are related to aortic blood flow and thus can be used to calculate SV and CO. CO derived from bioreactance is comparable to those from PAC, ICG, and Fick.[32]

ICG also uses a very small electrical current injected through skin electrodes placed at the base of the neck and the lower thorax (Fig. 17-52). These electrodes also sense the change in impedance due to pulsatile blood flow in the descending aorta during systole and diastole. The change in impedance over time (dZ/dt) directly reflects left ventricular contractility (Fig. 17-53) and is mathematically converted into SV and CO.[33]

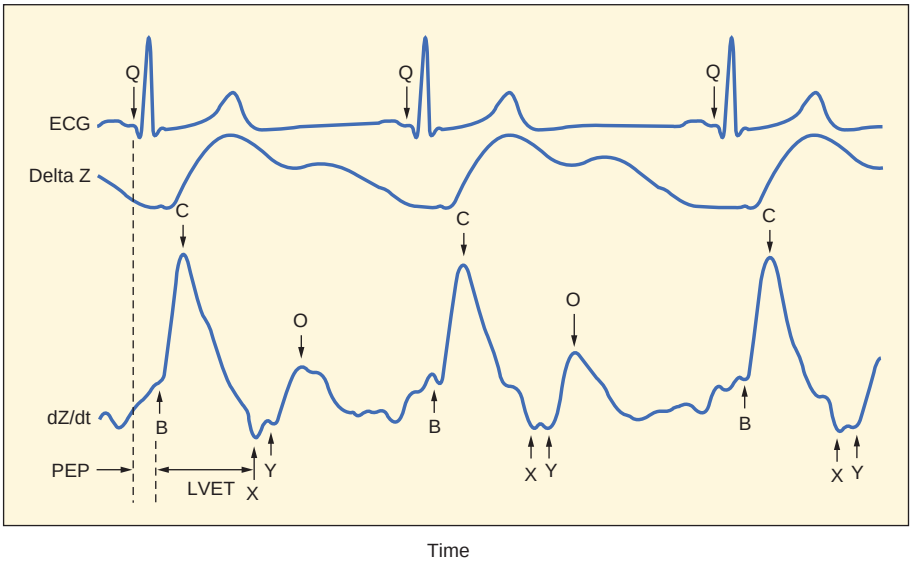


FIGURE 17-53 ▲ ECG and normal change in impedance over time (dZ/dt) waveform. Q, start of ventricular depolarization; B, opening of aortic and pulmonic valves; C, maximum deflection of dZ/dt (dZ/dt_{max}); X closure of aortic valve; Y, closure of pulmonic valve; O, mitral opening snap and early ventricular diastolic filling. (Courtesy of Cardiodynamics International, Inc.)

Both bioreactance and ICG monitors provide traditional hemodynamic determinations such as CO, SV, and SVR, if a blood pressure and CVP or RAP are entered into the monitor. Since aortic blood flow causes the most significant change in impedance, these technologies provide more direct indices of left ventricular contractility that are not available from a PAC. They also provide a baseline impedance, the thoracic fluid content, which reflects all of the fluid in the thorax (interstitial, intravascular, or intracellular). In some instances, when thoracic fluid content is very high, ICG determination of SV and CO may be affected.[33] CO values from bioreactance technology appear to be less influenced by pleural fluid, pulmonary edema, and chest wall movement than ICG.[32]

Nursing Considerations

Because ICG and bioreactance are noninvasive technologies, nurses in any inpatient or outpatient setting can initiate this type of hemodynamic monitoring.[33] Thus, the clinical applications are broad. For example, ICG parameters are used to evaluate patients with heart failure, hypertension, and permanent pacemakers in the emergency department, outpatient clinic, or physician office.

Because electrical impedance is reduced in the presence of fluid, the thoracic fluid content measurement is useful in the differential diagnosis of heart failure or chronic obstructive pulmonary disease as well as in the assessment and management of patients with heart failure who may have pulmonary congestion or pulmonary edema. Adjustment in diuretics, inotropic agents, and vasodilators can be fine-tuned based on impedance parameters. Similarly, patients with chronic and resistant hypertension can be managed more closely and more aggressively on an outpatient basis using ICG or bioreactance hemodynamic parameters as opposed to using blood pressure alone. Continuous noninvasive CO and hemodynamic parameters are also used to optimize the settings of atrial–ventricular (A-V) sequential cardiac pacemakers to adjust the time for A-V delay to allow appropriate ventricular filling to achieve optimal SV and CO.

Doppler-Based Cardiac Output Determination

Doppler-based technology uses the aortic blood flow velocity waveform to calculate SV and CO. The pulsatile velocity waveform directly reflects left ventricular contractility as well as the patient's intravascular volume status (preload).

The esophageal Doppler monitor (EDM) is a minimally invasive hemodynamic monitoring device that incorporates a Doppler transducer into a nasogastric tube. When placed in the esophagus, EDM monitors the descending aortic blood flow velocity[31,34] (Fig. 17-54). Continuous CO and SV determinations are calculated in real time relative to changes in blood flow using the Doppler waveform configuration.

USCOM is a noninvasive hemodynamic monitor that determines CO by continuous-wave Doppler ultrasound.[31,35] Data is obtained using a probe placed on the chest in either the left parasternal position to measure transpulmonary blood flow or the suprasternal notch to measure transaortic blood flow. USCOM does not provide continuous hemodynamic data. Information is obtained intermittently as desired by the clinician.

Both EDM and USCOM provide traditional parameters such as CO, SV, and SVR, if a blood pressure and CVP or RAP is entered into the monitor. Additional parameters derived from the waveform include: peak velocity, an indicator of myocardial contractility, and flow time, which reflects systolic ejection time and thus changes in preload.

Nursing Considerations

A valuable aspect of Doppler-based technology is use of the waveform shape to determine changes in myocardial contractility and intravascular volume (preload) because the waveform displayed on the monitor reflects the volume and velocity of blood in the aorta. The normal waveform is triangular, consisting of the beginning of systole, peak systole, and end-systole (Fig. 17-55). As flow from the left ventricle increases, the shape of the waveform changes, becoming a larger, higher, and wider triangle. Conversely, decreased contractility is reflected in a smaller waveform; hypovolemia causes the waveform to become more narrow at the base. The baseline shape of the waveform, as well as changes in response to therapy, can significantly contribute to hemodynamic assessment. Thus Doppler-based data and waveform analysis can guide the clinician in evaluating the patient's need for therapy and responses to fluid administration and titration of vasopressors and inotropes.[34]

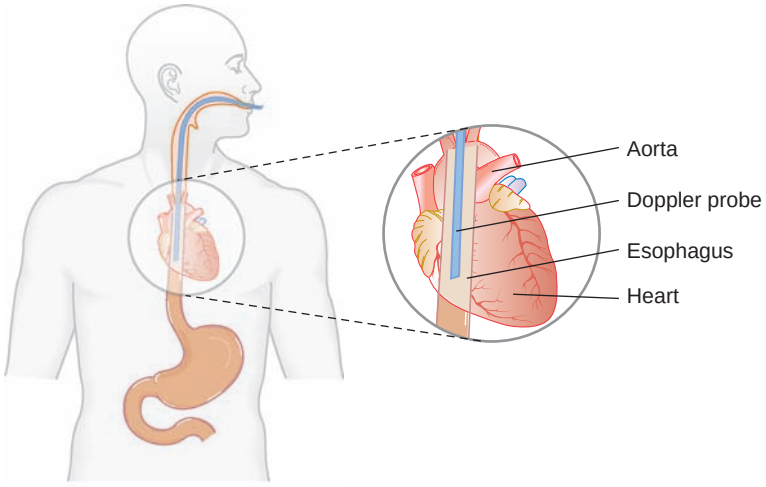


FIGURE 17-54 ▲ Location of the esophageal Doppler probe in the esophagus in relation to the heart and descending aorta. (Courtesy of Deltex Medical, Inc.)

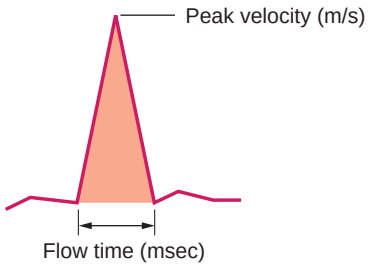


FIGURE 17-55 ▲ Esophageal Doppler waveform showing peak velocity and flow time. (Courtesy of Deltex Medical, Inc.)

EDM usually requires the patient to be sedated; therefore it is most often used in the critical care unit, operating room, postanesthesia care unit, and the emergency department. USCOM is noninvasive technology; clinicians in any inpatient or outpatient setting can use this type of hemodynamic monitoring. However, training is required and the data obtained depends on proper technique.

▲ Evaluation of Oxygen Delivery and Demand Balance

One of the primary objectives of hemodynamic monitoring is to use the data in the evaluation of oxygen delivery or transport and the consumption of oxygen by the tissues and organs. Adequate oxygen delivery to the body's organs is essential for maintenance of cell, tissue, and ultimately organ function. Insufficient oxygen delivery and consumption to meet the cellular requirements for oxygen, or oxygen demand, result in hypoxia and the accumulation of an oxygen deficit. Persistent oxygen deficit causes cell and organ dysfunction and eventually leads to cell death and organ failure.[36] Table 17-19 lists the parameters that are used to evaluate oxygen delivery and demand balance, the formulas, and normal values.

Determinants of Oxygen Delivery

Arterial oxygen delivery (DaO_2) is the amount of oxygen transported to the tissues. DaO_2 depends on arterial oxygen content and CO.

Oxygen Content

Oxygen content is the total amount of oxygen in the blood that is available to the cells. The two primary determinants of oxygen content are hemoglobin and oxygen saturation. Most of the available oxygen in arterial blood (>95%) is reversibly bound to hemoglobin in the form of oxyhemoglobin and is measured by arterial oxygen saturation (SaO_2). A very small amount of oxygen (<5%) is dissolved in plasma and measured as PaO_2. A sufficient amount of hemoglobin is required to ensure adequate oxygen-carrying capacity.

Cardiac Output

CO is required to deliver oxygenated blood to the cells of the body. DaO_2 is assessed by evaluating the adequacy of CO and arterial oxygen content. In nonstressed states, normal DaO_2 is 1,000 mL O_2/min, or indexed to BSA, 600 mL O_2/min/m^2. Increases in the body's oxygen demand associated with injury or illness are initially and primarily met by a compensatory increase in CO. Deficiencies of hemoglobin, arterial saturation, or CO decrease DaO_2 to cells and threaten the adequacy of cellular oxygenation.[37]

Determinants of Oxygen Consumption

Oxygen consumption (VO_2) is the amount of oxygen used by the tissues of the body. The primary determinants of VO_2 are the cellular demand for oxygen, the delivery of adequate amounts of oxygen, and the extraction of oxygen from the blood for use by the cells.

Oxygen Demand

Oxygen demand is the requirement of cells for oxygen and is not directly measurable. Any stress increases the oxygen demand (eg, surgery, infection, mobilization, pain, anxiety). Reduced oxygen demands are associated with lower metabolic rates (eg, hypothermia, sedation, pharmacological paralysis). Oxygen demands are met through adequate delivery of oxygen and cellular extraction of oxygen.[36,37]

Oxygen Delivery

Cellular use of oxygen depends on an adequate supply of oxygen. This is termed delivery-dependent oxygen consumption (Fig. 17-56). As oxygen delivery increases, oxygen consumption also increases to meet the oxygen demand. When the requirement for oxygen is met, further increases in oxygen delivery do not increase consumption. The level of critical oxygen delivery is the point at which a decrease in oxygen delivery results in decreased VO_2 because of an insufficient oxygen supply.[36,37]

Oxygen Extraction

Oxygen extraction ($CaO_2 - CvO_2$) is the amount of oxygen removed from the blood for use by the cells. It is measured by comparing the arterial oxygen content to venous oxygen content. Like arterial oxygen content, venous oxygen content (CvO_2) is primarily determined by the amount of hemoglobin that is saturated with oxygen. Venous saturation is obtained by withdrawing a mixed venous blood gas sample from the distal port of the PA catheter, or by using an SvO_2 or $ScvO_2$ monitoring central venous catheter or PA catheter, as discussed later in this section.

In normal circumstances, provided that oxygen is supplied in adequate amounts, the cells extract the oxygen they need to support tissue and organ function. Increased demand for oxygen results in a compensatory increase in oxygen extraction as more oxygen is "unloaded" from the hemoglobin for cellular use. The decreased amount of oxygen in venous blood means that the $CaO_2 - CvO_2$ difference is larger. Conversely, as oxygen demands decrease, less oxygen is required and extracted from the blood, and the $CaO_2 - CvO_2$ difference becomes smaller.[37]

Oxygen Supply and Demand Imbalance

An imbalance of oxygen supply and demand occurs whenever oxygen delivery is inadequate to meet cellular demand or the cells are unable to extract sufficient quantities of

Table 17-19 Oxygen Utilization Variables

Parameter	Definition	Formula	Normal Values
Arterial Oxygen Content (CaO_2)	The amount of oxygen carried by hemoglobin in a deciliter of arterial blood.	$(Hb \times 1.37 \times SaO_2) + (0.003 \times PaO_2)$	20 mL O_2/dL
Venous Oxygen Content (CvO_2)	The amount of oxygen carried by hemoglobin in a deciliter of venous blood.	$(Hb \times 1.37 \times SvO_2) + (0.003 \times PvO_2)$	15 mL O_2/dL
Arterial Oxygen Delivery Index (DaO_2I)	The amount of oxygen transported in the blood from the left ventricle through the arteries and capillaries to tissues/organs, in 1 min, indexed to the patient's BSA.	$CI \times CaO_2 \times 10$	500–600 mL O_2/min/m^2
Venous Oxygen Delivery Index (DvO_2I)	The amount of oxygen in the blood returned to the right ventricle in 1 min, via the veins from tissues/organs, indexed to the patient's BSA.	$CI \times CvO_2 \times 10$	375–450 mL O_2/min/m^2
Mixed Venous Oxygen Saturation (SvO_2)	The oxygen saturation of venous blood, measured in the pulmonary artery.	Direct measurement	60%–80%
Central Venous Oxygen Saturation ($ScvO_2$)	The oxygen saturation of venous blood, measured in the superior vena cava.	Direct measurement	65%–85%
Partial Pressure of Oxygen, venous blood (PvO_2)	Reflects the amount of oxygen dissolved in the plasma of venous blood.	Direct measurement	35–45 mm Hg
O_2 extraction	Amount of oxygen that is removed (extracted) from hemoglobin for use by cells/tissues/organs.	$CaO_2 - CvO_2$	3–5 mL O_2/dL
Oxygen Extraction Ratio (OER)	The percent of oxygen delivered that is removed (extracted) from hemoglobin for use by cells/tissues/organs.	$\frac{(CaO_2 - CvO_2)}{CaO_2}$	22%–30%
Oxygen Consumption Index (VO_2I)	The amount of oxygen used by cells/tissues/organs every minute, indexed to the patient's BSA.	$(CaO_2 - CvO_2) \times CI \times 10$	120–170 mL/min/m^2
Arterial pH (pHa)	The acidity (pH) of arterial blood.	Direct measurement	7.35–7.45
Base Excess/Base Deficit (BE/BD)	Amount of base required to titrate one liter of arterial blood to a pH of 7.40. Decreases with metabolic acidosis.	Direct measurement	–2 to +2
Lactate	A metabolic byproduct of Krebs cycle that increases with anaerobic metabolism.	Direct measurement	0.5–2.2 mmol/L

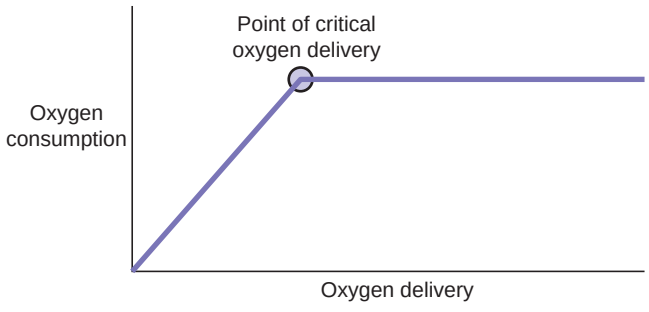


FIGURE 17-56 ▲ Delivery-dependent oxygen consumption curve reflecting the change in oxygen consumption related to oxygen delivery. At the point of critical oxygen delivery, oxygen delivery is sufficient to meet oxygen demand, and oxygen consumption does not increase further. However, any decrease in oxygen delivery from this point results in a decrease of oxygen consumption due to an inadequate supply of oxygen.

oxygen. Specific threats to the balance of oxygen supply and demand are decreased CO, hemoglobin, or arterial saturation; impaired cellular extraction of oxygen; or oxygen demands that are so great that they cannot be met by increased oxygen delivery or extraction.

Metabolic Indicators of Oxygen Delivery and Utilization Imbalance

Inadequate oxygen consumption causes an anaerobic state and cellular hypoxia. Cells deprived of oxygen become hypoxic and dysfunctional. Over time, cell damage becomes irreversible and cell death results. Cellular hypoxia is a major cause of multisystem organ dysfunction and failure. If an oxygen deficit is identified before irreversible cell injury has occurred, it may be reversed by increasing oxygen availability.

Several metabolic parameters can be measured to evaluate cellular hypoxia. When these indicators are used in conjunction with hemodynamic monitoring of oxygen delivery and consumption, therapies may be more specifically directed to achieve a balanced oxygen supply and demand.

Because hypoxia and oxygen debt are associated with anaerobic metabolism, the byproducts of anaerobic metabolism can be used to assess the presence of an oxygen deficit and cellular hypoxia. Lactic acid accumulation causes a metabolic acidosis in a hypoxic state. Therefore, laboratory measurement of lactate levels, serum pH, and base deficit/excess are means to evaluate if oxygen supply is sufficient to meet cell requirements. Serum pH and base deficit/excess are routinely measured and reported with blood gas analysis. Elevated lactate levels (>2.2 mm/L) or metabolic acidosis (pH < 7.35 with normal $PaCO_2$) correlate with oxygen debt, particularly when the patient has a low or even normal level of DaO_2 and VO_2. As with all assessment parameters, lactate levels, pH, and base deficit should not be viewed in isolation; they should be evaluated in conjunction with other assessment parameters.[20–22]

Monitoring of Mixed Venous and Central Venous Oxygen Saturation

Mixed venous oxygen saturation (SvO_2) reflects the level of oxyhemoglobin in desaturated blood returning to the right ventricle and PA. Venous oxygen saturation can also be measured in the superior vena cava ($ScvO_2$). SvO_2 or $ScvO_2$ can be monitored at the bedside by specialized central venous catheters or PACs containing fiber-optic filaments in one of the lumens ending at the distal end. The information is updated every few seconds; thus, a continuous SvO_2 or $ScvO_2$ reading is obtained.

Both SvO_2 and $ScvO_2$ are useful to evaluate the global balance of oxygen supply, oxygen utilization, and oxygen demand. The SvO_2 or $ScvO_2$ is significantly lower than arterial saturation because of the extraction of oxygen by the cells and the unloading of oxygen from hemoglobin.

SvO_2 or $ScvO_2$ is influenced by the degree of arterial saturation, the quantity of hemoglobin, the CO (the determinants of oxygen delivery), and the amount of oxygen extracted and consumed by the cells. Under normal conditions of oxygen delivery, oxygen consumption, and oxygen demand, approximately 25% of the available oxygen is extracted and used to meet demand. In this situation, the SvO_2 or $ScvO_2$ is in the normal range: SvO_2 of 60% to 80%, or $ScvO_2$ of 65% to 85%. If oxygen delivery is reduced by a decrease in arterial saturation, hemoglobin, or CO, then more oxygen is extracted from the blood to meet cellular demand. The blood returning to the right side of the heart and PA has had a greater quantity of oxygen removed and is more desaturated, which is reflected by a decrease in $ScvO_2$ or SvO_2. Similarly, if oxygen demand increases but oxygen delivery does not increase to meet this requirement, additional oxygen is extracted from the blood and consumed by the cells. Therefore, oxyhemoglobin is reduced in the venous blood, decreasing SvO_2 or $ScvO_2$. Persistently low SvO_2 or $ScvO_2$ is a warning that cellular hypoxia and an oxygen debt may be developing because of inadequate oxygen delivery or a high oxygen demand not met by the oxygen supply.[37,38]

Three general conditions result in increasing SvO_2 or $ScvO_2$:

- An oxygen delivery that is much greater than the oxygen demand; only a small percentage of the delivered oxygen is extracted, causing SvO_2 or $ScvO_2$ to increase.
- A low metabolic rate and oxygen demand; the need for oxygen is reduced, and less oxygen is extracted and consumed. SvO_2 or $ScvO_2$ reflects the decrease in extraction as greater amounts of oxyhemoglobin are returned to the right side of the heart.
- Pathological states in which the cells cannot extract oxygen from the blood or in which tissue beds are not well perfused with oxygenated blood; oxygen is not extracted from the blood despite the cellular oxygen demand. The SvO_2 or $ScvO_2$, returning to the right side of the heart and PA, is therefore higher because of the decreased oxygen consumption.[37,39]

Although the SvO_2 or $ScvO_2$ may be in the normal range, cells in the body may not use or receive the oxygen they require. In these instances, cells become reliant on anaerobic metabolism because of the reduced cellular oxygen extraction or the shunting of oxygenated blood past tissue beds. Thus, a normal SvO_2 or $ScvO_2$ may be misleading when viewed in isolation. Table 17-20 summarizes factors that cause SvO_2 or $ScvO_2$ to increase or decrease.

Lactate, base deficit and excess, and SvO_2 or $ScvO_2$ are measures of global tissue oxygenation status. In shock states, blood is shunted from the splanchnic tissue bed and from the extremities to vital organs. Therefore, assessment of perfusion

Table 17-20 Examples of Causes of Increasing or Decreasing Central Venous Saturation

↓ $ScvO_2$	↑ $ScvO_2$
Increased oxygen extraction	*Decreased oxygen extraction*
1. Increased oxygen demand Causes: Stress, pain, anxiety, fever	1. Decreased oxygen demand Causes: Sedation, pain relief, hypothermia
2. Oxygen delivery insufficient to meet oxygen demand Causes: Decreased CO, Hgb, SaO_2	2. Increased oxygen delivery Causes: Increased CO, Hgb, SaO_2
	3. Impaired cellular oxygen extraction Causes: cytotoxicity, sepsis, cell death

CO, cardiac output; Hgb, hemoglobin; SaO_2, arterial saturation.

in these specific tissue beds can be useful in the early detection of reduced oxygen delivery and/or utilization.

Near-infrared spectroscopy is a noninvasive technology to monitor tissue oxygen saturation (StO_2) in muscle. It uses an infrared light emitting and sensing "patch" over the thenar muscle (located at the base of the thumb on the palm of the hand) to measure oxygen saturation in the microcirculation below the sensor. (Fig. 17-57) As tissue perfusion is reduced, especially when associated with hypovolemia or decreased CO states, StO_2 decreases. StO_2 values that trend downward and are less than 75% are associated with higher morbidity and mortality.[40] In patients with sepsis and septic shock, StO_2 is lower compared with patients who meet SIRS criteria[41] but has not been shown to be superior to $ScvO_2$ in the sepsis population.[42]

Gastric tonometry and sublingual CO_2 monitoring are methods to evaluate perfusion of specific tissue beds that have early susceptibility to hypoperfusion.[43] In early shock or shock states, compensatory diversion of blood flow from the splanchnic bed and digestive tract to vital organs causes the gastric mucosa and upper gastrointestinal tract to be underperfused. Anaerobic metabolism produces increased amounts of CO_2 and lactate; thus, measuring the CO_2 level or pH of these tissue beds can provide an early indicator of oxygen supply and demand mismatch.

Gastric tonometry uses a nasogastric tube with a gas-permeable balloon near the distal end. CO_2 diffuses from the gastric wall into the balloon. Sampling the contents allows measurement of the PCO_2 and gastric mucosal pH. Normal gastric mucosal pH is 7.35 to 7.45, and normal gastric mucosal PCO_2 is 35 to 45 mm Hg. Decreasing gastric mucosal pH or an increasing gastric PCO_2 out of the normal range suggests hypoperfusion and is an indication that oxygen delivery and consumption should be analyzed and optimized. Medications that neutralize gastric pH, such as H-2 blockers may also affect the gastric tonometer values.

Sublingual capnometry is based on the same physiological principles as gastric tonometry. Blood flow to the upper digestive tract, including the area under the tongue, is reduced in response to shock or hemorrhage. Sublingual capnometry uses a handheld device similar to a thermometer to measure PCO_2 using a sensor that is placed under the tongue.

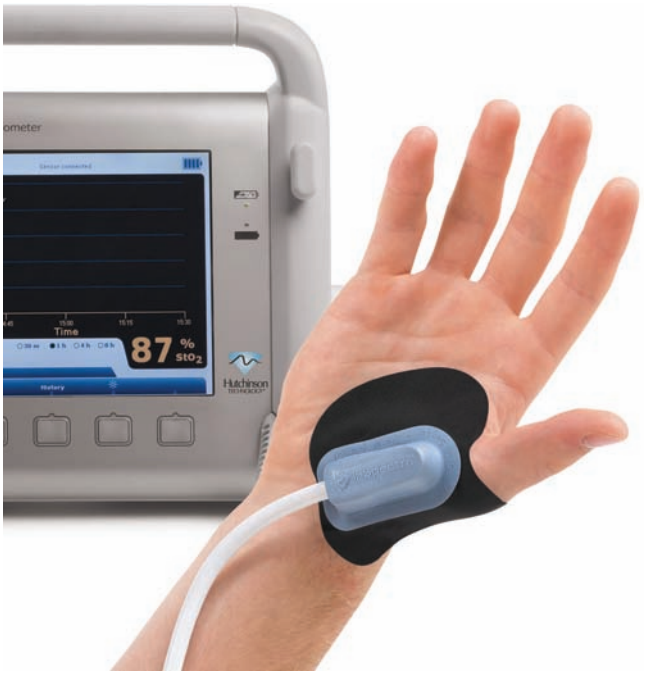


FIGURE 17-57 ▲ Placement of the StO_2 sensor over the thenar muscle.

Nursing Considerations

When patients are critically ill, careful evaluation of the adequacy of oxygen delivery, oxygen extraction, and consumption with respect to oxygen demand is paramount. Scrutiny of each determinant of CO (heart rate, preload, afterload, and contractility parameters), oxygen content (arterial saturation and hemoglobin), oxygen consumption (VO_2 and $CaO_2 - CvO_2$), and oxygen debt (lactate, pH, base deficit and excess, SvO_2 or $ScvO_2$) is important to critical care nursing.

Numerous interventions are used to enhance oxygen delivery. Measures to increase CO include the addition of intravascular volume to increase preload as well as the administration of positive inotropic agents to improve contractility and vasodilating agents to reduce afterload. Interventions that may increase arterial oxygenation and oxygen content include changes in mechanical ventilator settings; chest physiotherapy; positioning and mobilization; and, in nonmechanically ventilated patients, coughing and deep-breathing exercises. Administration of packed red blood cells increases hemoglobin and oxygen-carrying capacity. In all cases, it is necessary to manage both the treatment modalities and assess the patient's response to therapy.

Many of the interventions used to decrease oxygen demand and increase oxygen consumption are important tenets of nursing care. For example, appropriate management of the environment, pain, and anxiety reduces stress, thus decreasing the demand for oxygen. Maintaining normothermia by control of the patient's temperature may decrease oxygen requirements associated with fevers and facilitate impaired perfusion and oxygen consumption associated with hypothermia.

SvO_2 or $ScvO_2$ monitoring may be a helpful guide in nursing interventions. For example, endotracheal suctioning may cause a temporary decrease in arterial oxygenation and increase discomfort and anxiety. Monitoring SvO_2 or $ScvO_2$ allows the nurse to judge the impact of this activity on the patient's oxygen supply and demand. A decreasing SvO_2 or $ScvO_2$ during suctioning is usually caused by increased oxygen demand and decreased arterial oxygenation. Hyperoxygenating and hyperventilating before, during, and after suctioning helps lessen the negative effects on oxygen demand and arterial oxygenation. Before proceeding to another activity such as repositioning, the nurse should monitor the SvO_2 or $ScvO_2$ and wait until the value normalizes, thereby avoiding an additional stressor and further increase on oxygen demand.

$ScvO_2$ monitoring has been incorporated into the early, goal directed management guidelines for patients with sepsis and septic shock. A goal is to maintain an $ScvO_2$ of at least 70% by increasing oxygen delivery.[44] The use of a sepsis bundle and protocol that includes $ScvO_2$ is associated with improved morbidity and mortality.[45]

▲ Clinical Applicability Challenges

CASE STUDY

Mr. T. is a 69-year-old male, 6′1″, 97.6 kg (BSA 2.22) whose medical history revealed questionable hypertension with noncompliant use of medication and absent regular medical treatment.

Mr. T. arrived at the emergency department with complaints of increasingly severe abdominal pains. The abdomen was tender upon palpation and distended. Initial vital signs were: blood pressure 190/104 mm Hg, heart rate 86 beats/min, normal sinus rhythm, respiratory rate 22 breaths/min, and SpO_2 96%.

Laboratory results showed Hgb 11 g/dL, Hct 33%, white blood cell (WBC) 9,840, and lactate 2.2 mmol/L.

A preliminary differential diagnosis of acute appendicitis versus abdominal aortic aneurysm was made. The physical examination and laboratory test findings were inconclusive so a computed axial tomography scan was performed. Results of the scan showed a 9-cm abdominal aortic aneurysm.

Mr. T.'s condition worsens rapidly after the scan with complaints of increased abdominal pain, severe respiratory distress with an SpO_2 value decreasing to 89%, and a sudden decrease in his blood pressure to 108/64 mm Hg. Respiratory rate increased to a rate of 36 and he exhibited shallow breathing. Heart rate remained in a sinus rhythm, however it increased to 98.

Due to Mr. T.'s worsening respiratory status and blood pressure, he was intubated and placed on controlled mechanical ventilation. To assist with ventilation synchronization management, he was sedated. Mr. T. was subsequently admitted to the intensive care unit (ICU) for stabilization and additional monitoring.

Upon admission to the ICU an arterial line was placed and he was attached to a less invasive CO monitoring device to measure and monitor CO and other dynamic variables, such as stroke volume variation (SVV). In addition, to provide ready access to his central circulation, a central line was placed in his right internal jugular vein. The specific catheter used was one that monitors continuous central venous oxygen saturation values to provide an assessment of his overall oxygenation balance status.

Additional hemodynamic and oxygenation values obtained in the ICU were: cardiac index 2.8 mL/min/m^2, SV variation 13%, and $ScvO_2$ 72%. All of these variables are within the normal value range. Care included continued monitoring of Mr. T.'s status.

About an hour after admission to the ICU Mr. T.'s condition continued to deteriorate. His reprofiled parameters were: blood pressure 98/48 mm Hg, HR 126 beats/min, CI 2.1 L/min/m^2, SVV 24% and $ScvO_2$ 54%.

The profile Mr. T. now presents can be one of acute hypovolemia, hemorrhage, or potentially both. The cardiac flow indices are low. With a cardiac index of 2.1 and a heart rate of 126, the SV is only 37. These are significant changes from the earlier set of parameters that showed a SV of 63. The $ScvO_2$ value in addition decreased from 72% to 54%, again a clinically significant change. Factors to assess when the $ScvO_2$ value changes are those conditions that alter either oxygen delivery; arterial oxygen saturation, hemoglobin, or CO, and those that affect oxygen consumption; metabolic demand, pain, shivering, and fever.

SVV as a dynamic indicator of fluid responsiveness suggested that since Mr. T. is on controlled mechanical venation, he would respond favorably to fluid.

A repeat laboratory hemoglobin and hematocrit sample showed Mr. T.'s blood count to have dropped from Hgb 11/Hct 33 to Hgb 9/Hct 27. This significant drop along with the indicators of a hypovolemic state resulting from potential bleeding from the aortic aneurysm led the surgical team to perform an emergent abdominal aortic aneurysm repair.

The remaining course of hospitalization was unremarkable and Mr. T. did well. Discharge was within 5 days.

1. What were the symptoms that caused concern on initial assessment?

2. How did the additional parameters, CI, SVV, and $ScvO_2$, assist in the identification of the clinical problem?

3. What would be a course of action for improving CO, $ScvO_2$, and SVV?

References

1. American Heart Association: Risk Factors and Coronary Heart Disease. Retrieved June 27, 2011, from http://www. AHA.org
2. American Heart Association: Heart and Stroke Facts. Retrieved June 27, 2011, from http://www.AHA.org
3. Bickley L: Bates' Guide to Physical Examination and Health History, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009
4. Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010
5. Antman EM, Anbe DT, Armstrong PW, et al: ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction. Executive summary: A report of the American College of Cardiology/American Heart Association task force on practice guidelines (Committee to revise the 1999 guidelines for the management of patients with acute myocardial infarction). Circulation 110:588–636, 2004
6. Kushner FG, Hand M, Smith SC, et al: 2009 Focused updates: ACC/AHA Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction (Updating the 2004 Guideline and 2007 Focused Update) and ACC/AHA/SCAI Guidelines on Percutaneous Coronary Intervention (Updating the 2005 Guideline and 2007 Focused Update): A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 54:2205–2241, 2009
7. Grundy SM, Cleeman JI, Merz NB, et al: Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III Guidelines. Circulation 110:227–239, 2004
8. Thygesen K, Alpert J, White HD: Universal definition of myocardial infarction. Circulation 116:2634–2653, 2007
9. Sabatine MS, Morrow DA, Jablonski KA, et al: Prognostic significance of the Centers for Disease Control/American Heart Association high sensitivity C-reactive protein cut points for cardiovascular and other outcomes in patients with stable coronary artery disease. Circulation 115:1528–1536, 2007
10. Casas JP, Shah T, Hingorani J: C-reactive protein and coronary heart disease: A critical review. J Intern Med 264(4):295–314, 2008
11. Torbiki A, Perrier A, Konstantinides S, et al: Guidelines on the diagnosis and management of acute pulmonary embolism. Eur Heart J 29(18):2276–2315, 2008
12. Bosen D: Beyond ECG's: Understanding electrophysiology testing. Nursing2011 Critical Care 5(3):38–44, 2010
13. Thomas GS, Thompson RC, Miyamoto MI, et al: The RegEx trial: A randomized double blind, placebo and active-controlled pilot study combining regadenoson, a selective A2A adenosine agonist, with low-level exercise, in patients undergoing myocardial perfusion imaging. J Nucl Cardiol 16(1):63–72, 2009
14. Folsom A, Kronmal R, Detrano R, et al: Coronary artery calcification compared with carotid intima-media thickness in the prediction of cardiovascular disease incidence. Arch Intern Med 168(12):1333–1339, 2008
15. Greenland P, Bonow R, Brindage B, et al: American College of Cardiology/American Heart Association Expert Consensus Document on Coronary Artery Calcium Scoring by Computer Tomography in Global Cardiovascular Risk Assessment and in Evaluation of Patients with Chest Pain. J Am Coll Cardiol 49(3):378–402, 2007
16. Tonino P, De Bruyne B, Pijils N, et al: Fractional flow reserve versus angiography for guiding percutaneous coronary intervention. N Engl J Med 360:213–224, 2009
17. Nottingham F: Diagnosis and treatment of atrial fibrillation in the acute care setting. J Am Acad Nurse Pract 22:280–287, 2010
18. Wann LS, Curtis AB, January CT, et al: Writing on behalf of the 2006 ACC/AHA/ESC guidelines for the management of patients with atrial fibrillation writing committee. ACCF/AHA/HRS focused update on the management of patients with atrial fibrillation (Updating the 2006 guidelines): A report of the American College of Cardiology Foundation/American Heart Association task force on practice guidelines. Circulation 123:104–123, 2011
19. Drew BJ, Ackerman MJ, Funk M, et al: On behalf of the American Heart Association Acute Cardiac Care Committee of the Council of Clinical Cardiology, The Council on Cardiovascular Nursing, and the American College of Cardiology Foundation. Prevention of Torsade de Pointes in hospital settings: A scientific statement from the American Heart Association and the American College of Cardiology Foundation. Circulation 121:1047–1060, 2010
20. McGee WT, Headley JM, Frazier JA: Quick Guide to Cardiopulmonary Care, 2nd ed. Irvine, CA: Edwards Lifesciences LLC, 2009
21. Hardin SR, Kaplow R (eds): Cardiac Surgery Essentials for Critical Care Nursing. Sudbury, MA: Jones and Bartlett Publishers, 2009
22. Woods SL, Froelicher ESS, Motzer SAU, et al (eds): Cardiac Nursing, 6th ed. Philadelphia, PA: Wolters Kluwer/Lippincott Williams & Wilkins, 2010
23. AACN Practice Alert Pulmonary Artery/Central Venous Pressure Monitoring. Revised 12/2009. Retrieved June 16, 2010, from http://www.aacn.org
24. Naomi P, O'Grady NP, Alexander M, et al: Guidelines for the Prevention of Intravascular Catheter-Related Infections. MMWR Recomm Rep 51(RR10):1–26, 2002. Retrieved June 16, 2010, cdc.gov/mmwr/preview/mmwrhtml/rr5110a1.htm
25. Headley JM: Arterial pressure-based technologies: A new trend in cardiac output monitoring. Crit Care Nurs Clin N Am 18:179–187, 2006
26. Marik P, Cavallazzi R, Vasu T, et al: Dynamic changes in arterial waveform derived variables and fluid responsiveness in mechanically ventilated patients: A systematic review of the literature. Crit Care Med 37(9):2642–2647, 2009
27. Oren-Grinberg A: The PiCCO Monitor. Int Anesthesiol Clin 48(1):57–85, 2010
28. Sundar S, Peter Panzica P: LiDCO Systems. Int Anesthesiol Clin 48(1):87–100, 2010
29. Brian Hashim B, Lerner A: The FloTrac System—Measurement of stroke volume and the assessment of dynamic fluid loading. Int Anesthesiol Clin 48(1):45–56, 2010
30. Cannesson M, Aboy A, Hofer CK, et al: Pulse pressure variation: Where are we today? J Clin Monit Comput 25(1):45–56, 2011. DOI:10:1007/s10877-010-9929-1. Published on-line April 2010. Retrieved June 16, 2010
31. Mohammed I, Phillips C: Techniques for determining cardiac output in the intensive care unit. Crit Care Clin 26:355–364, 2010
32. Ravel N, Squara P, Cleman M, et al: Multicenter evaluation of noninvasive cardiac output measurement by bioreactance technology. J Clin Monit Comput 22:113–119, 2008
33. Bayram M, Yancy C: Transthoracic impedance cardiography: A noninvasive method of hemodynamic assessment. Heart Fail Clin 5(2):161–168, 2009
34. Mowatt G, Houston G, Hernández R, et al: Systematic review of the clinical effectiveness and cost-effectiveness of oesophageal Doppler monitoring in critically ill and high risk surgical patients. Health Technol Assess 13(7):1–95, 2009
35. Corley A, Adrian G, Barnett A, et al: Nurse-determined assessment of cardiac output. Comparing a non-invasive cardiac output device and pulmonary artery catheter: A prospective observational study. Int J Nurs Stud 46(10):1291–1297, 2009
36. Nichols D, Nielsen D: Oxygen delivery and consumption: A macrocirculatory perspective. Crit Care Clin 26:239–253, 2010
37. Von Rueden KT, Bolton PA, Vary T: Traumatic shock and multisystem organ dysfunction. In McQuillan K, Makic MB, Whalen E (eds): Trauma Nursing: Resuscitation Through Rehabilitation, 4th ed. Philadelphia, PA: WB Saunders Co., 2009, pp 200–227
38. Maddirala S, Khan A: Optimizing hemodynamic support in septic shock using central and mixed venous oxygen saturation. Crit Care Clin 26:323–333, 2010
39. Pope J, Jones A, Gaieski D, et al: Multicenter study of central venous oxygen saturation ($ScvO_2$) as a predictor of mortality in patients with sepsis. Ann Emerg Med 55(1):40–46, 2010
40. Creteur J. Muscle StO_2 in critically ill patients. Curr Opin Crit Care 14(3):361–366, 2008
41. Nanas S, Gerovasili V, Renieris P, et al: Non-invasive assessment of the microcirculation in critically ill patients. Anaesth Intensive Care 37(5):733–739, 2009

42. Napoli AM, Machan JT, Forcada A, et al: Tissue oxygenation does not predict central venous oxygenation in emergency department patients with severe sepsis and septic shock. Acad Emerg Med 17(4):349–352, 2010
43. Strehlow MC: Early identification of shock in critically ill patients. Emerg Med Clin North Am 28(1):57–66, 2010
44. Levy M, Dellinger RP, Townsend S, et al. The Surviving Sepsis Campaign: Results of an international guideline based performance improvement program targeting severe sepsis. Crit Care Med 38:367–374, 2010
45. Castellanos-Ortega A, Suberviola B, García-Astudillo L, et al: Impact of surviving sepsis campaign protocols on the hospital LOS and mortality in septic shock patients: Results of a 3-year follow-up quasi-experimental study. Crit Care Med 38:1036–1043, 2010

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

18 Patient Management: Cardiovascular System*

Barbara Leeper, Marla J. De Jong, Vicki J. Coombs, Kenneth J. Rempher, Dulce Obias-Manno, and Conrad Gordon

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Compare and contrast commonly used fibrinolytics, anticoagulants, and platelet inhibitors used to affect the thrombotic process.
2. Describe the four classes of antiarrhythmic drugs.
3. Explain how inotropic drugs improve myocardial function.
4. Discuss the rationale for using phosphodiesterase III inhibitor, angiotensin-converting enzyme inhibitor, and vasodilator drugs for patients with cardiovascular disease.
5. Compare and contrast the four major classes of antihyperlipidemic drugs.
6. Compare and contrast the indications and contraindications for percutaneous coronary interventions (PCIs), including percutaneous transluminal coronary angioplasty and intracoronary stenting.
7. Summarize interventions for complications associated with PCI procedures.
8. List potential nursing diagnoses and the interventions for each diagnosis in the patient undergoing an interventional cardiology procedure.
9. Discuss the indications for percutaneous balloon valvuloplasty.
10. Describe the physiological effect of intra-aortic balloon pump (IABP) counterpulsation therapy.
11. Explain indications for and contraindications to IABP therapy.
12. Describe a ventricular assist device and its indications and mechanism of action.
13. Discuss nursing interventions for the patient receiving IABP therapy or ventricular circulatory assistance.
14. Describe the indications, procedure, and nursing management for electrical cardioversion.
15. Explain the indications, procedure, and nursing management for radiofrequency catheter ablation.
16. Describe the indications for a permanent pacemaker.
17. Explain the components, functions, and modes of a pacemaker.
18. Explain complications of pacing and appropriate interventions.
19. Discuss the nursing management of the patient with a pacemaker.
20. Describe the indications, components, and function for an implantable cardioverter–defibrillator (ICD).
21. Explain the nursing management of a patient with an ICD.
22. Describe causes of cardiopulmonary arrest.

(continued)

*Opinions or assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the Department of the Air Force or the Department of Defense.

LEARNING OBJECTIVES *(Continued)*

23. Explain steps of cardiopulmonary resuscitation and the role of each member of the resuscitation team.
24. Explain indications, procedure, and nursing management for defibrillation.
25. Discuss the rationale for using hypothermia as part of cardiopulmonary arrest management.
26. Describe pros and cons of having family members present in a cardiopulmonary arrest situation.

PHARMACOLOGICAL THERAPY

Cardiovascular disease continues to be the leading cause of disease-related death for men and women in the United States. However, recent and remarkable pharmacological advances have reduced morbidity and mortality related to cardiovascular disease.

Critical care nurses are responsible for administering medications that affect the patient's cardiovascular function. Furthermore, nurses continuously evaluate the effects of these drugs and use detailed patient assessment data to guide the titration of these drugs.

This section provides a summary of medications used in critical care settings to treat patients with cardiovascular disease. Critical care nurses need to know drug indications, effects, contraindications, dosage, method of administration, and adverse effects. Additionally, many patients require treatment with numerous cardiovascular drugs; therefore, it is important to consider how drugs interact with other drugs.

▲ Fibrinolytics, Anticoagulants, and Platelet Inhibitors

Atherosclerotic plaque rupture or vascular endothelium damage initiates platelet activation resulting in platelet aggregation and adhesion. This process initiates the production of thrombin through the activation of the coagulation cascade. Thrombin converts fibrinogen to fibrin resulting in the formation of a nonsoluble fibrin thrombus. For further information about the coagulation process, see Chapter 45. An arterial thrombus may transiently or persistently occlude coronary artery blood flow, causing acute coronary syndrome (ACS). For further information about ACS, see Chapter 21. Fibrinolytic, anticoagulant, and platelet inhibitor drugs affect different phases of the thrombotic process.

Fibrinolytics

Fibrinolytic agents are indicated for patients with acute ST-segment elevation myocardial infarction (STEMI). The drugs are not effective and should not be administered to patients without ST-segment elevation or to patients with nonspecific electrocardiogram (ECG) changes.[1,2] Fibrinolytic agents either directly or indirectly convert plasminogen to plasmin, which in turn lyses the thrombus. Early fibrinolytic therapy has been shown to dissolve the thrombus, reestablish coronary blood flow, minimize infarction size, preserve left ventricular (LV) function, and reduce morbidity and mortality.[1] Table 18-1 summarizes commonly used fibrinolytic agents.

The decision to administer fibrinolytic therapy is based on the patient's cardiovascular physical assessment data and ECG. Unless contraindicated (Box 18-1), fibrinolytics should be given to patients with acute STEMI whose symptoms began within the previous 12 hours and who have ST-segment elevation greater than 0.1 mV in two or more contiguous (adjacent) leads, or who have a new-onset left bundle branch block (BBB).[1] Fibrinolytic therapy produces the greatest reduction in mortality when initiated within the first 0 to 4 hours of symptom onset; however, fibrinolytics may be administered up to 12 hours after symptom onset. The goal is to administer a fibrinolytic drug within 30 minutes of the patient's arrival in the emergency department. Patients are at risk for recurrent thrombus formation in the coronary artery; therefore, aspirin and heparin are given to most patients who receive fibrinolytic therapy.[1,3]

Reperfusion may be manifested by decreased or resolved ST-segment elevation, abrupt cessation of chest pain, early peak of serum cardiac biomarkers, and reperfusion dysrhythmias, such as premature ventricular contractions, ventricular tachycardia (VT), accelerated idioventricular rhythm, and atrioventricular (AV) blocks. In contrast, reocclusion may be evidenced by recurrent chest pain and ST-segment elevation, further myocardial ischemia or infarction, lethal dysrhythmias, cardiogenic shock, or death. The most common adverse effects of fibrinolytic therapy are bleeding, intracranial hemorrhage, stroke, and reperfusion dysrhythmias. For more details about the use of fibrinolytic therapy for acute myocardial infarction (AMI), see Chapter 21.

Anticoagulants

Anticoagulants, such as unfractionated heparin, low-molecular-weight heparins (LMWHs), direct thrombin inhibitors, and warfarin (Coumadin) limit further fibrin formation and help prevent thromboembolism.[3]

Unfractionated heparin, the most commonly used anticoagulant drug for acute conditions, is indicated for ACS, venous thromboembolism, percutaneous coronary interventions (PCIs), and patients receiving reteplase or tenecteplase. Heparin prevents clot formation by combining with antithrombin III and inhibiting circulating thrombin. However, unfractionated heparin does not lyse thrombi and is not an optimal anticoagulant because of its narrow therapeutic range, its low bioavailability, the varied anticoagulant response, the requirement for parenteral administration, the need for monitoring the activated partial thromboplastin

Table 18-1 Fibrinolytic Drugs

	Action	Indications	Dose	Half-Life (min)
Alteplase	Binds to fibrin in a thrombus and converts plasminogen to plasmin	Acute ischemic stroke	0.9 mg/kg (max 90 mg) IV over 60 min. 10% of total drug administered via bolus and the remaining dose via infusion.	<5
		Acute massive PE	100 mg infused over 2 h	
		Peripheral thrombus	100 mg infused over 2 min	
		Cathflo Activase only: to restore function of a central venous access device (for patients 30 or more kg, instill 2 mg in 2 mL solution into blocked catheter; dose may be repeated if first dose is not effective after 2 h)		
Reteplase	Catalyzes the cleavage of plasminogen to generate plasmin	AMI	10 U + 10 U IV bolus (each 10 U given over 2 min; second bolus given 30 min after first bolus)	13–16
Tenecteplase	Binds to fibrin and converts plasminogen to plasmin	AMI	Weight-based dose IV over 5 s: >60 kg = 30 mg 60 or more to <70 kg = 35 mg 70 or more to <80 kg = 40 mg 80 or more to <90 kg = 45 mg 90 kg or more = 50 mg	20–24
Streptokinase	Binds with plasminogen to produce a complex that converts plasminogen to plasmin	Acute arterial thrombosis or embolism (slowly instill 250,000 IU in 2 mL solution into occluded cannula and clamp for 2 h)		
		Occluded arteriovenous cannulas		

AMI, acute myocardial infarction; DVT, deep venous thrombosis; IV, intravenous; PE, pulmonary embolism.

time (APTT), the risk for bleeding, possible heparin-induced thrombocytopenia (HIT), and hypersensitivity reactions.

The dosage for unfractionated heparin varies according to its indication and administration route. When used with reteplase (Retavase) or tenecteplase (TNKase), the recommended heparin dosage is 60 U/kg intravenous (IV; maximum 4,000 U) bolus given when the fibrinolytic infusion is started, followed by an infusion of 12 U/kg/h (maximum 1,000 U/h) for ST-segment elevation AMI.[1,4] When IV heparin is administered for non–ST-segment elevation myocardial infarction (NSTEMI) and unstable angina, an initial IV bolus of 60 to 70 U/kg (maximum 5,000 U) followed by a 12 to 15 U/kg/h infusion is recommended.[4] The heparin infusion rate is adjusted to maintain an APTT of 50 to 70 seconds for 48 hours. Protamine sulfate reverses the effects of heparin; however, protamine may cause a life-threatening anaphylactic reaction.

BOX 18-1 Contraindications for Fibrinolytic Therapy

- Active internal bleeding
- Any history of intracranial hemorrhage
- Ischemic stroke within 3 months
- Intracranial neoplasm, arteriovenous malformation, or aneurysm
- Recent intracranial or intraspinal surgery
- Recent closed-head or facial trauma within 3 months
- Suspected aortic dissection
- Severe uncontrolled hypertension
- Bleeding diathesis

LMWHs, such as enoxaparin (Lovenox) and dalteparin (Fragmin), are small fragments derived from unfractionated heparin and are alternatives to heparin for patients with unstable angina, NSTEMI, or deep venous thrombosis. Table 18-2 summarizes these drugs, which inhibit clot formation by blocking Factor Xa and thrombin. Investigators have shown that enoxaparin is superior to unfractionated heparin for patients with STEMI, unstable angina, and NSTEMI.[5–8]

The advantages of LMWHs are their longer half-life, more predictable anticoagulation effect, greater bioavailability, and cost-effectiveness. In addition, LMWHs are administered subcutaneously twice daily and do not require APTT monitoring.

The most common adverse effects of LMWHs include bleeding, thrombocytopenia, elevated aminotransferase levels, and pain, erythema, ecchymosis, or hematoma at the injection site. Because LMWHs have different molecular weight distribution profiles, activities, and plasma clearance rates, they must not be used interchangeably with each other or unfractionated heparin.

Bivalirudin (Angiomax), a direct thrombin inhibitor, may be administered as an alternative to unfractionated heparin in low-risk patients who undergo PCI or patients with HIT.[9] The IV bolus dose for bivalirudin is 0.75 mg/kg, followed by an infusion of 1.75 mg/kg/h for the duration of the PCI procedure. An additional bolus dose of 0.3 mg/kg may be given

Table 18-2 Low-Molecular-Weight Heparins

	Indications	Absolute Bioavailability	Dose for Patients With USA or Non–Q-Wave AMI	Peak Effect	Half-Life
Dalteparin	USA (with aspirin) Non–Q-wave AMI (with aspirin) Prophylaxis of DVT	87%	120 IU/kg (maximum 10,000 IU) SC every 12 h	4 h	3–5 h
Enoxaparin	USA (with aspirin) Non–Q-wave AMI (with aspirin) Prophylaxis and treatment of DVT	100%	1 mg/kg SC every 12 h	3–4.5 h	4.5 h

AMI, acute myocardial infarction; DVT, deep venous thrombosis; SC, subcutaneous; USA, unstable angina.

in 5 minutes depending on results of the activated clotting time (ACT). The infusion may be continued for 4 hours after the PCI procedure.

Lepirudin (Refludan) is a direct thrombin inhibitor used to prevent thromboembolic complications for patients with HIT and thromboembolic disease. After an IV bolus dose of 0.4 mg/kg (maximum 44 mg), an infusion is initiated at 0.15 mg/kg/h (maximum 16.5 mg/h). The infusion rate is adjusted to maintain an APTT ratio of 1.5 to 2.5 times the baseline value. As with other anticoagulants, the major adverse effects of lepirudin are bleeding complications.

Argatroban (Acova) is another direct thrombin inhibitor indicated for prophylaxis or treatment of thrombosis in HIT. The recommended initial dose is 2 mcg/kg/min given as a continuous infusion. Dose adjustments are made to maintain an APTT ratio of 1.5 to 3.0 times the baseline value.

Warfarin, an oral drug used for chronic anticoagulation therapy, interferes with the synthesis of vitamin K–dependent clotting factors, such as factors II, VII, IX, and X. The most common cardiovascular indications for warfarin include post-AMI anticoagulation for high-risk patients, dilated cardiomyopathy, atrial fibrillation (AF), heart failure (HF), venous thromboembolism, mobile mural thrombus, and presence of a prosthetic heart valve. Studies show that warfarin plus aspirin combination therapy is associated with decreased recurrent AMI, stroke, and revascularization but with increased major bleeding.[10] Although high-intensity oral anticoagulation (international normalized ratio [INR]: 3.0 to 4.0) and moderate-intensity anticoagulation (INR: 2.0 to 3.0) plus aspirin lower the risk for untoward outcomes such as AMI, stroke, and death, warfarin treatment regimens are more inconvenient and place patients at greater risk for significant bleeding.[11] Thus, oral anticoagulants are not routinely administered after infarction.

Contraindications for warfarin include uncontrolled hypertension; severe hepatic or renal disease; bleeding diathesis; gastrointestinal (GI) or genitourinary (GU) bleeding; cerebral or dissecting aortic aneurysm; recent central nervous system, eye, or other major surgery; recent trauma; pregnancy (first and third trimesters); pericarditis; pericardial effusion; spinal puncture; and recent diagnostic procedures with the potential for uncontrolled bleeding. Patients must be able and willing to adhere to this somewhat complicated therapy.

Warfarin is usually started at 5 mg daily but should be decreased for the elderly and patients with liver or renal impairment and HF. The dose is titrated according to the patient's INR. Because warfarin levels do not peak for 3 to 4 days, acute anticoagulant therapy is continued until the INR is at the desired level for the patient's condition, usually 2.5 to 3.5. Once the INR is therapeutic on a stable warfarin dose, less frequent INR monitoring is appropriate. Elevated INR levels predispose the patient to bleeding, warfarin's most common adverse effect.[12]

Patient education is an important part of warfarin therapy. Warfarin interacts with numerous drugs and foods; safe treatment depends on the patient's knowledge of therapy.

A new class of anticoagulants called Factor Xa inhibitors is emerging as an alternative to warfarin therapy. These inhibitors seem to have fewer adverse events related to bleeding. Routine frequent blood testing is not required to monitor the effects.

Two recently approved Factor Xa inhibitor agents are rivaroxaban (Xarelto) and dabigatran (Pradaxa). There are no laboratory tests that will evaluate the effectiveness of the Factor Xa inhibitors. If bleeding occurs, laboratory coagulation tests can be used as a qualitative assessment to determine if the medication is contributing to the event. Additionally, there are no reversal agents should severe bleeding occur. Supportive care and control of bleeding are the cornerstones of therapy in this situation.

Platelet Inhibitors

Aspirin, the most widely used platelet inhibitor, inhibits thromboxane A_2, a platelet agonist, and prevents thrombus formation and arterial vasoconstriction. Aspirin is used to decrease mortality for patients with AMI; to reduce incidence of nonfatal AMI and mortality for patients with stable angina, unstable angina, or previous myocardial infarction (MI); and to prevent graft closure after coronary artery bypass graft (CABG) surgery and coronary artery thrombus after PCI. Aspirin is also indicated to reduce the risk for nonfatal stroke and death in patients with a history of ischemic stroke or transient ischemia resulting from platelet emboli. Aspirin is not indicated for primary prevention of AMI. Patients with a history of aspirin intolerance, GI or GU bleeding, peptic ulcers, severe renal or hepatic insufficiency, or bleeding disorders should not take aspirin.

Common aspirin dosages range from 75 to 325 mg daily. Depending on the indication, patients may take aspirin for a few weeks or indefinitely. Unless contraindicated, patients with symptoms of ACS should immediately chew 160 to 325 mg of non–enteric-coated aspirin. A 325-mg aspirin suppository is recommended for patients unable to take oral drugs or for patients with severe nausea, vomiting, or upper GI disorders. Aspirin may cause stomach pain, nausea, vomiting, GI bleeding, subdural or intracranial hemorrhage, thrombocytopenia, coagulopathy, and a prolonged prothrombin time (PT).

The adenosine diphosphate receptor antagonists clopidogrel (Plavix) and ticlopidine (Ticlid) prevent platelet

activation and platelet aggregation, resulting in an irreversible inhibition of platelet function.

- Clopidogrel is indicated to reduce new AMI, new stroke, and vascular death in patients with ACS (both STEMI and NSTEMI) or atherosclerosis as documented by recent stroke, recent AMI, or established peripheral arterial disease (PAD).
- Ticlopidine is used generally for patients who cannot tolerate aspirin.

Guidelines recommend that patients receive a loading dose of clopidogrel at least 6 hours before PCI.[9,10] Patients with a drug-eluting stent (DES) should take clopidogrel for a minimum of 12 months after PCI and in some cases, longer.[9] The dosage for clopidogrel is 75 mg daily with or without food. A loading dose of 300 to 600 mg is often used to achieve a rapid onset of action. The effects of clopidogrel begin immediately; steady-state platelet inhibition is achieved after 3 to 7 days of therapy. Once clopidogrel is discontinued, bleeding times and platelet function normalize within 3 to 7 days. Recently, a subgroup of patients has been identified with altered metabolism, which causes a suboptimal clinical response to clopidogrel. The Federal Drug Administration has identified the need for pharmacogenomic testing to identify patients' altered clopidogrel metabolism.[13] Major adverse effects include bleeding disorders, GI upset, thrombotic thrombocytopenic purpura, and neutropenia. Patients receiving clopidogrel have less GI upset, hemorrhage, and abnormal liver function than patients receiving aspirin. If CABG is planned within 1 week, clopidogrel should be withheld.

With ticlopidine, a dosage of 250 mg is administered twice daily with food to increase absorption and minimize GI irritation. A loading dose of 500 mg can be given to achieve platelet inhibition more quickly. Maximal platelet aggregation inhibition occurs after 4 to 7 days of therapy. Once ticlopidine is discontinued, bleeding times and platelet function normalize within 2 weeks. Major adverse effects include bleeding, neutropenia, agranulocytosis, thrombotic thrombocytopenic purpura, elevated liver aminotransferases, and GI irritation.

Three GP IIb/IIIa inhibitors include abciximab (ReoPro), tirofiban (Aggrastat), and eptifibatide (Integrilin; Table 18-3). These drugs inhibit the GP IIb/IIIa receptor, the final common pathway for platelet aggregation, preventing platelet aggregation and thereby inhibiting thrombus formation. These three drugs are all administered with either enoxaparin or unfractionated heparin. Box 18-2 lists the contraindications to GP IIb/IIIa inhibitors. Adverse effects for this class of drugs include bleeding, thrombocytopenia, stroke, and allergic reactions.

Practice guidelines for PCI intervention recommend a GP IIb/IIIa inhibitor for patients with ACS or NSTEMI who undergo PCI.[14] Guidelines for managing patients with STEMI recommend abciximab as early as possible before PCI.[10]

▲ Antiarrhythmics

Antiarrhythmic drugs are used to restore the heart to a regular rhythm. Many of these medications have serious side effects. Caution must be used to prevent complications. Antiarrhythmics are classified by their effect on the cardiac action potential—whether they block β-receptors; or sodium, potassium, or calcium channels. The action of these drugs is complex; drugs within the same class can work differently, and actions of those in different classes may overlap (Table 18-4). Table 18-5 on page 300 summarizes antiarrhythmic drugs that are commonly used in critical care settings. Refer to Chapter 16 for more specific information regarding the cardiac action potential.

Class I Antiarrhythmic Drugs

Class I antiarrhythmics stabilize the cell membrane by blocking the influx of sodium into the cell. Class I drugs are further categorized on the basis of the action and effects of the specific medications.

Class IA antiarrhythmics include quinidine (Quinate), procainamide (Pronestyl), and disopyramide (Norpace). These drugs are effective for treating atrial rhythms in the short term but may cause life-threatening dysrhythmias by prolonging the QTc interval. They also interact with other drugs commonly used for cardiovascular disease.

Class IB antiarrhythmics are lidocaine and mexiletine (Mexitil). Lidocaine, a less effective but acceptable alternative to procainamide for ventricular dysrhythmias, is no longer used routinely to prevent ventricular dysrhythmias.

Class IC antiarrhythmics are flecainide (Tambocor) and propafenone (Rythmol). Because these drugs are prodysrhythmic and may increase mortality, they are not commonly prescribed.

In general, research data do not support the effectiveness of class I antiarrhythmics. The current trend is to treat ventricular dysrhythmias with class II and class III antiarrhythmics, cardioversion, ablative techniques, and implantable cardioverter–defibrillators (ICDs).[15]

Class II Antiarrhythmic Drugs

β-Adrenergic blockers are class II drugs that interfere with sympathetic nervous system stimulation, contributing to decreased heart rate, prolonged AV node conduction, decreased myocardial contractility, and decreased myocardial oxygen demand. This class of drugs has a broad spectrum of activity and an established safety record and is currently the best class of antiarrhythmics for *general* use.[16] This is the only class of medications shown to reduce the incidence of sudden cardiac death following AMI and HF.

β-Blockers are categorized as cardioselective (inhibition of β_1 receptors) or nonselective (inhibition of β_1 and β_2 receptors). Inhibition of β_1 receptors causes decreased heart rate, slowed conduction through the AV node, and depressed cardiac function. Inhibition of β_2 receptors causes bronchoconstriction, vasoconstriction, and decreased glycogenolysis. Table 18-6 on page 302 indicates the β activity of selected β-blockers.

Unless contraindicated, β-blockers should be given indefinitely to all patients with a history of AMI, ACS, or LV dysfunction with or without symptoms of HF.[14] Other indications include tachydysrhythmias, unstable angina, hypertension, and HF. Acebutolol, esmolol, propranolol, and sotalol are approved to treat dysrhythmias. All β-blockers, except esmolol and sotalol, are indicated for hypertension.

Unless contraindicated, β-blockers should be a part of early treatment for patients with ACSs.[17,18] Metoprolol

Table 18-3 Glycoprotein IIb/IIIa Inhibitors

	Indications	Dose	Concurrent Aspirin and Heparin Therapy	Half-Life
Abciximab	Adjunct to PCI USA that does not respond to conventional therapy and when PCI is planned within 24 h	PCI: IV bolus of 0.25 mg/kg 10–60 min before PCI; then continuous IV infusion of 0.125 mcg/kg/min (maximum 10 mcg/min) for 12 h USA with planned PCI: IV bolus of 0.25 mg/kg; then 10 mcg/min IV infusion for 18–24 h, concluding 1 h after PCI	Yes	First phase, <10 min; second phase, 30 min; remains in circulation up to 10 d in a platelet-bound state
Eptifibatide	ACS: Non–Q-wave AMI or USA, including patients who are managed medically or with PCI PCI	ACS: 180 mcg/kg bolus IV, then 2 mcg/kg/min infusion up to 72 h, discharge, or CABG; if PCI performed, continue IV infusion up to discharge or for up to 18–24 h post-PCI (whichever comes first), allowing up to 96 h of therapy; for patients with a creatinine between 2 and 4 mg/dL, reduce infusion to 1 mcg/kg/min PCI: 180 mcg/kg IV bolus immediately before PCI; then 2 mcg/kg/min IV infusion and a second 180 mcg/kg bolus IV 10 min after the first bolus; continue infusion until discharge or for up to 18–24 h; a minimum of 12 h of infusion is recommended; for patients with a creatinine level between 2 and 4 mg/dL, 180 mcg/kg IV bolus immediately before PCI, then 1 mcg/kg/min IV infusion, and a second 180 mcg/kg bolus IV 10 min after the first bolus	Yes	2.5 h; platelet function returns to normal approximately 4 h after stopping the infusion
Tirofiban	ACS: Non–Q-wave AMI or USA, including patients who are managed medically or with PCI	0.4 mcg/kg/min IV for 30 min, then 0.1 mcg/kg/min IV infusion through angiography or for 12–24 h after PCI; for patients without signs of refractory ischemia who do not proceed to angiography and angioplasty, continue infusion for at least 48 h; for patients with severe renal insufficiency, give half the usual rate of infusion.	Yes	1.4–2.2 h; platelet function returns to near baseline 4–8 h after stopping the infusion

ACS, acute coronary syndrome; CABG, coronary artery bypass grafting; IV, intravenous; PCI, percutaneous coronary intervention; USA, unstable angina.

(Lopressor), atenolol (Tenormin), timolol, and nadolol (Corgard) are approved for angina, whereas metoprolol and atenolol are indicated as first-line drugs for AMI. The first dose may be given intravenously, and successive doses are usually given orally.

β-Blockers are contraindicated in patients with severe asthma or bronchospasm, severe chronic obstructive pulmonary disease, cardiogenic shock, severe LV failure, bradycardia (<60 beats/min), or second- and third-degree heart block. Cardioselective β-blockers are sometimes used with caution for patients with pulmonary disease. It is important to remember that cardioselective drugs lose their selectivity at higher doses.

Adverse effects of β-blockers include bradycardia, heart block, hypotension, HF, bronchospasm, cold extremities, insomnia, fatigue, decreased libido, and depression. Some patients who experience these adverse effects may respond better to a different β-blocker.

Class III Antiarrhythmic Drugs

Class III antiarrhythmic drugs include amiodarone (Cordarone), sotalol (Betapace), ibutilide (Corvert), and dofetilide (Tikosyn). It is important to know each drug's unique properties because individual agents contain unique properties not shared by other class III drugs.

Amiodarone is indicated for treating VT as well as AF and flutter. The advanced cardiac life support (ACLS) algorithms include amiodarone as a first-line option for treating ventricular fibrillation (VF), pulseless VT, wide-complex tachycardia, and AF associated with Wolff-Parkinson-White (WPW)

BOX 18-2 Contraindications for Glycoprotein IIb/IIIa Inhibitors

- Internal bleeding
- Bleeding diathesis within 30 days
- Intracranial neoplasm, arteriovenous malformation, or aneurysm
- Stroke within 30 days or any hemorrhagic stroke
- Thrombocytopenia with prior exposure to tirofiban
- Aortic dissection
- Major surgery or severe trauma within the previous month
- Severe hypertension
- Pericarditis (tirofiban)
- Concurrent use of another glycoprotein IIb/IIIa inhibitor
- Dependence on dialysis or serum creatinine 4.0 mg/dL or more (eptifibatide)

syndrome.[19] Limitations of amiodarone include its variable onset of action, long half-life, intolerable adverse effects, dangerous drug interactions, and life-threatening complications associated with chronic therapy.[15,16]

Ibutilide (Corvert) and dofetilide (Tikosyn) are class III drugs that are indicated for the pharmacological conversion of AF and atrial flutter. Ibutilide inhibits potassium current and enhances sodium current, prolonging repolarization. Dofetilide blocks the rapid potassium current channel, prolonging the action potential duration and refractory period. These drugs may cause a prolonged QT interval and torsades de pointes. Therefore, close monitoring of the QTC is required upon initiation of the medication. These medications have fewer systemic adverse effects than amiodarone and sotalol.

Class IV Antiarrhythmic Drugs

The class IV calcium channel blocker antiarrhythmics, verapamil (Calan) and diltiazem (Cardizem), decrease automaticity of the sinoatrial (SA) and AV nodes, slow conduction, and prolong the AV nodal refractory period. These agents have negative inotropic and peripheral vasodilation effects. In addition, they have antiplatelet and anti-ischemic effects. Calcium channel blockers are primarily indicated for angina, hypertension, and supraventricular tachycardia (SVT). Verapamil and diltiazem are contraindicated for usual forms of VT, severe sinus bradycardia, sick sinus syndrome, WPW syndrome with AF, digoxin toxicity, hypotension, HF, AV conduction defects, and severe aortic stenosis, and they are not standard therapies for AMI. Adverse effects include hypotension, AV block, bradycardia, headache, dizziness, peripheral edema, nausea, constipation, and flushing.

Calcium channel blockers do not decrease mortality after AMI, and in some cases, these medications may be harmful. Calcium antagonists, in general, should be used only in the setting of AMI when β-blockers are contraindicated or maximal dosage has been reached without effect.[20]

Unclassified Antiarrhythmic Drugs

Adenosine is a first-line antiarrhythmic that effectively converts narrow-complex paroxysmal supraventricular tachycardia (PSVT) to normal sinus rhythm by slowing conduction through the AV node. This agent is effective in terminating dysrhythmias due to reentry involving the SA and AV nodes; however, it does not convert AF, atrial flutter, or VT to sinus rhythm. It is also used to differentiate between VT and SVT, treat rare forms of idiopathic VT, and reveal latent pre-excitation in patients with suspected WPW syndrome.[16] The dose is 6 mg rapid IV bolus followed by a rapid saline flush. If the 6-mg dose is ineffective, a dose of 12 mg may be administered twice. The half-life of adenosine is less than 10 seconds; therefore, adverse effects are short-lived.

Magnesium sulfate is the drug of choice for treating torsades de pointes. Magnesium is also used for refractory VT and VF, as well as for life-threatening dysrhythmias from digitalis toxicity. Its mechanism of action is unclear; however, it has calcium channel blocking properties and inhibits sodium and potassium channels. The dose for patients in

Table 18-4 Classification of Antiarrhythmic Medications

Class	Action	Medication Examples
IA	Inhibits fast sodium channel, decreases automaticity, depresses phase 0, and prolongs the action potential duration	Quinidine Procainamide Disopyramide
IB	Inhibits fast sodium channel, depresses phase 0 slightly, and shortens action potential duration	Lidocaine Mexiletine
IC	Inhibits fast sodium channel, depresses phase 0 markedly, slows His-Purkinje conduction profoundly leading to a prolonged QRS duration	Flecainide Moricizine (plus IA and IB effects) Propafenone
II	Depresses phase 4 depolarization, blocks sympathetic stimulation of the conduction system	Esmolol Propranolol Sotalol (plus Class III effects) Acebutolol
III	Blocks potassium channel, prolongs phase 3 repolarization, prolongs action potential duration	Amiodarone Sotalol Ibutilide Dofetilide
IV	Inhibits inward calcium channel, depresses phase 4 depolarization, lengthens repolarization in phases 1 and 2	Verapamil Diltiazem

Table 18-5 Selected Antiarrhythmic Medications

Drug	Antiarrhythmic Indications	Antiarrhythmic Dose	Route	Effect on ECG	Major Adverse Effects
Procainamide	VT, VF; SVTs including WPW syndrome, AF, atrial flutter	IV: 20 mg/min IV infusion (maximum total dose 17 mg/kg). Then infusion of 1–4 mg/min to maintain therapeutic drug level PO: up to 50 mg/kg/d given in divided doses every 3 h to maintain therapeutic drug level	IV, PO	→ QRS → QTI	Hypotension with IV use, asystole, ventricular fibrillation, positive antinuclear antibody test, lupus syndrome, rash, fever, heart block, torsades de pointes, headache, agranulocytosis
Lidocaine	VT, VF	1.0–1.5 mg/kg IV bolus; may repeat 0.5–0.75 mg/kg IV every 5–10 min for total of 3 mg/kg. Then infusion of 1–4 mg/min	IV; ETT if no patent IV (2–4 mg/kg)	None	Bradycardia, blurred vision, hypotension, tremors, dizziness, tinnitus, convulsions, mental status changes
Flecainide	AF and PSVTs (AVNRT, AVRT) in patients without structural heart disease; life-threatening ventricular dysrhythmias (VT)	100–200 mg PO every 12 h	PO	→ PRI → QRS 0/→ QTI	Ventricular dysrhythmias, dizziness, dyspnea, headache, fatigue, nausea, palpitations
Esmolol	SVT including AF and atrial flutter; noncompensatory ST	500 mcg/kg/min IV loading dose for 1 min. Then 50 mcg/kg/min infusion for 4 min. Repeat loading dose every 5 min and increase infusion by 50 mcg/kg/min increments until desired therapeutic effect or maximum of 300 mcg/kg/min.	IV	↓ HR 0/→ PRI 0/← QTI	Hypotension, nausea, diaphoresis, dizziness, headache, weakness, somnolence, heart block, bronchospasm, thrombophlebitis from extravasation
Sotalol	Life-threatening ventricular dysrhythmias (VT, VF); maintenance of NSR in patients with symptomatic AF or atrial flutter who are currently in NSR	80 mg PO twice a day; may be increased to 240–640 mg/d given in two to three divided doses	PO	↓ HR → PRI 0/→ QTI	Bradycardia, AV block, dizziness, HF, bronchospasm, gastric pain
Ibutilide	AF, atrial flutter	1 mg IV infusion over 10 min (<60 kg, 0.01 mg/kg). May repeat either dosage in 10 min	IV	→ QTI	Hypotension, torsades de pointes, VT, BBB, bradycardia, nausea
Dofetilide	AF, atrial flutter; maintenance of NSR after conversion	125–500 mcg PO twice a day depending on creatinine clearance	PO	→ QTI	Torsades de pointes, bradycardia
Amiodarone	Recurrent VF or hemodynamically unstable VT in patients refractory to other drugs; unlabeled uses: AF and maintenance of NSR; SVTs including WPW syndrome; rate control in AF or atrial flutter when other therapies ineffective	IV: Loading infusion of 150 mg over 10 min; 360 mg over next 6 h; 540 mg over next 18 h. May follow with maintenance infusion of 0.5 mg/min. For breakthrough VF or VT, supplemental infusions of 150 mg IV over 10 min PO: Loading dose of 800–1,600 mg daily for 1–3 wk. Then 600–800 mg daily for 1 mo. Maintenance dose 100–400 mg/d	IV PO	→ PRI → QTI	Heart block, cardiac arrest, bradycardia, hypotension, VT, pneumonitis, liver disease, hypothyroidism or hyperthyroidism, photosensitivity, solar dermatitis, blue discoloration of skin, malaise, paresthesias, nausea, vomiting, constipation, visual disturbances, anorexia

Verapamil	PSVTs including WPW syndrome; ventricular rate control in AF, atrial flutter	IV: 5–10 mg over 2 min; may give 10 mg 30 min after first dose PO: 240–480 mg/d in 3–4 divided doses	IV PO	↓ HR → PRI	Hypotension, heart block, HF, bradycardia, headache, dizziness, edema, nausea, constipation
Diltiazem	Ventricular rate control in AF, atrial flutter; PSVT including WPW syndrome	IV: 0.25 mg/kg over 2 min; may give 0.35 mg/kg after 15 min. May follow with an infusion of 5–15 mg/h for up to 24 h	IV for antiarrhythmic indications	↓/0 HR → PRI	Bradycardia, heart block, edema, hypotension, nausea, dizziness, flushing, headache, fatigue
Adenosine	PSVT including WPW syndrome; idiopathic VT; used diagnostically to evaluate VT, SVT, latent pre-excitation	6 mg IV over 1–2 s followed by rapid saline flush. After 1–2 min, may give 12 mg. A second 12-mg dose may be given in 1–2 min if needed.	IV	→ PRI	Facial flushing, light-headedness, headache, bradycardia, dyspnea, heart block, asystole, chest pain, nausea
Atropine	Symptomatic sinus bradycardia, AV block, asystole, bradycardic PEA	Asystole or PEA: 1 mg IV push; repeat every 3–5 min to maximum of 0.04 mg/kg; bradycardia: 0.5–1.0 mg IV every 3–5 min to maximum of 0.04 mg/kg	IV; ETT if not patent IV (1–2 mg)	↑ HR	Palpitations, tachycardia, blurred vision, dry mouth, altered taste, nausea, urinary retention
Digoxin	Ventricular rate control in AF	IV: loading dose of 0.4–0.6 mg with additional doses of 0.1–0.3 mg every 4–8 h. Maintenance dose of 0.125–0.5 mg/d PO: loading dose of 0.5–0.7 mg with additional doses of 0.125–0.375 mg every 6–8 h. Maintenance dose of 0.125–0.5 mg/d	IV PO	↓ HR → PRI ← QTI	Heart block, bradycardia, weakness; toxicity: dysrhythmias, anorexia, nausea, vomiting, headache, fatigue, depression, confusion, hallucination

AF, atrial fibrillation; AV, atrioventricular; BBB, bundle branch block; ECG, electrocardiogram; ETT, endotracheal tube; HF, heart failure; HR, heart rate; IV, intravenous; NSR, normal sinus rhythm; PEA, pulseless electrical activity; PO, oral; PSVT, paroxysmal supraventricular tachycardia; ST, sinus tachycardia; SVT, supraventricular tachycardia; VF, ventricular fibrillation; VT, ventricular tachycardia; WPW, Wolff-Parkinson-White; ↑, increased; ↓, decreased; →, prolonged; ←, shortened; 0, little or no effect; AVNRT, atrioventricular nodal reentrant tachycardia; AVRT, atrioventricular reentrant tachycardia.

Table 18-6 Selected β-Blockers

Medication	Cardioselective	Nonselective
Acebutolol	X	
Atenolol	X	
Betaxolol	X	
Bisoprolol	X	
Carvedilol		X
Esmolol	X	
Labetalol		X
Metoprolol	X	
Nadolol		X
Pindolol		X
Propranolol		X
Sotalol		X
Timolol		X

cardiac arrest is 1 to 2 g diluted in 10 mL of D_5W given by IV push. Adverse effects include hypotension, nausea, depressed reflexes, and flushing.

Atropine, a parasympatholytic agent, is a first-line drug used to treat symptomatic bradycardia and slowed conduction at the AV node. It is also indicated for asystole or bradycardic pulseless electrical activity (PEA). Atropine reduces the effects of vagal stimulation, thereby increasing heart rate and improving cardiac function. It is important not to increase the heart rate excessively in patients with ischemic heart disease because this may increase myocardial oxygen consumption and worsen ischemia.

Digoxin (Lanoxin) is a mild positive inotrope with antidysrhythmic and bradycardic actions. It inhibits the sodium–potassium pump, causing a rise in intracellular sodium. This rise promotes calcium influx and ultimately enhances myocardial contractility. Digoxin also activates the parasympathetic system, decreasing heart rate and increasing AV nodal inhibition. Digoxin is primarily indicated for patients with both HF and chronic AF.[21] In addition, digoxin may be used to control a rapid ventricular rate associated with non-pre-excitation AF or atrial flutter and in combination with verapamil, diltiazem, or β-blockers for patients without HF.[19,21] Digoxin is not currently used for paroxysmal AF, acute SVTs, or acute LV failure, or as part of inotropic therapy regimens.[21]

The doses and therapeutic blood levels for digoxin are controversial. No longer is it common to administer loading doses.[21] Most patients on digoxin benefit from a low dose, which also reduces the incidence of toxicity.[21] Toxicity is a common occurrence and is frequently associated with serious dysrhythmias. Routine doses are individualized based on the patient's diagnosis, symptoms, underlying disease processes, age, response to therapy, and blood levels. Levels of 0.5 to 1.0 ng/mL are recommended for patients with HF, and levels of 0.8 to 2 ng/mL for those with dysrhythmias.

Signs and symptoms of digitalis toxicity include palpitations, syncope, dysrhythmias, elevated digoxin level, anorexia, vomiting, diarrhea, nausea, fatigue, confusion, insomnia, headache, depression, vertigo, facial pain, and colored or blurred vision. Digitalis levels may be increased by the concurrent use of quinidine, verapamil, amiodarone, captopril, diltiazem, esmolol (Brevibloc), propafenone (Rythmol), indomethacin (Indocin), quinine, or ibuprofen (Motrin). Finally, hypokalemia, hypomagnesemia, and hypothyroidism may predispose patients to digitalis toxicity. Blood serum levels are analyzed if toxicity is suspected.

▲ Inotropes

Cardiovascular function is regulated by two divisions of the autonomic nervous system, the sympathetic and parasympathetic systems. Refer to Chapter 32 to review this information.

Adrenoreceptor stimulation leads to a variety of effects; therefore, it is important to understand which receptors each drug stimulates (Table 18-7).

Inotropic drugs are used to increase the force of myocardial contraction and cardiac output. Inotropic drugs include sympathomimetics, such as dopamine (Intropin), dobutamine (Dobutrex), epinephrine, isoproterenol (Isuprel), and norepinephrine, and the phosphodiesterase inhibitor milrinone (Primacor). These drugs are commonly given to patients with impaired myocardial contractility or cardiogenic shock. Enhanced ventricular contraction increases stroke volume, cardiac output, blood pressure, and coronary artery perfusion. As the ventricles empty more completely, ventricular filling

Table 18-7 Adrenergic Receptors Affecting Cardiovascular Function

Receptor	Location	Effects of Stimulation
β_1	Heart	Positive inotropic (increases contractility) and chronotropic action (increases rate)
β_2	Bronchial smooth muscle	Bronchodilation
	Vascular smooth muscle	Vasodilation
	AV node	Positive dromotropic action (increases conduction velocity)
α_1	Vascular smooth muscle	Vasoconstriction
	Heart	Weak positive inotropic and chronotropic actions
α_2	Presynaptic sympathetic nerve endings	Inhibition of norepinephrine release
Dopaminergic	Kidney and splanchnic vessels	Renal and splanchnic vessel vasodilation

pressures, preload, and pulmonary congestion are decreased. However, as contractility and heart rate increase, myocardial oxygen demand also increases. Myocardial ischemia can occur if a myocardial oxygen supply–demand mismatch develops. The nurse must closely monitor the patient for evidence of ischemia, angina, and onset of dysrhythmias.

Dopamine

Dopamine, the most widely used inotropic drug, is administered to patients with conditions that cause hypotension, decreased cardiac output, and oliguria. Dopamine directly stimulates dopaminergic, β-adrenergic, and α-adrenergic receptors and promotes release of norepinephrine from sympathetic nerve terminals. Dopamine is given by continuous IV infusion, and its dose is titrated to achieve the desired effect. Increased myocardial contractility results from dosages of 3 to 10 mcg/kg/min. Higher dosages predominantly cause vasoconstriction and increased blood pressure. Dopamine is usually given through a central line to enhance its distribution and to avoid extravasation, which may cause local vasoconstriction and tissue necrosis. Adverse effects include tachycardia, palpitations, dysrhythmias, angina, headache, nausea, vomiting, and hypertension.

Dobutamine

Dobutamine acts on β_1 receptors and increases myocardial contractility. Dobutamine also stimulates the β_2 receptors as well as the α_1 receptors. The result is slight vasodilation. Dobutamine is used after cardiac surgery, during some cardiac diagnostic stress procedures, and for patients with HF, shock, or other conditions that cause poor cardiac contractility or a low cardiac output. The dosage for dobutamine is 2 to 20 mcg/kg/min by continuous IV infusion. Adverse effects include tachycardia, dysrhythmias, blood pressure fluctuations, headache, and nausea.

Epinephrine

Epinephrine stimulates α_1, β_1, and β_2 receptors and is given for a variety of indications, including cardiac arrest, symptomatic bradycardia, severe hypotension, anaphylaxis, and shock. In the intensive care unit (ICU), epinephrine is given by continuous IV infusion through a central line, as an IV bolus, or through an endotracheal tube. Continuous IV dosages of 1 to 2 mcg/min stimulate β_1 receptors to increase cardiac output by increasing heart rate and myocardial contractility. At higher dosages, epinephrine stimulates α receptors, causing profound vasoconstriction, increased blood pressure and systemic vascular resistance (SVR), and decreased renal and splanchnic perfusion. Epinephrine may cause dysrhythmias, tachycardia, cerebral hemorrhage, pulmonary edema, headache, dizziness, nervousness, myocardial ischemia, and angina.

Vasopressin is now used as an alternative to epinephrine for treating shock-refractory VF, asystole, or PEA.[19] Vasopressin promotes smooth muscle contraction and increases peripheral vascular resistance. The dosage for patients with cardiac arrest is 40 U given by IV push. The drug may also be given as an infusion. Adverse effects include dysrhythmias, myocardial ischemia, angina, MI, tremors, vertigo, sweating, and water intoxication.

Isoproterenol

Isoproterenol (Isuprel) stimulates β_1 and β_2 receptors to increase myocardial contractility, cardiac output, heart rate, and blood pressure. Currently, isoproterenol is used mainly to increase heart rate after cardiac transplantation. Other indications include refractory torsades de pointes, β-blocker overdose, and symptomatic bradycardia when an external pacemaker is not available. The IV dosage is 0.5 to 10 mcg/min by continuous infusion. Isoproterenol causes a variety of adverse effects, including dysrhythmias, tachycardia, palpitations, myocardial ischemia, hypotension, pulmonary edema, bronchospasm, headache, nausea, vomiting, and sweating.

Norepinephrine

Norepinephrine (Levophed) primarily affects α_1 receptors, causing peripheral vasoconstriction, increased blood pressure, and increased SVR. The increased SVR may actually increase myocardial oxygen demand and work, thus decreasing cardiac output. Norepinephrine is used for patients with cardiogenic shock and significant hypotension accompanied by a low SVR. The dosage is 2 to 12 mcg/min by continuous IV infusion. Adverse effects include tachycardia, bradycardia, dysrhythmias, headache, hypertension, and tissue necrosis from extravasation.

▲ Phosphodiesterase III Inhibitor

The phosphodiesterase III inhibitor milrinone (Primacor) increases contractility, venous vasodilation, and peripheral arterial vasodilation by inhibiting an enzyme that breaks down cyclic adenosine monophosphate. There is a reduction of ventricular filling pressures and a slight reduction of arterial pressure; however, there is minimal affect on the heart rate.

Milrinone is frequently used for the short-term treatment of acute HF. Some patients with HF may be on a milrinone infusion long term at home. The IV bolus dose of 50 mcg/kg is given over 10 minutes and is followed by a maintenance IV infusion of 0.375 to 0.75 mcg/kg/min. Patients who receive milrinone may experience ventricular dysrhythmias, hypotension, headache, bronchospasm, and thrombocytopenia.

▲ Vasodilators

Vasodilators decrease preload and afterload. Preload is the distending force that stretches the ventricular muscle at the end of filling. The greater the stretch, the better the contraction. However, if the cells are overstretched, contractile force decreases. Afterload is the force against which the heart has to work to eject its contents. If afterload is too low, blood pressure and tissue perfusion may be low. If afterload is too high, the heart has to work harder.

Nitrates

Patients with myocardial ischemia or infarction may have an increased preload and afterload, which further strains their

hearts. Nitrates cause peripheral vasodilation, which in turn decreases venous return to the heart and reduces preload. These drugs promote coronary artery vasodilation, improve collateral blood flow, reduce platelet aggregation, enhance perfusion to ischemic myocardium, and decrease myocardial oxygen demand, thus reducing ischemia, chest pain, and infarct size. Nitrates reduce blood pressure and previously elevated pulmonary vascular resistance, SVR, and central venous and pulmonary artery occlusion wedge pressures. At high doses, nitrates reduce afterload by arterial vasodilator effects.

Nitrates are indicated for unstable angina; large anterior AMI; AMI associated with acute and chronic HF, acute pulmonary edema, or hypertension; angina unresponsive to other therapies; and prophylaxis of effort angina. Nitroglycerin has been shown to raise the threshold for VF in the setting of AMI. Contraindications to IV nitrates include, but are not limited to, hypotension, uncorrected hypovolemia, hypertrophic obstructive cardiomyopathy, and pericardial tamponade. When a right ventricular (RV) AMI is suspected, nitrates are used with extreme caution because these patients require an adequate venous return to maintain cardiac output and blood pressure. Patients should not receive nitrates for 24 hours after sildenafil (Viagra), vardenafil (Levitra), or tadalafil (Cialis) use because the resulting drug interactions predispose patients to life-threatening hypotension.

Nitrates are available in a variety of dosage forms. In the ICU, nitrates are often given by the IV, sublingual, or topical routes. An IV nitroglycerin drip is initiated at 5 to 20 mcg/min and increased every 5 to 15 minutes, up to 200 mcg/min, to achieve the desired effects. When used to treat or prevent angina, a 0.3- to 0.6-mg tablet is placed under the patient's tongue and may be repeated twice at 5-minute intervals. The usual dose for nitroglycerin ointment is 1 to 2 inches every 8 hours; however, treatment is often initiated with 0.5 inch and increased gradually to achieve the desired effects.

The adverse effects of nitrates include headache, hypotension, syncope, and tachycardia. Tolerance may develop to the antianginal, hemodynamic, and antiplatelet effects of nitrates, especially with continuous or high-dose therapy; however, dosing regimens that allow for nitrate-free intervals for at least 12 hours may prevent this occurrence.[4]

Sodium Nitroprusside

Nitroprusside (Nipride) is a potent arterial and venous vasodilator that is used to treat severe LV HF, hypertension after CABG, hypertensive crisis, and dissecting aneurysm. Nitroprusside decreases SVR and increases cardiac output. The usual IV infusion dosage is 0.5 to 10 mcg/kg/min; however, to prevent cyanide toxicity, the maximal dose should not be given for longer than 10 minutes. The dose is titrated to effect; if blood pressure does not respond after 10 minutes, the drug is discontinued. Because nitroprusside is sensitive to light, the infusion bag must be covered with an opaque material to prevent the drug's degradation. Adverse effects include hypotension, myocardial ischemia, nausea, vomiting, abdominal pain, and cyanide toxicity.

Nesiritide

Nesiritide (Natrecor), a recombinant form of human B-type natriuretic peptide, is identical to the hormone produced by the left ventricle in response to volume overload and increased wall stress. A venous and arterial vasodilator, nesiritide reduces preload and afterload and increases cardiac output without increasing heart rate. Nesiritide is indicated for acutely decompensated HF with dyspnea at rest or with minimal activity and is often used with IV diuretics. The bolus dose is 2 mcg/kg/min, followed by an IV infusion of 0.01 to 0.03 mcg/kg/min. Contraindications include cardiogenic or distributive shock, valvular stenosis, constrictive pericarditis, and restrictive or obstructive cardiomyopathy. Adverse effects include hypotension, bradycardia, ventricular dysrhythmias, angina, dizziness, and apnea. Nesiritide should not be infused through heparin-coated catheters or IVs containing furosemide (Lasix), insulin, hydralazine (apresoline), enalapril (Vasotec), and bumetanide (Bumex).

▲ Angiotensin-Converting Enzyme Inhibitors

Angiotensin-converting enzyme (ACE) inhibitors are indicated to treat HF, hypertension, AMI with or without LV dysfunction or failure, and asymptomatic LV dysfunction. They are also used to decrease morbidity and mortality for patients at high risk for AMI, stroke, or cardiovascular death. Unless contraindicated, patients with a STEMI with anterior infarction, pulmonary congestion, or LV ejection fraction (EF) less than 40% should receive an ACE inhibitor within 24 hours of admission to the hospital.[1]

The ACE inhibitors block the conversion of angiotensin I to the potent vasoconstrictor angiotensin II, reduce aldosterone synthesis, and may promote fibrinolysis.[22–29] As a result, these agents mitigate LV remodeling, increase cardiac output, and decrease sodium retention, blood pressure, central venous pressure, SVR, pulmonary vascular resistance, and pulmonary capillary wedge pressure. Numerous research trials conducted in the late 1980s through the early 2000s showed that ACE inhibitors prevent HF, prevent hospitalization due to HF, and decrease mortality.[23–30]

All ACE inhibitors are contraindicated in pregnancy, angioedema, bilateral renal artery stenosis, and pre-existing hypotension. They should be used with caution for patients with renal failure or hyperkalemia. Patients with impaired renal function, hypotension, or concurrent diuretic use should receive a lower dosage. Adverse effects of ACE inhibitors include hypotension, dizziness, angioedema, cough, headache, fatigue, nausea, vomiting, diarrhea, hyperkalemia, and impaired renal function.

▲ Antihyperlipidemics

Cholesterol reduction is an important part of therapy for patients with cardiovascular disease. Patients are encouraged to modify their diet and lifestyle before reverting to drug therapy. The pharmacological management of hyperlipidemia decreases morbidity and mortality from coronary heart disease (CHD).[30–32] Recent evidence suggests

that intensive statin therapy can regress coronary atherosclerosis. The primary target of antihyperlipidemic therapy is low-density lipoprotein (LDL) cholesterol.[32] The goal LDL cholesterol level is (1) less than 160 mg/dL for patients with zero or one risk factor, (2) less than 130 mg/dL for patients with two or more risk factors, and (3) less than 100 mg/dL for patients with documented CHD or diabetes, stroke, and or PAD.[32] An LDL cholesterol goal of less than 70 mg/dL may be preferred for patients at highest risk.

A total cholesterol level of less than 200 mg/dL is desirable, and a high-density lipoprotein (HDL) cholesterol less than 40 mg/dL is considered low. Drug therapy with LDL cholesterol–lowering drugs is recommended for patients with (1) zero or one risk factor and an LDL cholesterol level greater than or equal to 190 mg/dL, (2) two or more risk factors and an LDL cholesterol level greater than or equal to 130 mg/dL, and (3) documented CHD or CHD risk equivalents and an LDL cholesterol level greater than or equal to 100 mg/dL.[32] Drug therapy is also appropriate when triglycerides are 200 mg/dL or greater.[32] Finally, patients with borderline-high triglycerides (150 to 199 mg/dL) and CHD or CHD risk equivalents may receive drugs to raise the HDL cholesterol.

The four major classes of antihyperlipidemic drugs are the following:

- Hydroxymethylglutaryl coenzyme-A reductase inhibitors (statins) decrease total and LDL cholesterol, decrease triglycerides, and increase HDL cholesterol by inhibiting the rate-limiting enzyme that promotes cholesterol biosynthesis.
- Nicotinic acid inhibits lipolysis in adipose tissue and inhibits hepatic production of very-low-density lipoprotein (VLDL) cholesterol, thus decreasing cholesterol, triglycerides, VLDL cholesterol, and LDL cholesterol, as well as increasing HDL cholesterol.
- The bile acid sequestrants bind bile acids in the intestine and form an insoluble complex that is excreted in the feces. Because bile acids are not absorbed, there is ultimately an increased hepatic synthesis of bile acids from cholesterol that may be evident by a slightly increased triglyceride level. However, plasma total and LDL cholesterol actually decrease owing to an increased clearance rate.
- The fibrates inhibit peripheral lipolysis and decrease the hepatic extraction of free fatty acids, which reduces triglyceride production. These agents decrease total cholesterol, triglycerides, and VLDL cholesterol, and they increase HDL-C.

Ezetimibe (Zetia) is a cholesterol absorption inhibitor that selectively inhibits the intestinal absorption of cholesterol. It is used more effectively when combined with a statin to further reduce LDL cholesterol.

Combination drugs have become increasingly popular; therefore, it is essential that nurses know which drugs may be contained within one tablet or capsule. For example, currently available combinations include aspirin and pravastatin (Pravachol), lovastatin (Mevacor) and extended-release niacin, and ezetimibe (Zetia) and simvastatin (Zocor). Future "polypills" may include one pill that contains a statin, an ACE inhibitor, and aspirin.

PERCUTANEOUS CORONARY INTERVENTIONS AND PERCUTANEOUS BALLOON VALVULOPLASTY

▲ Percutaneous Coronary Interventions

Historical Background

Cardiovascular disease is the number one cause of death in the United States. The American Heart Association (AHA) estimates that 81,100,000 Americans have one or more types of cardiovascular disease.[1] The estimated treatment cost of cardiovascular disease to Americans is $503.2 billion per year.[1]

The first major advance in the palliative treatment of coronary artery disease (CAD) was the implantation of an aortocoronary saphenous vein bypass graft in 1967. Since that time, the CABG procedure has been refined and has been the treatment of choice for many patients with CAD. However, the first percutaneous transluminal coronary angioplasty (PTCA), performed by Andreas Gruentzig in 1977, marked another major innovation in CAD treatment.

Since the late 1970s, techniques to treat CAD have expanded beyond PTCA. Today, the term PCI is used to describe less invasive procedures to treat CAD and includes PTCA, laser angioplasty, atherectomy, and stenting. These interventions are described in this chapter.

The path to PCI began in 1964, when Dotter and Judkins introduced the concept of mechanically dilating a stenosis in a blood vessel with a technique of inserting a series of progressively larger catheters to treat peripheral vascular disease. After experimenting with this technique, Gruentzig modified the procedure by placing a polyvinyl balloon on the tip of a catheter; the balloon was passed into a narrowed vessel and then inflated. This revised procedure produced a smoother luminal surface with less trauma than the Dotter-Judkins approach and reduced the risk for complications, including vessel rupture, subintimal tearing, and embolism. Gruentzig performed successful dilation of more than 500 peripheral lesions. He subsequently designed a smaller version of the dilation catheter for use within the coronary arterial tree. Gruentzig performed the first human PTCA in 1977.[2] Improvements in technique and device technology during the past three decades have made PCI the treatment of choice for managing CAD. In 2006, some 1,313,000 PCIs (76% utilizing drug-eluting stents) and 448,000 CABGs were performed in the United States.[1] PTCA is a nonsurgical technique used as an alternative to CABG in treating obstructive CAD. When indicated and if successful, PTCA can alleviate myocardial ischemia, relieve angina pectoris, and prevent myocardial necrosis. PTCA is the hallmark procedure and serves as the basis of almost all other percutaneous intracoronary interventions. During PTCA, a coaxial catheter system is introduced into the coronary arterial tree and advanced into an area of coronary artery stenosis. A balloon attached to the catheter is then inflated, increasing the luminal diameter and improving blood flow through the dilated segment. Several inflations ranging from 30 to 300 seconds may be performed.

Physiological Principles

The process that leads to successful dilation is complex and not clearly defined. Angiographic evaluation and animal and human histological studies indicate that PTCA stretches the vessel wall, leading to fracture of the inelastic atherosclerotic plaque and to tearing or cracking within the intima and media of the vessel. This cracking or slight dissection of the inner lumen of the vessel may be necessary for successful dilation.[2]

Comparisons Between PCI and CABG

As an alternative treatment for CAD, PCI compares favorably with CABG in terms of risk, success rate, the patient's physical capacity after the procedure, length of hospital stay, and cost.[3]

Mortality rates associated with first-time PCI and CABG are somewhat similar. According to the National Healthcare Cost and Utilization Project Statistics in 2007, the in-hospital death rate for patients undergoing PCI was 0.80% as compared to CABG in-hospital death rates of 1.95%.[1] In the event that a second surgical procedure becomes necessary to alleviate the symptoms of progressive CAD, the mortality and complication rates for the bypass procedure are significantly greater than for a second PCI. Seven-year survival data in the Bypass Angioplasty Revascularization Investigation trial revealed that CABG offers a survival benefit to diabetic patients compared with PTCA (76.4% vs. 55.7%). There was no difference in the survival rates of nondiabetic patients with CABG versus PTCA (86.4% vs. 86.8%).[4,5] Three-year outcome data after stenting compared with CABG for the treatment of multivessel disease revealed similar mortality rates.[6,7]

Successful PCI, which is defined as a significant reduction of the luminal diameter stenosis without in-hospital death, MI, or CABG, ranges from 80% to 100%, depending on the severity of the patient's angiographic and clinical presentation. In a study by Bentivoglio and colleagues, the cumulative 2-year survival rates were 96% and 95% among patients with stable and unstable angina, respectively, with event-free survival (ie, no death, MI, or CABG) in 79% and 76%, respectively.[8] Among patients with multivessel PCI, the actuarial survival rates were 97% at 1 year and 88% at 5 years in a study by O'Keefe et al.[9] Seven years after PTCA, Dorros et al[10] reported a survival rate of 90% in patients with simple single-vessel angioplasty and 95% in patients with simple multivessel angioplasty. Long-term survival data in the era of stenting revealed that survival rates remain approximately the same (92% to 97%)[7,11] Data regarding long-term survival in patients who have received DES are being collected at this time.

In the Coronary Artery Surgery Study, graft patency after CABG was 90% at 2 months, 82% at 18 months, and 82% at 5 years. The 10-year survival rate was 82%.[12]

Restenosis or patency data differ greatly between CABG and PCI. Within 6 months after angioplasty, 20% to 30% of lesions recur or restenose. Bare metal intracoronary stenting reduces the incidence of restenosis by an additional 5% to 10%. DES placement further reduces the risk for restenosis to approximately 2%.[13–15] Recently, late loss, defined as late restenosis following DES, has been observed. Stent manufacturers are addressing the concern of late loss through DES platform design and a variety of drug coatings applied directly to the stent. The mean occlusion rate for bypass grafts is approximately 18% during the first 5 years and 4% to 5% in 5 to 10 years.[7]

Psychological advantages of PTCA over surgery may argue favorably for the less invasive procedure. The emotional stress of awaiting dilation is less than that of awaiting surgery. However, this reduction in anxiety is partly offset by the risk for psychological crisis if the angioplasty fails and surgery—especially emergent (immediate) surgery—is needed. The psychological impact of this discouraging situation is significant, but it occurs in a relatively low percentage of cases.

Barring complications with either procedure, PCI requires a hospital stay of 8 to 24 hours, whereas CABG requires a stay of 3 to 7 days. Because the average hospital stay is shorter with PCI and because it is performed in the cardiac catheterization laboratory with the patient receiving local anesthesia, the average cost of PCI may be substantially lower than that of CABG. However, the following factors can increase the cost of PCI:

- Complications occurring during the procedure that necessitate emergency surgery (eg, coronary perforation, acute closure)
- Lesions that recur, requiring repeat dilation, or bypass surgery
- Lesions that require multiple devices to alleviate the lesion
- Complications associated with the anticoagulation regimen or arterial and venous access
- Long-term anticoagulation or antiplatelet therapy

In general, after a PCI, patients can expect a faster return to work (5 to 7 days) as opposed to patients who have had CABG (6 to 8 weeks). Depression in patients following CABG is common, although reports of quality of life in both groups are similar.[16]

In conclusion, the major advantages of PCI compared with CABG may include reduced mortality and morbidity, shorter convalescence, and lower cost to the patient and third-party payers.

Diagnostic Tests for Patient Selection: PCI and CABG

Before deciding between PCI and CABG, all objective evidence of coronary insufficiency must be documented. Noninvasive methods of evaluation that may be used before and after PCI include standard treadmill stress testing and thallium stress and redistribution myocardial imaging. These tests allow the physician to discover the areas of ischemia in the myocardium when the patient is subjected to stress (ie, exercise; see Chapter 17 for a discussion of these tests). It is necessary that nurses familiarize themselves with the results of thallium stress tests because an understanding of the patient's diagnosis, related symptoms, and indications for PCI promotes informed patient care.

Coronary angiography performed by cardiac catheterization, another method of documenting coronary insufficiency, is performed if the previous tests suggest the presence of CAD. Although this procedure is more invasive than treadmill testing and thallium imaging, it is the gold standard test to pinpoint the location of any stenoses and the degree of involvement of the artery or arteries (see Chapter 17 for a

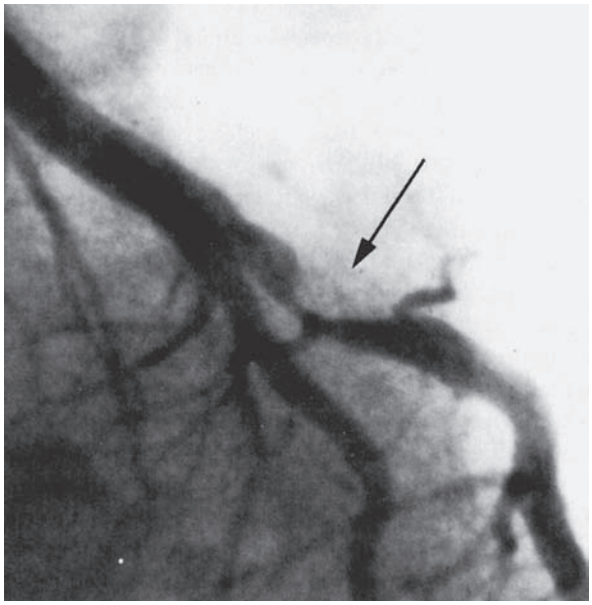


FIGURE 18-1 ▲ An eccentric stenosis in the left anterior descending artery. The term *eccentric* defines a plaque involving only one side of the intraluminal wall. (Courtesy of John B. Simpson, MD, Palo Alto, CA.)

discussion of this test). This procedure yields a 35-mm or digital image of the coronary artery anatomy. The physician can then analyze the areas of narrowing (stenosis) and gain precise information to decide the appropriate treatment (Figs. 18-1 and 18-2).

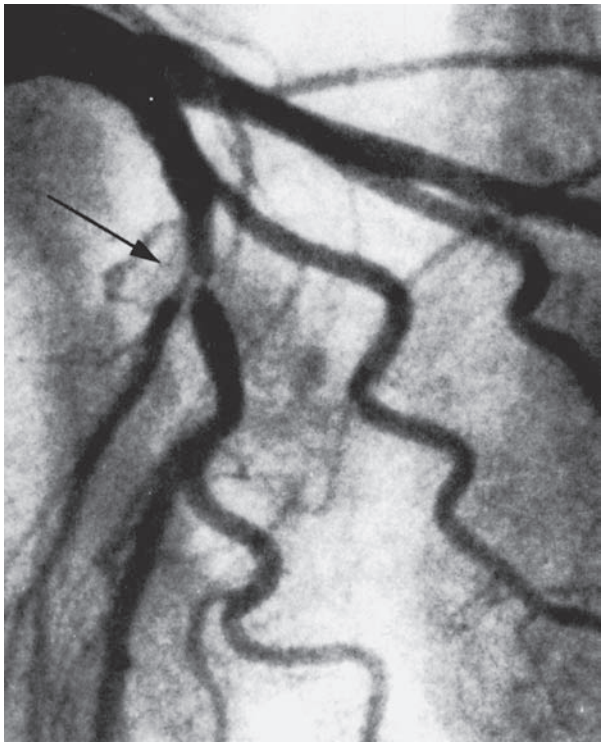


FIGURE 18-2 ▲ A coronary arteriogram of the circumflex artery illustrating a concentric stenosis. The term *concentric* defines a plaque involving the intraluminal wall circumferentially, giving a dumbbell appearance. (Courtesy of John B. Simpson, MD, Palo Alto, CA.)

Equipment Features

Since the introduction of the PCI procedure, the device technology has been refined and improved, resulting in fewer contraindications, lower rates of mortality, and fewer incidences of emergent bypass surgery. The guiding catheters used to direct and support the advancement of the dilation catheter into the appropriate coronary artery ostium have an outer diameter of 5 to 10 French (Fr). The tips of the guiding catheters have curves that are preshaped for selective access to either the right or left coronary artery.

Balloon dilation systems have evolved since Gruentzig's original design, in which the guide wire tip and catheter shaft were integral. In the early days of PTCA, physicians were limited by catheter performance and could address lesions only in the proximal anatomy. In 1982, Simpson introduced a coaxial "over-the-wire" system, an improvement that has become predominant in current catheter designs. The main innovation is an independently movable guide wire within the balloon dilation catheter. This guide wire can be manipulated to select the correct vessel despite side branches and permits safe advancement of the dilation catheter across the lesion. Currently, the available guide wires measure between 0.010 and 0.018 inch in diameter and thus usually pose little threat of interference with the blood flow through a stenosis.

Coronary balloon dilation catheter shafts range in size from 2.0 to 4.2 Fr, small enough for easy passage through the guiding catheter and for visualization around the catheter during contrast injection. Figure 18-3 shows the contrast injection through the guiding wire to verify position. The balloon dilation catheter has one or more radiopaque markers that can be imaged by fluoroscopy, allowing the interventional cardiologist to position the balloon accurately across the lesion. The inflated balloon size ranges from 1.5 to 5 mm wide and from 10 to 40 mm long.

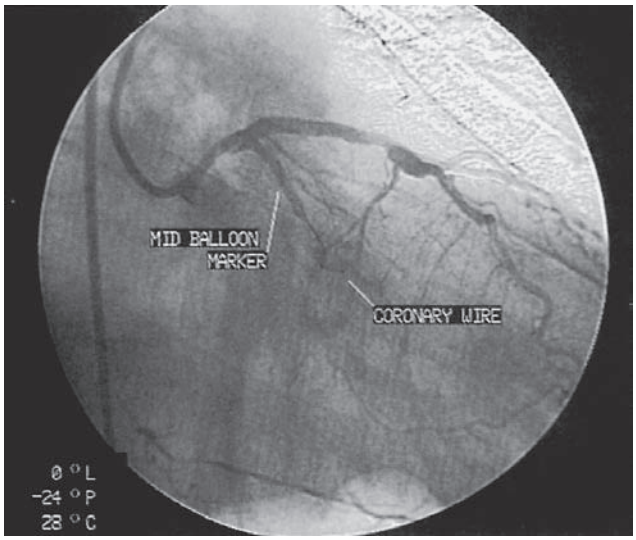


FIGURE 18-3 ▲ Contrast injection through the guiding catheter to verify position. The coronary guide wire tip is located at the occlusion of the circumflex artery, and the coronary balloon is positioned in the proximal vessel. (Reprinted with permission of Advanced Cardiovascular Systems [ACS] Inc., Santa Clara, CA.)

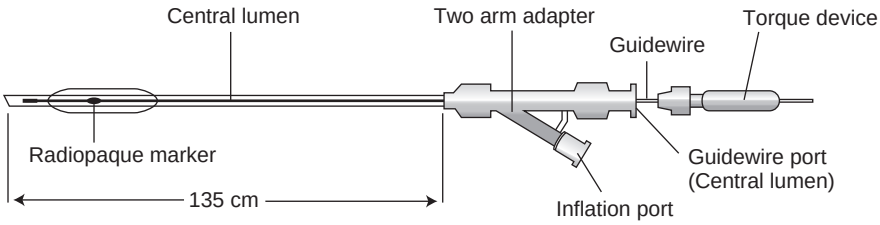


FIGURE 18-4 ▲ Percutaneous transluminal coronary angioplasty (PTCA) balloon dilation catheter illustrating the key components of the system. (Reprinted with permission of Advanced Cardiovascular Systems [ACS] Inc., Santa Clara, CA.)

The size (inflated diameter) of the balloon to be used for a particular PCI procedure is usually the same as the smallest-diameter segment of the coronary artery proximal or distal to the stenosis (ie, 3-mm vessel, 3-mm balloon). Lesion and balloon length also are approximated. Figure 18-4 shows the key components of the PTCA balloon dilation catheter.

The interventional cardiologist manually inflates the balloon with a contrast-filled, disposable inflation device that connects to the side arm or balloon lumen of the coronary dilation catheter. The device incorporates a pressure gauge that indicates the amount of pressure exerted against the balloon wall during inflation. Balloon pressure is measured in pounds per square inch (psi) or atmospheres (atm). The average initial inflation is between 60 and 150 psi or 4 to 10 atm and lasts from 30 to 180 seconds. Longer inflations may promote a smoother, more regular vessel wall as assessed by angiography and are used primarily for treating major dissections and abrupt closure. Extended inflations are performed safely with perfusion catheters that simultaneously dilate and perfuse the coronary artery.

Many factors must be considered when selecting the most appropriate equipment for performing PCI. Technological advances in balloon dilation catheter systems have improved the success and safety associated with PCI and have expanded the clinical and anatomical indications for these procedures.

Many interventional cardiologists consider the coaxial "over-the-wire" system a workhorse catheter because it can approach any anatomy well. However, the interventional cardiologist might select a rapid-exchange system to accomplish more easily the dilation of a bifurcation lesion. This type of device incorporates a "rail" system that facilitates the exchange process. A fixed-wire catheter is used to reach and dilate lesions in distal, tortuous anatomy, and its small shaft also makes it an option for the use of two coronary dilation catheters in one guiding catheter when the strategy calls for side-by-side balloons; this is also referred to as a "kissing balloon" technique.

Each PCI intervention also encompasses an inflation strategy. The main elements of an inflation strategy are the duration and pressure of balloon inflation required to open a lesion. Today, balloons that can withstand greater pressure for treating calcific lesions are available.

The outcome of any PCI procedure is greatly affected by (1) the selection of a guiding catheter that provides a platform for the advancement of the dilation system while preserving flow to the coronary artery and (2) the selection of a balloon dilation system and intracoronary DES that best address the vessel's anatomy and the lesion's location and characteristics.

Indications for and Contraindications to PCI

Indications

When choosing to treat with PCI, the physician's purpose is to alleviate angina pectoris unrelieved by medical treatment and to reduce the risk for MI in symptomatic patients and asymptomatic patients with significant stenosis. Indications for PCI have expanded as device technology, techniques, and operator experience have improved.

PCI may be indicated in coronary arteries that have at least a 70% narrowing. Arteries with less narrowing may not be considered appropriate for PCI because they are equally at risk for abrupt closure, which can have serious consequences. Patients with surgical risk factors, such as severe underlying noncardiac diseases, advanced age, and poor LV function, are particularly suited for PCI because successful dilation obviates the need for an operation that would be poorly tolerated.

An example of the wide spectrum of candidacy for PCI is the accepted practice of treating patients with multivessel disease. The common technique for dilating multiple lesions is to dilate the most critical stenosis first. With successful dilation of this "culprit" lesion, remaining lesions are dilated in stages (ie, at different intervals during the procedure or over several days). However, dilation of multiple vessels is technically more demanding and carries a higher risk for complications.

Another expanded indication is the approach to treating the patient with a totally occluded vessel. Early in PCI practice, acute and chronic total occlusions disqualified a patient for the procedure because the stenosis could not be crossed with the guide wire and balloon dilation catheter without causing severe trauma to the artery. Refinement of device technology and the increased physician experience have allowed dilation of total occlusions attempts in appropriate candidates. Total occlusions of short duration (ie, 3 months or less) are easier to cross and dilate successfully than total occlusions of longer duration (chronic total occlusions).

Additional candidates for PCI are those who have undergone CABG in whom symptoms have recurred because of stenosis and graft closure or progression of coronary disease in the native vessels or in vein grafts. For these candidates, successful PCI makes second surgery, with its increased potential for complications, unnecessary. It is thought that the proliferative disease in the graft wall generates fibrous stenosis that is much less dense than most fibrotic tissue in the native vessels, so certain vein graft stenoses respond favorably to percutaneous intervention.

Historically, patients experiencing an AMI as documented by significant ST-segment elevation, increased cardiac enzyme levels, and pain unrelieved by thrombolysis,

surgery, or pharmacological treatment were treated with complete bed rest in a coronary care unit. Today, if thrombus and underlying stenosis are causing the infarction, thrombolytic therapy, PCI, or both offer alternatives. If a blood clot has impeded flow to the distal myocardium and precipitates an ischemic episode, a thrombolytic agent can be administered intravenously or directly into the coronary artery. On successful lysis of the thrombus, dilation of the underlying stenosis often further enhances blood flow to the reperfused myocardium, reducing the risk for rethrombosis or critical narrowing caused by normal or spastic vasomotion superimposed on an organic stenosis.

Primary PCI is a dilation of an infarction-related coronary artery during the acute phase of an AMI without prior administration of a thrombolytic agent. Meyer et al[17] first used PTCA in the AMI setting in 1982. They reported an 81% success rate in PTCA of the infarction-related artery after intracoronary thrombolytic therapy. In 2006, the TRITON TIMI 38 trial reported a PCI with stenting success rate of 95% and a patency rate of 53% 1 year post PCI.[18] Parameters routinely assessed in patients selected to receive primary angioplasty are depicted in Box 18-3.

In the setting of AMI, PCI may benefit patients deemed ineligible for traditional medical therapy. Such patients include those in cardiogenic shock, those believed to be at high risk for bleeding complications (CVA, prolonged cardiopulmonary resuscitation [CPR], bleeding diathesis, severe hypertension, or recent surgery), and those of advanced age (>75 years). Primary PCI does not preclude the use of thrombolytic therapy if residual thrombus is observed. In fact, AMI patients deemed high risk (extensive ST-segment elevation, new-onset left BBB, previous AMI, Killip Class 2 or greater, anterior MI, or EF ≤ 35%) who receive fibrinolytic therapy at a non-PCI hospital should be transferred as soon as possible to a PCI-capable facility for evaluation and possible intervention. Patients who are not deemed to be at high risk and who receive fibrinolytic therapy at a non-PCI hospital may also be transferred as soon as possible to a PCI-capable facility.[19]

Primary PCI may offer distinct advantages in reducing the length of hospital stay and eliminating the need for additional intervention in many cases. Indications for PCI are summarized in Box 18-4.

Complications of primary PCI include retroperitoneal or vascular hemorrhage, other bleeding requiring transfusion, late restenosis, and early acute reocclusion (subacute thrombosis). These complications occur at approximately the same rate as those experienced in routine elective PCI.

BOX 18-3 Parameters Evaluated in Patients Selected to Receive Primary Angioplasty

- Age
- Hemodynamic status
- Angiographic anatomy:
  - Single-, double-, or triple-vessel disease
  - Vessel involvement: left anterior descending artery (LAD), right coronary artery (RCA), left circumflex artery (LCX)
  - Lesion location ostial: proximal, mid, or distal disease
  - Percent grade stenosis
  - Thrombolysis in myocardial infarction flow: 0, I, II, III
- Left ventricular (LV) ejection fraction (EF) (%)
- Presence of chest pain consistent with acute myocardial infarction (AMI)
- Electrocardiogram (ECG) evidence of AMI:
  - 1-mm ST-segment elevation in two contiguous leads
  - or
  - 1-mm ST-segment depression believed to represent reciprocal changes to an area of infarction

BOX 18-4 Indications and Contraindications for Percutaneous Coronary Intervention

Indications	Contraindications
Clinical	
Symptomatic (angina unrelieved by medical therapy)	
Asymptomatic but with severe underlying stenosis	
Stable/unstable angina	
Acute myocardial infarction	
High-risk surgical candidates	
Anatomical	
Severe stenosis (70% or more)	Mild stenosis (<70%)
Proximal and distal lesions	
Single and multivessel disease	
Bifurcation lesions	
Ostial lesions	
Totally occluded vessels	
Bypass graft lesions	
"Protected" and unprotected left main coronary artery (previous LAD or LCX coronary artery bypass graft)	

LAD, left anterior descending artery; LCX, left circumflex artery.

Contraindications

There are very few contraindications to PCI. Patients with left main CAD were once not considered candidates for PCI. The obvious drawback of PCI in left main artery disease is the possibility of acute occlusion or spasm of the left main artery during the procedure, which would result in severe LV dysfunction. Patients who have a "protected" left main artery (ie, have had previous bypass surgery to the left anterior descending or circumflex arteries with patent grafts present) are often candidates for PCI. One-year clinical outcomes of protected and unprotected left main coronary artery stenting revealed that those who had unprotected left main stenting had increased major adverse cardiac events, and their survival was decreased at 1 year. However, left main stenting should be considered in the absence of other options.[20] For high-risk patients (ie, patients with left main vessel disease, severe LV dysfunction, or dilation of the last remaining patent artery), percutaneous support devices may improve the safety of PCI. These devices include perfusion balloons, intra-aortic balloon counterpulsation, coronary sinus retroperfusion, and cardiopulmonary support.

Procedure

The PCI procedure is carried out in a sterile fashion, with the use of local anesthesia and either the Judkins (percutaneous

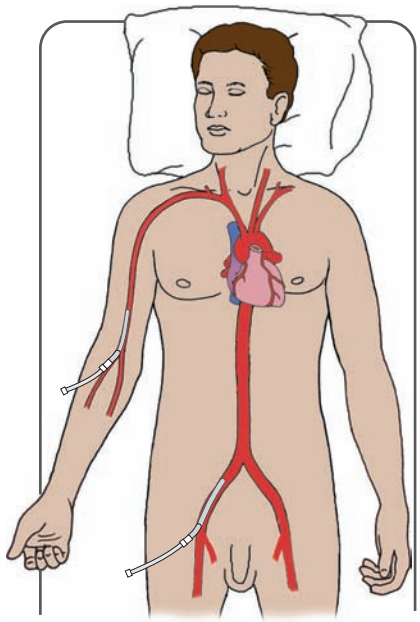


FIGURE 18-5 ▲ Two approaches to left heart catheterization. The Sones technique uses the brachial artery, and the Judkins technique uses the femoral artery. With either method, the catheter is passed retrograde through the ascending aorta to the left ventricle. (Reprinted with permission of Advanced Cardiovascular Systems [ACS] Inc., Santa Clara, CA.)

femoral) approach or, less often, the Sones (brachial cutdown) approach (Fig. 18-5). With the Judkins approach, the interventional cardiologist cannulates the femoral vein and artery percutaneously by inserting a needle (usually 18-gauge) containing a removable obturator. The obturator is then removed to confirm by the presence of blood flow that the outer needle is within the lumen of the vessel. Once proper placement is established, a guide wire is introduced through the outer cannula into the artery to the level of the diaphragm. The cannula then is removed and replaced by a valved introducer sheath. The sheath provides hemostasis and support at the puncture site in the groin and reduces potential arterial trauma if multiple catheter exchanges are necessary. The guiding catheter is preloaded with a 0.038-inch J-wire and introduced into the sheath. The 0.038-inch J-wire is advanced over the arch, and the guiding catheter is advanced over the wire. The 0.038-inch J-wire is removed, and the guiding catheter is rotated precisely to the appropriate coronary ostium. The procedure also may be accomplished by the Sones approach, in which a brachial cutdown is used to isolate the brachial vein and artery. A small arteriotomy is made, and the catheter is passed to the level of the aortic arch.

Regardless of the mode of access, coronary angiography is then carried out in both the left anterior oblique (30 degrees) and right anterior oblique (60 degrees) views. These views allow for visualization of the heart along its transverse and longitudinal planes. Opposing views provide a thorough assessment of both the lesion and the anatomical approach. A "freeze frame" of each view is obtained as a road map or guide throughout the procedure. A final lesion assessment is made, confirming lesion severity and vessel diameter for appropriate balloon and stent sizing.

If PCI is indicated, the patient may be anticoagulated with 5,000 to 10,000 U of heparin to prevent the formation

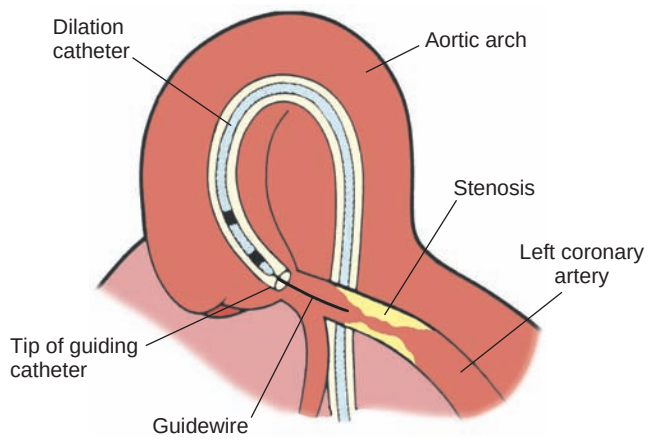


FIGURE 18-6 ▲ The advancement of the coronary dilation catheter to the tip of the guiding catheter, which is positioned in the left coronary artery, is facilitated by fluoroscopy. (Reprinted with permission of Advanced Cardiovascular Systems [ACS] Inc., Santa Clara, CA.)

of clots on or in the catheter system during the procedure. Intracoronary nitroglycerin is kept on the sterile field throughout the procedure and given intermittently as needed for vasospasm and for dilation to facilitate visualization of the culprit coronary artery.

The balloon dilation catheter and intracoronary stent system is introduced into the guiding catheter through a bifurcated adapter that provides access and is a port for contrast injections and aortic pressure measurement. The balloon dilation catheter, stent, and guide wire are advanced to the tip of the guiding catheter while their position is checked by fluoroscopy (Fig. 18-6). The guide wire then is advanced and manipulated to negotiate the branches of the coronary artery. Proper advancement can be confirmed by injecting contrast through the guiding catheter and fluoroscopically visualizing the coronary tree.

Once the guide wire is positioned safely beyond the stenosis, the balloon dilation catheter (with or without a stent) can be advanced slowly over the guide wire into the narrowing without risk for injury to the intima (Fig. 18-7).

Exact placement of the dilation balloon and stent in the stenosis is facilitated under fluoroscopy by the radiopaque marker on the balloon and by contrast injections for visualization. Initially, the balloon is inflated at 1 to 2 atm of pressure to confirm its position. Many PTCA balloon catheters expand at both ends and not in the center, where they are pinched by the stenosis (Figs. 18-8 and 18-9). The central indentation usually disappears as the stenosis is dilated. After each inflation, the interventional cardiologist injects a small bolus of contrast medium to assess any changes in coronary blood flow through the stenosis and to assess any increase in luminal diameter. At this time, the need for additional inflations is determined. Complications, such as vessel recoil and abrupt closure, occur most often during this early phase; however, their incidence is low, and redilation can be performed readily at this time. After dilation is complete, the guiding catheter, balloon dilation catheter, and stent delivery platform are removed. Postdilation angiography is performed to define more clearly the results of the PCI procedure.

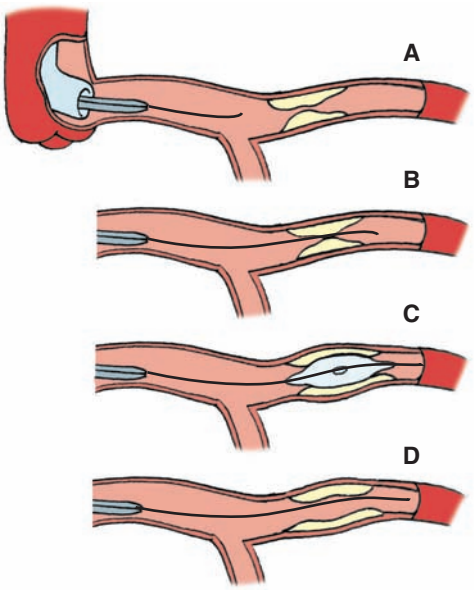


FIGURE 18-7 ▲ **A:** PTCA dilation catheter and guide wire exiting the guiding catheter. **B:** Guide wire advanced across the stenosis. **C:** Dilation catheter advanced across the stenosis and inflated. **D:** Dilation catheter pulled back to assess luminal diameter. (Reprinted with permission of Advanced Cardiovascular Systems [ACS], Inc., Santa Clara, CA.)

Reasons for failure to complete a PCI procedure include inability to cross the target lesion with a guide wire or dilation catheter due primarily to chronic total occlusions, inability to dilate the lesion due to rigid lesions or severe dissection, and embolization of friable vein graft material or of thrombus.

Successful dilation of a lesion commonly is defined as a reduction of the luminal diameter stenosis by about 40% or 50%. Clinical success commonly is defined as angiographic success with clinical improvement and without significant in-hospital complications, such as death, MI, or CABG or repeat PCI for abrupt closure.

Angiography after successful PCI demonstrates an immediate increase in the intraluminal diameter of the involved vessel (Fig. 18-10). Clinical improvement of the patient is demonstrated by improved or normalized myocardial perfusion deficits, as shown by comparison of a post-PCI thallium stress image with the pre-PCI stress image. Post-PCI treadmill test results compared with the preprocedure test results reveal increased exercise endurance and a decrease in exercise-induced angina or angina equivalent.

Results

Excellent short- and long-term results have been achieved in patients undergoing PCI. The results vary depending on the patient's clinical presentation (ie, stable or unstable angina) and angiographic characteristics (ie, subtotal or total occlusion). Among patients undergoing either single-vessel dilation or multivessel dilation, in-hospital clinical success has ranged from 85% to 100%.[8–10] In-hospital complications have been low, with a reported mortality rate of 1% to 2% in both these patient groups.[13] Long-term survival rates have been high, although repeat PCI may be necessary for recurrent or progressive disease. The frequency with which this occurs with DES has decreased dramatically.

Patients with high-risk clinical or angiographic presentations have lower success rates. However, PCI is often

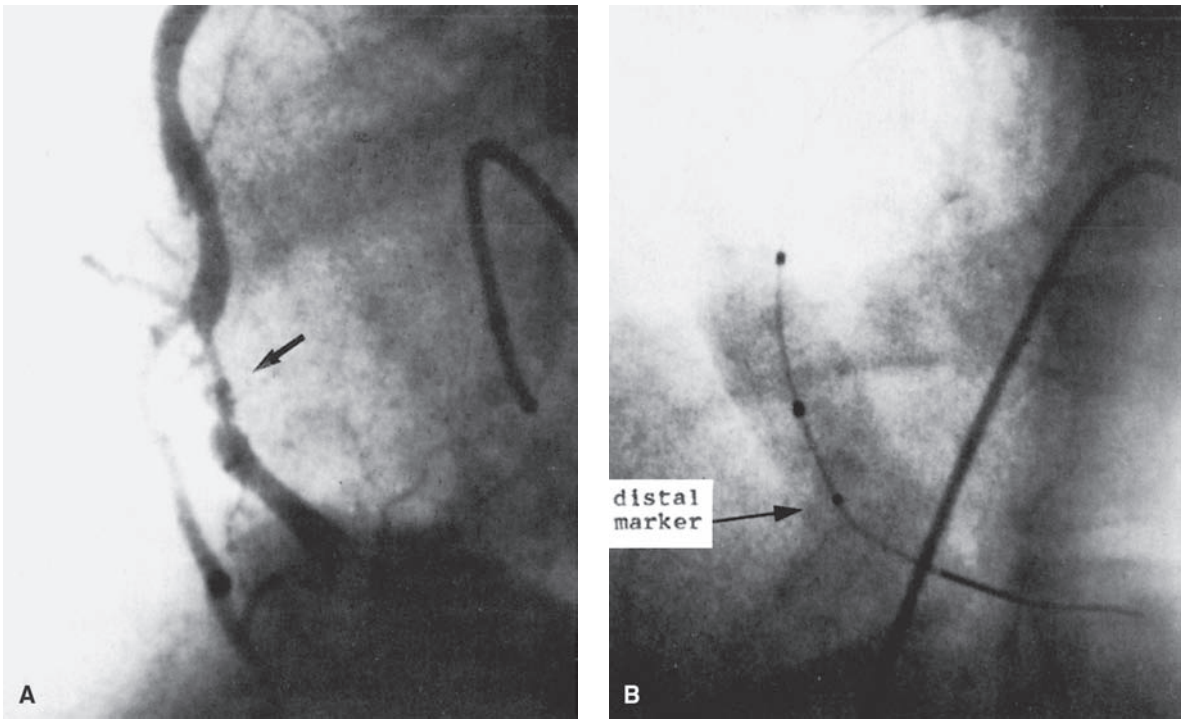


FIGURE 18-8 ▲ Thirty-five-spot frames showing (**A**) stenosis involving the midright coronary artery and (**B**) the first and second radiopaque markers revealing the position of the dilation balloon across the stenosis, with the distal marker referring to the tip of the catheter beyond the narrowing. (Courtesy of John B. Simpson, MD, Palo Alto, CA.)

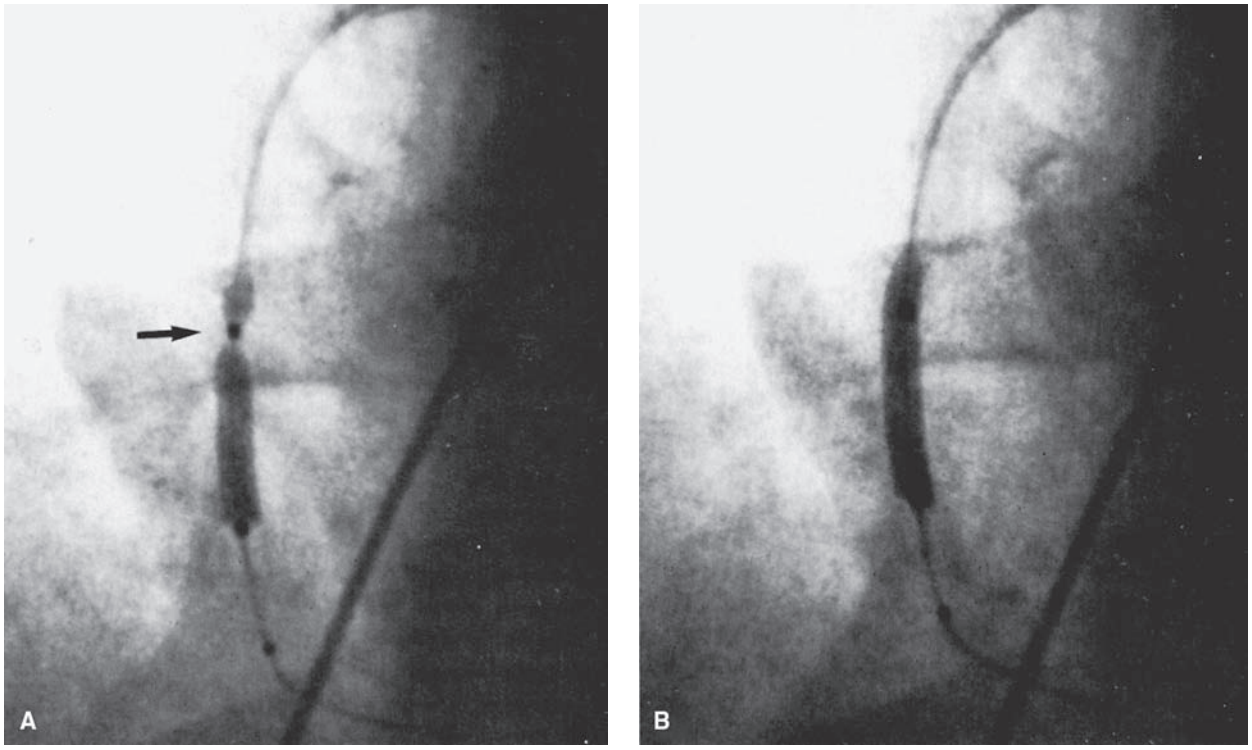


FIGURE 18-9 ▲ Thirty-five-spot frames showing (**A**) inflation of the balloon, revealing the position of the stenosis by the "dumbbell" effect, and (**B**) absence of stenosis after dilation. (Courtesy of John B. Simpson, MD, Palo Alto, CA.)

preferable to surgical revascularization because of the latter's increased risk for mortality in patients, such as older adults or those with depressed LV function. On the other hand, success of a PCI procedure can be defined angiographically, procedurally, or clinically.

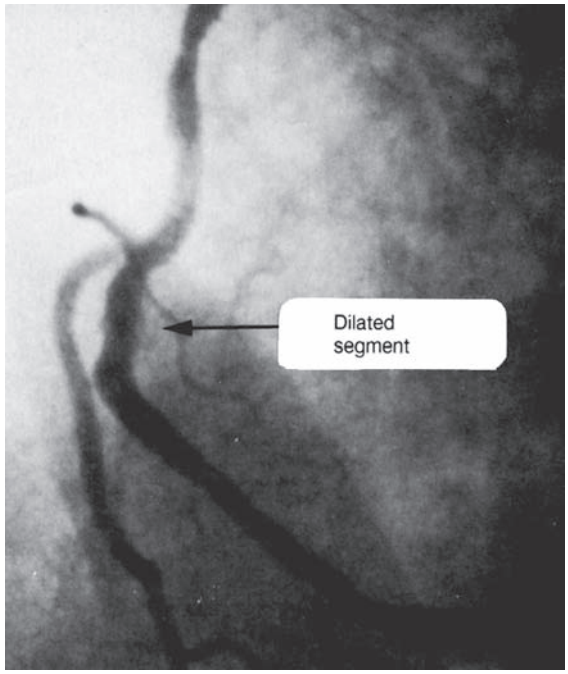


FIGURE 18-10 ▲ Repeat angiography after PTCA of a right coronary artery stenosis showing increased flow and increased diameter of the dilated segment. (Courtesy of John B. Simpson, MD, Palo Alto, CA.)

According to a joint statement by the American College of Cardiology (ACC) and the AHA[19] (2009), these successes are defined as:

1. Angiographic success: A successful PCI procedure is one in which the minimum residual vessel stenosis is less than or equal to 20%.
2. Procedural success: Angiographic success without complications (ie, death, CABG, MI) during the procedure or initial hospitalization.
3. Clinical success: Anatomical and procedural success with relief of the signs of symptoms of myocardial ischemia. Long-term success requires that the relief of the signs and symptoms persists for more than 6 months after the procedure. Restenosis is the principal cause of failure to achieve long-term success.[20]

Assessment and Management

Patient Preparation

When the decision has been made to proceed with any PCI procedure, the patient typically is admitted to the hospital the day of the procedure.

LABORATORY TESTS. The nurse monitors all preliminary laboratory tests, including cardiac enzymes, serum electrolytes, and coagulation studies (PT and partial thromboplastin time), serum potassium, creatinine, and blood urea nitrogen (BUN) levels.

Potassium levels must be within normal limits because low levels result in increased sensitivity and excitability of the myocardium and subsequent dysrhythmias. The cardiac

muscle also is sensitive and becomes irritable when the flow of oxygen-rich blood decreases, as it does for a controlled period of time during placement and inflation of the dilation balloon and stent across the lesion. The irritability arising from hypokalemia, ischemia, or both can give rise to life-threatening ventricular dysrhythmias.

Elevation in the levels of serum creatinine, BUN, or both may indicate problems in kidney function. Good kidney function is important because during PCI, radiopaque contrast material (which permits fluoroscopic visualization of the coronary anatomy and of catheter placement) is introduced into the bloodstream.[21] This contrast material is a hyperosmotic solution that the kidneys must filter and excrete from the blood. High levels of creatinine and BUN may reflect decreased renal filtration capability and vulnerability of the kidney in processing the extra load of radiopaque solution. Instances of acute renal failure have resulted from high doses of radiopaque contrast agent. A study by Rihal et al reported a 3.3% incidence rate of contrast-induced renal failure following PCI. Contrast-induced renal failure occurs more frequently in patients who are diabetic, in patients who are dehydrated, and in patients with higher baseline creatinine levels.[22] The nurse ensures that the patient is adequately hydrated, either orally or with IV solutions, to avoid falsely high electrolyte levels. Trends in creatinine and BUN levels, in conjunction with measurement of urine output, can be used to monitor kidney function.

INFORMED CONSENT. The informed consent for the PCI procedure is obtained from the patient before the procedure after a detailed discussion of the potential complications, anticipated benefit, and alternative therapies. This discussion should be conducted before any preoperative sedation. The nurse plays an important role in answering any questions that the patient and his or her family may have regarding the procedure and follow-up care.

PREOPERATIVE MEDICATIONS. Twenty-four hours before the procedure, the patient's medications should include aspirin, 325 mg once a day, for its antiplatelet effect. Diabetic patients taking metformin should be advised to discontinue this medication before their procedure because it is contraindicated with intravascular contrast agents. Anticoagulants, such as warfarin, are often withheld for a number of days before the PCI procedure. Studies show that administration of clopidogrel before and after a PCI decrease adverse events such as acute closure and subacute thrombosis.[23,24]

SURGICAL STANDBY. Surgical standby for PCI is controversial at this time. Surgical availability is required, but the degree to which the operating room is held for availability varies according to the patient's risk factors, patient acuity, and hospital policies. Many smaller community hospitals across the United States are performing PCI procedures without in-house surgical standby. These patients are typically low risk and reside near larger academic centers that can accept the patient by immediate transfer if complications arise during the PCI. A comparison of patients treated with PCI at hospitals without on-site cardiac surgery with those treated only with thrombolytic therapy reveals that the former group has better clinical outcomes at 1, 3, and 6 months.[25]

Nursing Management During PCI

Both before and during the procedure, nurses in the cardiac catheterization laboratory are responsible for understanding all aspects of equipment use and patient care. They should be experienced in ACLS and be knowledgeable about the proper administration of emergency medications and the correct application of emergency equipment, including the defibrillator, the intra-aortic balloon pump (IABP), the ventilator, and the temporary pacemaker. They observe and communicate with the patient intermittently and report any changes in patient status to the physician. The nurse monitors the ECG and arterial pressure, noting significant changes that may accompany the administration of drugs, symptoms of ischemia, or chest pain. The nurse must recognize signs and symptoms of contrast sensitivity, such as urticaria, blushing, anxiety, nausea, and laryngospasm. The nurse should understand the proper assembly and use of all PCI equipment and should be able to troubleshoot any situation that might arise.

The patient's anticoagulation status during the PCI procedure is of utmost importance. Subtherapeutic levels may result in serious complications, including acute closure or thrombotic events. An ACT should be measured in the catheterization laboratory at baseline (before the PTCA), 5 minutes after the heparin bolus (usually 5,000 to 10,000 U), and every 30 minutes thereafter for the duration of the procedure. ACT levels of 250 to 300 seconds are desirable after the initial heparin bolus. Subsequent boluses of 2,000 to 5,000 U of heparin may be required to achieve and maintain these ACT levels during the PCI procedure.

Patients at high risk for abrupt closure or with unstable lesions, such as in the setting of AMI, may be administered a platelet GP IIb/IIIa antagonist in addition to aspirin and heparin. This is referred to as a "facilitated PCI." These agents are typically initiated just before or during PCI. Eptifibatide (Integrilin) or tirofiban (Aggrastat) should be administered, in addition to aspirin and either unfractionated heparin or LMWH, to patients with continuing ischemia, an elevated troponin, or other high-risk features in whom an invasive management strategy is not planned.[25]

After the PCI is complete, the nurse instructs the patient in the precautions necessary to prevent bleeding from the puncture site (see Box 18-5).

After the procedure, the patient is transferred to a telemetry unit or catheterization recovery area for observation. Common nursing diagnoses for patients undergoing PCI are listed in Box 18-6.

BOX 18–5 TEACHING GUIDE *Precautions for Patients Post-Percutaneous Transluminal Coronary Angioplasty*

The nurse needs to remind the patient to:
- Remain on bed rest for 4 to 6 hours.
- Maintain the involved leg in a straight position (for Judkins technique).
- Avoid an upright position.
- Avoid vigorous use of the abdominal muscles, as in coughing, sneezing, or moving the bowels.

BOX 18–6 EXAMPLES OF NURSING DIAGNOSES

For the Patient Having PCI or Percutaneous Balloon Valvuloplasty (PBV)

- Decreased Cardiac Output related to mechanical factors that affect preload, afterload, and LV function
- Decreased Cardiac Output related to electrical factors affecting rate, rhythm, or conduction
- Decreased Cardiac Output related to structural changes (dissection, thrombus, or arterial spasm at PCI site), resulting in myocardial ischemia or infarction
- Decreased Cardiac Output related to increased preload and pulmonary congestion related to temporary mechanical factors (eg, balloon inflation during PBV)
- Decreased Cardiac Output related to left-to-right shunt with mitral PBV or late cardiac tamponade
- Ineffective Peripheral Tissue Perfusion related to hematoma, thrombus formation, or infection associated with cannulation site
- Acute Pain related to angina or stretching of the valve during dilation
- Deficient Fluid Volume related to renal sensitivity to contrast material or diuretic therapy
- Deficient Knowledge related to illness and impact on patient's future
- Anxiety/Fear related to lack of knowledge of PCI/PBV, acute care environment, and potential for surgery

Nursing Management After PCI

The nurse in the catheterization laboratory recovery area, coronary care, or telemetry unit, plays an important role in observing and assessing the patient's recovery. Post-PCI care is designed to monitor the patient closely for signs and symptoms of myocardial ischemia. The most overt symptom of a possible complication, early recurrence of angina pectoris, requires prompt nursing action.

As soon as possible on receiving the patient from the cardiac catheterization laboratory, the nurse attaches the ECG monitor, which allows a quick initial cardiac assessment and establishes a baseline if the patient's condition should change suddenly. The nurse assesses the patient's status from head to toe, noting the overall skin color and temperature and carefully observing the level of consciousness. After the patient is transferred to the bed and attached to the monitor, the nurse listens closely to heart and breath sounds. The nurse evaluates the peripheral circulation by noting peripheral skin color and temperature and the presence and quality of dorsalis pedis and posterior tibial pulses.

Because the Judkins technique is used most often in PCI to access the vasculature, most patients have an entry port in either the right or the left groin through which sheaths have been placed percutaneously in a vein and artery. If the Sones technique is used, there is an arterial catheter in the brachial area (see Fig. 18-5, p. 310). A variety of mechanical devices and clamps may be used to facilitate hemostasis after sheath removal. The insertion of collagen plugs or the application of a surgical suture around the opening of the blood vessel is also routinely performed to obtain hemostasis. After sheath removal, the nurse pays careful attention to the area distal to the puncture site, checking pulses frequently and reporting immediately to the physician any changes that may indicate bleeding. Bleeding at the sheath site may result in a major hematoma that can require surgical evacuation or compromise distal blood flow to the lower extremity. To prevent excessive bleeding and to aid hemostasis, the physician may order that a 5-pound sandbag be placed over the puncture site after sheath removal if hemostasis is achieved with manual compression.

The nurse instructs the patient on the importance of keeping the involved leg straight and the head of the bed angled at 45 degrees or less. To prevent clotting in the lumens of the introducing sheaths, an IV infusion is attached to the venous sheath, and a pressurized arterial flush is attached to the arterial line. This arrangement also ensures patency should an immediate return to the cardiac catheterization laboratory be necessary because of a complication. The physician chooses both the type of solution to be infused through the venous sheath and the rate of infusion. His or her decision depends on the patient's fluid volume status.

Although initial post-PCI laboratory blood tests vary by institution, they may include coagulation studies, cardiac enzymes, and serum electrolytes. Elevation of the cardiac enzymes can indicate that a silent MI has occurred (ie, infarction unannounced by chest pain). If a cardiac enzyme laboratory value is abnormal, the nurse notifies the physician immediately because the patient's postoperative care might need to be modified to prevent further injury.

The nurse plays a significant role in observing and assessing angina that recurs soon after a PCI procedure. Any chest pain demands immediate and careful attention because it may indicate either the start of vasospasm or impending subacute thrombosis. The patient may describe angina as a burning, squeezing heaviness or as sharp midsternal pain. Other signs and symptoms of myocardial ischemia include ischemic ECG changes (elevation of the ST segments or T-wave inversion), dysrhythmias, hypotension, and nausea. The nurse notifies the physician immediately of any such change in the patient's condition because it is impossible to tell merely by observation whether the change indicates a transient vasospastic episode, which can be resolved with vasodilation therapy, or an acute occlusion requiring emergent intervention (repeat PCI or CABG).

If vasodilation therapy is indicated, it may be administered as described subsequently unless the patient is hypotensive; in that case, vasodilation is contraindicated. At the first sign of vasospasm, the nurse gives oxygen by mask or nasal cannula. For fast, temporary (and possibly permanent) relief, 0.4 mg of nitroglycerin, 5 mg of isosorbide (Imdur), or 10 mg of nifedipine (Procardia) is administered sublingually. In addition, the IV drip of nitroglycerin should be titrated to maintain a blood pressure adequate to ensure coronary artery perfusion and to alleviate chest pain.

With the onset of the chest pain, a 12-lead ECG reading is recorded to document any acute changes. If the angina resolves and any acute ECG changes caused by medical therapy disappear, it is safe to assume that a transient vasospastic episode occurred; however, if the angina continues and the ECG changes persist, redilation or emergency bypass surgery should be considered.

If the post-PCI course is uncomplicated, the sheaths are removed after 2 to 4 hours, and a pressure dressing is applied to the site. A variety of mechanical clamps or hemostasis

BOX 18-7 CONSIDERATIONS FOR THE OLDER PATIENT

Before and After PCI

- Assess whether the patient will have assistance in the home with meals, cleaning, self-care, and transportation to medical appointments.
- Closely monitor kidney function before and after PCI because elderly patients may be sensitive to small amounts of radiocontrast material.
- Monitor vital signs frequently, including temperature, because elderly patients are prone to excessive body heat loss.
- Assess all preexisting comorbidities: arthritis, peripheral vascular disease, diabetes, and so forth.
- Provide clear, precise, written instructions in preparation for discharge.
- Assess patient's ability to purchase/afford required medications.

devices may be used to facilitate hemostasis after sheath removal. Many times, the sheaths are removed before the patient leaves the cardiac catheterization laboratory, and a hemostasis device is utilized. The patient must continue complete bed rest for 4 to 6 hours after the sheaths are removed. A normal, low-sodium, or low-cholesterol diet may be resumed, depending on the preference of the physician and the needs of the patient.

During the recovery period, the nurse can introduce the patient to the rehabilitation process, emphasizing lifestyle modifications to combat the advance of CAD. Efforts should be made during this instruction to reinforce the importance of aerobic conditioning with regular, moderate exercise. Risk factor and secondary prevention are also discussed, including stress reduction, weight loss, and smoking cessation. See Box 18-5 for instructions for the patient after PCI. Box 18-7 describes implications for the older patient.

After PCI, the patient is asked to take medications that help prevent thrombus formation and maintain maximal dilation at the culprit lesion site. Routinely, when all patients are sent home, they should be taking aspirin for the antiplatelet effect. Aspirin is continued indefinitely. Clopidogrel (Plavix) should be administered to patients who receive one or more DESs. Clopidogrel should be continued for at least 12 months and may be administered indefinitely.[23–25] However, in patients taking clopidogrel in whom elective CABG is planned, the drug should be withheld for 5 to 7 days. Often, long-acting nitrates, calcium channel blockers, ACE inhibitors, and lipid-lowering agents are added to the medical regimen. The nurse may be responsible for explaining to the patient the indications for the specific medications ordered by the physician, including side effects and signs of overdose. The nurse should also answer any questions that the patient may have regarding his or her follow-up care. Box 18-8 summarizes medications currently associated with PCI.

Four to six weeks after the patient's discharge, an exercise treadmill stress test and a thallium imaging study may be performed to test the efficacy of the PCI. Compared with the pre-PCI tests, an increase in exercise capacity and a decrease in or disappearance of exercise-induced chest pain (without ST-segment changes) suggest improved blood flow and normalization of cardiac function in the previously hypoperfused muscle. Treadmill stress testing should be repeated at 6 months and then annually after PCI.

Complications

The indications for PCI have expanded to include patients with more severe CAD (ie, total occlusions, multivessel disease, recent or ongoing MI, poor LV function). The rate of complications associated with PCI has not increased. Major complications that can result in ischemia and possible severe LV dysfunction necessitating emergent CABG include angina unrelieved by maximal administration of nitrates and calcium channel blockers (Box 18-8), MI, coronary artery spasm, abrupt closure of a dilated segment, coronary artery dissection leading to occlusion, and restenosis. See Box 18-6 on page 314 for nursing diagnoses for the patient having PCI or percutaneous balloon valvuloplasty (PBV).

Angina, Myocardial Infarction, and Vasospasm

Some degree of angina is anticipated during the PCI procedure owing to the temporary occlusion of the involved vessel during dilation. This angina is handled with intracoronary nitroglycerin or removal of the balloon dilation catheter while the guide wire is left across the lesion. Evidence of persistent chest pain after PCI, reflected in changes in heart rate and blood pressure and elevated ST segments, indicates ischemia predisposing to an insult to the myocardium and requiring immediate intervention. Coronary artery spasm sometimes requires emergent surgical intervention (CABG) when the vasoconstriction, occlusion, or ischemia cannot be reversed by administering nitrates.

Abrupt Closure of Dilated Segment

Abrupt closure is a serious complication of coronary artery dilation that occurs in approximately 3% of those undergoing angioplasty.[15] An estimated 70% to 80% of abrupt closures occur while the patient is still in the cardiac catheterization laboratory. Approximately one third to one half of those patients whose vessel abruptly closes undergo a successful repeat dilation. Abrupt closure can be caused by coronary artery dissection, coronary artery spasm, and thrombus formation. Treatment options include immediate repeat dilation, emergent CABG surgery, or pharmacological therapy. To maintain blood flow through the occlusion while the patient is being prepared for emergent CABG surgery, the physician can use a perfusion balloon catheter, which has side holes along its shaft to allow blood to flow through the catheter at the site of occlusion and perfuse the distal myocardium.

Coronary Artery Dissection

Coronary artery dissection or an intimal tear in the coronary artery can be visualized in the form of intraluminal filling defects or extraluminal extravasation of contrast material. Mild interruptions in the intraluminal wall are an expected result of the splitting and stretching of the intima on inflation of the balloon dilation catheter at the lesion site. However, a dissection may cause a major luminal obstruction associated with coronary artery occlusion, leading to deterioration in

BOX 18-8 Summary of Medications Most Often Associated With PCI

Anticoagulants/Antiplatelets

Aspirin
Indications: Prophylaxis of coronary and cerebral arterial thrombus formation
Actions: Blocks platelet aggregation
Dosage: 80 to 325 mg daily, PO
Adverse effects: (Usually well tolerated) nausea, vomiting, diarrhea, headache, and vertigo occasionally

Heparin (fractionated)
Indications: Prophylaxis of impending coronary occlusion and prophylaxis of peripheral arterial embolism
Actions: Inhibits clotting of blood and formation of fibrin clots; inactivates thrombin, preventing conversion of fibrinogen to fibrin; prevents formation of a stable fibrin clot by inhibiting the activation of fibrin stabilizing factor; inhibits reactions that lead to clotting but does not alter normal components of blood; prolongs clotting time but does not affect bleeding time; does not lyse clots
Dosage: Varies with indications; IV or intra-arterial: 10,000 U at start of PCI
Adverse effects: Uncontrollable bleeding, hypersensitivity

Low–Molecular-Weight Heparin (Enoxaparin Sodium, Dalteparin Sodium)
Indications: Treatment of unstable angina and myocardial ischemia, complete and non–Q-wave myocardial infarction (MI).
Action: Prevents clotting of blood and formation of thrombin.
Dosage
Enoxaprin: 1 mg/kg SC every 12 hours for 2 to 8 days
Dalteparin sodium: 120 mcg/kg SC every 12 hours for 5 to 8 days
Adverse effects: Thrombocytopenia, hematoma, pain or reaction at the injection site, rash, hemorrhage, fever.

Glycoprotein IIb/IIIa Antagonists (Abciximab, Eptifibatide, Tirofiban)
Indications: Prevention of clotting and abrupt closure during interventional procedures and prevention of restenosis.
Action: Blocks the receptor on the platelet membrane that leads to the final common pathway of platelet aggregation.
Dosage
Abciximab: 0.25 mg/kg IV bolus followed by 0.125 mcg/kg/min infusion for 12 to 24 hours after PCI
Eptifibatide: 135 mcg/kg IV administered immediately before PCI followed by 0.5 mcg/kg/min for 20 to 24 h
Tirofiban: 180 mcg/kg IV bolus followed by 1.2 to 2 mcg/kg/min infusion for 72 to 96 hours after PCI
Adverse effects: Thrombocytopenia, hemorrhage, nausea, hematoma

Clopidogrel (Plavix)
Indications: Reduction of atherosclerotic events (AMI, stroke, and vascular death) in patients with atherosclerosis documented by a recent stroke or AMI or established peripheral arterial disease
Action: Blocks platelet aggregation
Dosage: 75 mg once daily
Adverse effects: Diarrhea, rash, gastrointestinal (GI) disturbances, hemorrhage, neutropenia.

Coronary Vasodilators

Isosorbide Dinitrate (Isordil, Sorbitrate)
Indications: Prophylaxis of angina
Actions: A nitrate that acts as a smooth muscle relaxant; causes coronary vasodilation without increasing myocardial oxygen consumption; secondary to general vasodilation, blood pressure decrease
Dosage
Sublingual: 2.5 to 10 mg every 2 to 3 hours PRN angina
Oral: 5 to 40 mg four times daily.
Sustained-action oral: 40 mg every 6 to 12 hours
Adverse effects: Cutaneous vasodilation that can cause flushing; headache, transient dizziness, and weakness; excessive hypotension

Nitroglycerin
Indications: Control of blood pressure and angina pectoris
Actions: Potent vasodilator; affects primarily the venous system; selectively dilates large coronary arteries increasing blood flow to ischemic subendocardium
Dosage
Sublingual: 0.3 to 0.4 mg PRN chest pain
Topical (patch): 2.5 to 10 mg/d; indicated for primary, secondary, or nocturnal angina due to more sustained effect
Intravenous: 5 mcg/min to start—titrate to patient response (no fixed dose due to variable response in different patients)
Adverse effects: Excessive and prolonged hypotension; headache; tachycardia, palpitations; nausea, vomiting, apprehension; retrosternal discomfort

Calcium Channel Blockers

Nifedipine (Procardia), Diltiazem (Cardizem)
Indications: Treatment of angina pectoris resulting from coronary artery spasm and fixed vessel disease; hypertension; dysrhythmias
Actions: Inhibits calcium ion flux across the cell membrane of the cardiac muscle and vascular smooth muscle without changing serum calcium concentration; decreases afterload through peripheral arterial dilation and
1. Reduces systemic and pulmonary vascular resistance
2. Vasodilates coronary circulation
3. Decreases myocardial oxygen demands and increases myocardial oxygen supply

Dosage
Nifedipine: 10 to 30 mg three to four times daily, PO
Diltiazem: 30 to 90 mg three to four times daily, PO
Adverse effects: Contraindicated in patients with sick sinus syndrome; hypertension after IV use; GI distress; headache, vertigo, flushing; peripheral edema, occasional increase in angina, tachycardia

See pages 297–302 for full discussion of antiarrhythmic medications.

blood flow with resultant severe ischemia or MI that requires emergent bypass surgery.

Stent Thrombosis

As the number of stent implantations increased, so has the incidence of stent thrombosis. The Food and Drug Administration (FDA) met in 2006 and concluded that there appeared to be an issue of late stent thrombosis with drug-eluting stents, but the magnitude was deemed uncertain, and off-label use of the DES, as with bare metal stents, is associated with increased risk when compared with on-label use. The panel also agreed that, in the future, new DES studies should have longer follow-up, enroll greater numbers of patients, and include stent thrombosis as a study end point. The advisory panel concurred with the joint clinical practice guideline recommendation for 12 months of dual antiplatelet therapy after placement of a DES in patients who are not at high risk of bleeding.[23]

There is some evidence, however, that DESs may be susceptible to an event known as "late stent thrombosis." Late stent thrombosis is defined as a blood clot inside the stent that occurs 1 or more years after stent implantation. While this has been seen rarely in both the Taxus and Cypher stents, thrombosis is extremely dangerous, even fatal. To prevent subacute thrombosis (SAT), poststent antiplatelet therapy is crucial, and patients should be carefully and repeatedly instructed not to stop taking aspirin, Plavix, or Ticlid without consulting their interventional cardiologist. The FDA concluded that more information is needed, especially in the use of devices in off-label settings. However, when DESs are used as directed, no greater risks of death or heart attack are reported. Current recommendations and guidelines suggest that antiplatelet medications (aspirin and Plavix or Ticlid) must be continued for a minimum of 1 year assuming the patient has no history of bleeding complications.

The development of devices to remove atherosclerotic plaque (atherectomy catheters) and implantable devices to maintain the opening mechanically (stents) has provided effective adjuncts or alternatives to PTCA for the problem of recurring lesions. Restenosis of de novo lesions after atherectomy is similar in character and prevalence to that in PTCA; however, intracoronary stenting has resulted in a lower restenosis rate in native and vein graft lesions of approximately 10%.

The cause of restenosis still is unclear. It appears to be the result of an excessive healing response to balloon dilation that exposes the subintimal structures of the vessel to circulating blood. These exposed areas are then potential sites for platelet adhesion and aggregation and for thrombus formation. The degree of this "healing" response varies from lesion to lesion and may be influenced by the clinical and angiographic factors associated with restenosis that were discussed previously. Factors associated with increased incidence of restenosis are listed in Box 18-9.

Other Complications

Other major complications of PCI requiring medical intervention are coronary perforation, which may be treated with a sheathed stent to stop the leak of blood into the pericardium; bradycardia, which requires temporary pacing; VT or VF, which requires immediate defibrillation; and a central nervous system event causing transient or persistent neurological deficit.

Peripheral vascular complications occurring primarily at the catheter site include arterial thrombosis, excessive bleeding that causes a significant hematoma, pseudoaneurysm, femoral arteriovenous fistula, and arterial laceration. If any of these complications persists or compromises distal blood flow to the involved extremity, surgical intervention may be required.

Table 18-8 summarizes the complications that may result from PCI, including general signs of the complications and possible interventional actions.

Box 18-10 provides a complete outline of care for the patient undergoing PCI.

BOX 18-9 Factors Associated with Increased Incidence of Restenosis

Clinical Factors
Severe angina
Noncompliance with antiplatelet regimen
Diabetes
Smoking cigarettes
Substance abuse
Uncontrolled hyperlipidemia

Angiographic Factors
Lesion location
Lesion length
Lesion severity before and after PCI
Adjacent arterial diameter
Gaps between overlapping stents

Other Interventional Cardiology Techniques

The immediate and long-term efficacy of PCI in treating symptomatic patients with single-vessel disease has been well established. In many centers, PTCA also is routinely and successfully used in patients with multivessel disease. The safety and efficacy with which angioplasty has been used have fostered research into treating patients with unstable angina, AMI, and cardiogenic shock.

Technologies have been developed to address the challenges associated with complex PCI. These include laser angioplasty, thrombectomy devices, atherectomy devices, DES, brachytherapy, and distal protection devices.

Laser Angioplasty

The acronym LASER stands for "light amplification through stimulated emission of radiation." Through a series of mirrors and lenses, the laser beam is directed into a catheter containing numerous glass fibers. These fibers transmit the light energy through the catheter to the plaque that is to be ablated.[26] The laser is used to ablate plaque or as an adjunct to other PCI procedures to make a pathway in total occlusions to facilitate the passage of a PTCA balloon or stent.

Laser angioplasty is performed much like a standard PCI procedure. The guide catheter is advanced to the ostium

Table 18-8 Complications of PCI

Complications	General Signs/Symptoms	Possible Interventions
Angina	Chest pain or anginal equivalent	CABG or repeat PCI
Myocardial infarction (MI)	Dysrhythmias: tachycardia, bradycardia, VT/fibrillation, ST elevation	Redo PCI Oxygen
Abrupt reclosure Dissection/intimal tear	Marked hypotension Acute ECG changes (ST-segment change)	Medications: vasodilators (nitrates), calcium channel blockers, analgesics, anticoagulants, vasopressors
Hypotension	Nausea/vomiting	Intra-aortic balloon pump (IABP)
Coronary branch occlusion	ST-segment elevation	Possible repeat PCI
Restenosis	Angina pectoris Positive exercise test	Redo PCI Coronary artery bypass graft
Marked change in heart rate: bradycardia, VT, ventricular fibrillation	Rate below 60 beats/min Rate above 250 beats/min No discernible cardiac rhythm Pallor Loss of consciousness Hypotension	Temporary pacemaker Defibrillation Medications: antiarrhythmics, vasopressors
Vascular: excessive blood loss	Hypotension Decreased urine output (from hypovolemia) Decreased hemoglobin/hematocrit Pallor Hematoma at puncture site	Possible surgical repair Fluids Transfusion Oxygen Flat in bed
Allergic	Hypotension, urticaria, nausea/vomiting, hives, laryngospasm, erythema, shortness of breath	Medications: antihistamines, steroids, antiemetics Clear liquids/NPO Oxygen With anaphylaxis: fluids for volume expansion, epinephrine, vasopressors for hypotension
Central nervous system events	Changes in level of consciousness Hemiparesis Hypoventilation/respiratory depression	Oxygen Discontinue/withhold sedatives Medication: narcotic antagonist as a respiratory stimulant Computed tomography, magnetic resonance imaging

Miscellaneous complications: conduction defects, pulmonary embolism, pulmonary edema, coronary air embolism, respiratory arrest, febrile episode, nausea, minor bleeding.

BOX 18–10 *COLLABORATIVE CARE GUIDE for the Patient Undergoing PCI*

Outcomes	Interventions
Oxygenation/Ventilation	
Patient will maintain normal arterial blood gases, or pulse oximeter reading.	• Provide supplemental oxygen per face mask or nasal cannula per hospital post-PCI protocol. • Monitor blood gases/pulse oximeter per protocol. • Auscultate breath sounds when taking vital signs. • Monitor for signs of pulmonary edema or respiratory distress.
Circulation/Perfusion	
The patient will have stable vital signs following PCI.	• Monitor blood pressure, heart rate, respiration rate, arterial puncture site, distal pulses, and distal motor function and sensation: every 15 min × 4, every 30 min × 4 every 1 h × 4, then every 4 h
There is no evidence of post-PCI myocardial ischemia or infarction due to coronary reocclusion (eg, no ECG changes or angina).	• Monitor cardiac rhythm in leads specific to myocardium most affected by PCI location. • Administer medications to treat coronary artery spasms (eg, nifedipine and nitroglycerin).
There is no evidence of cardiac dysrhythmias after PCI.	• Administer heparin per protocol. • Report type and frequency of dysrhythmias. • Administer antiarrhythmic medication as indicated and ordered. • Temporary transvenous or external pacemaker and defibrillator are readily available.

(*continued on page 319*)

BOX 18–10 *COLLABORATIVE CARE GUIDE for the Patient Undergoing PCI (Continued)*

Outcomes	Interventions
There is no evidence of bleeding at the puncture site.	• Monitor site for hematoma as above with vital signs. • Assess for tenderness, ecchymosis, and warmth over puncture site. • Apply direct pressure to puncture site for 15–30 min after sheath is removed. • Apply sandbag to puncture site if oozing continues, per hospital protocol. • Apply a pressure dressing to puncture site when oozing has stopped. • Monitor activated clotting time, prothrombin time, partial thromboplastin time, and platelets, reporting coagulopathies per protocol.
There is no evidence of arterial occlusion at puncture site.	• Monitor involved extremity with vital signs for mottling, coolness, pallor, diminished pulses, numbness, tingling, pain, and so forth.
Fluids/Electrolytes	
Patient is euvolemic.	• Monitor intake and output. • Obtain type and cross-match, complete blood count, electrolytes prior to PCI. • Maintain IV patency.
Renal function is maintained after administration of radiographic IV contrast agent.	• Obtain pre-PCI and post-PCI blood urea nitrogen, creatinine, and electrolyte levels. • Closely monitor urine output; report if <30 mL/h. • Monitor urine specific gravity or osmolarity for clearance of IV contrast. • Administer diuretic agents as ordered.
Mobility/Safety	
	• The patient is on bed rest for 4–6 h post-PCI per hospital protocol. • While sheath is in place and while on bed rest, keep head of bed <45 degrees.
Skin Integrity	
Patient's skin will remain intact.	• Assess skin immediately after PCI for pressure areas. • Reposition to relieve pressure from bony prominences, maintaining alignment of extremity involved in procedure. • Consider pressure relief/reduction mattress.
Nutrition	
Nutritional intake is reestablished.	• Resume oral fluids and diet per protocol. • Monitor swallowing and protective airway reflexes while patient is receiving sedatives or narcotics.
Patient does not experience nausea or vomiting after PCI.	• Monitor nausea and vomiting. • Administer antiemetic medication as appropriate.
Comfort/Pain Control	
Patient will not experience anginal pain.	• Instruct patient to verbally report discomfort and pain. • Evaluate severity and location of pain, distinguishing angina from other causes of discomfort. • Administer nitrates or narcotics per order or protocol for angina. • Evaluate patient response to medication.
Patient will not experience pain from mobility restrictions.	• Reposition patient frequently, keeping involved extremity straight. • Use mattress overlay or egg crate for comfort. • Administer analgesics as appropriate, after distinguishing joint or muscular pain from angina.
Psychosocial	
Patient and family state risks associated with PCI. Patient uses personal support systems to reduce anxiety.	• Provide information for informed procedural consent. • Encourage verbalization of questions, concerns, and fears. • Encourage significant other to visit in early postprocedural recovery phase. • Validate patient/significant others' understanding of surgery and illness. • Initiate referrals to social services, clergy, and so forth as necessary.
Teaching/Discharge Planning	
Patient and family are prepared for possibility of emergent repeat PCI or cardiac surgery. Patient cooperates with post-PCI mobility restrictions. Patient states lifestyle changes required to reduce risk for worsening coronary artery disease.	• Preprocedure teaching includes discussion regarding causes for coronary reocclusion or perforation and rationale for surgery or repeat PCI. • Provide preprocedure and postprocedure instruction and rationale for bed rest and limited movement of involved extremity. • Provide verbal and written instruction/information regarding risk factors and pathophysiology, activity, diet, stress reduction, medication administration, and appropriate times/indications to seek medical attention.

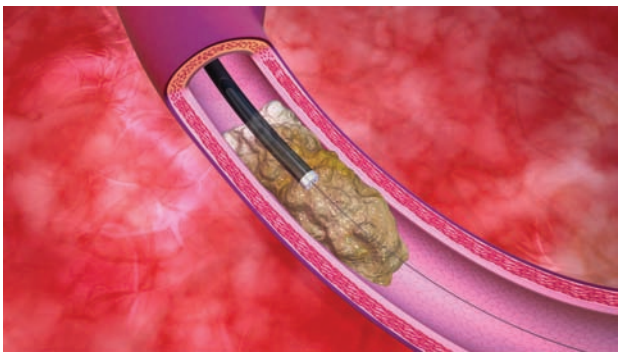


FIGURE 18-11 ▲ LASER ablation of a coronary artery stenosis.

of the coronary artery targeted by fluoroscopy. Once the lesion location is ascertained through contrast injection, a guide wire is advanced up and through the lesion. Before the laser is activated, everyone in the room (including the patient) must don protective eyewear. The laser catheter is then advanced through the guide wire and brought into contact with the lesion. Depending on anticipated lesion morphology, energy settings are chosen that will presumably suffice to ablate the plaque. The laser settings include the fluency (millijoules per square millimeter) to be delivered and the repetition rate (pulses per second). The plaque is then vaporized by the laser energy. Several passes down the length of the lesion may be performed. Laser success is determined by fluoroscopy and coronary injections with contrast dye. If there is residual stenosis after use of the laser, adjunctive PCI procedures, including stenting, can be performed to achieve an optimal final result (Fig. 18-11).

Stenotic lesions best suited for laser angioplasty include those that are long and diffuse (longer than 15 to 20 mm), ostial in location, highly calcified, in vein grafts, and totally occluded. Risks associated with laser angioplasty include perforation of the coronary artery, dissections, and aneurysms. Now considered a "niche" procedure, laser angioplasty is performed less frequently in the percutaneous treatment of cardiovascular disease.

Atherectomy

Atherectomy is the process of removing atherosclerotic plaque from the coronary artery by cutting or ablating and thus "debulking" the lesion. Atherectomy devices include directional coronary atherectomy (DCA) and rotational ablation (Rotablator).

Potential complications of all atherectomy devices include perforation of the coronary artery, abrupt closure, embolization distal to the lesion site, and MI. The rate of restenosis and other complications is comparable to that with standard balloon angioplasty and less successful than those results achieved with DESs.

Directional Coronary Atherectomy

The directional coronary atherectomy (DCA) device is a cutting catheter that is inserted over a guide wire into the coronary artery across the stenotic lesion. It is positioned so that the opening for the blade faces the lesion. A low-pressure balloon on the opposite side of the catheter is inflated, thus forcing the atherosclerotic plaque into the opening near the cutting blade. The cutting blade turns at approximately 1,200 revolutions/min (rpm) and is then slowly advanced along the length of the lesion, cutting the plaque and collecting it in the catheter nosecone. The DCA catheter is turned a complete 360 degrees in the artery to shave all sides of the atherosclerotic plaque with repeated passes. The procedure is repeated until the atherosclerotic plaque is sufficiently removed. The catheter, laden with plaque, is then withdrawn from the patient.

Rotational Ablation Device

The Rotablator device (Boston Scientific, Natick, MA) is a high-speed rotating, abrasive, burr-tipped catheter that ablates the atherosclerotic plaque in the coronary artery. The Rotablator has proved especially effective for complex stenotic lesions that are calcified, tortuous, small in diameter, ostial, or diffuse in character. The device consists of a football-shaped, diamond-studded burr attached to a drive shaft. The Rotablator is advanced over a guide wire to the lesion site. The burr rotates at 160,000 to 190,000 rpm and pulverizes the atherosclerotic plaque into microparticles that are absorbed into the patient's circulatory system. The spinning burr is advanced across the lesion several times to debulk the stenotic lesion. Adjunctive balloon angioplasty may be performed after use of the Rotablator device.

The AngioJet device (Possis) is a thrombectomy system used to extract clot from coronary arteries, saphenous vein grafts, or peripheral arteries. The system consists of three components: (1) the drive unit (Fig. 18-12A); (2) the pump set, which achieves isovolumetric balance between the fluid and thrombus that is removed from the artery and the fluid that is delivered (see Fig. 18-12B); and (3) the catheter, which is disposable and 4- to 6-Fr compatible (see Fig. 18-12C). The AngioJet System has been shown to be safe and effective in removing fresh clot from patients undergoing PCI for AMI[27] and in instances in which there is clot in saphenous vein grafts.[28]

Stents

Intracoronary stents are hollow stainless steel tubes that act as "scaffolding" in the coronary artery. After predilation with a PTCA balloon catheter, most stents are premounted on a balloon catheter and inserted through the guide catheter along a guide wire to the lesion site. Once placed across the stenotic lesion, the balloon is inflated, and the stent is expanded and left in the coronary artery.

Traditional and older stent designs are bare metal. Because many bare metal stent designs use stainless steel, they are potent thrombogenic prostheses. Stent thrombus is a major short- and long-term complication. Success of the stenting procedure depends on endothelialization of the stent to provide a smooth flow of blood in the coronary artery and through the stent yet controlled to prevent stent thrombosis. Anticoagulation and antiplatelet medication regimens are crucial to successful stenting and long-term prognosis. Stenting has been shown in numerous trials to reduce restenosis rates and improve long-term prognosis. Stents made of newer alloys and compounds are currently undergoing investigation. There are three main

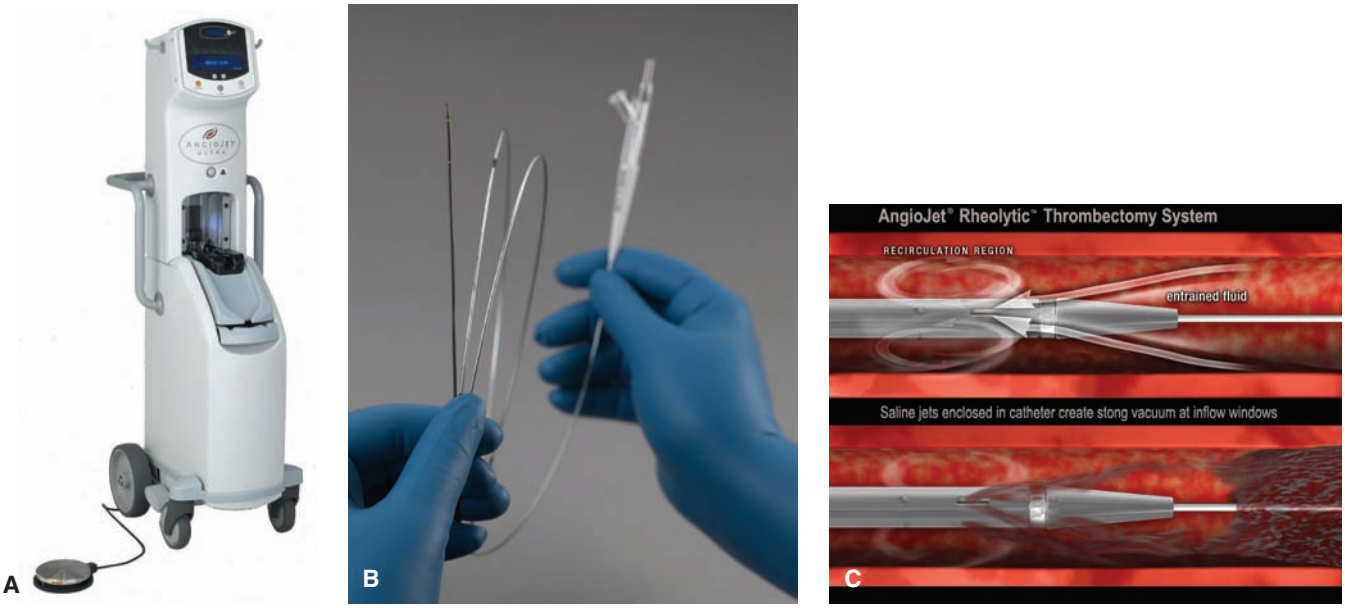


FIGURE 18-12 ▲ **A:** AngioJet Ultra Console. Power and control console for AngioJet Rheolytic Thrombectomy System. **B:** The AngioJet Spiroflex Thrombectomy Catheter is a 4-Fr thrombectomy catheter with a spiral-cut shaft for tracking. **C:** The mechanism of action of the AngioJet Thrombectomy Catheter. (Courtesy of Possis Medical Inc., Minneapolis, MN.)

components to a DES: (1) the type of stent that carries the drug coating, (2) the method by which the drug is carried (eluted) to the blood vessel wall, and (3) the drug itself. DESs are coated with drugs such as heparin, paclitaxel, sirolimus, or rapamycin. It is believed that the gradual release of these drugs into the coronary vasculature at the site of the atherosclerotic plaque inhibits restenosis by limiting smooth muscle cell proliferation and inflammation but allowing reendothelialization to proceed normally. At this time, the FDA-approved DESs include a sirolimus-coated stent known as Cypher (Johnson & Johnson, Cordis) (Fig. 18-13). Another stent, known as Endeavour (Medtronic), uses a cobalt chrome Driver stent with a phosphorylcholine coating of zotarolimus. Xience V (vee) (Guidant, Abbott) uses an L605 cobalt chrome ML Vision stent and adds a fluoropolymer multilayer coating with the drug everolimus and the paclitaxel-coated stent called Taxus (Boston Scientific) (Fig. 18-14).

The interventional cardiologist must make several important decisions that lead to a successful stent implantation. These include:

- Correct sizing of the stent length to match the length of the lesion
- Correct sizing of the stent diameter to match the thickness of the normal sections of the coronary artery

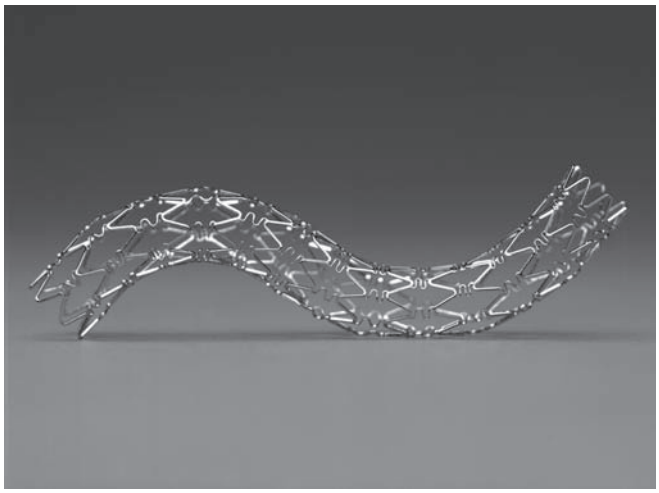


FIGURE 18-13 ▲ Fully expanded Cypher stent. Used with permission of Cordis Corporation.

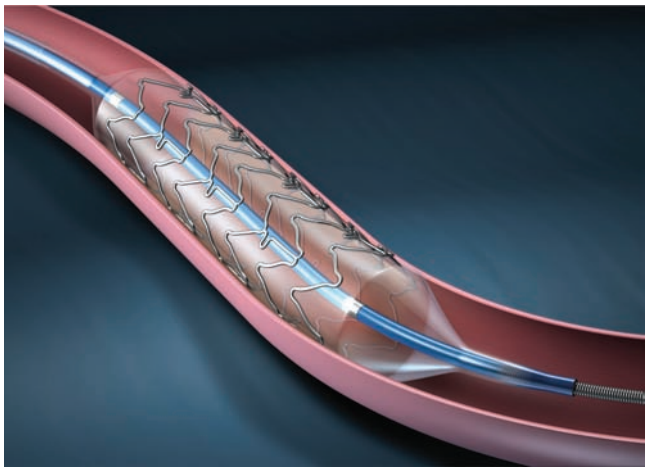


FIGURE 18-14 ▲ XIENCE V everolimus-eluting coronary stent. (Courtesy of Abbott Vascular. © Abbott Laboratories. All rights reserved.)

- Accurate and complete deployment of the stent. Underexpansion of the stent can result in tiny gaps between the stent and arterial wall, which can lead to serious problems such as SAT. Other complications following implantation of bare metal stents or DESs may include bleeding at the access site, stent migration, coronary artery dissection, and abrupt closure.

Brachytherapy

Intracoronary radiation (brachytherapy) is potentially a potent antiproliferative therapy that is currently being investigated for use with PCI and might therefore provide a means for effective reduction of restenosis. The radiation therapy is emitted in the form of temporarily implanted or inserted radioactive sources, such as seeds, radioactive stents, or radioactive liquid–filled balloons. Radiation works particularly well in inhibiting new growth by attacking the newer, more aggressive neoplastic cells, while often having little effect on normal tissue.[29] In brachytherapy, endovascular low-dose radiation is applied at the site of balloon dilation or stent implantation by a catheter system. Two types of radiation are used to treat restenosis: gamma and beta emitters. Gamma emitters create a radiation field for a considerable distance away from their source. This requires that the treatment be conducted in a heavily lead-shielded cardiac catheterization laboratory. Gamma-emitter intensity is lower than that of beta emitters, and gamma emitters must be left in place 14 to 45 minutes, depending on the strength of the source used. Beta sources, with a higher intensity of radiation near the source, can be more concentrated, enabling the brachytherapy to last only 3 to 10 minutes. The beta source can be shielded only with approximately 0.5 inch of polymerized methyl methacrylate (Lucite). The FDA currently approves the use of brachytherapy only for in-stent restenosis.

Distal Protection Devices

Distal embolization of particulate matter can complicate PCI and peripheral interventional procedures. Tiny microemboli can be showered distally (downstream of the lesion) during revascularization procedures. This can cause end-organ ischemia, AMI, serum cardiac enzyme elevation, stroke, and LV dysfunction. Distal protection devices are designed to reduce or eliminate distal embolization during PCI and peripheral interventions. Distal protection devices are often used during PCI of saphenous vein grafts and during carotid procedures.

To date, the only distal protection devices approved by the FDA are the PercuSurge GuardWire (Medtronic, Santa Rosa, CA) and the FilterWire (Boston Scientific) (Fig. 18-15). The PercuSurge device consists of a guide wire with a low-pressure occlusive balloon on the distal end. The balloon is inflated to prevent distal embolization, and an aspiration catheter removes the debris from the treated vessel before the balloon is deflated and antegrade flow is restored. The FilterWire device contains a low-profile filter mounted on an angioplasty wire. The filter contains small holes that permit antegrade blood flow while trapping microemboli and thus providing distal protection. Because these distal protection devices are quickly becoming the standard of care for degenerated saphenous veins grafts and carotid stenting, device manufacturers may seek indications for use in ACS interventions and other peripheral procedures in the near future.

FIGURE 18-15 ▲ Distal protection devices. Deployed filter wire device. (Photograph courtesy of Boston Scientific Corporation, 2006.)

▲ Interventions for Peripheral Arterial Disease

PAD is a condition that affects approximately 12 million Americans.[30] The disease results from the accumulation of plaque in the arteries. It constricts normal blood flow and can result in heart attack, stroke, extremity amputation, and death if left untreated. Patients with PAD have a 5-year mortality rate of 30%.[31] Refer to Chapter 19 for a discussion of aortic aneurysms and PAD and Chapter 22 for surgical management of carotid disease (endarterectomy). Until recent advances in technology made minimally invasive and percutaneous approaches possible, medical or surgical intervention for these cardiovascular diseases was the only option.

Remote endarterectomy is a minimally invasive endovascular procedure for complete superficial femoral artery revascularization. It provides treatment of lower extremity arterial disease and serves as an alternative to bypass surgery. The benefits of the remote endarterectomy approach are (1) preservation of the native vessel, (2) less invasive than surgery, (3) no limitation of future surgical options, (4) potentially faster and easier recovery for the patient compared with bypass procedures, and (5) comparable long-term clinical outcomes to surgical endarterectomy.

Percutaneous treatment is an emerging approach in PAD management. Angioplasty, atherectomy, and stenting of the carotid arteries, aorta, renal arteries, iliac and femoral arteries, and upper extremities are routinely performed in many medical institutions. Before undergoing a percutaneous intervention, most patients require magnetic resonance angiography, arterial duplex mapping, or angiography. Percutaneous

transluminal angioplasty of the peripheral arteries involves placing a balloon in the blood vessel at the site of the blockage and inflating the balloon to open the blood vessel. Stents can also be inserted into the blocked vessel to serve as scaffolding in opening it. Thrombolytic therapy can also be delivered to the site of the blockages before initiating angioplasty or stenting. Atherectomy, or debulking the peripheral blood vessels, has also been shown to be beneficial in some cases before angioplasty or stenting.

Abdominal aortic aneurysms and thoracic aortic aneurysms can also be treated percutaneously. Endovascular stent grafts are metal-lined fabric tubes that reinforce an aneurysm in a blood vessel. It essentially relines the blood vessel and decreases the incidence of aneurysm rupture. The stent graft seals tightly above and below the aneurysm (Fig. 18-16). The graft is stronger than the weakened aorta and permits blood to pass through without exerting pressure on the aneurysm. Patients are candidates for endovascular stent grafting if aneurysms measure 5 cm wide, the aneurysm and aorta contour are conducible to stent grafting, and the blood vessels are large enough to pass guiding catheters, angioplasty balloons, and the stent graft. Potential complications of endovascular stent grafting include endoleaks (leaking of blood around the graft), migration of the stent graft, infection, and restenosis.

Postprocedure care of the patient with PAD is similar to that of patients undergoing PCI of the coronaries. Groin incision observation, extremity assessment, and vital sign measurement are important components of postprocedure care. Patient education and discharge planning should include aggressive management of cardiovascular risk factors, such as smoking cessation, reduction of blood glucose levels, exercise regimens, and lowering of blood pressure and cholesterol level. Antiplatelet therapy is also indicated for the patient undergoing percutaneous treatment of PAD.

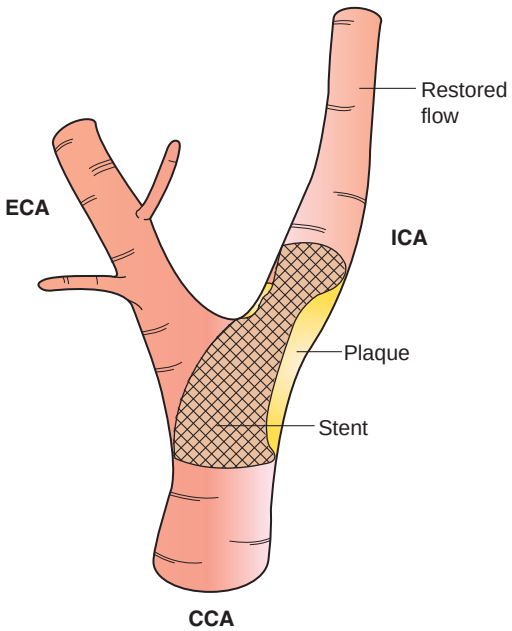


FIGURE 18-16 ▲ Endovascular stent graft (AAA). CCA, common carotid artery; ECA, external carotid artery; ICA, internal carotid artery.

With the various tools and technologies available to the interventional cardiologist, as well as improved adjunctive pharmacological therapy, the future should bring further improvement in the efficacy and predictability of PCI and in the long-term patency of involved atherosclerotic vessels in the coronary system and in the peripheral arterial system.

▲ Percutaneous Balloon Valvuloplasty

PBV is a nonsurgical technique for increasing blood flow through stenotic cardiac valves using dilation catheters. This procedure is similar to PCI procedures in that a catheter system is inserted percutaneously and advanced to the region of narrowing using fluoroscopic guidance. A dilation catheter then is inflated to increase the valvular opening and improve blood flow.

Historical Background

The first cases of balloon dilation of stenotic cardiac valves were reported in 1979 and 1982, when physicians successfully dilated pulmonary valve stenoses. This technique was considered an effective alternative to open heart surgery, although long-term results could not yet be evaluated. Because surgical commissurotomy was successful in treating mitral valve stenoses and because of the initial success with pulmonary valve dilation, physicians began percutaneous dilation of mitral valves in 1984 to avoid the need for thoracotomy. These procedures improved cardiac function with no serious procedural complications.

The number of PBVs does not approach the volume of PCI procedures. This is due partly to the lesser incidence of valve disease compared with CAD.

Assuming patients have long-term clinical improvement associated with PBV, the advantages compared with surgery are similar to those of PCI versus CABG. PBV is less traumatic, requires no anesthesia, is associated with lower morbidity and a shorter hospital stay, causes no scarring, and is less expensive. Minimally invasive surgical procedures are also available and include mini-thoracotomy approaches.

Pathophysiology of Stenotic Valves

Stenotic valves are caused by calcific degeneration, congenital abnormalities, or rheumatic heart disease. Calcific aortic and mitral valve degeneration now appears to be the most frequent causes of valve disease requiring surgical treatment. Refer to Chapter 22 for a discussion of the pathophysiology and surgical management of specific stenotic valves.

Diagnostic Tests for PBV and Valve Replacement

Before deciding on the appropriate intervention, the physician evaluates the patient for evidence and severity of valvular stenosis. A variety of noninvasive tests allow the physician to determine the degree of left atrial or LV hypertrophy, pulmonary venous congestion or hypertension, valvular rigidity,

and transvalvular gradient. In a 12-lead ECG, the magnitude of the R wave in the left precordial leads reflects the presence of LV hypertrophy associated with AV stenosis. The presence of broad, notched P waves in leads I, II, and aVL reflects left atrial hypertrophy associated with mitral valve stenosis. A chest radiograph illustrates the presence of calcium in or around the valve, LV or atrial hypertrophy, and pulmonary venous congestion or HF. A two-dimensional echocardiogram is used to scan the cardiac valves and chambers. A Doppler ultrasound study allows measurement of the transvalvular gradient, indirect calculation of valve area, and assessment of valvular regurgitation. With this information, the physician is able to (1) estimate the size of the valve orifice, (2) visualize the degree of valve leaflet movement, and (3) determine the extent of LV or atrial hypertrophy.

Right and left heart catheterization is performed if the previous tests indicate valvular disease. Although this procedure is invasive, it is required to determine the pressures within each of the cardiac chambers and to confirm transvalvular gradients. Once the pressures and gradients are obtained, a series of radiographs may be taken by injecting radiopaque contrast medium, either in the aorta to visualize aortic regurgitation or in the left ventricle to visualize mitral regurgitation. This procedure yields a cineangiogram illustrating the function of the cardiac valves and chamber sizes.

After this series of tests, the physician can analyze the valves closely, gaining precise information with which to decide the treatment mode. The nurse should be familiar with the results of these tests because a better understanding of the patient's diagnosis and related symptoms, and thus of the reasons for intervention, promotes better care.

Equipment Features

Although PBV and PCI catheters are based on similar designs, there are important differences, primarily because of the larger diameters of heart valves compared with coronary arteries. One major difference is the outer diameter of the catheters. PBV catheter shafts range from 7 to 9 Fr PBV balloons range from 15 to 25 mm in diameter when inflated. A 10- to 14-Fr introducing sheath may be used at the arterial or venous puncture site to allow for introduction of the valve dilation catheter.

A large guide wire, 0.035 to 0.038 inch, also is used to provide the added stiffness and support required to introduce the dilatation catheter. PBV dilation catheters have radiopaque markers similar to PCI catheter systems for fluoroscopic imaging.

Indications for and Contraindications to PBV

The use of PBV initially was limited by the fear of embolization of calcific debris, disruption of the valve ring, acute valvular regurgitation, and valvular restenosis. The incidence of these complications continues to be a concern. Both major and minor complications have been reported in numerous early studies; however, these complications must be assessed in terms of the patient population in which the procedure is performed.

BOX 18-11 Indications and Contraindications for PBV

Clinical Indications
- High-risk surgical patients (advanced age, severe pulmonary hypertension, renal failure, pulmonary dysfunction, LV dysfunction)
- Unstable presurgical patients
- Patients not candidates for chronic anticoagulation

Anatomical Indications
- Moderate to severe valvular narrowing
- Moderate to severe valvular calcification
- Mild valvular regurgitation

Anatomical Contraindications
- Inability to access vasculature
- Thrombus
- Severe valvular regurgitation
- History of embolic events

Although surgical valve replacement is an effective treatment for those with aortic valve stenosis and operative mortality rates are low, the operative mortality rate significantly increases in patients with multisystem disease (often, these are older patients). PBV initially has been proved a safe and efficacious alternative for these patients. It also is an effective therapy for children who are high surgical risks because it delays the need for surgery until the child is older and can better tolerate an operation. In addition, the longevity of both mechanical and bioprosthetic valves is approximately 10 to 20 years, so PBV delays or prevents the need for a second operation. Also, the long-term anticoagulation therapy required with mechanical valve prostheses is undesirable in younger patients and pregnant women. PBV also is effective for stabilizing those with poor LV function before surgery; it is contraindicated in patients with moderate to severe valvular regurgitation due to a small but significant risk for increasing valvular insufficiency with the procedure (Box 18-11).

A complication associated with PBV is excessive bleeding at the puncture site due to the large catheters required to perform dilation. The development of smaller catheters may reduce the incidence of bleeding. As with PCI, PBV catheters are being refined continually to increase procedural safety, time, and efficacy.

Procedure

The procedure is performed in the cardiac catheterization laboratory and involves many of the same steps as PCI (see earlier section on PCI procedure). Right and left heart catheterization is repeated to evaluate hemodynamic status and to obtain baseline transvalvular gradients. Coronary angiography, when indicated, is repeated to determine whether the patient still meets the criteria for valvuloplasty. Thorough repeat evaluation is necessary because a patient's status can change, precluding treatment with this intervention.

The angiographic catheter is replaced either by an introducing sheath or a dilation catheter. In mitral PBV, a venous puncture is made in the right femoral vein. During both aortic and mitral PBV, maintaining patent IV and radial or

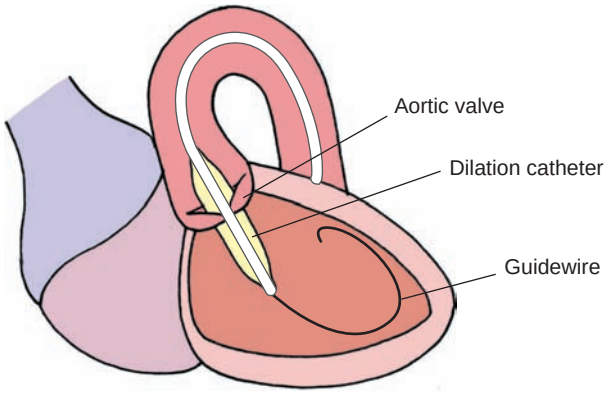


FIGURE 18-17 ▲ Cross-sectional view of heart illustrating guide wire and dilation catheter positions across the aortic valve. The guide wire is curved to prevent ventricular dysrhythmias or puncture.

femoral arterial lines is important to administer medications and draw blood samples.

In aortic PBV, once the sheaths are in place, the patient is anticoagulated with 5,000 to 10,000 U of heparin to prevent clot formation in the catheter system. The dilation catheter and guide wire then are advanced to the root of the ascending aorta. The guide wire is advanced across the stenotic aortic valve, and the dilation catheter is advanced over the guide wire (Fig. 18-17). Exact placement of the dilation catheter is facilitated by fluoroscopy and radiopaque markers on the balloon.

In mitral PBV, a pacing catheter may be positioned through a separate venous sheath at the level of the inferior vena cava or right atrium and placed on standby. The mitral valve then is approached either by way of the femoral artery and aortic valve or, in most cases, through the right heart by perforating the atrial septum to enter the left atrium. Once the mitral valve has been accessed, the patient is anticoagulated with 5,000 to 10,000 U of heparin. The dilation catheter is then advanced over the guide wire through the atrial septal puncture and across the mitral valve (Fig. 18-18). Again, exact placement of the dilation catheter in the valve is facilitated by fluoroscopy and radiopaque markers on the balloon.

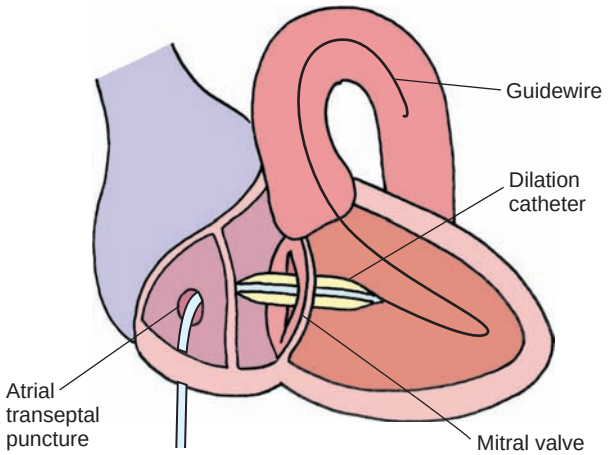


FIGURE 18-18 ▲ Cross-sectional view of heart illustrating guidewire and dilation catheter placed through an atrial transseptal puncture and across the mitral valve. The guide wire is extended out the aortic valve into the aorta for catheter support.

Average inflation time of the dilation catheter is 15 to 60 seconds in aortic valvuloplasty and 10 to 30 seconds in mitral valvuloplasty. During dilation of either valve, the nurse monitors blood pressure closely because of the imposed decrease in cardiac output. Once the dilation catheter has been deflated, blood pressure should return to normal. During dilation of the mitral valve, there is a temporary increase in the pulmonary artery occlusion pressure (PAOP; formerly known as pulmonary artery wedge pressure [PAWP]). Once the dilation catheter has been deflated, the PAOP should return to baseline. Dysrhythmias, such as VT, VF, or sinus bradycardia, also may occur during dilation.

Once maximal dilation has been obtained, the catheter is removed. Hemodynamic measurements, including transvalvular gradients, are repeated to determine efficacy of the procedure. Repeat angiography is performed to assess for valvular regurgitation. When the procedure is complete, the anticoagulant effects of heparin are reversed to prevent bleeding complications associated with the large puncture site.

Results

Aortic PBV is associated with a decrease in pressure gradient and end-systolic volume and an increase in aortic valve area, EF, and cardiac output. Although there is an increase in the aortic valve area, it is not as great as with surgical valve replacement. In addition, the restenosis rate associated with PBV is high. Therefore, aortic valvuloplasty is indicated primarily for older and high-risk surgical patients and generally is considered a palliative, not a curative, procedure.

Results of mitral valvuloplasty are more dramatic. There is a more significant increase in valve area and cardiac output and a decrease in valve gradient, PAOP, and mean pulmonary arterial pressure. Three mechanisms have been postulated for improving valvular function due to PBV: (1) fracture of calcific nodules adherent to leaflets (most frequent), (2) separation of fused commissures, and (3) stretching of the annulus and leaflet structure.

Assessment and Management

Patient Preparation

The patient is admitted to the hospital the day of the PBV procedure. The goal of nursing care is to reduce the cardiac workload, monitor fluid and electrolyte balance, and reduce psychological stress so that the patient remains hemodynamically stable.

In most cases, the patient does not have invasive pressure monitoring lines in place before the procedure. The nurse therefore carefully monitors signs and symptoms of HF: narrowing in the arterial pulse pressure, more frequent increases in heart rate during activity, peripheral edema, presence of a cough, complaints of dyspnea, or rales in lung fields. The nurse also must note any changes in sensorium, color, skin temperature, and pulse volume, and any decrease in urinary output. To monitor fluid and electrolyte balance, the nurse obtains a baseline serum electrolyte level and baseline body weight. In addition, daily fluid intake and output are recorded.

The patient's medications before admission may have included diuretics, digoxin, and anticoagulants. Before the procedure, any anticoagulant medication is discontinued because of the possibility of emergency surgery. Therefore, patients with chronic AF who have the potential for systemic embolization due to thrombus should be monitored closely. The nurse also monitors preliminary laboratory tests and notifies the physician of any abnormalities. (See the section on patient preparation for PTCA for further information on these tests.)

After the patient fully understands the procedure, the physician must obtain an informed consent for PBV, anesthesia, and surgery. Surgical standby usually is provided during PBV due to possible complications requiring emergency valve replacement.

Nursing Assessment and Management During PBV

The nurse continuously monitors pulmonary artery pressure and PAOP and is aware of changes in tracings that may suggest symptoms of HF or pulmonary edema. In the presence of severe hypotension, the nurse should be prepared to start an IV infusion of dopamine or norepinephrine (Levophed). In the case of ventricular dysrhythmias, a lidocaine drip should be available for infusion.

Nursing Assessment and Management After Percutaneous Balloon Valvuloplasty

The nurse is important in the patient's recovery. The goal of postvalvuloplasty nursing care is to maintain adequate cardiac output, maintain fluid and electrolyte balance, and verify hemostasis at the puncture site. Alterations in cardiac output can be caused by dysrhythmias secondary to valve manipulation, resulting in edema near the bundle of His; left-to-right atrial shunt through the transseptal puncture created during mitral valvuloplasty; cardiac tamponade; alteration in circulating fluid volume; or blood loss. Alteration in fluid and electrolyte balance results from diuretic therapy and contrast medium used during catheterization. Bleeding at the puncture site is secondary to the combined effect of systemic anticoagulation and the large diameter of catheters used.

Because fluids are important in the hemodynamic balance of the patient with valvular disease, the volume of IV fluids is recorded to establish an accurate intake and output. The decreased circulating volume from diuretic medications given before PBV, combined with improved stroke volume after successful PBV, can be reflected as a decrease in cardiac output. Therefore, careful monitoring of central venous pressure, pulmonary artery pressure, PAOP, and blood pressure, in addition to heart rate, urinary output, and electrolyte balance, is essential in the evaluation and assessment of circulating fluid volume and cardiac pumping status.

In addition, the nurse assesses the patient's status from head to toe, noting overall skin color and temperature and carefully observing the level of consciousness and neurological signs. The nurse also listens closely to heart and breath sounds. Circulation distal to the puncture site is evaluated by noting peripheral skin color and temperature in addition to the presence and quality of the dorsalis pedis and posterior tibial pulses.

Finally, any drainage appearing on the puncture site dressing or tenderness during palpation should be noted to establish a baseline for the possibility of increased pericatheter bleeding. The nurse reports immediately any changes that may indicate excessive bleeding. Bleeding at the sheath site may result in a hematoma requiring surgical evacuation. To prevent excessive bleeding and to aid hemostasis, the physician may order a sandbag or clamp placed over the puncture site.

If the patient has documented CAD, the physician also may request a serum cardiac enzyme panel. Particular attention should be paid to creatine kinase (CK) and CK isoenzymes (see Nursing Management After Percutaneous Coronary Interventions). The nurse should be aware of the signs and symptoms of myocardial ischemia in addition to the appropriate interventions.

The nurse instructs the patient about the importance of keeping the involved leg straight for the first few hours after valvuloplasty.

Post-PBV laboratory evaluation may include PT, hemoglobin and hematocrit, coagulation studies, serum electrolytes, CK, ECG, and chest radiograph. Box 18-6, under Nursing Management During Percutaneous Coronary Interventions, lists nursing diagnoses and collaborative problems for patients undergoing PBV. Teaching points for home care for the patient after PCI or PBV are described in Box 18-12.

Complications

A common in-hospital complication associated with PBV is bleeding at the arterial puncture site due to the large diameter of the catheters needed to dilate the valve annulus. In addition, in mitral PBV, a common complication is left-to-right shunting secondary to septal dilation, again due to the large diameter of the dilation catheters. Systemic

BOX 18–12 TEACHING GUIDE *Advising the Cardiac Patient After PCI or PBV*

Physiological Teaching Points

- Restrict physical activities during the first week after PCI/PBV.
- Avoid lifting more than 10 pounds the first 2 weeks after PCI.
- Resume exercise program after undergoing an exercise stress test.
- Follow prescribed low-fat diet.
- Consider cardiac rehabilitation.
- Limit alcohol to three drinks per week.
- Notify physician of any oozing, bleeding, or pain at puncture site.
- Notify physician of fever or other signs of infection.
- Notify physician or call 911 for any chest discomfort not relieved with three nitroglycerin tablets taken 5 minutes apart.
- Begin a weight loss program if indicated.

Psychosocial Teaching Points

- Stop smoking, if appropriate, and avoid exposure to second-hand smoke.
- Resume sexual activities after completing an exercise stress test.
- Begin stress management activities.
- Recognize signs of depression.
- Comply with medication regimen.
- Schedule and keep medical appointments

BOX 18-13 PATIENT SAFETY

Serious Complications of PBV That Need Intervention

- Embolization of calcific debris
- Valve ring disruption
- Valvular regurgitation
- Valvular restenosis
- Bleeding at arterial puncture site
- LV perforation
- Severe hypotension
- Transient ischemia
- Vascular trauma
- Atrial septal defect (with mitral PBV)
- Aortic dissection
- Aortic rupture
- Cardiac tamponade
- Chordae tendineae rupture

embolization in both mitral and aortic PBV is a potential and significant complication, although its incidence is low. There have been few reports of significant increases in valvular regurgitation. Complications associated with PBV are listed in Box 18-13.

INTRA-AORTIC BALLOON PUMP COUNTERPULSATION AND MECHANICAL CIRCULATORY SUPPORT

▲ Intra-Aortic Balloon Pump Counterpulsation

Harken and colleagues of Boston originally described the concept of counterpulsation in 1958 when, in an attempt to increase coronary artery perfusion, they used femoral access to remove blood during systole and replace it during diastole. IABP counterpulsation was first introduced clinically by Kantrowitz and associates in 1967. This therapeutic approach was instituted for treatment of two patients with LV failure after AMI. Since that time, IABP has become a standard treatment for medical and surgical patients with acute LV failure that is unresponsive to pharmacological and volume therapy.

IABP counterpulsation is designed to increase coronary artery perfusion pressure and blood flow during the diastolic phase of the cardiac cycle by inflation of a balloon in the thoracic aorta. Deflation of the balloon, just before systolic ejection, decreases the impedance to ejection (afterload) and thus LV work, with subsequent decreased myocardial oxygen consumption. Inflation and deflation counterpulse each heart beat. With improved blood flow and effective reduction in LV work, the desired results are increased coronary artery perfusion and decreased afterload with subsequent increase in cardiac output. Goals are directed toward increasing oxygen supply to the myocardium, decreasing LV work, and improving cardiac output. Before IABP, no single therapeutic agent was capable of meeting these three goals.

The ACC/AHA guidelines for management of AMI consider IABP counterpulsation therapy a class I recommendation for the following conditions: (1) hypotension, defined as systolic blood pressure less than 90 mm Hg, or 30 mm Hg below baseline mean arterial pressure (MAP), in patients with STEMI who do not respond to other interventions; (2) low output state in patients with STEMI; and (3) cardiogenic shock that has not been quickly reversed with pharmacological agents in patients with STEMI.[1] The ACC/AHA guidelines also regard IABP counterpulsation to be a class I recommendation when used with other medical therapy for patients with STEMI and recurrent ischemic-type chest discomfort with signs of hemodynamic instability, poor LV function, or a large area of myocardium at risk.[1]

While IABP therapy remains an ACC/AHA class I recommendation, recent studies challenge the current recommendations and suggest that a review and update of the clinical practice guidelines are necessary.[2]

Physiological Principles

Greater work is required to maintain cardiac output in the failing heart. With this added work requirement, oxygen demand increases. These circumstances may occur at a time when the myocardium already is ischemic and coronary artery perfusion is unable to meet the oxygen demands. As a result, LV performance diminishes even further, resulting in decreased cardiac output. A vicious cycle ensues that is difficult to interrupt (Fig. 18-19). Without interruption of the cycle, cardiogenic shock may be imminent. This cycle can be broken with IABP therapy by increasing aortic root pressure during diastole through inflation of the balloon. With increased aortic root pressure, the perfusion pressure of the coronary arteries is increased.

Effective therapy for the patient in LV failure also involves decreasing myocardial oxygen demand. Four major determinants of myocardial oxygen demand are afterload, preload, contractility, and heart rate. IABP counterpulsation therapy can have an effect on all these factors. It decreases afterload directly and affects the other three determinants indirectly as cardiac function improves. Because IABP therapy assists the left heart, only the left ventricle is discussed here.

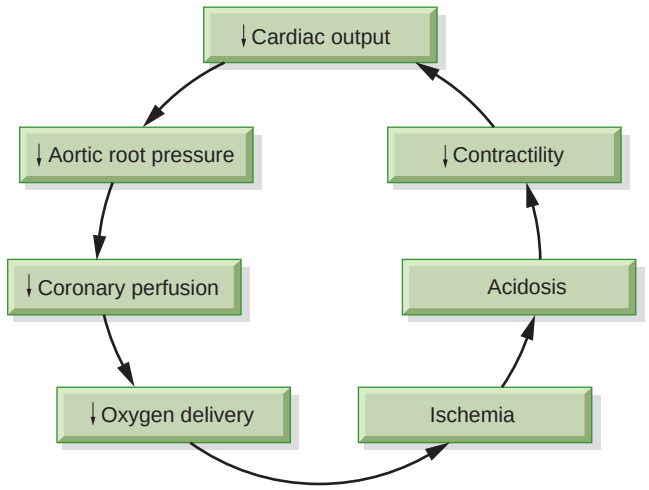


FIGURE 18-19 ▲ Cycle leading to cardiogenic shock.

Afterload and Preload

The greatest amount of oxygen required during the cardiac cycle is for the development of afterload (see Chapter 16). With greater impedance to ejection, afterload increases, resulting in increased myocardial oxygen demand. Impedance to ejection is caused by the aortic valve, aortic end-diastolic pressure, and vascular resistance. Greater aortic end-diastolic pressures require higher afterload to overcome impedance and ejection. Vascular resistance increases impedance when vessels become vasoconstricted. Vasodilation or lower vascular resistance decreases afterload by decreasing impedance to ejection. Deflation of the balloon in the aorta just before ventricular systole lowers aortic end-diastolic pressure. This decreases impedance to ejection and decreases LV workload. In this way, IABP can effectively decrease the oxygen demand of the heart.

A person in acute LV failure has increased volume in the ventricle at end-diastole (preload; see Chapter 16) as a result of the heart's inability to pump effectively. This excessive increase in preload increases the workload of the heart. IABP therapy helps decrease excessive preload by decreasing impedance to ejection. With decreased impedance, there is more effective forward flow of blood and more efficient emptying of the left ventricle.

Contractility

Contractility refers to the velocity and vigor of contraction during systole. Although vigorous contractility requires more oxygen, it is a benefit to cardiac function because it ensures good, efficient pumping, which increases cardiac output. In the patient with HF, cardiac contractility is depressed. The biochemical status of the myocardium directly affects contractility. Contractility is depressed when calcium levels are low, catecholamine levels are low, and ischemia is present with resultant acidosis.

IABP counterpulsation can increase oxygen supply, thereby decreasing ischemia and acidosis. In this way, IABP therapy contributes to improved contractility and better cardiac function (see Fig. 18-19).

Heart Rate

Heart rate is a major determinant of oxygen demand because the rate determines the number of times per minute the high pressures must be generated during systole. Normally, myocardial perfusion takes place during diastole. Coronary artery perfusion pressure is determined by the gradient between aortic diastolic pressure and myocardial wall tension. PAOP estimates wall tension and resistance to perfusion by approximating LV end-diastolic volume. It can be expressed by the following equation:

Coronary perfusion pressure =
aortic diastolic pressure – myocardial wall tension

Tension in the muscle retards blood flow, which is why approximately 80% of coronary artery perfusion occurs during diastole. With faster heart rates, diastolic time becomes shortened, with very little change occurring in systolic time. A rapid heart rate not only increases oxygen demand but decreases the time available for oxygen delivery. In acute ventricular failure, a person may not be able to maintain cardiac output by increasing the volume of blood pumped with each beat (stroke volume) because contractility is depressed. Cardiac output is a function of both stroke volume and heart rate:

Cardiac output = stroke volume × heart rate

If stroke volume cannot be increased, heart rate must increase to maintain cardiac output. This increase is very costly in terms of oxygen demand.

By improving contractility, IABP therapy helps improve myocardial pumping and the ability to increase stroke volume. Decreasing afterload also increases pumping efficiency. With improved myocardial function and cardiac output, the need for compensatory tachycardia diminishes. IABP counterpulsation increases coronary artery perfusion pressure by increasing aortic diastolic pressure during inflation of the balloon, resulting in improved blood flow and oxygen delivery to the myocardium.

The physiological effects of IABP therapy are summarized in Box 18-14. Proper inflation of the balloon increases oxygen supply, and proper deflation of the balloon decreases oxygen demand. Timing of inflation and deflation is crucial and must coincide with the cardiac cycle.

BOX 18-14 Direct Physiological Effects of Intra-Aortic Balloon Pump (IABP) Therapy

Inflation
↑ Aortic diastolic pressure
↑ Aortic root pressure
↑ Coronary perfusion pressure
↑ Oxygen supply

Deflation
↓ Aortic end-diastolic pressure
↓ Impedance to ejection
↓ Afterload
↓ Oxygen demand

Equipment Features

The intra-aortic balloon catheter and the balloon mounted on the end are constructed of a biocompatible polyurethane material. Filling of the balloon is achieved with a pressurized gas that enters through the catheter. Because of its low molecular weight, helium is the pressurized gas of choice. Balloon size should be determined by the patient's physical stature to optimize counterpulsation (Table 18-9). With inflation, the addition of the balloon volume into the aorta acutely increases aortic pressure and retrograde blood flow

Table 18-9 IABP Balloon Size Guidelines

Patient Height	Balloon Volume	Body Surface Area
<5′4″	30 mL	1.8 m^2 or less
5′4″–6′0″	40 mL	<1.8 m^2
>6′0″ (or aortic diameter >20 cm)	50 mL	>1.8 m^2

BOX 18-15 Indications for IABP Therapy

- Cardiogenic shock after acute infarction
- LV failure in the postoperative cardiac surgery patient
- Severe unstable angina
- Postinfarction ventricular septal defect or mitral regurgitation
- Short-term bridge to cardiac transplantation

back toward the aortic valve. With deflation, the sudden evacuation of the balloon volume acutely decreases aortic pressure. Catheters have a central lumen with which aortic pressure can be measured from the tip of the balloon.

Indications for Intra-Aortic Balloon Pump Counterpulsation

Two major applications of IABP therapy are for treatment of cardiogenic shock after MI and for low cardiac output following cardiac surgery. Other applications of IABP therapy for patients with cardiac pathophysiological conditions are noted in Box 18-15.

Cardiogenic Shock

Treatment of cardiogenic shock is complicated, and the mortality rate remains high. Cardiogenic shock develops in approximately 15% of patients with MI.

Initially, patients are treated with various inotropic drugs, vasopressors, and volume. A lack of, or minimal response in, cardiac output, arterial pressure, urine output, and mental status after this therapy indicates a need for assisted circulation with IABP therapy. Once hypotension is present, the self-perpetuating process of injury is in effect. Control of further injury and improvement in survival require early reversal of the shock state.

After IABP therapy is instituted, improvement should be observed within 1 to 2 hours. At this time, steady improvement should be seen in cardiac output, peripheral perfusion, urine output, mental status, and pulmonary congestion. With improved cardiac function, a decrease in central venous pressure and PAOP also should be seen. Average peak effect should be achieved within 24 to 48 hours.

Postoperative Low Cardiac Output

The primary indication for use of IABP therapy in the perioperative cardiac surgery patient is low cardiac output refractory to traditional inotropic support.[3] IABP therapy is also used preoperatively in patients who have sustained mechanical injury resulting from AMI as well as those with refractory angina.

IABP counterpulsation therapy can be used to wean patients from cardiopulmonary bypass (CPB) and to provide postoperative circulatory assistance until LV recovery occurs. In these situations, early recognition of failure is evidenced by the heart's inability to support circulation after CPB. Early recognition and treatment are crucial if LV failure is to be reversed.

In addition to providing circulatory assistance, outcomes in cardiac surgery patients have been positively influenced by IABP through other properties as well. For example, the pulsatile blood flow produced by IABP therapy during CPB has been shown to decrease activation of the systemic inflammatory response through inhibition of endothelial activation.[4] IABP therapy has also been associated with improvements in whole body perfusion during CPB.[5]

Unstable Angina

IABP counterpulsation therapy may be used during PCI for patients with unstable angina or mechanical problems. In this situation, PCI procedures usually are followed by emergency cardiac surgery. Patients in this category include those with unstable angina, postinfarction angina and postinfarction ventricular septal defects, or mitral regurgitation from papillary muscle injury with resultant cardiac failure. IABP counterpulsation therapy has been used successfully to control the severity of angina in patients in whom previous medical therapy has failed. The use of IABP therapy for patients with cardiac failure after ventricular septal rupture or mitral valve incompetence aids in the promotion of forward blood flow, which decreases shunting through the septal defect and decreases the amount of mitral regurgitation.

Contraindications to IABP Counterpulsation

There are few contraindications to the use of IABP therapy. A competent aortic valve is necessary if the patient is to benefit from IABP therapy. With aortic insufficiency, balloon inflation would only increase aortic regurgitation and offer little, if any, augmentation of coronary artery perfusion pressure. In fact, the patient's HF could be expected to become worse.

Severe peripheral vascular occlusive disease also is a relative contraindication to the use of IABP therapy. Occlusive disease would make insertion of the catheter difficult and possibly interrupt blood flow to the distal extremity or cause dislodgment of plaque formation along the vessel wall, resulting in potential emboli. In patients who absolutely require IABP therapy, insertion can be achieved through the thoracic aorta, thus bypassing diseased peripheral vessels. Any previous aortofemoral or aortoiliac bypass graft contraindicates femoral artery insertion.

In addition, the presence of an aortic aneurysm is a contraindication to the use of IABP therapy. A pulsating balloon against an aneurysm may predispose the patient to dislodgment of aneurysmal debris with resultant emboli. A more serious complication is rupture of the aneurysm; it is possible for the catheter to perforate the wall of the aneurysm during insertion.

Procedure

Insertion

Proper positioning of the balloon is in the thoracic aorta just distal to the left subclavian artery and proximal to the renal arteries (Fig. 18-20). The most commonly used method of catheter placement is percutaneous insertion using a Seldinger technique, although other approaches have been

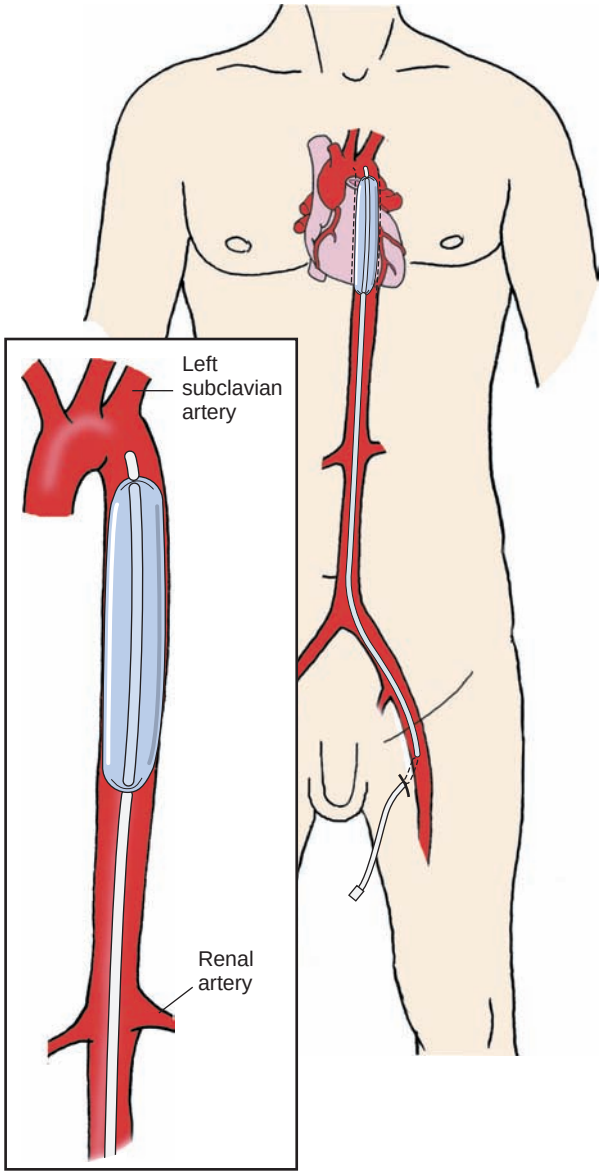


FIGURE 18-20 ▲ Proper position of the balloon catheter; illustrating percutaneous insertion.

described. The most common alternative is direct insertion into the thoracic aorta. Because this requires a median sternotomy incision, it is restricted to cardiac surgical patients whose chests have been opened for the surgery.

Once in place, the catheter is attached to a machine console that has three basic components: a monitoring system, an electronic trigger mechanism, and a drive system that moves gas in and out of the balloon. Monitoring systems have the capability of displaying the patient's ECG and an arterial waveform showing the effect of balloon inflation–deflation. Consoles also are capable of displaying a balloon waveform that illustrates the inflation and deflation of the balloon itself. The standard trigger mechanism for the balloon pump is the R wave that is sensed from the patient's ECG. This trigger signals the beginning of each cardiac cycle for the drive system. Other possible triggers include systolic arterial pressure or pacemaker spikes on the ECG. Adjustment of exact timing is controlled on the machine console. The drive system is the actual mechanism that drives gas into and out of the balloon by alternating pressure and vacuum.

Timing

Two primary methods of timing can be used with IABP therapy: conventional timing and real timing. Conventional timing uses the arterial waveform as the triggering mechanism to determine both inflation and deflation of the balloon. Real timing uses the same point of reference (the dicrotic notch on the arterial waveform) for balloon inflation but uses the ECG signal as the trigger for balloon deflation. Real timing is discussed briefly after conventional timing.

CONVENTIONAL TIMING. The first step to proper timing of the balloon pump using conventional timing is the identification of the beginning of systole and diastole on the arterial waveform. Systole begins when LV pressure exceeds left atrial pressure, forcing the mitral valve closed.

There are two phases of systole: isovolumetric contraction and ejection. Once the mitral valve is closed, isovolumetric contraction begins and continues until enough pressure is generated to overcome impedance to ejection. When ventricular pressure exceeds aortic pressure, the aortic valve is forced open, initiating ejection, or phase 2. Ejection continues until pressure in the left ventricle falls below pressure in the aorta. At this point, the aortic valve closes, and diastole begins.

Closing of the aortic valve creates an artifact on the arterial waveform that is called the dicrotic notch. The dicrotic notch is used as a timing reference to determine when balloon inflation should occur. Inflation should not occur before the notch because systole has not been completed.

After the aortic valve closes, two phases of diastole begin: isovolumic relaxation and ventricular filling. After the aortic valve closes, there is a period in which neither the aortic nor mitral valve is open. The mitral valve remains closed because LV pressure still is higher than left atrial pressure. This phase is isovolumic relaxation. When LV pressure falls below left atrial pressure, the mitral valve is forced open by the higher pressure in the left atrium. This begins the filling phase of diastole. Balloon inflation should continue throughout diastole. Deflation should be timed to occur at end-diastole, just before the next sharp systolic upstroke on the arterial waveform.

Figure 18-21 illustrates the cardiac cycle with left atrial, LV, and aortic pressures superimposed on one another.

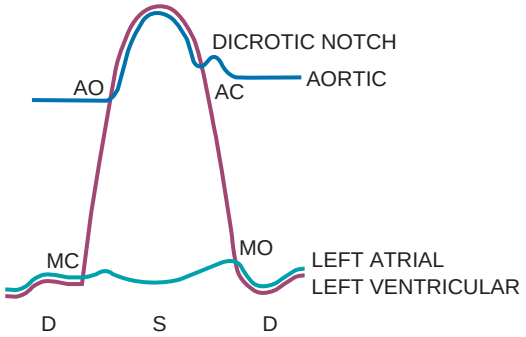


FIGURE 18-21 ▲ Cardiac cycle of the left heart with aortic, left ventricular (LV), and left atrial pressure waveforms. AC, aortic valve closure; AO, aortic valve opening; D, diastole; MC, mitral valve closure; MO, mitral valve opening; S, systole.

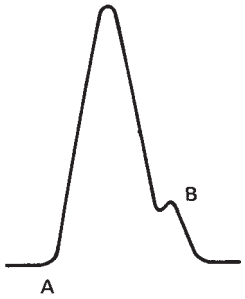


FIGURE 18-22 ▲ Arterial waveform, with *A* representing the point of balloon deflation before the systolic upstroke, and *B* representing balloon inflation at the dicrotic notch, at diastole.

Figure 18-22 illustrates a radial artery waveform with the beginning of systole and diastole marked.

REAL TIMING. The main difference between the two timing methods is balloon deflation and the triggering mechanism used. Real timing uses the ECG as the trigger signal for balloon deflation. The QRS complex is recognized as the onset of ventricular systole, and balloon deflation occurs at this time. Triggering off the R wave allows for balloon deflation to occur at the time of systolic ejection and not before (as with conventional timing). This timing mechanism is more effective in patients with irregular heart rhythms because balloon deflation occurs on recognition of the R wave (systolic ejection). It does not need to be approximated by the operator or an algorithm, as in conventional timing. Both a rapid deflation mechanism and a reliable ECG signal are necessary for IABP using real timing to augment blood pressure effectively. Balloon inflation with real timing occurs at the onset of diastole as triggered by the dicrotic notch on an arterial waveform, just as in conventional timing.

Advances in IABP technology have led to the development of automatic timing mechanisms currently available in some IABP models. Automatic timing therapy is possible because of special IABP catheters that have fiber optic pressure sensors in the tip.[3] These pressure sensors, capable of transmitting real-time pressure signals at the speed of light, use Windkessel model algorithms to calculate real-time aortic flow from aortic pressure.[6] This allows the balloon pump to determine the precise time when the aortic valve closes with each contraction of the heart, regardless of the patient's heart rhythm. The closure of the aortic valve signals the onset of diastole, and balloon inflation occurs.

Interpretation of Results

Waveform Assessment

Analysis of the arterial pressure waveform and the effectiveness of IABP therapy is an important nursing function. Nurses must be able to recognize and correct problems in balloon pump timing. Figure 18-23 illustrates the five points that are assessed on the waveform.

STEP 1. The first step in timing assessment is the ability to recognize the beginnings of systole and diastole on the arterial waveform, as shown in Figure 18-23. Systole begins at point A, where the sharp upstroke begins. Point B marks

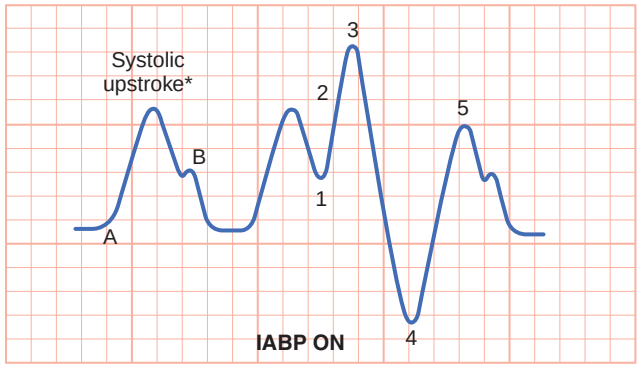


FIGURE 18-23 ▲ Inspection of the arterial waveform with intra-aortic balloon assistance should include observation of (*1*) inflation point, (*2*) inflation slope, (*3*) diastolic peak pressure/diastolic augmentation, (*4*) end-diastolic dip, and (*5*) next systolic peak.

the dicrotic notch, which represents aortic valve closure. At this point, diastole begins, and the balloon should be inflated. Balloon deflation occurs just before point A, at end-diastole.

Box 18-16 lists five criteria that can be used to measure the effectiveness of IABP therapy on the arterial pressure waveform. To evaluate the waveform effectively, the patient's unassisted pressure tracing must be viewed alongside the assisted pressure tracing. This can be accomplished through adjustment of the console so that the balloon inflates and deflates on every other beat (ie, a 1:2 assist ratio). Most patients tolerate this well for a brief period of time. Machine consoles are capable of freezing the waveform on the console monitor so that it is necessary to assist at a 1:2 ratio only for one screen. Another alternative is to obtain a strip recording of the 1:2 assistance for analysis.

STEP 2. After identification of the patient's dicrotic notch, a comparison is made with the assisted tracing to see that inflation occurs at the point of the dicrotic notch. Inflation before the dicrotic notch shortens systole abruptly and increases ventricular volume as ejection is interrupted. Late inflation, past the dicrotic notch, does not raise coronary artery perfusion pressure. The peak-diastolic pressure may not be as high as it would be with proper timing.

STEP 3. Next, the slopes of systolic upstroke and diastolic augmentation (also known as diastolic peak pressure) should be compared. The diastolic slope should be sharp and parallel

BOX 18-16 Criteria for Assessment of Effective IABP Therapy on the Arterial Pressure Waveform

- Inflation occurs at the dicrotic notch.
- Inflation slope is parallel to the systolic upstroke and is a straight line.
- Diastolic augmentation peak is greater than or equal to the preceding systolic peak.
- An end-diastolic dip in pressure is created with balloon deflation.
- The following systolic peak (assisted systole) is lower than the preceding systole (unassisted systole).

the systolic upstroke, as shown in Figure 18-23. The slope always should be a straight line. The greater the peak in diastolic pressure, the greater the increase in aortic root pressure. For this reason, balloon assistance is adjusted until the highest peak possible is achieved.

STEP 4. Deflation should occur just before systole, causing an acute drop in aortic end-diastolic pressure. This quick deflation displaces approximately 40 mL of volume. The result is an end-diastolic dip in pressure that reduces the impedance to the next systolic ejection. The end-diastolic pressure without the balloon assistance should be compared with the end-diastolic pressure with the dip created by balloon deflation. Optimally, a pressure difference of at least 10 mm Hg should be obtained. Better afterload reduction is achieved with the lowest possible end-diastolic dip.

The point of deflation also is crucial. Deflation that is too early allows pressure to rise to normal end-diastolic levels preceding systole. In this situation, there is no decrease in afterload. Deflation that is too late encroaches on the next systole and actually increases afterload because of greater impedance to ejection from the presence of the still-inflated balloon during systolic ejection. Figure 18-24 demonstrates possible errors in timing.

STEP 5. Finally, if afterload has been reduced, the next systolic pressure peak will be lower than the unassisted systolic pressure peak. This implies that the ventricle did not have to generate as great a pressure to overcome impedance to ejection. This may not always be seen because the systolic pressure peak also represents the compliance of the vasculature. If the vasculature is noncompliant due to atherosclerotic disease, the systolic peak may not change very much.

Balloon Fit

The fit of the balloon to any particular patient's aorta determines how well these criteria are met. Ideally, approximately 80% of the aorta is occluded with balloon inflation. In a dilated aorta, in which less than 80% occlusion occurs, the effect of inflation and deflation is not as dramatic on the waveform. When a patient is hypotensive or hypovolemic, the balloon does not have as pronounced an effect on the waveform because there is less volume displacement as the balloon inflates or deflates. See Table 18-9 on page 328 for a review of balloon size guidelines.

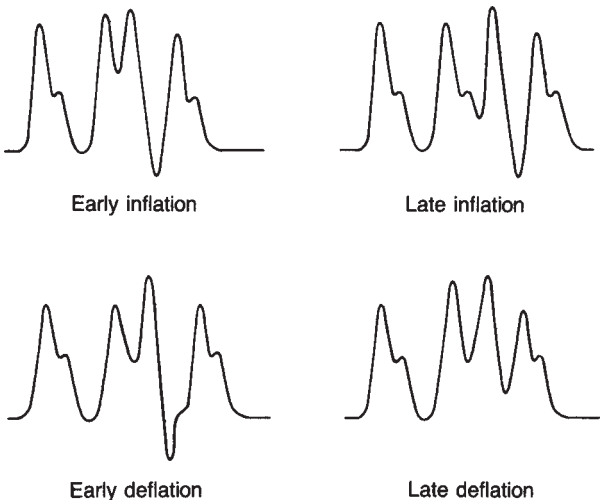


FIGURE 18-24 ▲ Illustration of possible errors occurring with timing.

Assessment and Management

Patients requiring IABP are managed much like any other critically ill patient in cardiogenic shock or acute LV failure. Nursing assessment and management of these conditions are discussed in Chapter 54. Additional nursing skills and assessment considerations specific to IABP therapy must be included in the care of these patients. These are summarized in Box 18-17. Nursing diagnoses for patients with an IABP are listed in Box 18-18.

Monitoring the Cardiovascular System

Monitoring the cardiovascular system is extremely important in determining the effectiveness of IABP therapy. The basis for this assessment includes vital signs, cardiac output, heart rhythm and regularity, urine output, color, perfusion, and mentation.

VITAL SIGNS. Three important vital signs with respect to IABP therapy are heart rate, MAP, and PAOP. Effective IABP therapy causes a decrease in all three parameters. Acute changes in the MAP may indicate volume depletion. Critically ill patients tolerate little change in their volume status. The PAOP is an important parameter for monitoring volume and provides the clinician with an early indication of volume depletion or overload.

Blood pressure readings require special consideration. Because the balloon inflates during diastole, peak-diastolic pressure may be higher than peak-systolic pressure. Most IABP consoles have monitoring systems capable of distinguishing systole from peak diastole; however, some monitoring equipment can distinguish only peak pressures from low-point pressures. For this reason, a monitor's digital display of systolic pressure actually may represent peak-diastolic pressure. It is advisable to record blood pressure as systolic, peak-diastolic, and end-diastolic, that is, 100/110/60. These pressures can be read from a strip recording of the arterial waveform.

HEART RHYTHM AND REGULARITY. Heart rhythm and regularity are important considerations. Early recognition and treatment of dysrhythmias are crucial for effective IABP support. Irregular dysrhythmias may inhibit efficient IABP therapy with some types of consoles because timing is set by the regular R-R interval on the ECG. A safety feature of all balloon pump consoles is automatic deflation of the balloon for premature QRS complexes. One particular IABP model tracks real time versus any average of beats, so it more effectively tracks dysrhythmias. If the dysrhythmia persists and timing is ineffective, another alternative might be use of the systolic peak on the arterial waveform as the trigger mechanism for balloon inflation. The primary goal is to treat the dysrhythmia.

OTHER OBSERVATIONS. Urine output, color, perfusion, and mentation all are important assessment parameters for determining the adequacy of cardiac output. All should

BOX 18-17 NURSING INTERVENTIONS

For IABP Counterpulsation and Ventricular Assist Devices (VADs)

IABP Nursing Interventions

- Verify correct timing using assist ratio of 1:2 and document settings hourly.
- Reevaluate timing for any change in the heart rate greater than 10 beats/min.
- Maintain proper balloon volume and refill as needed every 2 to 4 hours. Use automatic filling mode if available. Avoid hip flexion, which may impair gas movement in and out of IABP catheter.
- Maintain good arterial waveform and adequate ECG signal for evaluation of timing.
- Transduce aortic arterial line to the IABP per unit protocol.
- Reduce or eliminate situations that will interfere with the IABP's ability to maintain proper assist ratio. Notify physician of the development of tachycardias or irregular rhythms, and treat dysrhythmias with drug therapy or pacing as ordered. Use appropriate trigger (ie, ECG, arterial pressure, pacing).
- Use pacer modes only if the patient is 100% paced.
- Notify physician of significant changes in balloon pressure waveform.

VAD Nursing Interventions

- Assess and maintain adequate filling pressures during immediate postoperative phase.
- Monitor and assess heart rate, blood pressure, mean arterial pressure, pump flow, urine output, and neurological status hourly. Treat changes as ordered.
- Assess and change equipment level for devices that require specific placement of equipment for adequate pump flow.
- Evaluate pump flow and rate of VAD in relation to native heart rate and activity level of patient.
- Manage VAD function and volume status as ordered to maintain adequate device output.

General Nursing Interventions

- Monitor and record temperatures every 4 hours and PRN.
- Observe all insertion sites and incisions for signs of infection. Maintain sterile technique with dressing changes.
- Change any dressing that is wet or not intact.
- Change all infusion lines and infusion bags per unit protocol.
- Culture any site with suspicious drainage, redness, or swelling.
- Notify physician of elevation in white blood cell count.
- Treat patient with antibiotics as ordered.
- Auscultate and document breath sounds every 2 to 4 hours.
- Assist patient with pulmonary toilet (ie, coughing, deep breathing, frequent turning). Suction intubated patients as needed.
- Use pulse oximeter to monitor patients with abnormal blood gas levels, excessive secretions, or respiratory difficulty.
- Extubate patient and increase activity level as tolerated—particularly for patients with VADs.
- Document quality of peripheral pulses and neurological status before IABP or VAD insertion. Assess and document quality of pulses, skin perfusion, and neurological status per protocol. Evaluate peripheral perfusion with any complaints of leg or foot pain by patient.
- Notify physician of any changes in pulses or neurological status.
- Maintain anticoagulation as ordered.
- Avoid hip flexion, which might obstruct flow to the affected extremity, by keeping the cannulated leg straight and the bed at angle less than 30 degrees.
- Always maintain balloon motion to avoid thrombus formation on the balloon.
- Assess skin integrity, and document any redness and ulcerations over bony prominences.
- Use sheepskin, foam pads, and specialty beds as needed. Turn patient every 2 hours.
- Ensure that skin remains clean and dry.
- Maintain adequate nutrition by encouraging oral intake or implementing use of parenteral or enteral nutrition when necessary.
- Maintain alarm volumes, monitor noise at lowest level possible, and minimize unnecessary noise in the room.
- Talk with and orient patient to date and time frequently.
- Encourage family visits.
- Explain all procedures and activities to the patient.
- Organize care to allow for periods of uninterrupted sleep. Turn the lights off in room at night if possible.
- Sedate patient if necessary and as tolerated per physician orders.

BOX 18-18 EXAMPLES OF NURSING DIAGNOSES

For the Patient Requiring Circulatory Support With an IABP

- Decreased Cardiac Output related to alterations in preload
- Decreased Cardiac Output related to alterations in afterload
- Decreased Cardiac Output related to alterations in heart rate and rhythm
- Ineffective Peripheral Tissue Perfusion related to LV failure
- Ineffective Peripheral Tissue Perfusion related to unstable angina
- Ineffective Peripheral Tissue Perfusion related to improper IABP timing
- Risk for Infection related to invasive procedure
- Impaired Bed Mobility related to dependency on mechanical device
- Risk for Impaired Skin Integrity related to decreased perfusion
- Disturbed Sleep Pattern related to disruption in circadian rhythm
- Deficient Knowledge with IABP related to lack of history with device

improve in patients responsive to IABP therapy. Any deterioration in these signs also might indicate a fall in cardiac output. Cardiac output measurement is indicated when deterioration is evident, when a major change in volume or pharmacological therapy has been instituted, and during weaning from IABP support.

The left radial pulse and the cannulated extremity should be frequently assessed. A decrease, absence, or change in character of the left radial pulse may indicate that the balloon has advanced up the aorta and may be partially obstructing or has advanced into the left subclavian artery.

The presence of the balloon catheter in the femoral or iliac artery predisposes the patient to impaired circulation of the involved extremity. The affected extremity needs to be kept relatively immobile. Because flexion of the hip may kink the catheter and impair balloon pumping, it may be helpful to use a knee immobilizer to remind the patient to avoid hip flexion. The head of the bed also should not be elevated more than 30 degrees. Hip flexion also contributes to decreased perfusion to the distal extremity. Extremities should be checked hourly for

pulses, color, and sensation. Any deterioration in the affected extremity should be reported to the physician. Severe arterial insufficiency necessitates removal of the catheter.

Physicians advocate the use of heparin therapy to prevent possible thrombus formation around the catheter and vascular insufficiency, especially in medical patients. Each physician determines whether the risks of anticoagulation outweigh the benefits for the specific patient. Low-molecular-weight dextran is another possible choice of therapy to prevent thrombus formation. This agent impairs platelet function and prevents triggering of the coagulation cascade. Low-molecular-weight dextran is usually preferred in the cardiac surgical patient for the first 24 hours.

Monitoring the Pulmonary System

Many patients on IABP therapy require intubation and ventilatory assistance. Some of these patients may have respiratory insufficiency secondary to fluid overload associated with HF. The immobile, intubated patient is always at risk for respiratory infections and the development of atelectasis. Turning the patient is appropriate provided modifications are implemented to keep the extremity cannulated by the balloon catheter straight. Daily chest radiographs are needed to follow pulmonary status and to inspect IV catheter placement. The position of the balloon catheter also can be determined in this manner.

Monitoring the Renal System

Patients in cardiogenic shock or severe LV failure are at risk for the development of acute renal failure. In the shock state, the kidneys suffer the consequences of hypoperfusion; therefore, urine output and quality should be monitored closely. Serum BUN, creatinine, and creatinine clearance are monitored daily to assess renal function. Creatinine clearance indicates renal dysfunction and possible failure much earlier than elevated serum creatinine. Any acute, dramatic drop in urine output may be an indication that the catheter has slipped down the aorta and is obstructing the renal arteries.

Weaning

INDICATIONS FOR WEANING. Weaning patients from balloon assistance usually can begin 24 to 72 hours after insertion. Some patients require longer periods of support. Weaning can begin when there is evidence of hemodynamic stability that does not require excessive vasopressor support. Ideally, vasopressor support is minimal when weaning begins. After the balloon is removed, it is much easier to increase vasopressor support than to reinsert a balloon catheter for hemodynamic support.

The patient should exhibit signs of adequate cardiac function, demonstrated by good peripheral pulses, adequate urine output, absence of pulmonary edema, and improved mentation. Good coronary artery perfusion is indicated by an absence of ventricular ectopy and no ECG evidence of ischemia or injury.

Complications may require abrupt cessation of IABP. This may or may not result in reinsertion of another balloon catheter. Severe arterial insufficiency, evidenced by a loss of pulses in the distal extremity, pain, and pallor, is definitely an indication to remove the balloon catheter from that particular insertion site. Any balloon that develops a leak also requires removal. The physician may choose to reinsert the balloon catheter in another extremity or to replace the faulty balloon if the patient is hemodynamically unstable. Depending on the philosophy of the institution and physician, a deteriorating, irreversible situation also might be an indication for weaning or discontinuing balloon pump support. Box 18-19 lists major indications for weaning from IABP therapy.

BOX 18-19 PATIENT SAFETY

Indications for Weaning Patient From IABP

To ensure patient safety when weaning him or her from IABP, the nurse should be alert for the following:

- Hemodynamic stability
  - Cardiac index greater than 2 L/min
  - Pulmonary artery occlusion pressure less than 20 mm Hg
  - Systolic blood pressure greater than 100 mm Hg
- Minimal requirements for vasopressor support
- Evidence of adequate cardiac function
  - Good peripheral pulses
  - Adequate urine output
  - Absence of pulmonary edema
  - Improved mentation
- Evidence of good coronary perfusion
  - Absence of ventricular ectopy
  - Absence of ischemia on the ECG
- Severe vascular insufficiency
- Deteriorating, irreversible condition

APPROACHES TO WEANING. Weaning is commonly achieved by decreasing the assist ratio from 1:1 to 1:2 and so on until the minimal assist ratio is achieved on any particular console. A patient may be assisted at the first decrease for up to 4 to 6 hours. The minimal amount of time should be 30 minutes. During this time, the patient must be assessed for any change in hemodynamic status. An increase in heart rate, a decrease in blood pressure, and a decrease in cardiac output indicate deterioration in hemodynamic status. Weaning should be discontinued temporarily, and therapy should be adjusted before another weaning attempt is made. If the first decrease in assist ratio is tolerated, the assist ratio is decreased to minimum, with 1 to 4 hours allowed for each new assist ratio. The patient must be assessed continually for any indications of intolerance to the process. Although less common, weaning can also occur by decreasing balloon volume, which is controlled from the console in many models.

Complications Specific to IABP Therapy

Patients with IABP counterpulsation need to be monitored for development of poor blood flow to the cannulated extremity, which could lead to compartment syndrome. It may occur within the first 24 hours of support or not until several days after catheter insertion. Compartment syndrome is caused by a rise in the tissue pressure in one of the compartments of the affected lower extremity. Bone, muscle, nerve tissue, and blood vessels all are enclosed by a fibrous membrane called the fascia, and this enclosed space is called a compartment. It is nonyielding, so a rise in volume in the compartment increases the pressure in the compartment. The patient with IABP in whom

Table 18-10 Injuries Secondary to Balloons

Injury	Assessment Findings	Nursing Intervention
Balloon rupture	Presence of bright red blood or flecks of dried blood in the catheter or helium delivery line Gas alarm sounds Decreased augmentation Signs of embolic event Entrapment (may be the first indication)	Immediate removal of the catheter by the appropriate personnel Before removal: Turn pump off Clamp the line Place the patient on left side in Trendelenburg position
Balloon entrapment	Balloon pressure waveform indicates leaks Small amounts of blood in tubing or flecks of dried blood in tubing	Surgical removal is usually indicated Physician may consider pharmacological dissolution of clot with thrombolytics Physician may consider use of Fogarty embolectomy to remove fresh clot

limb ischemia develops from decreased capillary flow can suffer cellular and capillary damage that leads to increased capillary permeability. The resultant transudation of fluid into the closed compartment space increases tissue pressure to a level that can interfere with capillary blood flow. When this degree of tissue pressure is reached, tissue viability may be threatened. Treatment is directed at improving blood flow. Pressure release by fasciotomy may be needed to prevent tissue death.

Decreased circulating platelets in the first 24 hours of IABP therapy and a minimal decrease in red blood cell count have been reported; however, these problems are not thought to be significant. There is a low incidence of balloon leakage and rupture. These complications may result from balloon inflation against a calcific, atherosclerotic plaque in the aorta. This disruption in the balloon surface may be as small as a pinhole or may be a large tear. The associated danger is gas embolism. In addition, the risk for entrapment is minimal but still exists. Table 18-10 provides additional details about injury secondary to balloons.

Insertion of the catheter in cases of severe atherosclerotic vascular disease may result in arterial perforation or occlusion. Any leak is an indication for immediate balloon removal. Iatrogenic dissection of the aorta is rare but has been reported. Arterial insufficiency is the most common complication of IABP therapy. Arterial insufficiency may be permanent, or it may be relieved by aortofemoral or ileofemoral bypass grafting. Neuropathy in the catheterized extremity is another reported complication.

▲ Mechanical Circulatory Support

When there is profound myocardial injury, the augmentation of systemic blood pressure by IABP counterpulsation may not be adequate for patient survival. Use of IABP for circulatory support requires that a patient have a functioning left ventricle because IABP augments cardiac output only by 8% to 10%. Patients with severe, acute LV failure after a MI, after a surgical procedure, or from end-stage HF may need a mechanism for replacing LV function. Circulatory support with a ventricular assist device (VAD) has become a successful treatment for patients with cardiac failure refractory to pharmacological therapies, revascularization procedures, and IABP counterpulsation. These devices are capable of supporting circulation until the heart recovers or a donor heart is obtained for transplantation. As of 2008, 80% of all VAD placements were for bridge to transplant.[7]

Interest in the research and development of artificial circulatory support devices has existed since the 1930s. CPB, an early example of these efforts, was successfully implemented in the 1950s. A National Institutes of Health initiative helped organize and support these efforts on a national level. Michael DeBakey became the first clinician to support a postcardiotomy patient with a LV bypass pump successfully in 1966. An impetus for continued research during the 1960s and 1970s was the limited early success with heart transplantation. At that time, the focus of research was the development of a device that could support the failing heart until sufficient cardiac function had returned. Current research focuses on the use of these devices as a bridge to heart transplantation and as a method of permanent cardiac support for patients with end-stage cardiac disease.

Physiological Principles

Patients who are candidates for ventricular assistance suffer from HF resulting from ischemic or myopathic heart disease. Both disease processes lead to a reduction in cardiac output and oxygen delivery. The physiological response of the body to this low output state is vasoconstriction and increased SVR. Although these compensatory mechanisms are meant to protect and preserve cardiovascular function in the short term, a vicious cycle develops that is characterized by compromised cardiac contractility and a low ventricular EF. Hypotension ensues, leading to hemodynamic instability requiring the use of pharmacological agents and possibly IABP therapy for cardiovascular support. Should the patient continue to deteriorate despite drug therapy and IABP, a VAD may be necessary for survival. Hemodynamically, these patients usually demonstrate a cardiac index of less than 2 L/min/m^2, a PAOP of greater than 20 mm Hg, and a systolic blood pressure of less than 80 mm Hg despite pharmacological therapies and the use of IABP counterpulsation.

Restoration of adequate blood flow and preservation of end-organ function are the fundamental goals of short- or long-term VAD use. Hemodynamics and perfusion improve as the VAD assumes the workload of the failing ventricle. Ventricular assistance may involve supporting one or both ventricles depending on the extent of myocardial damage and ventricular failure.

LV support usually requires cannulation of the left ventricle with a conduit that leads to the device. The ascending aorta, which receives the output from the device, is also cannulated with a conduit. In certain situations, the left atrium may be cannulated instead of the left ventricle. Circulation in the patient supported by a left ventricular assist device (LVAD) is similar to the normal circulatory process. Venous blood returns to the right heart, passes through the lungs to be oxygenated, and then returns to the left atrium through the pulmonary veins. Blood then passes from the left atrium through the left ventricle and into the LVAD. The LVAD then ejects blood into the ascending aorta during pump systole.

In situations that necessitate biventricular support, two pump units function in synchrony to assume the roles of the native right and left ventricles. One pump supports right heart circulation while the other supports left heart circulation. The addition of RV assistance requires cannulation of the right atrium for inflow to the pump and the pulmonary artery for outflow from the right ventricular assist device (RVAD). During biventricular assistance, blood is diverted from the right atrium to the lungs through the RVAD, bypassing the right ventricle. Circulation continues to the left heart, where the LVAD undertakes support of systemic circulation. Univentricular or biventricular assistance relieves the ventricles of its workload by acting as the primary pump supporting pulmonary circulation or systemic blood pressure. Reducing ventricular workload decreases cardiac oxygen demand.

Devices

Several VADs are available for use. Certain devices are commercially available, whereas others require special exemption for investigational purposes. Although no universal classification system exists, the devices can be categorized according to four general functional characteristics: the intended duration of support (short term vs. long term), the type of support provided (univentricular vs. biventricular), the actual physical placement of the device (internal vs. external), and the type of blood flow produced (pulsatile vs. nonpulsatile). Short-term support usually refers to assistance for patients expected to recover from episodes of acute LV failure secondary to MI or surgical procedures. Long-term ventricular assistance may be an option for people awaiting heart transplantation, or it may provide an alternative method of permanent cardiac support.

Nonpulsatile Pumps

Centrifugal and roller pumps are examples of nonpulsatile VADs capable of providing univentricular (to either ventricle) or biventricular support. Centrifugal pumps introduce blood near the center of a rapidly spinning disk that accelerates blood toward the periphery of the disk. They are primarily used for short-term ventricular assistance when myocardial recovery is expected. These devices have been used, infrequently, as bridges to transplantation. Both types are approved by the FDA and are commercially available. Centrifugal and roller pumps are extracorporeal devices designed to support circulation of the patient's blood. Because these devices do not generate pulsatile blood flow, IABP is often used in conjunction with them to create a pulse. Blood is transported from the cannulated chamber to an external pump console that circulates the blood back to the corresponding great vessel by a separate cannula. Should right ventricular failure (RVF) be identified after placement of an LVAD, an RVAD can be added for additional support with these devices.

These devices can be inserted relatively quickly and are adequate methods of deploying short-term circulatory assistance. Methods of cannulation and physical placement of the equipment limit the mobility and activity level of the patient. Patients supported by these VADs are usually sedated and paralyzed. A commonly used centrifugal pump is the BioMedicus.

Axial pumps, another type of nonpulsatile pump, use corkscrew-type impellers that propel blood by rapid rotation. These pumps are much more compact than centrifugal pumps and can be used short or long term.[7] In addition, they weigh less than centrifugal pumps, are more compact, and therefore are more comfortable for patients.

Extracorporeal membrane oxygenation (ECMO) or CPB systems are alternative methods of temporary CPR involving circulatory support and oxygenation of the patient's blood. CPB is primarily used for operative situations but has demonstrated effectiveness as a mechanism of support for patients unable to wean from the pump perioperatively or for those requiring cardiopulmonary support refractory to conventional efforts. Circulation of blood between the patient and an external pump is supported by cannulation of the femoral vessels. Venous blood is diverted from central venous circulation; pumped through a membrane oxygenator, where oxygen and carbon dioxide are exchanged; and returned to the arterial circulation through the femoral artery cannula. A heating mechanism in the pump console helps maintain body temperature during circulatory support.

Rapid deployment without the need for surgical intervention and the ability to provide hemodynamic stabilization for a brief period are the major advantages of these resuscitative devices. CPB and ECMO allow time for further assessment and intervention during episodes of acute hemodynamic decompensation. Disadvantages include the need for continuous anticoagulation and the inability to provide extended circulatory support. The presence of occlusive peripheral vascular disease could be a contraindication to use of these devices.

Pulsatile Pumps

IMPLANTABLE PULSATILE PUMPS. Implantable pumps were designed with the intention of providing long-term LV support while allowing the patient a certain amount of physical independence. A few devices have successfully supported a patient for greater than 1 year while awaiting heart transplantation. Many patients with the implantable devices have been physically rehabilitated by participating in regular physical therapy programs and normal activities of daily living while being supported with a VAD. This might better prepare them physically to endure the transplantation process. Examples of the implantable devices are the HeartMate IP and the Novacor left ventricular assist system (LVAS). The Novacor device operates on electricity, whereas the HeartMate IP operates as a pneumatic unit.

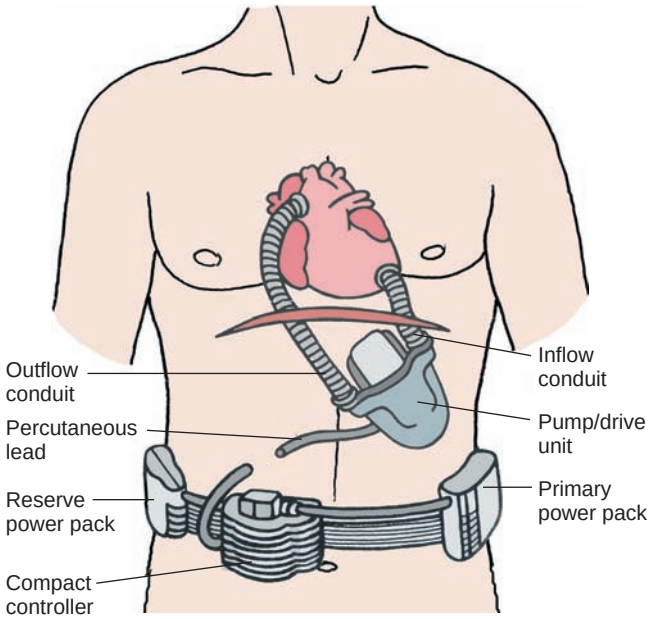


FIGURE 18-25 ▲ Portable, implantable LV assist device. (Artwork courtesy of the Novacor Division, Baxter Healthcare Corporation, Oakland, CA.)

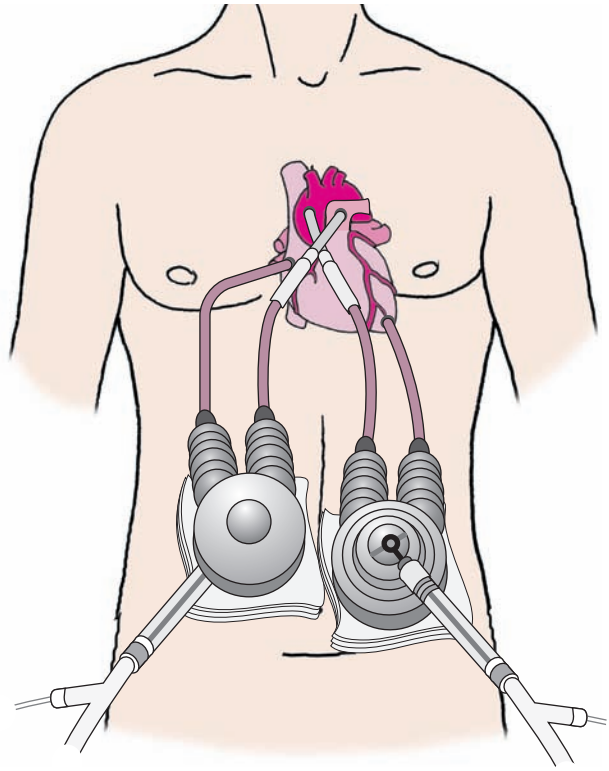


FIGURE 18-26 ▲ Thoratec pneumatic ventricular assist device. External placement with biventricular assist capabilities. (Courtesy of Kathy J. Vaca, RN, Department of Surgery, St. Louis Health Sciences Center, St. Louis, MO.)

Surgical implantation of the VAD necessitates a sternotomy and the use of CPB. Device placement is in an abdominal pocket just below the left diaphragm. Typically, the inflow conduit is tunneled through the diaphragm and anastomosed to the apex of the left ventricle. The outflow conduit is brought around the diaphragm and is anastomosed to the ascending aorta. Drivelines extending from the implanted device are tunneled through the patient's skin and connected to a portable, external power source. This power source may be a portable console or battery pack that is worn by the patient (Fig. 18-25). Either situation allows the patient mobility and independence during the recovery period.

Pump units of the implantable VADs are encapsulated in rigid housing and consist of a blood pump sac and single or dual pusher plates (depending on the particular device). Inflow and outflow conduits have valves that support unidirectional blood flow. These devices work on the principle of converting electrical or pneumatic energy to mechanical energy. This mechanical energy activates the pusher plates, causing them to compress the blood sac at the appropriate time. Blood sac compression causes ejection of the blood out of the pump sac and into the ascending aorta through the outflow conduit. These devices have stroke volumes of 70 to 83 mL and can support pump outputs of greater than 10 L/min. Depending on the device implanted, long-term anticoagulation may be necessary to prevent thromboembolic events.

EXTERNAL PULSATILE PUMPS. Two commonly used external pulsatile devices are the Thoratec VAD and the Abiomed pump. Both devices have successfully supported patients postcardiotomy and patients bridged to heart transplantation.

The Thoratec VAD is a pneumatically driven device that is positioned externally on the recipient's upper abdomen. Placement of this device requires a sternotomy incision and use of CPB. The structure of the pump drive, the inflow and outflow conduits, and the cannulation techniques of the chambers and great vessels are all similar to that of the implantable devices. A major difference is that the cannulas supporting the blood flow pass through the patient's chest wall to the externally positioned pump. One advantage of this device is the ability to provide univentricular or biventricular support, depending on the extent of HF. Figure 18-26 is an example of biventricular support. Another advantage is that due to its external placement, small patient body size is less of a contraindication when considering the need for ventricular assistance.

Another external VAD, the Abiomed pump, is designed for short-term univentricular or biventricular support. It has been used in patients when myocardial recovery is expected and as a bridge to transplantation. Components consist of cannulas for venous and arterial access, blood pumps to support unidirectional blood flow and systemic circulation, and a pneumatically driven console that provides the power source. Cannulation sites for this device are either atria, the pulmonary artery, and the ascending aorta. Filling of the blood pumps occurs passively by gravity; therefore, the blood pumps must be positioned securely below the level of the heart to promote adequate blood flow into their chambers (Fig. 18-27). The internal bladders operate in a fill-to-empty mode. Pumps positioned too high fill insufficiently, and pumps that are too low have a prolonged filling time, each adversely affecting patient hemodynamics.

Nursing interventions specific to the Abiomed include monitoring and adjusting the level of the blood pumps and monitoring filling pressures to ensure adequate volumes

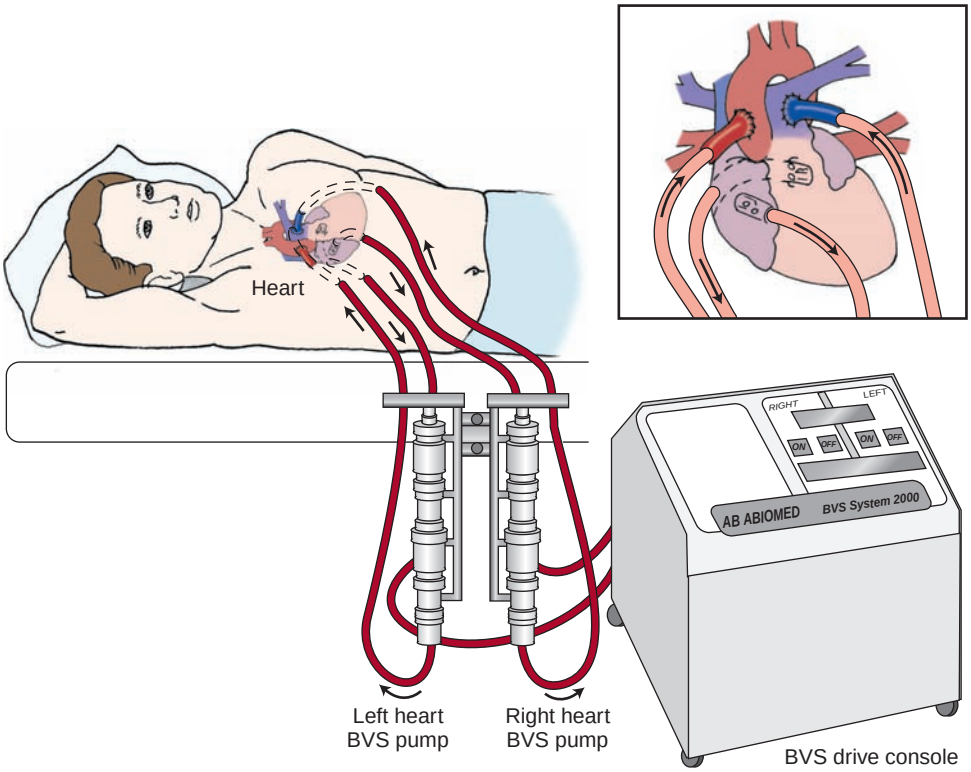


FIGURE 18-27 ▲ Abiomed biventricular support system. (Artwork courtesy of ABIOMED Cardiovascular, Inc., Danvers, MA.)

necessary to support optimal flow through the system. Use of this device significantly impairs patient mobility.

ADVANCES IN MECHANICAL CIRCULATORY SUPPORT. There have been many advances in mechanical circulatory support in the last decade. In November 2002, the FDA approved the Thoratec HeartMate SNAP-VE LVAS for permanent implantation, also known as "destination therapy," in patients with end-stage HF who are not candidates for traditional heart transplantation. Since October 2003, the Centers for Medicare and Medicaid Services have provided coverage for patients who meet specific criteria. The HeartMate SNAP-VE LVAS is an implantable electric pump that allows patients to ambulate and participate in cardiac rehabilitation programs, making it an appropriate system for in-home use by specific HF populations.

Additional advances in VAD technology have been introduced through third-generation devices that use rotary pumps to create a centrifugal flow with the help of magnetically levitated propellers. The use of magnetic bearings over traditional blood-coated bearings has positive effects including extended operating life, improved reliability, and decreased blood damage.[8]

Another recent advance in mechanical circulatory support has been the introduction of very small pumps that can be incorporated into a transvascular catheter. Major surgical procedures are required for implantation of all other models of VADs, whereas catheter-based LVADs can be placed percutaneously. Several such devices have been developed, including the Tandem Heart LVAD, which is an extracorporeal centrifugal LVAD. Table 18-11 provides a comprehensive review of VADs.

Table 18-11 Ventricular Assist Devices

Device	Manufacturer	Duration of Support	Physical Placement	Flow	Drive System
Abiomed BVS 5000	Abiomed	Short term Intermediate term	External	Pulsatile	Pneumatic
Biomedicus	Medtronic-Biomedicus	Short term	External	Nonpulsatile (centrifugal)	Electric
HeartMate IP	Thoratec	Long term	Implantable	Pulsatile	Pneumatic
HeartMate VE	Thoratec	Long term	Implantable	Pulsatile	Electric
Novacor	World Heart	Long term	Implantable	Pulsatile	Electric
TandemHeart	Cardiac Assist	Short term	Percutaneous	Nonpulsatile (centrifugal)	Electric
Thoratec ventricular assist device	Thoratec	Short term Long term	External	Pulsatile	Pneumatic

Table 18-12 Total Artificial Heart

Device	Manufacturer	Flow	Drive System
Abiocor	Abiomed	Pulsatile	Hydraulic
CardioWest	SynCardia	Pulsatile	Pneumatic

Artificial hearts have been approved in the United States by the FDA for investigational use (Table 18-12). Abiomed has developed the first fully implantable replacement heart known as the AbioCor TAH (totally artificial heart).[9] Devices such as the AbioCor are designed for use in patients ineligible to receive a VAD, such as those with both right- and left-sided HF. The AbioCor received Humanitarian Device Exemption from the FDA in September of 2006. In addition, SynCardia Systems has developed the CardioWest device, which is an implantable pneumatic artificial heart. Blood and air in each of the heart's ventricles are separated by a polyurethane sheath and triggered by compressed air from the external console.[8]

Whereas the AbioCor is a self-contained device, the CardioWest device is designed such that patients are connected to a large console by tubes through their chest wall.

MODES OF OPERATION. With the exception of the Abiomed device, the pulsatile pumps have several modes of operation. Two primary modes depend on the patient's ECG or the rate of blood flow through the pump during each cardiac cycle. In the ECG trigger mode, the pump initiates blood ejection in conjunction with the patient's QRS complex; the R wave acts as the trigger for pump systole. The second mode is a dynamic mode that allows the pump to respond to the changing heart rate, depending on patient activity level. Pump systole and cardiac output depend on the blood flow sensed by the device, which is programmed to respond to changes in pump filling rate as blood passes from the left ventricle into the blood sac of the pump drive. This ability is particularly important as a patient's level of activity increases during the recovery phase after implantation. A third mode of operation, rarely used clinically, is a fixed-rate mode that functions independently of the native heart.

Nursing Implications

Historically, VAD recipients have received care in the ICU, usually intubated and sedated. Evolution of the technology and the use of portable devices as bridges to transplantation have changed the mode of care. Now, patients are encouraged to be independent, pursue physical rehabilitation, and engage in normal activities of daily living when possible (Fig. 18-28). Certain patients may even be discharged from the hospital. Nurses have an opportunity to be instrumental in the coordination of patient care and outcome management in this new patient population.

During the immediate postoperative phase, the critical care nurse must be cognizant of the physiological responses expected and the common postoperative complications associated with device implantation. The nurse determines whether the equipment is functioning appropriately by monitoring parameters associated with adequate tissue perfusion and improved end-organ function because these are the primary goals of VAD implantation. Hemodynamic instability and the maintenance of adequate filling pressures are critical issues in the immediate postoperative period. Other issues the critical care nurse encounters include, but are not limited to, dysrhythmias, bleeding complications, infections, thromboembolic events, and possible mechanical problems associated with the devices.

FIGURE 18-28 ▲ Wearing the portable LV assist system, a patient is able to enjoy the independence of outdoor activities. Some patients may take day trips or be discharged from the hospital. (Photograph courtesy of Emory University.)

Psychosocial issues and patient education dominate the nursing focus during periods of extended support with a VAD; most of these patients require minimal direct nursing care once stabilized and discharged from the ICU. Increased independence in activities of daily living, continued physical rehabilitation, and patient education are emphasized. All aspects of the rehabilitation phase should include the recipient's family members or identified support person. As patients are discharged, they and their primary care givers need to be educated about the operation of the equipment and how to troubleshoot malfunctions. A person capable of operating the VAD needs to accompany the patient at all times. Nursing must facilitate the integration of the patient's lifestyle with the boundaries created by having a VAD implanted for extended support. Feelings of isolation may unfold because investigational device protocols governed by the FDA may restrict the patients' social activity and geographical mobility.

An advanced-practice nurse is in a pivotal position to assume the role of case manager facilitating the implementation of clinical paths, protocols, and procedures related to patient progress from the acute to chronic phases of rehabilitation. Nursing education facilitated by a clinical nurse specialist

is vital to patient care as increasing numbers of nurses on the general floors, and possibly the outpatient setting, are exposed to this patient population. As more patients receive the portable devices and approach the possibility of hospital discharge, case management will be a principal facet of patient care.

▲ Complications Associated With IABP Therapy and Circulatory Support

Bleeding

Prolongation of bleeding times is a side effect of exposure to CPB, which is normally reversed in the early postoperative period. With the use of mechanical circulatory support, the continued exposure of blood to an artificial surface causes trauma to platelets. A cascade of events involving the platelets, white blood cells, fibrinolytic system, and complement system occurs. The frequency and severity of bleeding associated with artificial circulatory devices have been reduced by improved surgical techniques and methods of maintaining hemostasis, the reversal of heparin, the infusion of coagulation factors (platelets, fresh frozen plasma), and continued experience with the equipment. Episodes of severe bleeding are usually corrected within the first 24 hours after surgical implantation of a VAD.

Factors associated with increased postoperative bleeding in VAD recipients are preoperative and postoperative use of anticoagulants; coagulopathies secondary to cardiogenic shock, HF, and extended CPB exposure; and the use of multiple cannulation sites. Hemodynamic instability, a reduction in native cardiac output and device output, a risk for ischemia to target organs, and possible cardiac tamponade are all deleterious events associated with uncontrolled bleeding in the patient supported with a VAD. In the patient receiving IABP therapy, bleeding is usually related to use of continuous anticoagulation or the development of coagulopathies. Bleeding commonly occurs at the insertion site of the balloon catheter. In both patient populations, nursing interventions include observing external cannulation sites for oozing, monitoring changes in vital signs (particularly hemodynamic parameters, such as filling pressures for VAD recipients) and laboratory values, and regularly assessing adequate tissue perfusion.

Thromboembolic Events

Placement of IABP puts a patient at risk for thromboembolic events. At the time of insertion, plaque may become dislodged from the vessel wall, or emboli may break off a thrombus that has formed on the indwelling catheter or balloon. Both situations can impair circulation to distal extremities and other vital organs or cause a stroke. Continuous anticoagulation with a heparin infusion is required during IABP therapy; dextran infusions may also be used.

The development of a thrombus and the migration of emboli have been reported with the use of mechanical circulatory support. Anticoagulation regimens and the prevention of embolic events are unresolved issues in the clinical management of VAD recipients. Currently, anticoagulation therapy is managed differently depending on the device that is inserted. Devices used for short-term support require prophylactic use of low-dose heparin infusions. Similar to IABP, dextran infusions may be used in conjunction with heparin. Patients who are supported with the Novacor, HeartMate, and Thoratec devices and who require long-term support are at greater risk secondary to extended periods of exposure to the device. These patients are usually managed with heparin infusions in the immediate postoperative phase. During the extended support period, the heparin is weaned, and warfarin (Coumadin) therapy is initiated to maintain the PT at an INR of 2.5 to 3.5.[10] Antiplatelet agents, such as dipyridamole (Persantine), may be used with warfarin therapy. Obtaining baseline and postimplantation neurological assessments; monitoring peripheral pulses, especially those distal to cannulation sites; and assessing tissue perfusion are critical to the early recognition and intervention of any embolic event.

Right Ventricular Failure

RVF is a significant contributor to morbidity and mortality in patients who have undergone LVAD placement. In an effort to detect risk for RVF in patients who are candidates for LVAD placement, researchers at the University of Michigan have developed a preoperative risk assessment instrument.[11] The risk score is calculated using preexisting clinical data and has been scientifically proven to effectively stratify the risk of RVF and death after LVAD implantation.[11]

RVF becomes a problem for patients with LVADs when the pumping capabilities of the device exceed those of the impaired left ventricle, systemic circulation and RV preload increase, subsequently increasing RV workload. RV output is increased in a patient with a healthy right ventricle. However, a patient with underlying RVF may not be able to handle this augmentation in circulatory volume. Evidence of primary RV dysfunction may not become apparent until the right heart is challenged by the cardiac output of the LVAD. When RVF develops after LVAD implantation, vasodilators and IV inotropes, such as prostaglandin E_1, isoproterenol (Isuprel), and epinephrine, are used to reduce pulmonary pressures and improve RV contractility. It may be possible to add an RVAD for additional support if pharmacological intervention is unsuccessful. Clinical practice has shown that the addition of an RVAD after LVAD placement is a poor prognostic indicator.[12]

Infection

People requiring mechanical circulatory assistance and IABP therapy are at increased risk for infection secondary to the surgical procedures and the presence of external cannulas, pumps, drivelines, and so forth. Many of these patients suffer from chronic illness that renders them more immunocompromised. Infection may be related to surgical wounds after device insertion, invasive monitoring lines, drain placement, pulmonary status, or nutritional status. Early recognition of signs and symptoms of infection and early intervention can prevent the development of sepsis. Early detection is particularly important because some of these patients await heart transplantation, and an infection could preclude transplantation. Diligent handwashing, changing or removing invasive lines or drainage tubes when appropriate, adherence to sterile

dressing techniques and schedules, and the use of appropriate prophylactic antibiotics are effective barriers to the development of infection. Early extubation and mobilization are goals for patients with the implanted devices. Primary nursing interventions include monitoring invasive sites for signs of infection, encouraging good pulmonary toilet, increasing activity level as tolerated, and promoting adequate nutrition.

Dysrhythmias

Most patients with cardiomyopathy who require some form of circulatory assistance experience dysrhythmias before insertion of a device. These dysrhythmias often continue after device implantation and may hinder device function, depending on the rhythm. Dysrhythmias should be treated when they occur, and attempts should be made to restore sinus rhythm.

Circulatory assistance with IABP is affected by dysrhythmias. Diastolic augmentation and systolic assistance decrease in the presence of irregular rhythms, such as AF or sinus rhythm with frequent ectopy. These rhythm changes make it difficult to manage the timing of balloon inflation and deflation. Lethal ventricular dysrhythmias need to be treated conventionally because IABP is designed only to augment existing cardiac output.

RV function and the maintenance of adequate pump output are of primary concern in LVAD recipients with lethal ventricular dysrhythmias. These patients may lack sufficient RV function to support cardiac output during ventricular dysrhythmia even though LV function has been assumed by the LVAD. Although LVAD flow and mean blood pressure have been known to decrease by approximately 20%, it has been demonstrated that patients with LVADs do tolerate sustained lethal ventricular dysrhythmias without the need for RVAD support. Symptoms associated with these rhythms and low-flow states are usually weakness and palpitations.[13] Patients receiving biventricular support should be able to maintain adequate device outputs despite the dysrhythmia because left and RV function has been taken over by the VAD. AF is usually tolerated by these patients even though it may have some effect on right heart function. Severe bradycardia and tachydysrhythmia need to be addressed because they will change pump flow and output. Cardiac rhythms require close monitoring for any acute changes.

Nutritional Deficits

Nutritional status is an important element of any recovery process. Many patients have had end-stage HF and are nutritionally depleted before any surgical intervention, placing them at a higher risk for nutritional deficits during the postoperative phase. Adequate nutrition is necessary for wound healing. Obtaining dietary consultation, encouraging increased oral intake, and providing flexibility with meals will assist these patients in meeting their nutritional goals. Patients who are supported by IABP and VADs and who require intubation and sedation require parenteral or enteral feedings. Those with implanted devices eventually progress to a regular diet but may need smaller, more frequent meals. Experiencing feelings of fullness or early satiety is not uncommon for these patients due to the abdominal placement of the device.

Psychosocial Factors

Balloon and VAD insertion are usually unplanned, emergent interventions for a deteriorating condition. Abundant monitoring is frightening for both patient and family; therefore, explanations of procedures and surroundings are very important. Family members need to be prepared before visiting their loved one immediately after device insertion. The goal is to alleviate anxiety and to help the patient and family feel more secure in a foreign environment. Honest communication is important. This helps the family members recognize changes in their loved one's condition and make informed, realistic decisions regarding the patient's care. Putting the family in contact with nonmedical personnel who can objectively provide emotional support is often beneficial. Issues that families and patients struggle with include fear, hopelessness, and death.

Critically ill patients often suffer from disorientation and sleep deprivation. Immobility and unfamiliar noises of the ICU tend to increase stress and anxiety. Mechanisms to help alleviate this stress and anxiety include frequent reorientation by the nursing staff and contact with family members. Better organization of time and procedures also reduces stress because it allows the patient longer periods of uninterrupted rest.

MANAGEMENT OF DYSRHYTHMIAS

▲ Electrical Cardioversion

Electrical cardioversion is used to convert sustained supraventricular or ventricular tachydysrhythmias to sinus rhythm, especially when the patient has an unstable rhythm that causes hemodynamic collapse. It may be used electively for recent-onset dysrhythmias that do not respond to antiarrhythmic drugs. As opposed to defibrillation, which delivers an unsynchronized current to the heart, cardioversion delivers a shock that is synchronized with the heart's activity. By setting the automated external defibrillator (AED) to the synchronized mode, the device detects the patient's R wave and delivers the shock during ventricular depolarization. As a result, there is no danger of causing VF, which can occur when a shock is delivered during ventricular repolarization (on the T wave). Indications for synchronized, external cardioversion and recommendations for initial joules (J) used are listed in Table 18-13.[1,2] Precautions and relative contraindications for cardioversion are listed in Table 18-14.

Table 18-13 Indications and Energy Requirements for Cardioversion

Indications	Energy in Joules (J) Monophasic Waveform*
Monomorphic VT with a pulse	100–360
Atrial flutter	50
Atrial fibrillation	200 initially

*The energy requirement when using a biphasic waveform defibrillator varies but is usually less compared with a monophasic waveform automatic external defibrillator.

Table 18-14 Precautions/Relative Contraindications to Cardioversion

Condition	Complications
Digitalis toxicity	Ventricular irritability, asystole
Hypokalemia	Ventricular irritability/fibrillation
AF with slow ventricular response	Postcardioversion asystole
AF of unknown duration with inadequate anticoagulation	Thromboembolization
Pacemaker dependency	Rise in thresholds with loss of capture
Low-amplitude R wave	Synchronization on T wave leading to ventricular fibrillation

The energy needed to convert monomorphic VT with a pulse may be as low as 100 J initially, followed by 200, 300, or 360 J, as necessary for conversion.[1] The energy required for conversion of atrial flutter may start at 5 to 50 J when a biphasic waveform defibrillator is used.[2] The energy required to convert AF is greater, starting at 200 J.[3] After conversion to sinus rhythm, antiarrhythmic therapy may be initiated for rhythm maintenance. Although recommendations are made for the amount of joules needed to convert various rhythms, the actual energy needed may vary depending on the duration of the dysrhythmia, transthoracic impedance, and the waveform morphology of the defibrillator (ie, monophasic vs. biphasic).[2]

Procedure

The steps for cardioversion follow:

1. Explain the procedure to the patient and obtain informed consent.
2. Restrict the patient's food and water for 6 to 8 hours before cardioversion, unless emergency cardioversion is required.
3. If the patient is on chronic digitalis, confirm that digoxin levels are therapeutic. Patients with digitalis toxicity should not undergo elective cardioversion until levels are normalized.
4. Record a 12-lead ECG and vital signs, establish an IV line, monitor blood oxygen saturation levels, and ready all necessary resuscitation equipment.
5. Turn on the defibrillator and monitor, and attach the monitoring electrodes to the patient's chest. Avoid placing the electrodes in the area where the defibrillation paddles will be positioned. Some devices permit both monitoring and defibrillation through disposable defibrillation patches.
6. Select a monitoring lead that provides a good ECG pattern with a tall R wave. If monitoring by way of the disposable defibrillator patches, select "paddles" lead.
7. Turn on the synchronizer mode button. The size of the R wave or the monitored lead may need to be adjusted until the synchronization marker appears on each R wave.
8. Sedate the patient, and maintain an adequate airway.
9. Remove paddles from the defibrillator and apply a generous amount of electrode gel to the metal surface. Take care not to smear electrode gel between the two paddles on the chest. Disposable pregelled defibrillator patches may be selected rather than standard paddles.
   a. If hands-free gel patches are used, disconnect the paddles from the defibrillator and connect the terminal pin of the patches to the defibrillator using an adaptor. Place the sternum patch to the right of the sternum, just below the clavicle, and the apex patch below the anterior/axillary margin of the left chest. Apply each patch firmly from the center to the periphery, ensuring that there are no air pockets, which can cause electrical arcing and skin burns.
   b. If using paddles, apply firmly, one just below the right clavicle and the other over the apex of the heart. Make sure paddles or patches are away from electrode wires or from an implanted pacemaker or ICD generator.
10. Set the desired energy level.
11. Press the charge button. A light will flash until paddles are fully charged.
12. Reconfirm the synchronization markers on the R waves on the monitor.
13. Call out "clear" and visually check to make sure no one is touching the patient or the bed.
14. While applying 25 pounds of firm pressure on the paddles, push and hold both paddle discharge buttons until the defibrillator discharges. Maintain contact on the chest wall until the machine has delivered the shock. There will be a momentary delay from the pressing of the discharge button to delivery of the shock because of the synchronization with the R wave. Failure to keep the paddles on the chest can result in failure to cardiovert and burns to the chest.
15. Assess the patient's rhythm, airway, and vital signs.
16. Subsequent shocks may need to be delivered. If so, be certain to select the synchronized mode.
17. If the patient's rhythm deteriorates to VF, turn off the synchronizer and immediately defibrillate the patient, starting with 200 J and increasing to 360 J as needed.
18. After cardioversion, observe the patient for changes in rhythm, blood pressure, and respirations. Patients with AF converting with sinus pauses may have underlying tachy–brady syndrome. Be prepared for transcutaneous pacing if needed, or have atropine sulfate readily available. If a patient has a pacemaker, the pacemaker may need to be interrogated or reprogrammed because a temporary rise in capture thresholds may follow cardioversion. Older pacemaker models may revert to a reset or backup mode.
19. Antiarrhythmic agents may need to be initiated to maintain sinus rhythm.
20. Monitor the patient's respiratory status and level of consciousness because sedation was delivered before the procedure. Inspect the chest wall for any signs of burns and treat appropriately.
21. Clean the paddles thoroughly before storing them.
22. Document the procedure, the outcomes of the procedure, and the patient's status in the medical record.

▲ Catheter Ablation

Catheter ablation is an invasive procedure used for treating tachydysrhythmias. The technique involves percutaneously inserting a catheter into the heart via a vein or artery, and applying radiofrequency or cryoablation. Delivery of the catheter electrode tip to targeted areas responsible for initiating or conducting the dysrhythmia limits the tissue damage.

Clinical use of catheter ablation of cardiac tissue started in the early 1980s with direct current shocks delivered through a catheter attached to a defibrillator. Because this technique was associated with significant risk, safer means to ablate tissue were investigated.

Radiofrequency energy, the primary source of energy used to ablate cardiac tissue, is produced by alternating current (AC) delivered at 500 kHz through the tip of the catheter in unipolar fashion. The circuit is completed through a grounding pad applied on the patient's skin. Resistive heat is created as the energy dissipates around the active electrode, which results in a small localized lesion in cardiac tissue. Tissue temperatures of 50°C or higher lead to irreversible tissue injury. If properly targeted, this localized area of damage can prevent the initiation of the dysrhythmia (the "focus") or interrupt the conduction (the "accessory pathway") of the dysrhythmia. The size of the resulting lesion depends on the electrode temperature, power delivered, and duration of the AC used. When tissue temperature exceeds 100°C, formation of coagulum and char at the electrode tissue interface prevents further energy delivery and adds the risk for steam venting to the endocardial tissue, possibly causing perforation. Electrode cooling (eg, through saline irrigation) reduces the risk for overheating and allows for larger lesion size delivered by higher power. The size, shape, and electrode material of the ablating catheter also influence the resulting lesion.[4]

Indications for Ablation

A PSVT can be treated with radiofrequency ablation. Most PSVTs are caused by either atrioventricular nodal reentrant tachycardia (AVNRT) or by atrioventricular reentrant tachycardia (AVRT). PSVT also can be caused by intra-atrial reentrant tachycardia. Recurrent symptomatic or life-threatening ventricular dysrhythmias also may be indications for ablation. Indications for catheter ablation procedures are included in the AHA and Heart Rhythm Society (formerly North American Society of Pacing and Electrophysiology; NASPE) policy statement on catheter ablation.[5]

The most common mechanism for SVT is reentry[3] which occurs when conduction of an impulse through myocardial tissue is initially blocked (or functionally refractory, unresponsive to stimuli) in one direction. The advancing wave front proceeds through an alternate slower route. As the previously refractory pathway recovers, the electrical impulses return through that pathway and then find their way back down to the alternate slower route. As a result, a circuitous reentrant pattern of conduction occurs.

Atrioventricular Nodal Reentrant Tachycardia

The compact AV node can utilize two functional pathways for conduction—slow and fast—setting the stage for AVNRT. When this phenomenon is observed in the electrophysiology (EP) laboratory, the AV node is described as having dual physiology. AVNRT, the most common type of PSVT, occurs when an AV node with dual physiology is stimulated by a premature atrial contraction. The fast pathway, which is preferentially used in normal sinus rhythm, has not recovered, so the impulse travels down the slow pathway and activates the ventricles. On surface ECG, this initiating rhythm would be viewed as a premature atrial contraction with a long PR interval. The impulse then returns back up to the atria from the ventricles through the fast pathway, which has now recovered excitability, then back down again to the ventricles through the slow pathway, causing the reentrant circuit to perpetuate. Selective ablation of the slow pathway is the preferred method of treating AVNRT. The fast-pathway ablation sites are closer to the compact AV node, and ablation of the fast pathway may be complicated by high-grade AV block.

Atrioventricular Reentrant Tachycardia

In the normal heart, the AV node and the bundle of His serve as the connection between the atria and the ventricles for the conducting system. AVRT rhythms are characterized by the presence of additional accessory pathways that link conduction between the atria and the ventricles. Conduction through accessory pathways may start from the atria to the ventricles (antegrade conduction), from the ventricles to the atria (retrograde conduction), or in both directions. AVRT rhythms result when circular movement of the impulse occurs because of the ability of the accessory pathways to conduct signals in either direction.

In WPW syndrome, an electrocardiographic pattern associated with PSVT and sometimes associated with Ebstein's anomaly of the tricuspid valve, the person has one or more anomalous conduction accessory pathways linking the atria and the ventricles. Because of these accessory pathways, the person with WPW syndrome is prone to AVRT and AF with rapid ventricular response. When rapidly conducting, these PSVTs may deteriorate into VF. Ablation of the accessory pathways is used to interrupt the rapid limb of the reentrant circuit and eliminate the offending dysrhythmias.

Atrial Fibrillation or Flutter

Ablation may be indicated for patients with AF or flutter with a rapid ventricular response that has not been controlled by pharmacological therapy. The AV junction may be ablated, completely disrupting communication from the atria to the ventricles. Successful ablation results in complete heart block with a ventricular rate of 40 to 60 beats/min. A permanent pacemaker is implanted after AV junction ablation to ensure the presence of a reliable rhythm and adequate rate as well as to reduce the risk for bradycardia-dependent torsades de pointes.

In addition, ablation for AF can be performed by creating lines of block around the anatomical triggers (eg, around the orifice of the pulmonary vein), or when the focus is identified, by electrically isolating the foci. The various techniques require special catheters and mapping equipment.[6] However, not all types of AF are amenable to this procedure; the etiology and trigger of the dysrhythmia need to be elucidated before a decision is made to carry out the ablation.

Ablation therapy for primary atrial flutter is indicated in those patients who have reentrant circuits within the right atrium. Ablation lesions are directed at creating a line of block usually along a narrow isthmus between the inferior vena cava and tricuspid annulus to interrupt the circuit.[7] When successful, atrial flutter ablation can provide a permanent cure. Unlike AV node ablation, atrial flutter ablation does not require permanent pacemaker implantation.

Ventricular Dysrhythmias

The success of ablation for the treatment of VT depends on the cause of the dysrhythmia. Radiofrequency ablation has been effective in patients with VT in structurally normal hearts and in patients with VT due to bundle branch reentry. The technique also has had some limited success in patients with hemodynamically stable, monomorphic VT associated with a healed myocardial scar. However, it is not unusual to have multiple morphologies (forms) of VT in this population, and some unstable morphologies may not be amenable to ablation.

Procedure

Before an ablation, the patient undergoes an electrophysiological study (EPS) to evaluate the electrical activity of the heart. The EPS is an invasive test in which catheters are placed in the heart to record intracardiac electrograms (IC-EGM). The test provides information about the sequence of electrical activation of the heart in sinus rhythm and any abnormal sequence of activation during an induced dysrhythmia. An electrical map is inferred from the electrical recordings to help identify the focus of a dysrhythmia or locate an accessory pathway. The map guides the placement of the ablating catheter.

After the catheters are positioned, ECG recordings are made from the surface electrodes on the patient's chest and electrograms (EGMs) from the intracardiac electrodes. Programmed electrical stimulation (PES) is then performed to induce the dysrhythmia so that its mechanism and pathway can be evaluated. Once a diagnosis of the dysrhythmia is confirmed, an ablating catheter is positioned in the targeted area of the heart. Additional catheters are positioned to stimulate atrial and ventricular tissue. The ablation catheter contains multiple electrodes designed to localize the site of the dysrhythmia and to deliver the ablation current. The distal tip of the catheter can be flexed to facilitate access to the tissue and to ensure direct contact. Fluoroscopy and the EGM pattern from the catheter, as well as special mapping equipment and intracardiac ultrasound, help the physician determine the appropriate target area. The clinical ECG of the tachycardia is a useful template of the target dysrhythmia when several morphologies are induced.

When the appropriate site is identified, the radiofrequency current is applied for several seconds until the target tip temperature is achieved. Longer application time is allowed when a cooled or irrigated catheter tip is used. Several lesions may be required to eliminate the abnormal conducting tissue. Successful elimination of the target site is determined by examining the ECG and EGM tracings and confirmed when the dysrhythmia is no longer inducible. When the procedure is finished, the intracardiac catheters and venous or arterial sheaths are removed, and efforts to attain hemostasis at the insertion site are implemented.

Nursing Management

The nurse plays a vital role in the care of the patient undergoing radiofrequency ablation. In consultation with the electrophysiologist, the nurse provides information to the patient and family about what to expect before, during, and after the procedure. The psychosocial support provided by the nurse may be key in helping the patient and family cope with the uncertainties of dysrhythmia management.

Preablation

The nurse participates in educating the patient and family about radiofrequency ablation (Box 18-20). During the preablation period, the nurse records a 12-lead ECG, continuously monitors the patient's cardiac rhythm, and treats any dysrhythmias per the physician's orders. Other baseline data obtained include vital signs, breath sounds, fluid status, serum chemistries, PT, and complete blood counts. Antiarrhythmic drugs may be discontinued 2 to 3 days before the procedure to allow provocation of the dysrhythmia during the procedure. The patient receives nothing by mouth for about 8 hours before the procedure. It is important to verify that a female patient is not pregnant because of x-ray exposure during the test. No activity restrictions are imposed before the procedure.

During Ablation

The nurse in the EP laboratory is responsible for monitoring the patient throughout the procedure and assisting the physician with necessary interventions. The nurse must be competent in ACLS so that an emergency situation can be handled appropriately.

In the laboratory, the nurse explains each intervention to the patient and helps put the patient at ease. The nurse

BOX 18-20 TEACHING GUIDE Preablation

Points the patient needs to know before the ablation procedure include the following:

- Purpose of the procedure
- The patient's dysrhythmia and how the procedure will help
- Interventions that will occur before transport to the electrophysiology (EP) laboratory
- The appearance of, equipment, and personnel in the EP laboratory
- The use of IV conscious sedation, including the amnesic/analgesic effect of conscious sedation and possible side effects, such as nausea, vomiting, or hypotension
- Sensations associated with the procedure, such as:
  - Cool sensation from cleansing agents
  - Pressure sensation from catheter insertion
  - Palpitations, dizziness, or other sensations when dysrhythmia is induced
  - possible mild burning sensation during ablation
  - Restlessness or back discomfort from lying immobilized
- Anticipated length of the procedure
- Potential for placement of a permanent pacemaker
- Anticipated after effects, such as:
  - Skipped beats or faster than usual resting rate may be felt initially
  - Mild chest discomfort or burning may occur for a few days
  - A "skin effect," a dark outline of the grounding or defibrillation pad, which may persist indefinitely

connects the patient to a cardiac monitor and physiological recorder and applies a grounding pad for the radiofrequency catheter, AED patches, automatic blood pressure device, and pulse oximeter. Oxygen is provided by nasal cannula. If not already in place, an IV line is inserted. IV conscious sedation is administered to ensure patient comfort. A urinary catheter is inserted if the procedure is anticipated to be lengthy. Both groins and the right subclavian vein sites are shaved and the skin prepared. A sterile field is established and maintained throughout the procedure. A lead apron may be placed under the patient's lower back to block fluoroscopy radiation from penetrating the reproductive system.

Throughout the procedure, the nurse monitors hemodynamic status, activated clotting time (ACT) if heparin is used, sedation level, and patient comfort. Communication with the patient is essential so that the patient is kept informed about the progress of the procedure, and anxiety and fear are minimized. The nurse also warns the patient that a burning sensation may be felt for a brief time during the actual ablation.

Postablation

Thorough assessment and monitoring of the patient are continued after the ablation procedure. Essential components of the assessment include vital signs, cardiac rhythm, catheter insertion sites, peripheral pulses, and level of consciousness. The patient may remain drowsy for several hours and experience nausea and vomiting as a result of the medications. When an arterial site has been used, leg immobilization and bed rest are maintained for about 6 hours. If only venous sites were used, the patient may begin ambulation in about 4 hours. The nurse assesses the patient for any pain or discomfort and provides comfort measures if indicated. Fluid volume status is checked, and when the patient's condition is stable, the urinary catheter is removed.

During the postablation period, the nurse carefully assesses the patient for any evidence of complications. Table 18-15 lists potential complications of radiofrequency ablation and associated signs and symptoms.

Table 18-15 Potential Complications of Radiofrequency Ablation and Associated Signs and Symptoms

Complications	Signs and Symptoms
Cardiac perforation	Tachycardia, hypotension, dyspnea, pleuritic chest pain
Cardiac tamponade	Hypotension, distended neck veins, muffled heart sounds, pulsus paradoxus, change in level of consciousness
Coronary artery spasm	Chest pain, ECG changes
Pneumothorax	Dyspnea, decreased oxygen saturation decreased breath sounds
Cerebral embolus	Slurred speech, blurred vision, headache, seizures
Pulmonary embolus	Chest pain, dyspnea, tachycardia
Femoral artery dissection	Bruit at site of pulse, hematoma, retroperitoneal bleed
Deep venous thrombosis	Swelling of leg at site of catheter insertion, calf pain

▲ Cardiac Pacemakers

Electrical stimulation of the heart was tried experimentally as early as 1819. In 1930, Hyman noted that he could inject the right atrium with a diversity of substances and restore a heartbeat. He devised an "ingenious apparatus" that he labeled an artificial pacemaker, which delivered a rhythmic charge to the heart. In 1952, Zoll demonstrated that patients with Stokes-Adams syndrome could be sustained by the administration of current directly to the chest wall. In 1957, Lillehei affixed electrodes directly to the ventricles during open heart surgery.

From 1958 to 1961, implantable pacemakers became accepted treatment for complete heart block. In the 1970s and 1980s, AV synchrony and "physiological" pacing became available. At the start of the first decade of the 21st century, clinical trials on right and LV (biventricular) pacing made tremendous progress. Biventricular pacing is achieved by positioning an additional lead in one of the coronary sinus branches to stimulate the left ventricle, nearly simultaneous with the right ventricle. Commonly referred to as cardiac resynchronization therapy (CRT), biventricular pacing is used for symptom improvement and treatment of HF in patients with moderate to severe LV dysfunction and BBB. Biventricular pacing corrects intra- and interventricular delay; CRT has been shown to improve functional class and quality of life in select HF populations.[8]

Currently, technological advances have resulted in smaller pacemakers with longer battery life and numerous programmable options in most models. Furthermore, the goal of individualized, physiological pacing has been achieved with recent advances.

Indications for Cardiac Pacing

Cardiac pacing is most commonly indicated for conditions that result in failure of the heart to initiate or conduct an intrinsic electrical impulse at a rate adequate to maintain perfusion. Pacemakers are necessary when dysrhythmias or conduction defects compromise the electrical system and the hemodynamic response of the heart. The original pacemakers were designed for antibradycardia. Today's pacemakers also monitor and treat tachydysrhythmias and facilitate electrical remodeling. Further research and advances in technology have allowed the use of pacemakers in heart conditions such as congestive heart failure, long QT syndrome, and neurocardiogenic syncope.[9]

Critical care nurses work with members of the health care team to assess potential pacemaker patients who may exhibit dysrhythmias, atherosclerotic heart disease, AMI, or other conditions that alter the conduction of the heart. To assist medical professionals in determining the clinical criteria for pacemaker implantation, a Joint Committee of the ACC, AHA, and Heart Rhythm Society (formerly NASPE) was formed to establish uniform criteria for pacemaker implantation.[9] The committee divided its recommendations for implantation into three classes of indications. Class I indication recommends implantation, includes conditions for which there is evidence or general agreement that treatment is

useful and effective. Class II indication includes conditions for which there is conflicting evidence or a divergence of opinion about the usefulness or efficacy of a procedure or treatment. (In class IIa conditions in which treatment is reasonable, and in class IIb conditions in which treatment may be considered) Class III includes conditions for which there is evidence or general agreement that the procedure or treatment is not useful or effective and in some cases may be harmful. The most common indications for pacemaker implantation with recommended pacing modes are summarized in Box 18-21.[9]

The Pacemaker System

The pacemaker system, which consists of a pulse generator and one to three leads with electrodes, performs two main functions: diagnosis and treatment. The diagnostic function is to sense intrinsic cardiac activity; the treatment function is to emit an electrical impulse that excites endocardial cells and produces a wave of depolarization in the myocardium. Clinical terminology related to pacemakers is listed in Box 18-22.

Permanent Pacing Systems

THE PULSE GENERATOR. The pulse generator for a permanent pacemaker is composed of a lithium iodide battery source and electronic circuits enclosed in a hermetically sealed metal container. The generator weighs 20 to 30 g and is 5 to 7 mm thick (Fig. 18-29). The longevity of most permanent pacemakers is about 6 to 12 years, depending on the percentage of pacing the heart requires over time. Most permanent pulse generators are inserted in a subcutaneous pocket in the pectoral region below the clavicle (Fig. 18-30).

BOX 18-21 Indications for Permanent Cardiac Pacing*

Acquired Atrioventricular (AV) Block in Adults

Class I: Third-degree and advanced second-degree AV block at any anatomical level, associated with any one of the following conditions: (1) symptomatic bradycardia; (2) asystole greater than or equal to 3.0 seconds or any escape rate less than 40 beats/min in awake, symptom-free patients; (3) dysrhythmias and other medical conditions that require drugs that result in symptomatic bradycardia; (4) post-AV node ablation; (5) postoperative AV block that is not expected to resolve after cardiac surgery; and (6) neuromuscular diseases with AV block, (7) atrial fibrillation with pause of 5 seconds or greater, and (8) exercise-induced AV block in the absence of myocardial ischemia

Class IIa: (1) Asymptomatic third-degree AV block at any anatomical site with average awake ventricular rates of 40 beats/min or faster without cardiomegaly (2) asymptomatic type II second-degree AV block with a narrow QRS; (3) asymptomatic type I second-degree AV block at intra- or infra-His levels found at EPS (4) first- or second-degree AV block with symptoms similar to those of pacemaker syndrome.

Class IIb: (1) Neuromuscular diseases, such as myotonic muscular dystrophy, Kearns-Sayre syndrome, Erb's dystrophy (limb-girdle), and muscular atrophy, with any degree of AV block (including first-degree AV block) with or without symptoms. (2) AV block in the setting of drug use when the block is expected to occur even after the drug is withdrawn

Class III: Asymptomatic first-degree AV block and type I second-degree AV block, transient AV block

Chronic Bifascicular Block

Class I: (1) Intermittent complete heart block, (2) type II second-degree AV block, and (3) alternating bundle branch block

Class IIa: (1) Syncope not demonstrated to be due to AV block when other likely causes have been excluded, specifically VT; (2) prolonged His–ventricular interval on EPS; (3) incidental finding of pacing-induced, nonphysiological infra-His block at EPS

Class IIb: (1) Neuromuscular diseases, such as myotonic muscular dystrophy, Kearns-Sayre syndrome, Erb's dystrophy (limb-girdle), and peroneal muscular atrophy, with any degree of fascicular block with or without symptoms

Class III: Fascicular block without AV block or symptoms and asymptomatic fascicular block with first-degree AV block

AV Block After AMI

Class I: (1) Persistent type II second-degree AV block with alternating bundle branch block (BBB) or third-degree AV block (2) transient advanced (second- or third-degree) infranodal AV block and associated BBB; (3) persistent and symptomatic second- or third-degree AV block.

Class IIb: (1) Persistent second- or third-degree AV block at the AV node level

Class III: Transient AV block

Sinus Node Dysfunction (SND)

Class I: SND (1) with documented symptomatic bradycardia (2) symptomatic chronotropic incompetence (3) occurring spontaneously or as a result of required drug therapy for a medical condition

Class IIa: SND (1) with heart rate less than 40 beats/min, (2) syncope of unexplained origin with provoked SND during EPS

Class IIb: In minimally symptomatic patients, chronic heart rate less than 40 beats/min while awake.

Class III: Asymptomatic SND; SND with symptomatic bradycardia due to nonessential drug therapy; symptoms occur in the absence of bradycardia

Hypersensitive Carotid Sinus Syndrome and Neurocardiogenic Syncope

Class I: Recurrent syncope caused by spontaneous carotid sinus stimulation and asystole of more than 3 seconds induced by minimal carotid sinus pressure.

Class IIa: Recurrent syncope without clear, provocative events and a hypersensitive cardioinhibitory response of 3 seconds or longer

Class IIb: Significantly symptomatic neurocardiogenic syncope associated with bradycardia documented spontaneously or at head-up tilt

Class III: (1) A hyperactive cardioinhibitory response to carotid sinus stimulation in the absence of symptoms or in the presence of vague symptoms such as dizziness, light-headedness, or both and (2) situational vasovagal syncope in which avoidance behavior is effective

*Box does not include indications for special population and specific conditions.

Adapted from Epstein AE, DiMarco JP, Ellenbogen KA, et al: ACC/AHA/HRS 2008 Guidelines for device-based therapy of cardiac rhythm abnormalities: Executive summary. A report of the American College of Cardiology/American Heart Association task force on practice guidelines (ACC/AHA/NASPE 2002 guideline update for implantation of cardiac pacemakers and antiarrhythmia devices). Heart Rhythm 5:e7–e19, 2008

BOX 18-22 Clinical Terminology Related to Pacemakers

Active fixation lead: A pacing lead with some design at the lead tip (corkscrew, coil) that allows the tip to be embedded in heart tissue, thus decreasing the likelihood of dislodgment
Asynchronous pacing: A pacemaker that fires at a fixed rate regardless of the intrinsic activity of the heart
Bipolar lead: A pacing lead containing two electrodes. One electrode is at the tip of the lead and provides stimulation to the heart. A second electrode is several millimeters proximal to the tip and completes the electrical circuit. Both electrodes provide sensing of the intrinsic cardiac activity.
Capture: The depolarization of a cardiac chamber in response to a pacing stimulus
Chronotropic incompetence: Inability of the sinus node to accelerate in response to exercise
Demand pacing (inhibited pacing): A pacemaker that withholds its pacing stimulus when sensing an adequate intrinsic heart rate
Dual-chamber pacing (physiological pacing): Pacing in both the atria and the ventricles to restore artificially AV synchrony
Electromagnetic interference: Electrical or magnetic energy that can interfere with or disrupt the function of the pulse generator
Milliamperage (mA): The unit of measure used for the electrical stimulus (output) generated by the pacemaker
Multisite pacing: The ability to stimulate more than one site in a chamber (eg, right ventricle and left ventricle stimulation in biventricular pacing/cardiac resynchronization therapy [CRT])
Overdrive pacing: A method to suppress a tachycardia by pacing the heart at a rate faster than the patient's intrinsic rate
Oversensing: Inhibition of the pacemaker by events other than those that the pacemaker was intended to sense. These may include tall T waves and EMI.
Pacing threshold: The minimal electrical stimulation required to initiate atrial or ventricular depolarization consistently
Passive fixation lead: A pacing lead that lodges in the trabeculae of the heart without actually penetrating the cardiac wall
Rate-responsive (rate-adaptive, rate-modulated) pacing: A pacemaker that alters pacing rate in response to detected changes in the body's metabolic demand
Sensing: The ability of the pacemaker to detect intrinsic cardiac activity and respond appropriately. How the pacemaker responds depends on the programmed mode of pacing.
Sensing threshold: The minimal atrial or ventricular intracardiac signal amplitude required to inhibit or trigger a demand pacemaker
Situational vasovagal syncope: Syncope associated with bradycardia by vagal stimulation during coughing, micturition, or severe pain
Triggered: A response to sensing in which the pacemaker fires a stimulus in response to intrinsic cardiac activity. In pacemaker terms, triggered is the opposite of inhibited.
Undersensing: Failure of the pacemaker to sense the heart's intrinsic activity. As a result, the pacemaker fires inappropriately.

THE LEAD SYSTEM. The lead is a wire that provides the communication network between the pulse generator and the heart muscle. One or more electrodes are at the distal end of the lead and provide sensing and pacing of the heart muscle. In a bipolar lead, the negative electrode (cathode) is at the tip, and the positive electrode (anode) is about 1 to 3 cm proximal to the tip (Fig. 18-31).

The permanent pacemaker lead is typically inserted either through a subclavian vein or a cephalic vein through the chest wall. Alternate insertion sites include the external jugular, internal jugular, and rarely, femoral vein. The lead is then positioned with fluoroscopic guidance and affixed in the right atrial appendage or in the apex of the right ventricle, or in both locations. A third lead may be inserted in a coronary sinus branch to stimulate the left ventricle for biventricular pacing. The leads must provide adequate electrical stimulation, sufficient insulation, and the endurance to withstand pulsatile turbulence.

The permanent pacemaker lead tip can be affixed to the myocardium with a lead fixation mechanism. Over time, fibrotic tissues anchor the tip to the myocardium securing placement and ensuring proper function of the electrode.

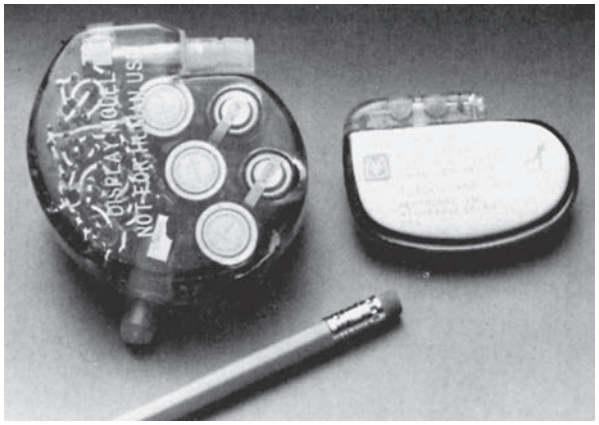


FIGURE 18-29 ▲ Permanent pulse generators, old (*left*) and new (*right*) models. Note the decrease in size and weight that has been achieved over the years. The smaller unit is an example of an activity-responsive pulse generator and is four times smaller than the larger 1,968 U.

Temporary Pacing Systems

Temporary pacemaker systems are used in emergency and elective situations. In life-threatening situations, a temporary pacemaker serves as a bridge to permanent pacemaker

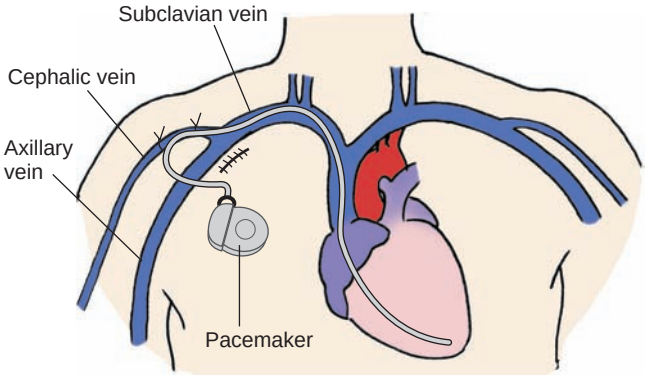


FIGURE 18-30 ▲ Transvenous installation of a permanent pacemaker. For dual-chamber pacing, a separate pacing wire would be in the atrium.

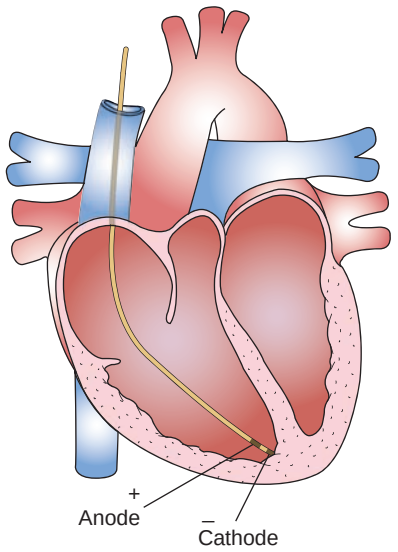


FIGURE 18-31 ▲ Transvenous bipolar pacing catheter in place.

implantation or until resolution of a reversible cause. Electively, temporary pacemakers may be used for overdrive or pace-termination of tachydysrhythmias. Temporary pacing systems can be transvenous, epicardial, transcutaneous, or transthoracic.

TRANSVENOUS TEMPORARY PACING SYSTEMS. A transvenous pacemaker system consists of an external pulse generator and a temporary transvenous pacing lead. The lead system usually includes the use of a bipolar catheter with a paired negative distal and positive proximal electrode at the endocardial surface. The lead terminal pins are attached to a connecting cable and the corresponding negative and positive ports of the pulse generator.

For temporary transvenous pacing, the clinician uses local anesthesia and then introduces the catheter/lead into a superficial vein. The brachial, internal or external jugular, subclavian, and femoral veins may be used. The subclavian and internal jugular sites allow greater lead stability and patient mobility. The transvenous lead is threaded through a sheath in the vein, into the vena cava and the right atrium, through the tricuspid valve, and into the right ventricle. The lead tip is placed in contact with the endocardial surface of the RV apex for stability and reliability. For atrial pacing, an atrial bipolar catheter is placed in the right atrial appendage. There are also pulmonary artery balloon flotation catheters with atrial and ventricular pacing ports for dual-chamber pacing. Balloon flotation catheters are useful in the critical care setting because they provide thermodilution and cardiac output determination and do not require fluoroscopy for positioning.

After placement, the leads are affixed at the skin entry site with nonabsorbable suture. The sheath should be sutured and attached to a continuous drip if used for drawing blood or administering drugs. To maintain sterility at the connection site and terminal tip of the catheter, a sterile protective sleeve over the catheter can be used before insertion and then connected to the end of the sheath after satisfactory position is confirmed. The sheath entry site should be covered with an antiseptic ointment and a self-adhesive, semipermeable transparent dressing. A small label above the dressing, initialed by the nurse, should indicate time and date of application.

EPICARDIAL TEMPORARY PACING SYSTEMS. Placement of epicardial wires provides another method for temporary pacing. This method can be accomplished by thoracotomy or through a subxiphoid incision with the placement of pacing electrodes directly on the outer surface of the heart. Epicardial wires often are used as a temporary adjunct during and after open heart surgery. The pacing wires are attached to the epicardial surface of the heart, and the proximal end is brought outside through the chest incision and either connected to a temporary pacemaker generator or capped and then connected if the need for pacing arises. The wires are extracted without reopening the incision, even after scar tissue has formed over the tips.

TRANSCUTANEOUS TEMPORARY PACING SYSTEMS. Another method of temporary pacing is known as external transcutaneous pacing. This method involves placing large gelled electrode patches directly on the chest wall. The cathode or negative electrode is applied anteriorly to the left of the sternum, and the anode or positive electrode is applied straight posteriorly on the patient's back and then connected to an external transcutaneous pacemaker (Fig. 18-32). Transcutaneous pacing can cause significant discomfort, and the patient should be informed and adequately sedated, if necessary. Transcutaneous pacing is used when temporary transvenous pacing is not immediately available. In patients with profound asystolic arrest, the transcutaneous pacemaker should not be relied on indefinitely.

TRANSTHORACIC TEMPORARY PACING SYSTEMS. Transthoracic pacing is a temporary pacing method used as a last resort in an emergency situation. This method involves introduction of a pacing needle in the anterior wall of the heart. Transthoracic pacing has limited success rates and a high potential for complications.

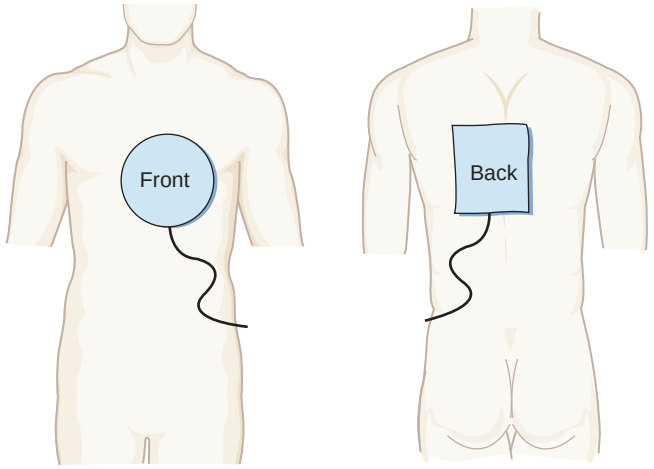


FIGURE 18-32 ▲ Transcutaneous pacing. Electrodes are placed on anterior and posterior chest walls and attached to the external pacing unit.

FIGURE 18-33 ▲ A dual-chamber temporary pacemaker. (Courtesy of Medtronic, Minneapolis, MN.)

EXTERNAL PULSE GENERATOR. The temporary pulse generator is an external device powered by a 9-V alkaline or lithium replaceable battery (Fig. 18-33). Often called a temporary pacemaker, the device contains several controls that regulate the current output, rate, sensitivity, and the mode of pacing; and for dual-chamber pacing, base and upper rate, AV interval and refractory period settings can be chosen. A dual-chamber pulse generator has separate terminals for the atrial and ventricular inputs. The use of a connecting cable allows the lead tips to tighten into the connector block and a locking mechanism to the pulse generator. Cables should be labeled appropriately near the distal lock: atrial or ventricular so as to avoid interchanging the leads when attaching to the atrial or ventricular port of the pulse generator. Nursing assessment and intervention guidelines for temporary transvenous pacemaker placement are summarized in Box 18-23.

Pacemaker Functioning

When the pacemaker system functions appropriately, it senses and treats the heart rhythm dysfunction. The sensing function is the ability of the pacemaker to detect the heart's intrinsic activity, and the sensing amplitude is the largest intrinsic signal that is consistently detected by the pacemaker electrode (eg, the R wave is usually the largest signal sensed by the ventricular lead). At the site of the sensing electrode, the amplitude of the intrinsic depolarization wave is measured in millivolts (mV). The smallest number on the sensor control represents the most sensitive setting, in mV, indicating the smallest signal the pacemaker will sense. If the heart's intrinsic amplitude is smaller than the sensitivity setting,

BOX 18-23 NURSING INTERVENTIONS

For the Patient With a Temporary Transvenous Pacemaker

Assessment

- During insertion:
  - Vital signs, O_2 saturation, peripheral pulses
  - Level of sedation/sedative agents used
  - Continuous cardiac rhythm monitoring
  - Date, time, method, and site of insertion
  - Location of wire inserted (atrial, ventricular, atrial and ventricular)
  - Measured values: capture (mA) threshold and intrinsic amplitude (mV)
  - Patient's tolerance of procedure
  - Complications
  - 12-lead ECG
  - Final settings: mode, rate, output, and sensitivity
- After insertion:
  - Rate setting, mV setting, mA setting, mode of operation (demand, asynchronous) and AV interval (if appropriate)
  - Pacemaker turned off or on
  - Rhythm strip, capture and intrinsic, if appropriate; 12-lead ECG
  - Status on insertion site and sutures (if present)
  - Chest x-ray performed, results on chart
- Every change of shift:
  - Pacemaker turned off or on
  - Pacemaker secured appropriately to patient
  - All connections are secure
  - Setting for rate, mA, sensitivity, mode of operation, AV interval (if appropriate)
  - Rhythm strip (and with any clinical change or intervention)
  - Sensing and capture thresholds (compare to baseline)
  - Presence/absence of hiccupping or muscle twitching
  - Status of insertion site and sutures (if present)
  - Signs of infection (redness, pain, fever, pus)
  - Pulse perfusion distal to insertion site (if appropriate)
  - Connective ends of pacer wires covered (as appropriate)

Intervention

- Continuous cardiac monitoring
- Pacemaker generator:
  - Verify replacement 9-volt battery available.
  - Verify connections are intact.
- Wear rubber/latex gloves when handling the connective ends of pacer wires.
- Cover connective ends of pacer wires to prevent microshock hazard.
- Label epicardial pacer wires *atrial* or *ventricular*.
- Clean and dress pacer wire insertion site(s) daily with gauze dressing or transparent dressing per institutional protocol. Label time and date of dressing change and initial.

Documentation

- Document in Critical Care Flow Sheet/Nursing Notes:
  - Assessments
- Instructions to patient/family
  - Pacing wire insertion site care
  - Pacing and sensing thresholds (print ECG strips)
  - Pacing problems, nursing interventions, and results of interventions
  - Complications/problems

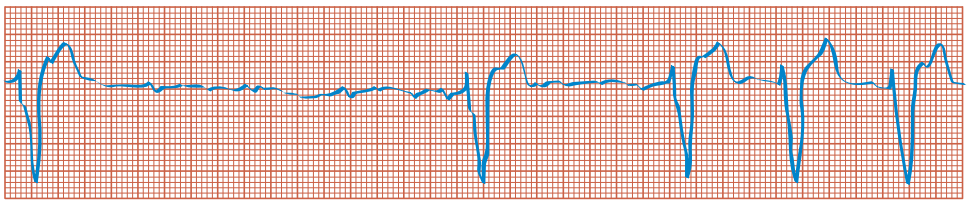


FIGURE 18-34 ▲ Failure to discharge or oversensing with pacing inhibition. In the first half of the strip, the sensing amplifier may have detected electrical noise (oversensing) causing pacemaker inhibition.

undersensing occurs. This can occur when the electrode has inadequate contact with the heart tissue. The drawback in setting pacemaker sensitivity at its most sensitive setting is that oversensing may occur, such as when the pacemaker senses extraneous signals (eg, T waves) or signals from the other chamber. If oversensing occurs, the pacemaker stimulus may be inhibited (Fig. 18-34).

When intrinsic heart rate is adequate, the pacemaker responds by inhibiting a pacing stimulus. When intrinsic heart rate drops to the programmed minimum rate, the pacemaker delivers a stimulus through the lead. When the pacemaker discharges, an artifact known as a pacing spike appears on the ECG, as shown in Figure 18-35. As a result of this stimulus, the cardiac chamber containing the pacemaker lead is depolarized. *Capture* is the term used to indicate depolarization of the atria or ventricle in response to a pacing stimulus. The minimal amount of voltage required from the pacemaker to initiate consistent capture is known

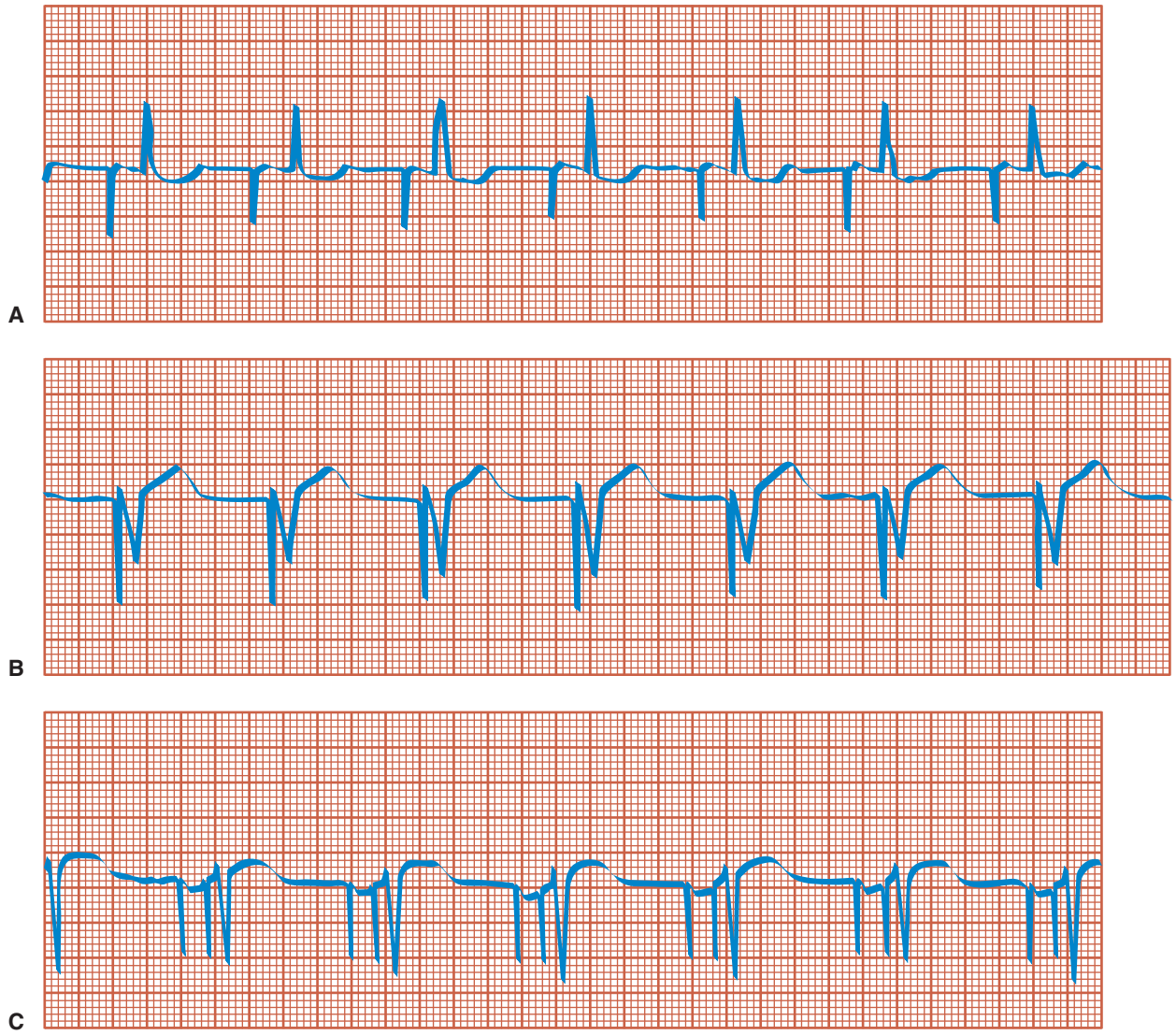


FIGURE 18-35 ▲ Strip **A** shows an atrial pacemaker. Note that each pacing stimulus is followed by a P wave. Strip **B** shows a ventricular pacemaker. Note that each pacing stimulus is followed by a wide QRS complex. Strip **C** shows a dual-chamber pacemaker. Note that the first spike is followed by a P wave and the second spike is followed by a QRS complex. All strips show 1:1 capture.

as the pacing threshold. This threshold level is determined by establishing successful pacing at higher energy and then gradually decreasing the energy output of the generator until capture ceases. The pacing threshold is expressed as milliamperage (mA) in the temporary generator and voltage (V) in the permanent pulse generator, within a given pulse width duration. The generator output is then set at two or three times the threshold level to allow for an adequate safety margin.

Many factors affect the pacing threshold. These include hypoxia, hyperkalemia, antiarrhythmic drugs, catecholamines, digoxin toxicity, and corticosteroids.

The Pacemaker Code

Since the initial use of cardiac pacemakers, the technology has become so complex and diverse that a coding system has been formed to identify the various modes of pacemaker operation. Initially developed in 1974, the pacemaker coding system has undergone several revisions. The most recent version of the code was developed in 2002 through the joint efforts of the AHA and Heart Rhythm Society (formerly NASPE) and the British Pacing and Electrophysiology Group (BPEG).[10] The NASPE/BPEG Generic (NBG) Pacemaker Code is shown in Table 18-16 and is simply called the NBG pacemaker code.

The first letter describes the chamber or chambers of the heart in which pacing occurs: A, atrium; V, ventricle; and D, dual chamber.

The second position of the code indicates the chamber or chambers in which intrinsic cardiac activity is sensed: A, atrium; V, ventricle; and D, dual chamber.

The third position of the code denotes the pacemaker's response to sensed intrinsic cardiac activity. The letter "I" means that the pacemaker is inhibited from firing in response to a sensed intrinsic event. For example, if the pacemaker is set to a rate of 70, and if the patient's intrinsic rate exceeds 70 beats/min, the pacemaker will not fire. The pacemaker fires only if the patient's intrinsic heart rate drops below the programmed rate. Thus, the pacemaker functions on demand and is known as a demand pacemaker. Because the pacemaker is inhibited by adequate intrinsic heart activity, there is no danger of the pacemaker firing at an inappropriate time that could initiate a dangerous cardiac dysrhythmia, such as VT. The letter "T" indicates a pacemaker that triggers pacing stimuli in response to a sensed intrinsic beat. In a patient with complete AV block, a dual-chamber pacemaker is capable of sensing intrinsic atrial activity and triggering ventricular pacing stimulus in response to a sensed atrial event. The letter "D" designates a dual response (inhibited pacing output and triggered pacing after sensed event). The letter "O" in the third position designates a mode in which the pacemaker does not respond to sensed intrinsic activity. Pacemaker insensitivity to intrinsic activity is known as asynchronous pacing. This can be achieved by setting the sensitivity to the highest number or programming to asynchronous mode (ie, DOO, VOO).

Permanent pacemakers also may be switched temporarily to an asynchronous mode by placement of a large magnet over the pulse generator. This maneuver causes the pacemaker to fire without regard to intrinsic rate (fixed pacing), allowing assessment of firing and capture while the patient's rhythm is overridden by the fixed pacing pulse.

The fourth position of the code describes the presence or absence of rate modulation. The letter "O" denotes no rate modulation, and the letter "R" means that rate modulation is active. This is a feature in which the pacing rate varies in response to a physiological variable reflecting activity levels. The physiological variables used are mechanical vibration, acceleration, or minute ventilation. When patients increase their activity, the pacer detects the physiological response (eg, muscle vibration, increased respiratory rate) and increases the pacing rate to meet increased metabolic demands.

The fifth position of the code describes whether multisite pacing is present: "A" in the atrium, "V" in the ventricle, "D" in both atrium and ventricle, and "O" means that no multisite pacing is present.

In clinical parlance, the absence of a fourth or fifth letter designation signifies no rate modulation and no multisite pacing. The first three positions are required when describing pacemaker mode, although all positions may be indicated for completeness.[8]

Pacing Modes

Knowledge of the pacemaker code helps the critical care nurse determine the type of implanted device, the intended mode of operation, and the actual mode of operation.

Table 18-16 The NBG Pacemaker Code

Position: Category				
I: Chamber(s) Paced	**II: Chamber(s) Sensed**	**III: Response to Sensing**	**IV: Rate Modulation**	**V: Multisite Pacing**
O = none	**O** = none	**O** = none	**O** = none	**O** = none
A = atrium	**A** = atrium	**T** = triggered	**R** = rate modulation	**A** = atrium
V = ventricle	**V** = ventricle	**I** = inhibited		**V** = ventricle
D = dual (A +V)	**D** = dual (A + V)	**D** = dual (T + I)		**D** = dual (A + V)

Adapted from North American Society of Pacing and Electrophysiology/British Pacing and Electrophysiology Group. The revised NASPE/BPEG generic code for antibradycardia, adaptiverate, and multisite pacing. Pacing Clin Electrophysiol 25(2):260–264, 2002.

Modes of operation can be classified as single- and dual-chamber modes.

AAI and VVI are single-chamber modes of operation in the atrium or ventricle. An AAI is a mode of operation for atrial pacemakers. With this mode of operation, there is **a**trial pacing, **a**trial sensing, **i**nhibited response to sensed events, and no rate modulation. Temporary atrial pacemakers most often are set to an AAI mode and are particularly useful in overdrive pacing of atrial dysrhythmias.

DDD mode provides **d**ual-chamber pacing, **d**ual-chamber sensing, **d**ual response to sensed events (inhibited or triggered). Dual-chamber modes allow physiological pacing, in which atria and ventricles are sequentially sensed or paced. DDDR mode has the additional feature of rate modulation. VDD mode paces only in the ventricle, but this mode senses atrial and ventricular events and has dual response to sensed events. Therefore, an atrial event (P wave) will trigger a ventricular event. If an intrinsic R wave is sensed, ventricular pacing is inhibited. This mode is particularly useful in patients with intact sinus node function but with high-grade AV block.

Pacemaker Malfunction

Permanent Pacemaker Malfunction

Pacemaker malfunction can be a result of inappropriate programming (pseudomalfunction) or true component malfunction. Although pacemakers are now manufactured to provide more complex capabilities and are generally considered to be more reliable, unanticipated pacemaker malfunction (eg, "advisories") do occur.[11] For this reason, it is important for the patient to know the manufacturer, model, and serial number of his or her pacemaker components (pulse generator and leads) and to ascertain that they have been appropriately registered with the manufacturer. This is usually provided to the patient after the implant procedure, and the patient is given an implanted device identification card.

Advisories are issued when devices of a certain lot or model are associated with component or battery failure. Most manufacturers have toll-free telephone numbers that can provide information on advisories. However, it is important that the patient contact his or her physician, who can provide counseling on appropriate interventions for the advisory. At times, simple programming or monitoring may be all that is needed.

Temporary Pacemaker Malfunction

Temporary pacemaker malfunction should be addressed systematically. First and foremost, immediate action is required to restore pacemaker capture when the patient has no underlying rhythm. These steps are to be followed:

1. Increase pulse generator output (in mA) to the highest setting, asynchronous mode (VOO, DOO).
2. Check patient hemodynamics, simultaneous multiple ECG lead recordings; intervene if appropriate with transcutaneous pacing, atropine sulfate, or isoproterenol.
3. Check all connections.
4. Replace pulse generator or battery; be prepared for transcutaneous pacing backup during change.

Proceed with troubleshooting if the patient's condition is stable. Table 18-17 describes troubleshooting strategies for temporary pacemaker malfunction.

Types of Malfunction

FAILURE TO DISCHARGE. If stimulus discharge from the pacemaker causes an artifact, or "spike," to appear on the ECG, failure to discharge may be manifested by absence of the artifact and unexplained loss of pacing. The cause of this failure may be within the generator itself, either processor or battery failure. Processor failure is not common, but battery failure is more prevalent among patients who are noncompliant with follow-up or unaware of the longevity of their pacemaker battery. This may be evaluated by measuring the output values of the generator through a programmer. In extreme cases, the generator fails to communicate with the programmer. If the situation is emergent, the physician may insert a temporary transvenous pacemaker to support the patient hemodynamically until the permanent pacemaker problem can be corrected.

FAILURE TO CAPTURE. Failure of the pacing stimulus to capture the ventricles or atria is noted by the absence of the QRS or P wave immediately after the pacemaker artifact on the ECG (Fig. 18-36). Failure to capture may be caused by elevated threshold, a lead dislodgment, or impending battery depletion. These conditions may create insufficient output delivery to meet the capture threshold. If the patient is pacemaker dependent and becomes symptomatic, drug therapy (atropine, isoproterenol), transcutaneous pacing, or CPR may be required until the cause of the problem is found and corrected.

OVERSENSING. Oversensing occurs when the pacemaker detects events other than those intended. For example, in VVI pacing, if large T waves are sensed in addition to the R wave, the pacemaker is inhibited, and pacing at a slower than programmed rate may be noted. Similarly, electromagnetic interference (EMI) may result in inappropriate sensing and as a result, incorrectly activate the inhibited or triggered mode of a dual-chamber pacemaker. Oversensing may be caused by electrode displacement, inappropriate sensitivity settings, EMI or impending lead fracture. A partially fractured lead often allows signals to saturate the sensing amplifier, causing oversensing and inhibition of pacing output in demand mode. On the surface ECG, oversensing mimics failure to discharge because oversensing may cause inhibition of the pacing stimuli; for example, in a DDD pacemaker, a sensed P wave usually triggers a ventricular stimulus after completing the timed P-V interval; however, if the ventricular lead senses an intrinsic signal (eg, oversensing noise), the ventricular stimulus is inhibited. The only way to confirm oversensing is to examine IC-EGM through a programmer. If noise is recorded on the IC-EGM, then the problem is due to oversensing.

To correct suspected oversensing in a temporary pacemaker system, the nurse should check the connection between the temporary pacemaker and the lead. Electrical noise from an improperly connected lead could cause oversensing. EMI should be investigated, and the grounding wires of all electrical equipment should be checked. Unipolar leads are particularly prone to EMI because of the broad sensing field from the tip of the lead (negative pole) to the generator (ground). The sensitivity may be decreased by turning the dial toward asynchronous, toward

Table 18-17 Troubleshooting a Temporary Pacemaker

Problem	Cause	Intervention
Failure to discharge: No evidence of pacing stimulus, patient's heart rate below programmed rate	Due to battery depletion or pulse generator failure, output or timing circuit failure	Replace battery or generator.
	Due to loose cable connection	Check all connections for tightness.
Failure to capture: Pacing stimulus not followed by ECG evidence of depolarization	Due to lead dislodgment	Review chest film, turn patient to left lateral decubitus position until lead can be replaced.
	Due to broken connector pins or fractured extension connecting cable	Connect wire directly to generator to diagnose cable problem, replace connecting cable.
	Due to incompatibility of wire pins with cable or to generator	Ascertain a secure fit of the exposed pin to the cable or the generator, adjust connection or replace pulse generator.
	Due to output setting (mA) too low	Check capture thresholds and adjust output to a two- to threefold safety margin.
	Due to perforation	Review 12-lead ECG, report signs of perforation, stabilize hemodynamics.
	Due to lead fracture without insulation break	Check intracavitary ECG; if evidence of fracture in one pole, unipolarize lead; if total fracture, replace lead.
	Due to increase in pacing threshold from medication or metabolic changes	Check laboratory test results, correct metabolic alterations, review medications and vital signs, increase output.
Oversensing: Device detects noncardiac electrical events and interprets them as depolarization	Due to oversensitive setting	Reduce sensitivity (value [in millivolts] should be larger to make pacer less sensitive); if patient is pacer dependent (no intrinsic R wave), program to asynchronous mode until problem is corrected.
	Due to device detecting tall T waves and interpreting them as R waves	Increase ventricular refractory period beyond T wave.
In dual-chamber pacing, cross-talk is a form of oversensing: The device detects signals from the other chamber and inhibits; in atrial channel, R waves are detected as P waves.	Caused by atrial lead dislodgment	Recheck atrial capture thresholds; if high, dislodgment is probable.
In ventricular channel, atrial pacing stimulus afterpotential is detected as an R wave, with V pacing inappropriately inhibited	Due to high output from atrial channel	Reduce output from atrial channel, decrease ventricular channel sensitivity (higher millivolt value).
	Due to electrical interference, improperly grounded electrical devices	Remove nongrounded equipment.
Undersensing: Device fails to detect intrinsic cardiac activity and fires inappropriately	Due to asynchronous mode setting (VOO, DOO, AOO)	Reprogram to synchronous mode (VVI, DDD, AAI).
	Due to small intrinsic amplitude	Increase sensitivity (turn sensitivity dial toward lower millivolt value).
	Due to lead dislodgment	Recheck capture thresholds; if high, lead probably dislodged and needs repositioning.
	Due to lead insulation break	Check lead with pacing system analyzer, if impedance too low (<200 Ohms), insulation break is likely, and lead needs to be replaced or can be temporarily placed in unipolar configuration

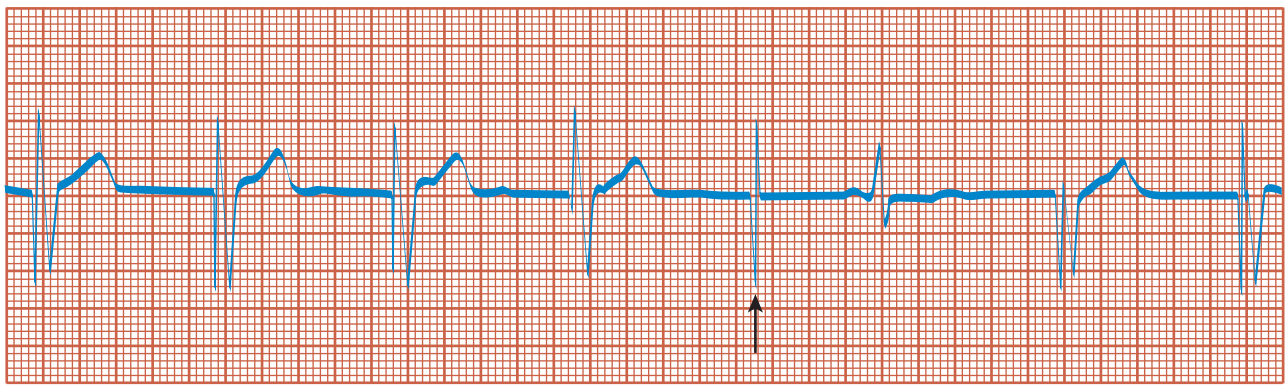


FIGURE 18-36 ▲ Electrocardiogram (ECG) strip showing evidence of failure to capture. Note that pacing stimulus is not followed by a QRS complex.

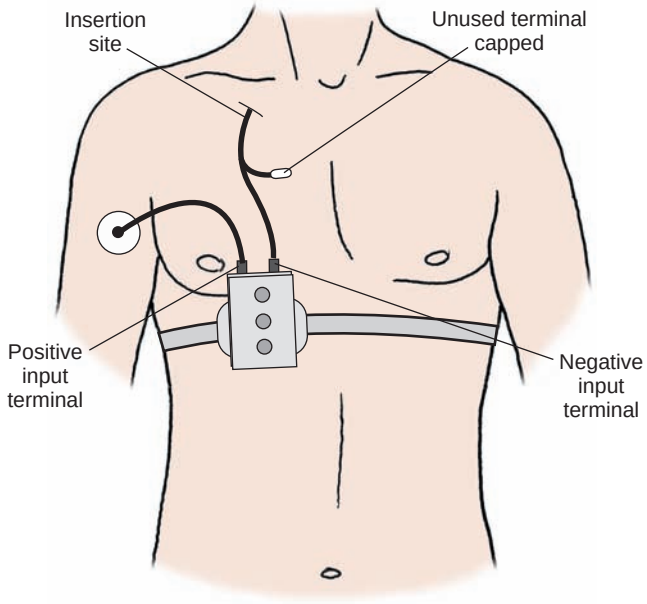


FIGURE 18-37 ▲ Unipolarizing a temporary bipolar lead. This uses an older-style generator with positive and negative terminal input and is for single-chamber pacers only.

a higher mV value. Oversensing due to partial wire fracture in a temporary transvenous bipolar catheter may also be corrected by converting to a unipolar system. To diagnose, an intracavitary ECG is obtained by connecting a single pole to a surface V lead using an alligator clamp connector and obtaining a unipolar V1 precordial recording. The recording should show a large voltage artifact amplitude and an elevated ST segment. A complete fracture shows a flat tracing. Noise on the recording that is not related to intrinsic ECG or diminished artifact amplitude demonstrates partial fracture. If one of the poles is intact, this lead is unipolarized by inserting the terminal tip of the intact pole into the negative port of the generator terminal. The tip of an external ECG single wire electrode applied on the skin is inserted into the positive port of the generator, serving as the ground (Fig. 18-37). The unused pole terminal of the lead is shielded with rubber tubing. Since a unipolarized bipolar lead in temporary pacemakers is subject to EMI, it should not be used indefinitely. The safest intervention for a fractured lead is replacement.

UNDERSENSING. Failure of the pacemaker to sense intrinsic beats is known as undersensing and results in inappropriately placed pacemaker artifacts on the ECG (Fig. 18-38). Undersensing may be a result of lead dislodgment, lead insulation defect, lead wire fracture, or inappropriately programmed sensitivity. Ventricular dysrhythmia caused by the pacemaker firing during the vulnerable phase of the T wave is of concern with undersensing. The most likely cause of sensing failure in the temporary pacemaker is lead displacement.

To correct undersensing problems in the temporary pacemaker, the nurse must first ascertain that the lead is properly connected to the temporary pacer. Undersensing may also be corrected by increasing the sensitivity of the device, which is done by turning the dial to a lower millivolt value (ie, detect a smaller signal). If problems persist, the physician may need to reposition or replace the lead. In permanent pacemakers, undersensing can sometimes be corrected by reprogramming to a more sensitive setting or by switching bipolar to unipolar mode.

Pacemaker Complications

Numerous possible complications are associated with cardiac pacemakers. The critical care nurse plays a vital role in the early detection and management of these complications.

Pneumothorax

Insertion of a transvenous lead through the subclavian vein can be complicated by traumatic injury to the lung by the exploring needle, allowing air to escape into the pleural cavity, because of the proximity of the subclavian vein to the apex of the lung. The symptoms may occur suddenly or may insidiously present up to 48 hours after the procedure. The symptoms include pleuritic pain, hypotension, respiratory distress, or hypoxia. A chest radiograph can reveal the extent of the trauma. When severe, placement of a chest tube allows for lung reexpansion.

Ventricular Irritability

Ventricular irritability at the site of the endocardial catheter tip is occasionally encountered in temporary pacing systems after initial catheter insertion. The premature ventricular complexes usually appear similar in configuration to the pacemaker complexes (Fig. 18-39). Irritability from the catheter as a foreign body usually disappears after a few hours. Persistent ventricular irritability may indicate lead dislodgment in both temporary and permanent pacing systems.

Perforation of Ventricular Wall or Septum

Perforation of the ventricular free wall or septum by the transvenous catheter occurs in a few patients. This may or may not result in cardiac tamponade. Elderly patients and patients on chronic corticosteroid or anticoagulant therapy are at highest risk. Perforation can be suspected if the patient demonstrates a change in precordial lead morphology on cardiac monitoring. RV apical pacing often results in a negative QRS in a V1 lead on a 12-lead ECG

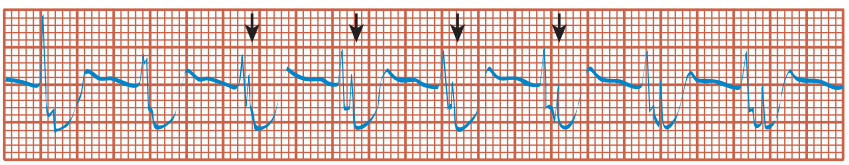


FIGURE 18-38 ▲ ECG strip showing evidence of undersensing. Failure of the ventricular demand pacemaker to detect the intrinsic rhythm is shown by pacemaker spikes at inappropriate intervals after spontaneous QRS complexes.

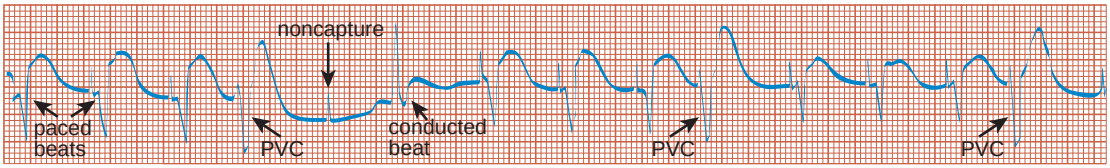


FIGURE 18-39 ▲ Ventricular demand pacemaker with premature ventricular contractions. This strip also shows one noncaptured pacemaker spike followed by a spontaneous conducted beat.

recording. Ventricular perforation may result in pacing from the left ventricle, and the QRS becomes positive in polarity. When ventricular wall perforation is suspected, pericardial tamponade, causing a decrease in blood pressure and an increase in sinus rate, can be confirmed by two-dimensional echocardiography.

Catheter or Lead Dislodgment

Dislodgment of the pacing catheter or lead may occur resulting in oversensing, undersensing, or failure to capture. A chest radiograph usually confirms the findings. Catheter or lead dislodgment usually requires repositioning.

Infection and Phlebitis or Hematoma Formation

Infection and phlebitis can occur at the temporary pacemaker insertion site, and infection or hematoma may occur at the site of permanent generator implantation. These sites must be inspected for swelling and inflammation and kept dry. Infection in permanent pacemakers needs immediate attention by a physician. In most cases, pacemaker pocket infection requires removal of the entire pacemaker system and replacement at a different site, after systemic antibiotics have been given. In temporary pacing sites, sterile technique must be used to prevent infection when dressings are changed.

Abdominal Twitching or Hiccups

Abdominal twitching or hiccups occur occasionally as a result of electrode placement against a thin RV wall and resultant electrical stimulation of the abdominal muscles or diaphragm. In patients with a LV lead for biventricular pacing, abdominal twitching may be due to phrenic nerve stimulation (PNS) by the LV lead in the lateral branch of the coronary sinus vein. PNS is usually uncomfortable for the patient but can sometimes be corrected by programming the lead polarity or reducing the output of the generator.[12]

After acute implantation, diaphragmatic stimulation may sometimes be associated with perforation. A drop in the patients' blood pressure and high capture thresholds accompanying diaphragmatic stimulation warrants critical observation and evaluation.

Pocket Erosion

Erosion at the implantation site occurs rarely in the early postimplantation period and is more often regarded as a late complication of permanent pacemaker implantation. At times, erosion heralds a fulminant infection. However, at other times, erosion may be due to poor skin integrity or pressure of the generator on thinning tissues. When pre-erosion is detected, prompt reoperation for pocket relocation can protect the patient from a potentially malignant cause of systemic infection and salvage the device. Once a pacemaker system erodes, an aggressive infection occurs throughout the lead system into the heart, making complete system removal a necessary course of action.

Nursing Management

Critical care nurses play a key role in caring for patients with a pacemaker. The nurse is responsible for comprehensive assessment of the patient, patient and family education, ECG monitoring, and patient safety. For intervention guidelines for patients with a temporary transvenous pacemaker, see Box 18-23 on page 349, and for a list of nursing diagnoses for the patient with a pacemaker, see Box 18-24 below.

Patient Assessment

The critical care nurse may be the first to detect the patient's dysrhythmia that necessitates pacing. Knowing the indications for pacing and how to initiate emergent transcutaneous pacing is essential for the critical care nurse. After a comprehensive assessment and stabilization of the patient, the critical care nurse may need to assist the physician in inserting a transvenous or permanent pacing system.

An important aspect of preimplantation of a pacemaker includes an assessment of the patient's medical and social history. For example, knowledge of a previous fracture to the clavicle may lead to avoidance of implantation on the same side of the fractured clavicle because of potential anatomical distortion. A subclavian approach may be avoided in a person with a history of a collapsed lung or previous lobectomy. A patient with a right arm arterial-venous fistula is best served with a left-sided implant. For social history, avocations such as hunting, professional sport activities, and even just preferential arm dexterity come into consideration. For example, the right pectoral region should not be used in a right-handed tennis player.

BOX 18-24 EXAMPLES OF NURSING DIAGNOSES

For the Patient With a Pacemaker

- Anxiety related to life-threatening cardiac disease requiring pacemaker
- Deficient Knowledge related to newly diagnosed condition
- Risk for Infection related to invasive procedure and presence of foreign body
- Decreased Cardiac Output related to pacemaker syndrome or absence of AV synchrony

To assess patients with pacemakers accurately, the nurse must understand the pacemaker code to know the type of pacer used and the intended mode of the device. The nurse must be aware of the patient's underlying rhythm so that if the pacemaker fails, the nurse is prepared to treat any life-threatening dysrhythmias.

A thorough assessment also helps the nurse determine the patient's physiological response to pacing therapy. Important parameters to assess include pulse rate, underlying cardiac rhythm, blood pressure, activity tolerance, and evidence of dizziness, syncope, dyspnea, palpitations, or edema. The nurse should be attentive to results of chest radiographs, blood tests, and other relevant laboratory tests. If a permanent pacemaker has been implanted, the incision is examined for swelling, redness, drainage, hematoma, and tenderness.

Psychosocial assessment is another essential component of comprehensive care of the patient with a cardiac pacemaker. Patients' psychosocial responses to the need for cardiac pacing may differ. Some may be relieved to have a device that supports the functioning of their heart, whereas others may be anxious about the technology and express fears of dying. If a permanent pacemaker is implanted, patients and families should be encouraged to join support groups where they can share their fears and concerns with others who depend on pacing technology.

Patient and Family Education

A planned and systematic approach to teaching the patient and family about cardiac pacing is a vital part of nursing care. Teaching a patient about pacemakers begins at the time the decision for pacemaker insertion is made. The nurse can begin by eliciting the patient's previous knowledge of pacemakers and clarifying any misconceptions. Nothing is assumed about the patient's understanding. If appropriate, the difference between heart block and heart attack is clarified. The patient may confuse cardiac monitoring with pacing and become anxious when the monitoring electrodes are removed.

The patient and family should be told why the pacemaker is necessary. The anatomy of the heart is discussed in general terms when explaining the need for pacing and how the pacemaker takes the place of or complements spontaneous rhythm. The insertion procedure and the immediate postinsertion care that can be expected are explained.

Many booklets and media presentations are available to aid the nurse in teaching the pacemaker patient. Visual and written guidelines are helpful for the patient and family to review after discharge from the hospital.

The depth of teaching that is appropriate and the teaching tools used may depend on the patient's age, intellect, attention span, vision, and interest in learning. Initial teaching should be confined to the positive aspects of life with a pacemaker. Knowledge of the function and care of the pacemaker are of no interest until the patient is able to accept it as part of life. Box 18-25 provides a guide for teaching patients and families about living with a pacemaker.

Electrocardiogram Monitoring

Careful monitoring of the ECG of the patient with a cardiac pacemaker is an essential component of comprehensive patient assessment. The first step in the analysis involves examining the strip for evidence of pacemaker stimulation. This evidence is noted by the presence of pacing spikes on the strip. Unipolar pacing spikes are usually tall and visible, but bipolar pacing spikes may not be visible in certain leads. Each pacing spike should result in capture. If the pacing lead is in the atria, a pacing spike is followed by a P wave. If the pacing wire is in the ventricle, the spike is followed by a wide QRS complex (Fig. 18-40). However, a narrower QRS following a pacing spike does not necessarily mean there is pacemaker malfunction. When fusion is present, the patient spike appears right before the intrinsic QRS (see Fig. 18-40A). Biventricular pacing (pacing both ventricles) for cardiac resynchronization in HF also results in a narrower QRS.

The sensing function of the pacemaker is evaluated next. If the pacemaker does not sense intrinsic cardiac activity (undersensing), inappropriate pacemaker spikes may appear throughout the underlying rhythm. An oversensing problem can be detected when the pacemaker senses events other than the intrinsic rhythm and is inappropriately inhibited in that chamber or causes a triggered response in the other chamber.

The third step in evaluating the ECG is to measure various intervals in milliseconds (ms). Each small box on the ECG paper represents 40 ms, and one large box represents 200 ms. The duration of each interval is compared with the programmed setting for that interval.

The first interval, the pacing interval, is the amount of time between two consecutive pacing spikes in the chamber being paced. This interval is used to determine the pacing rate. To calculate the pacing rate, the nurse counts the number of milliseconds between two consecutive atrial spikes or two consecutive ventricular spikes (Fig. 18-41). To convert from milliseconds to beats per minute, the following formula is used: 60,000 ms/min divided by the number of milliseconds between pacing spikes equals the pacing rate.

The next interval to measure is the AV interval, also known as the AV delay. This interval is analogous to the PR interval on the ECG. The AV interval is measured from the beginning of an intrinsic P wave or an atrial pacing spike to the beginning of the intrinsic QRS complex or the ventricular pacing spike (see Fig. 18-41).

The third interval to measure is the ventriculoatrial (VA) interval, also called the atrial escape interval. The VA interval is the amount of time from a ventricular paced or sensed event to the next atrial paced stimulus (see Fig. 18-41). The sum of the AV and the VA interval equals the pacing interval.

Patient Safety

Electrical safety precautions must be observed when the patient has a temporary pacemaker. Electrical equipment in the room is kept at a minimum and must be properly grounded. Use of a nonelectric bed is preferable. If an electric bed is used, it must be properly grounded or remain disconnected from AC. Only battery-operated electric shavers, toothbrushes, or radios are recommended. An AC-powered television may be used if it is operated by someone who is not in contact with the patient. The nurse should avoid simultaneous contact with the patient and any electrical equipment.

BOX 18-25 TEACHING GUIDE *Living With a Pacemaker*

Patient Activity
- Start passive and active range-of-motion exercises on the affected arm 48 hours after implantation to avoid "frozen shoulder." For those with new leads implanted, avoid abduction of the affected arm above the shoulder level for 4 to 6 weeks to prevent lead dislodgment.
- Avoid activities that may result in high impact or stress at the implantation site.
- Return to work at the discretion of your physician after discussing the type of work you do and what your job entails.
- Return to whatever degree of sexual activity you prefer.
- Your pacemaker will set off the alarm on metal-detector devices in airports, so avoid going through the detector gates. Show your pacemaker identification card. A manual search may be done or a magnetic wand may be used. Do not allow the wand to linger at the pacemaker site for an indefinite amount of time because the magnet in the wand may temporarily put the pacemaker into asynchronous mode. The metal detector or wand will not cause any permanent damage to your pacemaker.

Signs of Pacemaker Malfunction
- Be alert for symptoms of pacemaker malfunction: those associated with decreased perfusion of the brain, heart, or skeletal muscles. Be particularly mindful of return of symptoms you experienced before pacemaker implantation.
- Report any dizziness, fainting, shortness of breath, undue fatigue, or fluid retention. Fluid retention includes sudden weight gain, "puffy ankles," "tightness of rings," and so forth.
- Take your pulse once daily upon awakening. Report a pulse rate over 5 beats/min slower than that at which pacemaker is set.
- Be aware that your pulse may be somewhat irregular with a demand pacemaker and has some spontaneous beats and paced beats. This does not signify pacemaker malfunction.

Signs of Infection
- Report any redness, swelling, warmth, drainage, or increase in soreness at the implantation site.
- Report a fever of undetermined source.

Medications
- Antibiotics are usually given within 24 hours of pacemaker implantation. Report any unusual reactions to your physician.
- Medications that were withdrawn before pacemaker implantation may need to be restarted. Check with your physician about such medications as beta blockers, digitalis, or blood thinners. Know the name of the medication and the dose, frequency of administration, side effects, and use of each medication.
- If warfarin (Coumadin) therapy is restarted, warfarin blood levels should be rechecked after reinitiation of the medication.

Considerations for Home Care
- Carry a pacemaker identification card at all times. This card shows the brand and model of your pacemaker, the date of insertion, and the implanting physician.
- Wear a medical identification bracelet or necklace stating that you have a pacemaker.
- Adhere to a schedule of follow-up visits with your physician or clinic. The follow-up visit will include an interval history and ECG recording. Many pacemaker clinics have specialized equipment available to determine pacemaker and lead performance and to predict battery longevity. Some clinics have the capability for obtaining some of this information by telephone, reducing the necessity for travel to the clinic. However, when pacemaker follow-up is done by phone transmission, have the pacemaker checked at least once a year in the pacemaker clinic. Many problems related to the pacemaker pocket or intermittent failure of components cannot be picked up through telephone transmission tests.
- If you have any symptoms similar to those you had before pacemaker insertion, have the pacemaker checked. Be alert for other symptoms of malfunction, such as unexplained dizzy spells, fatigue, or a slow pulse.
- Inform any physician or dentist of the pacemaker and of the medications you are taking.

Pulse Generator Replacement
- Follow-up is intensified when the pacemaker battery approaches its elective replacement indicator. Avoid extended absences or vacations without consulting your physician at this time.
- Be aware that when the battery depletes, the generator stops working.
- The battery cannot be removed from the generator, so the entire generator is replaced when the battery is low.
- Generator replacement can be done within a 24-hour stay, as long as the leads are in good condition. Usually only the generator needs to be replaced.

Considerations for the Older Patient
- Report any changes in skin condition at the pacemaker site. Sudden weight loss or poor nutritional status may predispose elderly patients to pocket erosion.
- Report symptoms, such as fatigue, neck pulsations, and lack of energy. Loss of AV synchrony in patients with single-chamber VVI pacemakers may result in pacemaker syndrome.
- If the pacemaker feels like it is "flipping" inside the pocket, report it to your doctor and do not reposition it. When the skin is loose or when the patient "twiddles" with the pacemaker, the leads can become tangled and coiled and may fracture.

The patient's bed must be kept dry at all times. Diathermy and electrocautery equipment should not be used because their output may be sensed by and inhibit the demand pacemaker.

The literature on ensuring electrical safety in temporary pacemaker wires has been sparse; however, manufacturers have been guided by the FDA to increase vigilance over patient safety. Currently manufactured leads have no exposed areas after they are inserted tightly into the connecting cable. The use of rubber gloves to handle temporary pacing lead terminal pins is often recommended. Most manufacturers supply connecting cables with the pulse generator to ensure compatibility. Some connecting cables are nonsterilizable, and integrity may be compromised by resterilization. Care should also be taken to ensure that nonsterile cables are not in close proximity with the sterile field during insertion.

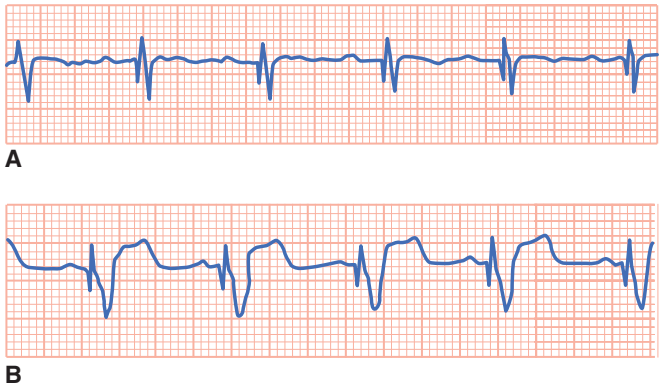


FIGURE 18-40 ▲ Pacemaker initiated beats. **A:** The pacemaker artifact is followed by intrinsic QRS complex deflection. **B:** Pacemaker capture (ventricular capture) beats with typical widening of QRS complex.

When using the older pulse generator model, the hard plastic cover over the dials of the temporary generator must be secured in place to prevent inadvertent change in settings. The generator should be attached to the patient's arm or visibly pinned to the patients' gown. The catheter should be taped securely to the patient's skin without direct tension on the catheter. Motion of the extremity nearest the catheter entry site should be minimized, especially if the femoral site has been used.

According to manufacturers of permanent pacemaker generators, very few electrical hazards are associated with the permanent generators currently in use. These generators are shielded from external electrical sources and usually are not affected by microwave ovens or small appliances. There have been rare reports of unipolar pacemakers being affected by large electromagnetic fields and radiofrequency signals, such as radio transmitters. Precautions should be taken to avoid large magnetic fields or use a cellular phone in close proximity (<6 inches) to the device.

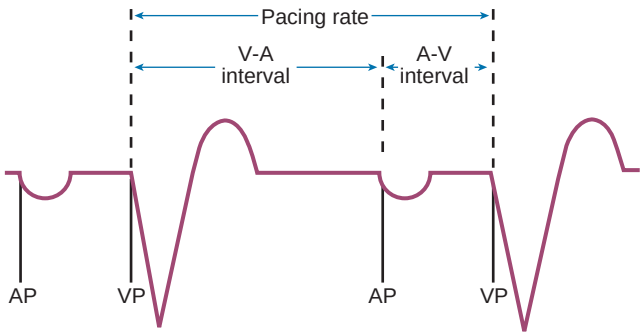


FIGURE 18-41 ▲ The intervals measured on an ECG strip for a patient with a pacemaker. The pacing interval is the amount of time between two consecutive atrial pacing spikes or two consecutive ventricular pacing spikes. The atrioventricular (AV) interval is measured from the beginning of a P wave or an atrial pacing spike to the beginning of an intrinsic QRS complex or the ventricular pacing spike. The ventriculoatrial (VA) interval is measured from a ventricular pacing or sensed beat to the next atrial pacing spike. The sum of the AV and VA intervals equals the pacing interval.

▲ Implantable Cardioverter–Defibrillators

Sudden cardiac arrest continues to be a leading cause of death in the United States.[13] Sudden death may be caused by the rapid loss of heart function due to VF. VT and fibrillation can be corrected when treated within minutes with electrical cardioversion or defibrillation.

In the late 1960s, Dr. Michel Mirowski and Dr. Morton Mower developed a device called an ICD to treat patients at risk for sudden death due to ventricular dysrhythmia. In 1980, the first device was implanted successfully in a person. The device was found to be safe and effective and, as a result, received FDA approval in 1985. Since its initial use in 1980, the ICD generator and lead technology have undergone many improvements in design and function. With these improvements, as well as with expanded indications and increased understanding of patients at risk, ICD implantation has rapidly increased.

Indications for Implantable Cardioverter–Defibrillators

Uniform criteria for ICD implantation were established by the Joint Committee of the ACC, AHA, and Heart Rhythm Society by class indications.[9] Class I indication recommends implantation. Class II includes conditions for which ICDs may be used, with less than sufficient evidence or divergence of opinion regarding the necessity of insertion. Class III includes conditions for which there is evidence that ICD implantation is unnecessary or may be harmful. Box 18-26 lists the indications for use of ICD therapy.

The Implantable Cardioverter–Defibrillator System

The purpose of an ICD is to monitor the patient's rhythm continuously, diagnose rhythm changes, and treat life-threatening ventricular dysrhythmias. Similar to a pacemaker, the ICD consists of a lead system and a pulse generator containing the battery, capacitors, and circuits. The lead system and the pulse generator have undergone significant changes in design and function since their initial use in 1980.

The Pulse Generator

Early ICD pulse generators were large and heavy in comparison to the pacemaker pulse generator. Because of their size and weight, these ICD pulse generators required implantation in the patient's abdomen. Currently used ICD pulse generators are not much larger than the earlier models of pacemakers and can be implanted in the pectoral area. The size of the device is shown in Figure 18-42. Lithium silver vanadium oxide (Li/SVO) batteries provide the power source for ICDs. Improved circuit design has expanded the capabilities and functions of the ICD.

The Lead System

Lead systems sense the life-threatening ventricular dysrhythmia and deliver a shock to convert the dysrhythmia. Initially,

BOX 18-26 Indications for Implantable Cardioverter–Defibrillators (ICDs)*

Class I: Conditions Where ICD is Effective/Beneficial

1. Survivors of cardiac arrest due to ventricular fibrillation (VF) or ventricular tachycardia (VT), after evaluation to exclude transient or reversible causes
2. Spontaneous sustained VT associated with structural heart disease
3. Syncope of undetermined origin with induced sustained VT or VF at electrophysiological study
4. LV dysfunction with EF less than 35% due to prior MI more than 40 days, and NYHA functional class II or III
5. Nonischemic dilated cardiomyopathy (ND-CM) with an EF less than or equal to 35% and in NYHA functional class II or III

Class IIa: Conditions Where ICD is Reasonable

1. Patients with unexplained syncope, significant LV dysfunction and ND-CM
2. Sustained VT and normal or near normal ventricular function
3. Hypertrophic cardiomyopathy with one or more major risk factors for sudden cardiac death (SCD)
4. Arrhythmogenic right ventricular dysplasia cardiomyopathy with one or more risk factors for SCD
5. Syncope and/or VT while on beta blockers in patients with long QT
6. Nonhospitalized patients awaiting transplantation
7. Brugada syndrome with syncope
8. Brugada syndrome with documented VT that has not resulted in cardiac arrest
9. Catecholaminergic polymorphic VT with syncope and/or sustained VT while on beta blockers
10. Cardiac sarcoidosis, giant cell myocarditis, or Chagas disease

Class IIb: Conditions Where ICD may be Considered

1. Nonischemic heart disease with an EF less than or equal to 5% with NYHA functional class I
2. Long QT syndrome and risk factors for SCD
3. Syncope and advanced heart disease in which thorough evaluations have failed to define a cause
4. Familial cardiomyopathy associated with sudden death
5. LV noncompaction

Class III: Conditions Where ICD is not Useful or may be Harmful

1. Conditions that meet Class I, IIa, and IIb but with expected survival less than 1 year
2. Incessant VT or VF
3. Patients with significant psychiatric illnesses that may be aggravated by device implantation or preclude follow-up
4. NYHA Class IV patients with drug-refractory congestive heart failure (HF) and not candidates for cardiac transplantation or CRT-D
5. Syncope of undetermined cause in a patient without inducible VT and without structural heart disease
6. VT or VF amenable to surgical or catheter ablation
7. VT due to reversible cause in the absence of structural heart disease

*Box does not include indications for children, adolescents, and patients with congenital heart disease.

Adapted from Epstein AE, DiMarco JP, Ellenbogen KA, et al: ACC/AHA/HRS 2008 Guidelines for device-based therapy of cardiac rhythm abnormalities: Executive summary. A report of the American College of Cardiology/American Heart Association task force on practice guidelines (ACC/AHA/NASPE 2002 guideline update for implantation of cardiac pacemakers and antiarrhythmia devices). Heart Rhythm e37–e38, 2008.

the lead system consisted of a pair of epicardial patches for energy delivery and epicardial coils for sensing. The leads were often implanted at the time of CABG surgery, when indicated, or by the subxiphoid approach. The sensing coils were later replaced with a long transvenous lead positioned in the RV endocardium. These leads were tunneled down from the subclavian insertion site to the generator in the abdomen. Improved lead design and smaller generators paved the way for prepectoral implantation. At this time, previously implanted epicardial leads are used only with replacement of ICD generators, when the leads are deemed to be usable. Newer implants use bipolar or tripolar transvenous leads for sensing and defibrillation. The sensing electrodes are bipoles at the tip of the lead. One unipolar coil in the distal portion of the ventricular lead serves as the defibrillation cathode, whereas another coil in the mid-proximal portion or the ICD generator serves as the defibrillation anode, giving rise to the term "active can" or "hot can." With dual-chamber ICDs, an additional bipolar electrode in the right atrium provides atrial sensing and pacing. In biventricular ICDs, a third lead is inserted in the coronary sinus and positioned in the lateral vein for LV stimulation and resynchronization of the ventricles.

FIGURE 18-42 ▲ Implantable cardioverter–defibrillators (ICDs), old and new models. Note the decrease in size and weight that has been achieved with the newer generation, allowing prepectoral implantation. (Courtesy of Medtronic Inc. Minneapolis, MN.)

Ideally, the ICD generator is implanted in the left pectoral area so that the heart is central to the vector of the defibrillation current (Fig. 18-43). Improvements in lead design have allowed ease of implantation not very dissimilar to that of permanent pacemaker implantation. It is no longer unusual for a patient to be discharged a day after ICD implantation.

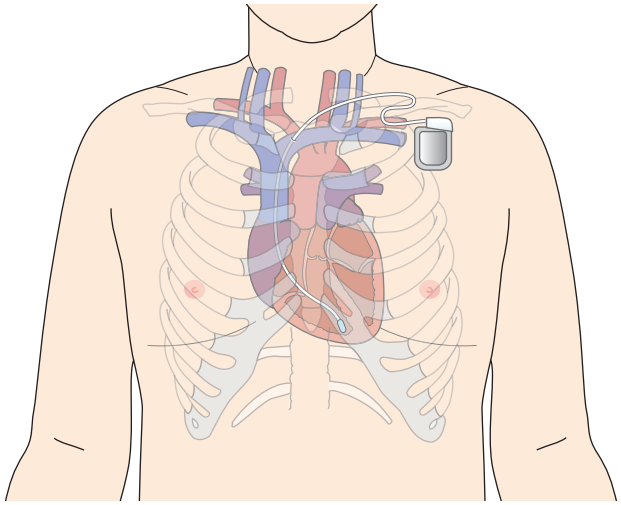


FIGURE 18-43 ▲ The ICD mechanical system consists of a generator and a sensing/pacing/defibrillating electrode.

Implantable Cardioverter–Defibrillator Functioning

ICDs have been categorized into "generations," based on their functionality. The first-generation ICDs were nonprogrammable devices that used a factory-specified rate criterion to detect ventricular dysrhythmias and delivered a shock at a preset energy level. In the mid-1980s, the second generation of the device became available and included programmable features, among them bradycardia and antitachycardia pacing and synchronized cardioversion. These features allowed the use of tiered therapy, the term used to describe different levels of therapy to treat a dysrhythmia. Table 18-18 illustrates the concept of tiered therapy. The first tier of therapy is usually antitachycardia pacing, which involves the carefully timed delivery of pacing stimuli. If antitachycardia pacing is not successful, the second tier of therapy is initiated by synchronized cardioversion. The joules for cardioversion can be programmed anywhere from 1 to 36 J, with the highest output dependent on device specifications. Current devices use ATP while charging to minimize painful shocks. If cardioversion is not successful, the third tier of therapy, defibrillation, is used. The energy delivered for defibrillation can be programmed to a maximum of 36 J, again depending on the model and capacity of a device. The number of defibrillation attempts varies with different devices, but six attempts is usually the maximum. If the patient is successfully converted to a life-compatible rhythm, but the rate is slow, ventricular demand pacemaker is initiated. Bradycardia pacing is usually intended for brief periods of pacing until normal rhythm resumes. However, with CRT, 100% biventricular pacing is desirable.

Table 18-18 Implantable Cardioverter–Defibrillator: Tiered Therapy

Type of Therapy*	Mode/Energy Level	Condition(s)
Antibradycardia pacing/Biventricular pacing	VVI/DDD/VDD	Bradycardia CHF
Antitachycardia pacing (ATP)	Burst/ramp ATP	AT†/VT (120–200 bpm)
Cardioversion‡	10–36 J	VT (180–230 bpm)
Defibrillation	30–36 J	VF (>230 bpm)

*Therapy and detection intervals (rate of tachycardia detected) are programmable.

†AT therapies limited to certain models.

‡ATP while charging allows painless therapy.

CHF, congestive heart failure; AT, atrial tachycardia; VT, ventricular tachycardia; VF, ventricular fibrillation.

Today's ICDs have many programmable features that allow the physician to tailor the device to the patient's needs. Bradycardia pacing therapies with biventricular pacing are common features of current ICDs. The availability of an atrial sensing lead allows for a more specific SVT discrimination algorithm. To improve discrimination of tachydysrhythmias, the device allows programming of discrimination algorithms, which withhold VT therapy when PSVT is confirmed. Some devices also have separate tiers of therapy for atrial tachycardia and AF or flutter.

All current ICDs are "noncommitted"; that is, therapy is aborted if the tachycardia terminates even while the ICD is charging. Patients with nonsustained VT need not suffer the discomfort of an inappropriate shock. Third-generation defibrillators have additional features, including memory and event retrieval. Event retrieval may involve successive R-wave analysis or the recording of an EGM during therapy. These methods document the dysrhythmia before and after the therapy, allowing the physician to analyze the problematic rhythm. These data can be correlated to the patients' symptoms to help further diagnose the dysrhythmia.

Current devices also have the ability to deliver PES using a programmer. PES, a noninvasive method similar to EPS, is used to induce a dysrhythmia to determine the device's ability to successfully terminate it with programmed therapies. It can also be used to ascertain the integrity of the shocking coil, to determine whether a suspected lead problem exists, and to define the defibrillation threshold (DFT) of a patient. The DFT is the lowest amount of energy tested that successfully converts VF. Certain antiarrhythmic drugs can increase DFTs. For the sake of patient safety, the device should be capable of delivering at least 10 J above the patients' DFT.

PES minimizes the need to test the device in a laboratory situation where catheters are placed in the patient's heart to induce the rhythm disturbance. Testing is done through the device itself, thus reducing the risk associated with the invasive procedure.

The Implantable Cardioverter–Defibrillator Code

The cardiac pacemaker code previously discussed has limited ability to describe modes of ICD function. As a result, in 1993, the AHA and Heart Rhythm Society (formerly NASPE) and BPEG developed the NASPE/BPEG Defibrillator Code.[14]

Table 18-19 The NBD Defibrillator Code

Position: Category			
I: Shock Chamber	**II: Antitachycardia Pacing Chamber**	**III: Tachycardia Detection**	**IV: Antibradycardia Pacing Chamber***
O = None	**O** = None	**E** = Electrogram	**O** = None
A = Atrium	**A** = Atrium	**H** = Hemodynamic	**A** = Atrium
V = Ventricle	**V** = Ventricle		**V** = Ventricle
D = Dual (A+V)	**D** = Dual (A+V)		**D** = Dual (A+V)

*Added in NBG code format (VVI, DDD).

Adapted from The NASPE/BPEG Defibrillator Code. Pacing Clin Electrophysiol 16(9):1776–1780, 1993.

Known as the NBD defibrillator code, it is similar to the NBG pacemaker code in describing ICD capabilities and operation. The four positions in the code are designated as: (I.) shock chamber, (II.) ATP location, (III.) means of tachycardia detection, (IV.) antibradycardia pacing chamber (Table 18-19).

Nursing Management

The critical care nurse plays a key role in the preimplantation and postimplantation management of patients with an ICD. Patient teaching is one of the most important tasks of the critical care nurse. Topics for discussion are included in the Box 18-27. Patients and families need to understand why an ICD is indicated, the purpose of an ICD, the basic parts of the ICD system, and how the ICD functions. Once the physician has determined the type of system to be used, the nurse reinforces the physician's explanation of how the device will be implanted and where the leads and pulse generator will be placed. The patient and family should be informed of the expected length of hospitalization and plans for follow-up care. Many resources for patient education are available from manufacturers of ICDs, including printed materials and videotapes. In addition, the patient and family may find it helpful to meet with a person who has an ICD. This person may be able to alleviate any fears or clarify misconceptions about living with an ICD.

BOX 18-27 TEACHING GUIDE *Implantable Cardioverter–Defibrillator*

When teaching patients receiving an ICD, be sure to include the following points:

- The purpose of an ICD and why it is indicated
- Components of an ICD
- How the ICD works
- How a shock feels
- How the ICD will be implanted
- The expected length of hospitalization
- Activities of daily living that can be tolerated postimplantation
- Rate cutoff and therapies programmed in the ICD
- Plans for follow-up care and when to call the doctor
- Importance of carrying an ICD identification card and/or wearing medical identification devices, such as a bracelet or necklace
- Need for carrying a list of current medications and dosages
- Safety precautions
- Importance of keeping emergency phone numbers readily available and calling the physician after receiving a shock, especially when not feeling completely recovered
- Importance of calling the physician immediately if you receive more than one shock or several in succession
- What the patient and family should do if a shock occurs
- Information the family, significant others, coworkers, and traveling companions should know about the ICD
- Precautions to be taken when traveling by air and informing airline security personnel of the ICD
- Encouraging of family members to take a cardiopulmonary resuscitation (CPR) course
- Benefits of support groups

In the postimplantation period, the nurse continuously monitors the patient for the development of any ventricular dysrhythmias and intervenes if necessary. If the patient experiences a sustained VT and no therapy is delivered, it may be that the rate of the tachycardia is below the programmed rate cutoff or that undersensing of the tachydysrhythmias is occurring. Knowledge of the parameters of the ICD and the rate of the patient's dysrhythmia can help the critical care nurse assess this situation correctly. A patient who has an ICD and who has a sustained, hemodynamically unstable rhythm should not be treated any differently from one without an ICD. External cardioversion can be given in an emergency in the absence of therapy from the patient's ICD. Care should be taken not to apply paddles near or above the ICD generator.

The nurse must be aware of the programmed settings and features of the patient's ICD to provide safe and competent care. Device information should be readily available at the bedside and clearly documented in the patient's chart. If the device fires, the status of the patient and the patient's rhythm is assessed and documented. When a device fires in the absence of dysrhythmias, there is a high probability of oversensing due to a dislodged lead, loose connection at the header, or an oversensitive setting. Immediate intervention by the EP service is necessary to avoid further discomfort to the patient.

Other immediate postoperative care (wound care, activity instructions) is very similar to that of the patient after pacemaker implantation (see Box 18-25, p. 357). Furthermore, because the operative approach is almost identical to that of a pacemaker, the complications that one might expect from a pacemaker implant can also be encountered after ICD implantation.

After consulting with the implanting physician, the nurse provides discharge instructions about resuming daily activities. Patients are usually cautioned against swimming or boating alone, climbing ladders, and operating equipment that may produce sparks or cause EMI. Patient and family teaching points (Box 18-27) should be reviewed with the patient and family with discharge instructions.

Discussion of psychosocial issues regarding living with an ICD also should be part of the discharge preparation. Although the emotional adjustment varies with each patient, many have fears about receiving their first shock. Other potential patient concerns include alterations in body image, return to work, participation in recreational activities, and reaction of family and friends to the device. If support groups are available, the patient and family should be encouraged to join.

CARDIOPULMONARY RESUSCITATION

The acuity of patients in an ICU requires the nurse to be especially vigilant for subtle signs of change in the patient's status. The numerous technologies and monitoring devices assist the nurse in delivering effective interventions, but physical assessment skills must be continually practiced and improved.

In any ICU, there is an increased chance that the patient's condition will deteriorate. The cessation of breathing and circulation is known as cardiopulmonary arrest. This condition is also referred to as sudden cardiac arrest, or SCA. When a patient is determined to be in cardiopulmonary arrest, seconds matter. Unless definitive action is taken within 4 to 6 minutes, the patient will suffer irreversible brain injury. Prompt intervention is necessary if the patient is going to have a chance of survival. Immediate and effective CPR often prevents fatal complications. CPR is divided into basic life support (BLS), which is discussed in this chapter, and ACLS.[1] The ACLS guidelines developed by the AHA can be found in Appendix A. This section of the chapter outlines assessment, procedures, interventions, and roles of the nurse in the cardiopulmonary arrest situation. Box 18-28 defines some common terms used during CPR.

▲ Causes of Cardiopulmonary Arrest

Box 18-29 outlines some of the causes of cardiopulmonary arrest. There are many additional causes of cardiopulmonary arrest. Determination of the cause of the arrest is secondary to rapid intervention. Once intervention to preserve life has been initiated, the cause of the arrest can then be ascertained, and any specific interventions designed to correct the underlying cause can be added to the BLS and ACLS measures.

BOX 18-28 Common Terms in Cardiopulmonary Resuscitation

Cardiac arrest: Abrupt cessation of effective cardiac pumping activity, resulting in cessation of circulation
Code: Informal term for emergency resuscitation
Crash cart: Emergency cart (see Table 18-20)
Resuscitation: Restoration of vital signs by mechanical, physiological, and pharmacological means
Clinical death: Absence of vital signs
Biological death: Irreversible cellular changes

BOX 18-29 Causes of Cardiopulmonary Arrest

Cardiac Causes
- Myocardial infarction
- Heart failure
- Dysrhythmia
- Coronary artery spasms
- Cardiac tamponade

Pulmonary Causes
- Respiratory failure secondary to respiratory depression
- Airway obstruction
- Impaired gas exchange, such as in acute respiratory distress syndrome
- Impaired ventilation, such as pneumothorax
- Pulmonary embolus

Electrolyte Imbalances
- Hyperkalemia
- Hypomagnesemia
- Hypercalcemia/hypocalcemia

Procedural Causes
- Pulmonary artery catheterization
- Cardiac catheterization
- Surgery

Miscellaneous
- Drug toxicity and drug side effects

▲ Assessment and Management of the Patient in Cardiopulmonary Arrest

Before implementing the resuscitative measures in a code situation, the patient must first be assessed. A myriad of technological monitoring devices are used in the ICU, but it is the everyday physical assessment skills used by nurses that are most accurate in determining a patient's status. The nurse needs to ensure that the alarm parameters of the bedside monitors are set accurately for each patient. The default settings are not always appropriate.

Determine Responsiveness

The nurse first determines the patient's responsiveness before initiating CPR. The nurse should take no more than 10 seconds to check for breathing and a pulse before initiating CPR. If the patient is unresponsive, the nurse calls for help ("initiate a code") and initiates BLS measures. In the new AHA Guidelines for CPR, the new acronym to guide resuscitative efforts is C-A-B, rather than A-B-C as in previous years. Box 18-30 summarizes the priority interventions of resuscitation.

BOX 18-30 Circulation, Airway, Breathing

Circulation	• Compress chest at rate of at least 100/min • Compress chest to a depth at least 2 inches • Place backboard or use CPR setting on bed • Watch ECG monitor to assist with ensuring rate • Palpate pulses (radial, femoral, pedal) to determine effectiveness of compressions • Measure blood pressure manually
Airway	• Open patients airway using head tilt–chin lift maneuver (jaw thrust for cervically injured patients) • Place oropharyngeal airway (if possible) • Provide suction as necessary
Breathing	• Use Ambu bag, set for 100% oxygen • Aim for compression to breath ratio 30:2 • Deliver breaths at rate of 6–8/min once advanced airway is in position • Maintain seal around patient's mouth and nose • Observe for chest rise and fall • Pulse goniometry • Auscultate for bilateral breath sounds

Circulation

Chest compressions provide blood flow to the brain and vital organs. Even with an apneic patient, there is still enough oxygen bound to hemoglobin to allow for oxygen delivery. This change in the interventions required is based on numerous studies compiled by the International Liaison Committee on Resuscitation (ILCOR) that have shown that in out-of-hospital cardiac arrest situations, early initiation of chest compressions have improved survival rates. Too much time was being lost assessing for breathing, positioning the patient, opening the airway and delivering two rescue breaths before initiating chest compressions. Additional changes to the CPR protocol are: (1) Compressions must be delivered at a rate of at least 100/min, and (2) the adult sternum should be depressed at least 2 inches for adult patients.

The arrival of a second rescuer allows for compressions to continue unabated while the second rescuer can open the airway and be ready to deliver breaths via a bag-valve device when the initial rescuer has completed a 30-chest compression cycle. External cardiac compression is a simple technique performed by standing at either side of the patient, placing the heel of one hand two to three fingerbreadths above the xiphoid process, and placing the heel of the other hand over the first. Firm compressions are applied directly downward, and the sternum is depressed *at least* 2 inches and released abruptly. The chest must be allowed to fully recoil between compressions. This rhythm is maintained at the rate of at least 100 times/min. A 30:2 compression–ventilation ratio is used, with a pause to provide the ventilation. If the patient is mechanically ventilated, there is no need for an interruption in compressions.[1] To be effective, these techniques must be learned correctly and applied skillfully[1] (Fig. 18-44). A recent study indicates that compressions-only CPR is favorable to conventional CPR in an out-of-hospital arrest situation.[2]

If one person must apply both ventilation and compression, the rescuer gives 30 chest compressions, then gives two breaths via a bag-valve device (also known as an Ambu bag). This routine may be maintained until additional members of the team arrive. When help arrives, one person delivers breaths using the bag-valve device, while another performs chest compressions. The patient's radial, carotid, or femoral pulse is checked at regular intervals to determine the adequacy of compressions. If compressions are being delivered effectively, a pulse should be felt in these regions. This circulation check should be performed by a third rescuer; there should be no interruption of CPR for a pulse check.[1] CPR considerations for the older patient are found in Box 18-31.

Position the Patient

The patient should be placed in a supine position on a firm, flat surface. This position enables the rescuer to open the

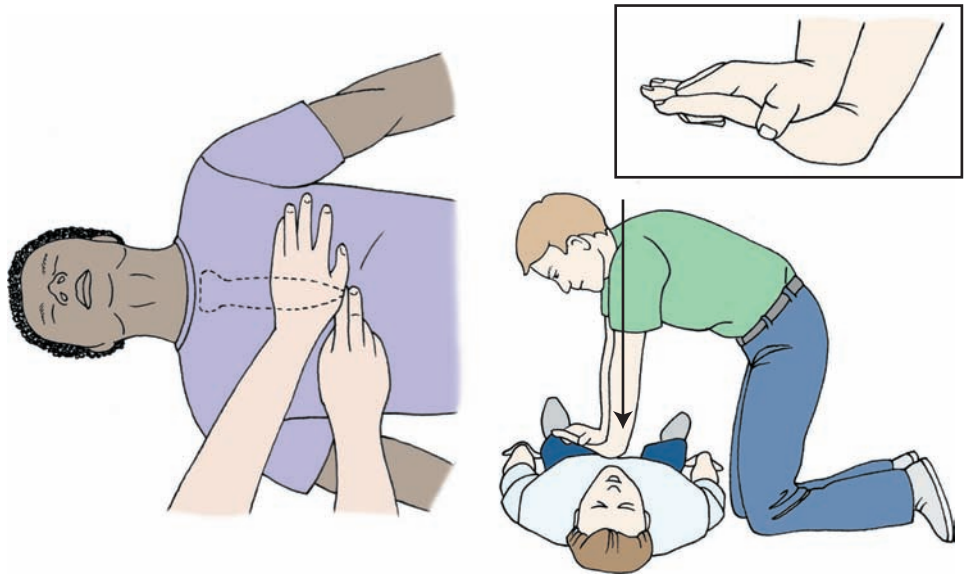


FIGURE 18-44 ▲ External chest compression. *Left,* proper hand position over lower portion of sternum. *Right,* correct rescuer position.

BOX 18-31 CONSIDERATIONS FOR THE OLDER PATIENT

Cardiopulmonary Resuscitation

- Assess for fractured sternum after CPR. Continue with CPR even if fracture occurs.
- Be certain the health care team implements the patient's desire for *do not resuscitate* or *do not intubate* orders.
- Consider family presence during code.
- Keep in mind the effect of medications due to delayed clearance and altered metabolic response.

airway and assess for the presence and effectiveness of any spontaneous breathing. If the patient is in a standard hospital bed, a resuscitation board is placed under his or her torso when help arrives. If the patient is in a specialty bed, the CPR setting on the bed is selected.

If the patient is found to be breathing effectively and there is no evidence of trauma, the patient is placed in the recovery position. The recovery position is used to reduce the possibility of airway obstruction by the tongue or by secretions or emesis. To place the patient in the recovery position, the rescuer kneels next to the shoulder of the patient. The rescuer lifts the arm of the patient nearest the rescuer and bends it at the elbow. The arm is then positioned so that the patient's palm of the hand is turned upward and moved toward the patient's face. The rescuer then lifts the leg of the patient furthest from the rescuer and crosses it over the patient's body, moving it toward the rescuer. One hand of the rescuer supports the patient's head during turning and the second hand is used to turn the patient's hips toward the rescuer (Fig. 18-45). Caution must be used when moving patients with suspected or actual spinal cord injuries. One rescuer should ensure that the patient's head remains in a neutral position.

Airway

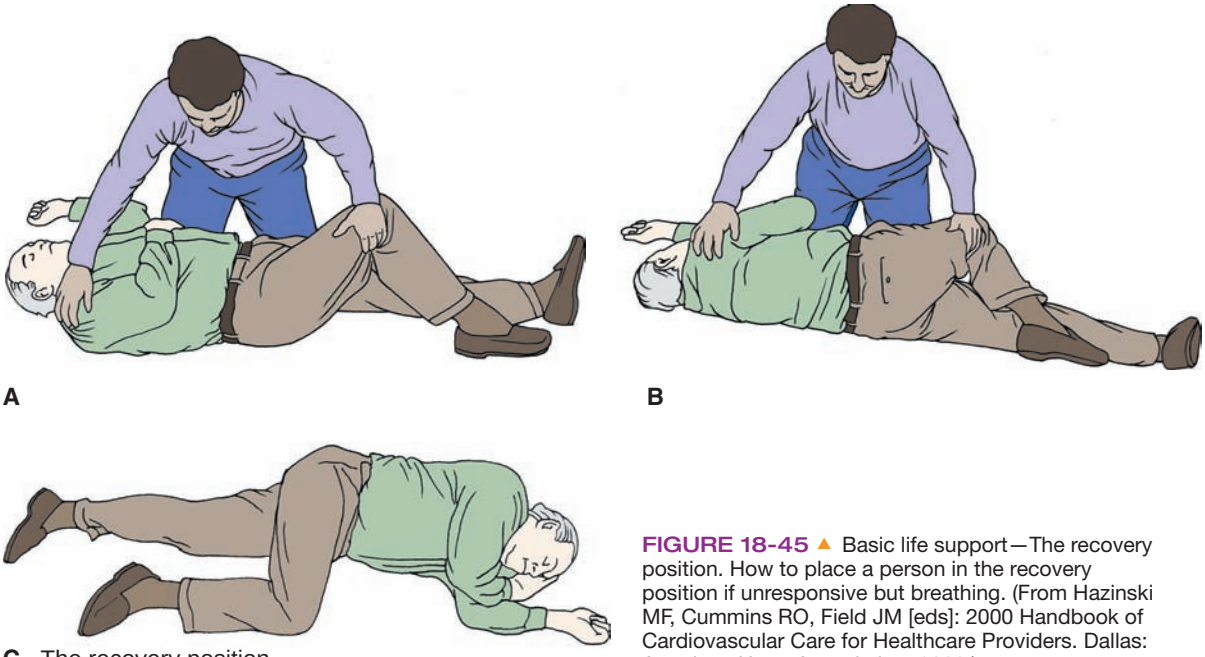
The nurse assesses for an adequate airway. The patient is positioned to ensure an open, patent airway. The patient is placed in the supine position, and the airway is opened using the head tilt–chin lift method. In this method, the head is tilted back, and the chin is raised to stretch the airway and advance the tongue in preparation for ventilation (Fig. 18-46).

In the case of patients with confirmed or suspected cervical spine injuries, the jaw thrust method is used (Fig. 18-47). The patient's head and neck must not be moved to ensure that no damage is done to the cervical spinal cord. Keeping the head in a neutral position, the rescuer places a hand on each side of the patient's head behind the temporomandibular joint and gently pushes the jaw forward. This will open the airway enough to allow for ventilation.

If spontaneous respirations have not returned once a patent airway has been established, then the patient must be assisted with breathing.

Breathing

Using a bag-valve device, the rescuer can deliver oxygen as rescue breaths. The bag-valve device is connected to 100% high-flow oxygen, and the mask portion is placed over the patient's mouth and nose. If the patient has an endotracheal tube or tracheostomy tube, there is an adapter that allows breaths to be delivered through an artificial airway. The bag reservoir is then squeezed to deliver the breaths. The adapter must be fitted properly; this allows the rescuer to deliver breaths using both hands to squeeze the Ambu bag. Observation of the patient's chest is necessary to determine whether the delivered breaths are actually ventilating the lungs. A second person assisting with CPR should auscultate all lung fields to confirm that the delivered breaths



A B C The recovery position

FIGURE 18-45 ▲ Basic life support—The recovery position. How to place a person in the recovery position if unresponsive but breathing. (From Hazinski MF, Cummins RO, Field JM [eds]: 2000 Handbook of Cardiovascular Care for Healthcare Providers. Dallas: American Heart Association, 2000.)

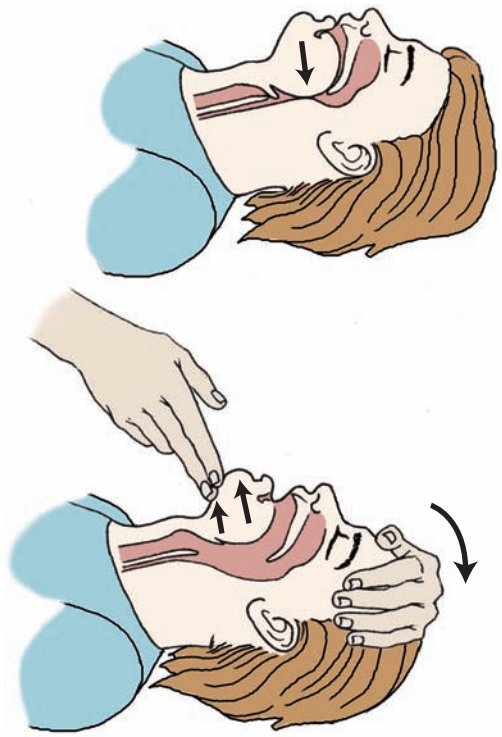


FIGURE 18-46 ▲ Opening the airway with the back head tilt–chin lift maneuver.

are reaching the lungs. Pulse oximetry is used to determine oxygenation.

When one person is performing CPR, two slow breaths are delivered initially. Caution must be used to ensure that the breaths are not going into the patient's stomach. Air in the stomach may lead to vomiting, which in turn could result in aspiration. The ratio of compressions to breaths is 30:2.

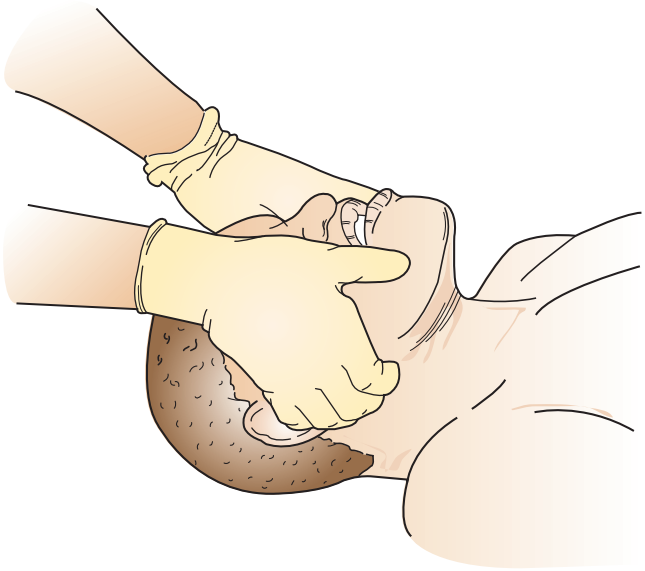


FIGURE 18-47 ▲ The jaw thrust maneuver without head extension is used if cervical spine trauma is suspected.

Once an advanced airway has been placed, and position of the artificial airway confirmed, the compressions and breaths may be delivered asynchronously. Accurate placement of the artificial airway and effectiveness of delivered breaths should be confirmed using an end tidal carbon dioxide monitor (see Chapter 25 for additional information on endotracheal intubation). The breaths are then delivered at a rate of 1 breath every 6 to 8 seconds (8 to 10 breaths/min).

▲ Role of Resuscitation Team Members

When a cardiopulmonary arrest ("code") is called, various members of the emergency response team are notified. Each institution has its own policies regarding who responds. In many teaching hospitals, residents, medical students, and other students may respond. Box 18-32 outlines the roles and responsibilities of the code team members. Some institutions now have Rapid Response Teams that respond in cases of cardiopulmonary arrest (see Chapter 14).

BOX 18-32 Roles and Responsibilities of Code Team Members

Director of the Code (Physician/Nurse Practitioner/ Advanced Cardiac Life Support (ACLS) Qualified Personnel)
- Make diagnosis.
- Direct treatment.

Primary Nurse
- Provide information to code director.
- Contact attending physician.

Second Nurse
- Coordinate use of emergency cart.
- Prepare medications.
- Assemble/pass equipment.
- Defibrillate.

Medication Nurse
- Administer medications.

Charge Nurse
- Coordinate personnel performing CPR.

Nursing Supervisor
- Control crowd.

Anesthesiologist/Nurse Anesthetist
- Intubate patient.
- Manage airway/oxygenation.

Respiratory Therapist
- Assist with manual ventilation.
- Draw arterial blood for blood gas analysis.
- Assist with intubation.
- Set up mechanical ventilator.

Recorder
- Record events and document personnel involved.

▲ Equipment Used in Cardiopulmonary Arrest

The equipment used in resuscitative efforts is kept in a central location in what is commonly referred to as the "crash cart." Most hospitals have rolling carts in easy-to-access locations throughout the hospital. These carts are stocked in a standard way so that all hospital personnel are familiar with the contents and layout of the equipment. The carts must be inventoried daily to ensure their contents are complete and available in the event of a cardiopulmonary arrest. The cart consists of several drawers, a flat top for the storage of larger equipment, an oxygen tank rack, and space for storing a backboard. The drawers and intubation tray are locked with a numbered lock in most cases to ensure that all vital equipment remains in place and undisturbed unless needed for an emergency situation. When any of the locks are broken to access the equipment, resupply of the cart must be performed at the earliest convenience.

The drawers are labeled to assist personnel in locating specific equipment. The intubation tray is separate from the rest of the crash cart because intubation may be the only measure required to treat an individual in respiratory distress, or in whom there is the potential for a compromised airway. In addition to the intubation tray, a cardiac monitor–defibrillator (preferably with transcutaneous pacing capabilities) is on the top of the cart. An oxygen tank, oxygen tubing, and a bag-valve device are found on the outside of the cart for the same reason, as is portable suctioning equipment. Table 18-20 details the contents and the rationale for the equipment and medications found inside the cart.

Table 18-20 Resuscitation Equipment Cart

Equipment	Rationale
Intubation equipment (usually a separate locked container) Straight and curved blades Endotracheal tubes Syringes Oropharyngeal airways Nasopharyngeal airways Suction catheters	• Provides adequate, patent airway, thus ensuring oxygenation of the lungs during resuscitation • Allows for patient to be placed on mechanical ventilation • Reduces chances for gastric distention, aspiration, or vomiting • Permits suctioning • Allows for the administration of oxygen in high concentrations • Provides route for certain medications (NAVEL)*
Oxygen source (separate tank)	• Ensures that oxygen is available if wall oxygen unavailable
Bag-valve device (Ambu bag)	• Provides seal over patient's mouth and nose; reduces risk to rescuer
Suctioning equipment	• Ensures suctioning available if wall suction unavailable
Suctioning source	• Clears oropharyngeal (nasopharyngeal) airway before intubation
Suctioning catheters	
Suction tubing	
IV fluids and tubing	• Improves hypotension
Nitroglycerin tubing	• Prevents precipitation of IV nitroglycerin
Medications (ACLS drugs as a minimum)	• Amiodarone • Lidocaine • Atropine • Epinephrine • Sodium bicarbonate • Calcium chloride • D_{50} • Premixed dopamine infusion
Drip chart (attached to outside of cart)	• Allows for rapid titration of ACLS/Critical Care drugs during and after resuscitation without having to perform complex calculations
Blood tubes	• Allows for the rapid drawing and sending of blood for analysis • Red—chemistries • Blue—coagulation studies • Purple—hematology (complete blood count) • Green—troponin
Arterial blood gas kits	• Allows for rapid drawing and sending of arterial blood gases
Peripheral IV supplies	• Ensures access for fluid and IV drug administration
Prefilled flush syringes (normal saline solution)	• Allows for faster flushing of IV lines
Needles	• Allows for drawing up of medications

(Continued on page 367)

Table 18-20 Resuscitation Equipment Cart *(Continued)*

Equipment	Rationale
Decompression (cardiac) needles	• Used in cardiac tamponade
Clipboard with paper and pen; code sheets	• Used to document the arrest
Pressure bags	• Used for rapid infusion of fluid boluses
Manual blood pressure cuff	• Provides dedicated equipment to monitor resuscitative effectiveness
Gloves (latex, nonlatex, sterile)	• Provides protection for rescuers • Provides sterile gloves for invasive/sterile procedures
Defibrillator/transcutaneous pacemaker	• Used in defibrillation, cardioversion, and temporary transcutaneous pacing

*NAVEL is a mnemonic for drugs that may be administered by endotracheal tube: naloxone, atropine, diazepam, epinephrine, lidocaine.
ACLS, advanced cardiac life support; IV, intravenous.

▲ Medications

Numerous pharmacological interventions are used during and immediately after a cardiopulmonary arrest situation. These drugs should be readily available in the crash cart and include antiarrhythmics, inotropes, vasoconstrictors, and electrolyte replacements. Table 18-21 lists these medications, the indications for their use, and their dosages.

▲ Defibrillation

All patients in an ICU are connected to a cardiac monitor that provides useful information during a cardiopulmonary arrest. Once BLS procedures (ie, CPR) have been initiated, further intervention may be necessary. The nurse assesses the ECG rhythms on the monitor throughout the resuscitation. If the patient is in VF or pulseless VT, preparations for

Table 18-21 Medications Used to Treat a Patient in Cardiopulmonary Arrest

Drug	Class	Uses	Dosages
Adenosine	Antiarrhythmic	SVT, AF	6 mg rapid IV followed by 10 mL NS flush Repeat twice with 12 mg Maximum dose: 30 mg
Amiodarone	Antiarrhythmic	VT, SVT, AF, VF	150–300-mg bolus, 1 mg/min for 6 h, then 0.5 mg/min for 18 h
Atropine	Anticholinergic	Bradycardia, PEA	0.5–1.0 mg IV Maximum dose: 3 mg
Bretylium tosylate	Antiarrhythmic	VT, VF	
Calcium chloride	Electrolyte	Hyperkalemia, hypocalcemia, calcium channel blocker toxicity	Syringe 10 mL of 10% solution (100 mg/mL), 2–4 mg/kg
Dobutamine	Inotrope; β_1 agonist	Decreased cardiac output	5–20 mcg/kg/min
Dopamine	Inotrope; β_1 agonist	Hypotension	5–20 mcg/kg/min
Epinephrine	Catecholamine	VF	Syringe 1:10,000, 1-mg bolus IV Repeat every 3–5 min
Isoproterenol	Catecholamine; β agonist	VT, VF	Drip 0.5–5 mcg/min
Lidocaine	Antiarrhythmic	VT, VF	Bolus 1–1.5 mg/kg Drip 20–50 mcg/kg/min
Magnesium sulfate	Electrolyte	Torsades de pointes	Drip 1–2 g/50 mL NS solution
Nitroglycerin	Coronary vasodilator	MI, angina	5–100 mcg/min
Procainamide	Antiarrhythmic	VT, VF	Bolus 5–10 mg/kg over 8–10 min Drip 20–30 mg/min
Sodium bicarbonate	Alkalinizer	Acidosis	50 mEq syringe Normal dose is 1 mEq/kg
Verapamil	Calcium channel blocker	SVT	2.5–5 mg IV over 2 min Repeat 5–10 mg in 15–30 min

AF, atrial fibrillation; NS, normal saline; PEA, pulseless electrical activity; SVT, supraventricular tachycardia; VF, ventricular fibrillation; VT, ventricular tachycardia.

defibrillation need to be started immediately to avoid death. It is paramount that compressions and ventilations continue during the defibrillation preparation time. The myocardium needs oxygen and electrolytes provided during compressions. The delivery of an electrical impulse by an external defibrillator simultaneously depolarizes most of the ventricular cells during VF and the reentry abnormalities of VT. If the conditions are right, and there has not been too much damage to the heart's intrinsic electrical conduction system, the SA node may resume its function as the pacemaker of the heart.

If indicated, an external countershock should be applied as soon as the instrument is available. The defibrillator paddles (or pads) are positioned so that the heart is in the current pathway. The anterior apex, also known as the anterolateral or sternum–apex position, is used most often. The anterior paddle is placed firmly on the patient's upper right chest below the clavicle and to the right of the sternum. The apex paddle is positioned firmly on the patient's lower left chest in a midaxillary line (Fig. 18-48).

A growing body of evidence indicates that early defibrillation can convert a patient's rhythm from VF more than 90% of the time. In a change to previous defibrillation protocols, defibrillation should be attempted using one shock, with immediate resumption of CPR. If a monophasic defibrillator is used, it should be set at 360 J. If a biphasic device is used, it should be set at either 120 or 200 J; this initial setting is device specific, so familiarity with which defibrillator is on the unit is very important.[1]

Following this first shock, five cycles of CPR should be performed. If the patient remains in a shockable rhythm, then a second shock should be delivered.[1] After each subsequent shock, five more cycles of CPR should be performed before assessing whether the patient has remained in a shockable rhythm. All personnel are advised to avoid touching the patient or bed when the shock is delivered. Immediate resumption of artificial circulation and ventilation (CPR) should occur after each countershock if no pulse returns. Box 18-33 outlines the indications and procedure for defibrillation.

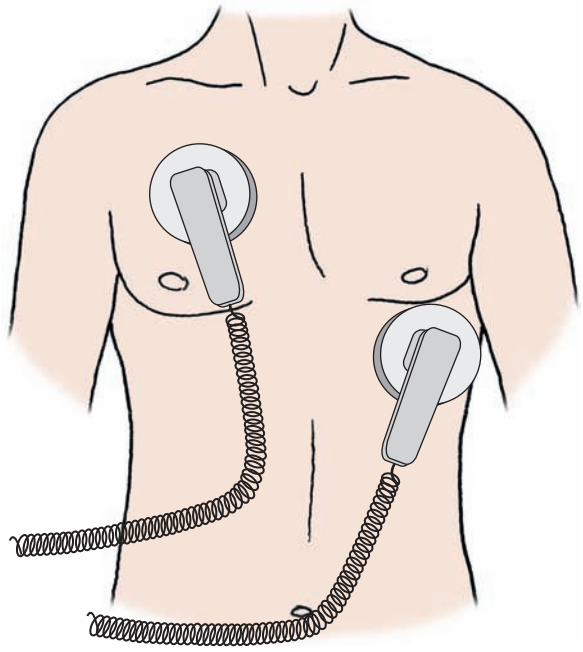


FIGURE 18-48 ▲ Standard positioning of defibrillator paddles.

BOX 18-33 Indications and Procedure for Defibrillation

Indications
- Pulseless VT
- Ventricular fibrillation

Procedure
1. Apply defibrillator pads to patient.
2. Turn on defibrillator.
3. Charge defibrillator to 200 J for *monophasic* device; for *biphasic* defibrillation, change to 120 or 200 J, depending on device.
4. Ensure all personnel are not touching patient or bed.
5. Deliver shock.
6. Determine effectiveness of treatment. Check pulse and observe patient's rhythm.
7. Continue CPR.
8. Be prepared to deliver subsequent shocks per ACLS protocol.

Defibrillators are classified based on the type of waveform used by the defibrillator. Since the early 1970s, monophasic defibrillators have been used. This type of defibrillator provides a shock that flows in one direction from one paddle or electrode pad to the other. In more recent years, newer technology has been developed that changes the way the electrical current flows during defibrillation. Known as a biphasic defibrillator, this newer type delivers the current in two phases. The current initially flows in one direction, then flows in the opposite direction. The biphasic wave uses less peak current, so there is less damage to the heart during defibrillation.[3] ICDs are devices that are designed to shock patients out of potentially fatal dysrhythmias. ICDs have used biphasic technology for more than a decade. Currently, transthoracic biphasic defibrillation is also possible.

▲ Automatic External Defibrillator

Studies show that the sooner a patient in VF is defibrillated, the greater the chance for survival.[5] The development of the AED has improved the survivability of individuals suffering from potentially life-threatening dysrhythmias. The AED allows for defibrillation to be performed in a variety of settings by personnel trained in the use of the AED but not BLS or ACLS.

The AED consists of a computerized detection system that recognizes the patient's inherent heart rhythm and delivers a defibrillatory countershock when necessary. This cycle of rhythm analysis followed by countershock lasts approximately 30 seconds. AEDs are now found in airports, train stations, sports stadiums, office buildings, and shopping malls. AEDs are also available in most hospitals in common areas, general floors, and laboratories. The widespread availability of these devices allows for a much quicker response time when a person experiences cardiac arrest.[4]

▲ Transcutaneous Pacing

A combination defibrillator–transcutaneous pacemaker is usually found on top of the crash cart. The large pacing electrodes ("combination pads") used in defibrillation can also be used to pace a patient transcutaneously. Transcutaneous pacing may be used as a "bridge" (temporary measure) until either a transvenous or permanent pacemaker can be placed.

A nurse, nurse practitioner, or physician quickly and easily initiates transcutaneous pacing. This procedure is noninvasive and therefore is low risk and saves time during an arrest situation. Indications for transcutaneous pacing include new complete (third-degree) heart block or symptomatic bradycardia (unresponsive to drug therapy). The pacing electrodes are also placed when the patient's rhythm changes to a new second-degree, Mobitz II heart block. Transcutaneous pacing also is used when invasive (transvenous) pacing is unsuccessful or contraindicated, such as after the use of thrombolytics and for the patient with sepsis.

The transcutaneous pacemaker is used in a "demand mode" for bradycardia and asystole; it paces the heart only when needed. This mode is safer because the chance of firing on the T wave ("R-on-T phenomenon") is greatly reduced. When the pacemaker is used in an asynchronous mode, the heart is paced at a fixed rate, regardless of the heart's intrinsic rate or rhythm. The pacemaker may fire on the T wave, producing either AF or VT. Box 18-34 outlines the indications and procedure for transcutaneous pacing.

The nurse must ensure that a conscious patient understands what is happening. Much technical jargon and various personnel are involved when a patient is having noninvasive temporary pacing. The nurse is responsible for placing the pacing pads in an anteroposterior configuration, which allows for more effective pacing. Figure 18-49 shows the proper placement of the electrodes. The skin is not shaved, which means that there is no skin irritation. No alcohol or adhesive should be used so that the electrical current is not compromised. The nurse sets the pacing rate and stimulation threshold. Blood pressure should be taken using the right arm to avoid interference from the pacemaker.

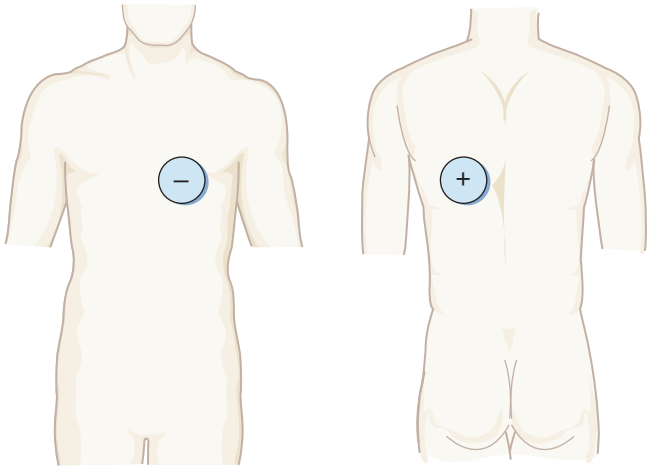


FIGURE 18-49 ▲ Standard position of transcutaneous electrodes.

Transcutaneous pacing requires diligent monitoring by the nurse. A loss of capture can occur if the electrodes fail to keep good contact with the skin. Inappropriate pacing may result if the pacemaker cannot detect the heart's intrinsic rhythm. In either case, the nurse must recognize the problem and reposition the patient or the electrodes to ensure efficacious transcutaneous pacing.

BOX 18-34 Indications and Procedure for Transcutaneous Pacing

Indication

- Complete (third-degree) heart block

Procedure

1. Explain procedure to patient.
2. Clip excess hair from chest. Ensure skin is dry.
3. Apply anterior electrode to chest at the fourth intercostal space to the left of the sternum.
4. Apply posterior electrode to patient's back in the area of the left scapula.
5. Connect pacing electrodes to transcutaneous pacemaker.
6. Set pacemaker mode, heart rate, and output.
7. Turn unit on.
8. Assess for effectiveness of pacing:
   - Observe for pacemaker spike with subsequent capture.
   - Assess heart rate and rhythm.
   - Assess blood pressure.
   - Check level of consciousness.
   - Observe for patient anxiety and/or pain and treat accordingly.

▲ Therapeutic Hypothermia

Unconscious adult patients who suffer out-of-hospital cardiac arrest caused by VF and are resuscitated in the field may benefit from induced mild hypothermia. During cardiac arrest, blood flow to the brain is compromised, and even prompt interventions may not counteract the deleterious effects of this hemodynamic compromise. The cerebral metabolic rate for oxygen, or $CMRO_2$, is reduced when the body temperature is reduced. Apoptosis (programmed cell death) and the production of free radicals are reduced in a hypothermic state. Studies have shown that the cooling of the patient after cardiac arrest may preserve neurological function. This therapy is currently contraindicated in children of any age, although research continues. There is evidence that supports hypothermic therapy for cardiac arrest caused by dysrhythmias.[5]

Many institutions are now implementing therapeutic hypothermia protocols. Common among these protocols is the systematic lowering of a patient's temperature to 32°C to 34°C. This can be accomplished by various methods, including ice packs, cooling blankets, and endovascular cooling devices. The patient remains in this hypothermic state between 8 and 12 hours. Rewarming is then allowed to occur over the next 8 to 12 hours.[5–7]

Nursing care during therapeutic is intense. In addition to the monitoring of cardiac rhythm and MAP, potassium levels and blood glucose must also be tracked. In most cases,

the patient is intubated and on mechanical ventilation, so arterial blood gases and ventilator settings must be evaluated periodically. The nurse must ensure that medications, such as sedatives, neuromuscular blockade medications, and analgesics, are administered in accordance with the established protocols. Care must include skin assessment to check for possible damage caused by the cooling devices, as cooling injuries are a realistic complication that is not often seen in an ICU. The patient's core body temperature is monitored, usually using a bladder catheter.

Research continues as to the optimal temperature, means of cooling, and method of rewarming. Investigative studies are now being conducted as to the efficacy of therapeutic hypothermia in cases of cerebral vascular accidents. One of the major controversies surrounding therapeutic hypothermia is the patient's quality of life following rewarming, as well as the long-term costs if the patient recovers, but with resulting neurological deficits.[8]

▲ Family Presence in Cardiac Arrest Situations

One aspect of the treatment of cardiopulmonary arrest that has gained attention in recent years is the issue of family presence during a code. Nurses have voiced strong opinions for both sides of the issue. Health care institutions have become more flexible, and they are more accommodating of families and visitors. Some emergency departments and ICUs have protocols in place regarding having loved ones at the bedside while resuscitation efforts are taking place. Every effort must be made to have a knowledgeable person explain to the family what measures are being implemented and the rationale. By involving the family in this manner, they can make more informed decisions about the continuation of resuscitation.[9]

Many family members express a desire to be with the patient during CPR for various reasons. Some want to be reassured that all resuscitative efforts were attempted. There are those who want to have a chance to say goodbye at the moment of death, rather than in the hours or days after death. One of the most often cited reasons for wanting to be with the patient is to make sure that the death is painless.

When discussing advanced directives with patients and their families, the techniques of resuscitation are often described. In the event of a cardiac arrest situation, some family members have seen these measures taking place and make the determination to terminate resuscitation.

The recognition of families after a successful resuscitation must be experienced to appreciate it. When the family sees a team of health care professionals working against time to save a patient, they express the realization that nurses, physicians, respiratory therapists, and others combine to provide excellent and compassionate care.

Many nurses and physicians believe that family presence during CPR detracts from their performance and that individuals who have an emotional attachment to the patient hinder their efforts. Rules must be in place to escort family members from the room if the rescuers cannot perform resuscitative measures effectively.

▲ Clinical Applicability Challenges

CASE STUDY

P.M., a 77-year-old female recovering from sudden cardiac arrest, myocardial infarction, and status post placement of an implantable cardioverter–defibrillator, is a "full code." She has visitors at this time, her daughter and son-in-law. The nurse is outside at the nurse's station, documenting morning assessments. The sound of a cardiac monitor alarm begins as the nurse hears P.M.'s daughter yell: "Someone come quick! There's something wrong with my mother!" The nurse enters the room and notes that the monitor is showing ventricular fibrillation.

1. What are the immediate actions the nurse must perform?
2. When more health care team members arrive, what are the various roles to be performed and actions to be taken?
3. What should be done with the family members in the room?

References

Pharmacological Therapy

1. Kushner FG, Hand M, Smith S, et al: 2009 Focused Updates: ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction: (Updating the 2004 guideline and 2007 Focused Update) and ACC/AHA/SCAI Guidelines on Percutaneous Coronary Intervention (Updating the 2005 Guideline and 2007 Focused Update): A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation 120:2271–2306, 2009
2. Armstrong PW, Collen D, Antman E: Fibrinolysis for acute myocardial infarction: The future is here and now. Circulation 107:2533–2537, 2003
3. Fox KA, Opie JJS, White HD, et al: Antithrombotic agents: Platelet inhibitors, anticoagulants, and fibrinolytics. In Opie LH, Gersh BJ (eds): Drugs for the Heart. Philadelphia, PA: Elsevier Saunders, 2009, pp 293–340
4. Fishman WH, Cheng-lai A, Nowarkas J: Current Cardiovascular Drugs, 4th ed. Philadelphia, PA: Current Medicine LLC, 2005, pp 98–135
5. Menon V, Berkowitz SD, Antman EM, et al: New heparin dosing recommendations for patients with acute coronary syndromes. Am J Med 110:641–650, 2001
6. Antman EM, Morrow DA, McCabe CH, et al: Enoxaparin versus unfractionated heparin with fibrinolysis for ST-elevation myocardial infarction. N Engl J Med 354:1477–1488, 2006
7. Antman EM, Cohen M, McCabe C, et al: Enoxaparin is superior to unfractionated heparin for preventing clinical events at 1-year follow-up of TIMI 11B and ESSENCE. Eur Heart J 23:308–314, 2002
8. Fox KA, Antman EM, Cohen M, et al: Comparison of enoxaparin versus unfractionated heparin in patients with unstable angina pectoris/non-ST-segment elevation acute myocardial infarction having subsequent percutaneous coronary intervention. Am J Cardiol 90:477–482, 2002
9. Goodman SG, Cohen M, Bigonzi F, et al: Randomized trial of low molecular weight heparin (enoxaparin) versus unfractionated heparin for unstable coronary artery disease: One-year results of the ESSENCE Study. Efficacy and Safety of Subcutaneous Enoxaparin in Non-Q Wave Coronary Events. J Am Coll Cardiol 36:693–698, 2000
10. Smith SC Jr, Feldman TE, Hirshfeld JW Jr, et al: ACC/AHA/SCAI 2005 Guideline Update for Percutaneous Coronary Intervention. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation 117:261–295, 2008
11. Rothberg MB, Celestin C, Fiore LD, et al: Warfarin plus aspirin after myocardial infarction or the acute coronary syndrome: Meta-analysis with estimates of risk and benefit. Ann Intern Med 143:241–250, 2005
12. Hirsh J, Fuster V, Ansell J, et al: American Heart Association/American College of Cardiology Foundation guide to warfarin therapy. J Am Coll Cardiol 41:1633–1652, 2003
13. Holmes DR, Dehmer GJ, Kaul S, et al: ACCF/AHA Clopidogrel alert: Approaches to the FDA "Boxed Warning": A report of the American College of Cardiology Foundation Task Force Expert Consensus Documents and the American Heart Association. Circulation 122:537–557, 2010
14. Smith SC Jr, Allen J, Blair SN, et al: AHA/ACC guidelines for secondary prevention for patients with coronary and other atherosclerotic vascular disease: 2006 Update. Circulation 113:2363–2372, 2006
15. Kowey PR, Yan G, Crojins H: Antiarrhythmic drugs. In Fuster V, Alexander RW, O'Rourke RA (eds): Hurst's The Heart, 12th ed. New York, NY: McGraw-Hill, 2008, pp 1077–1130
16. Fox KA, White HD, Opie JJS, et al: Antiarrhythmic drugs and strategies. In Opie LH, Gersh BJ (eds): Drugs for the Heart. Philadelphia, PA: Elsevier Saunders, 2009, pp 218–274
17. 2005 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care: Part 7.4—monitoring and medications. Circulation 112:IV-78–IV-83, 2005
18. Anderson JL, Adams CD, Antman EM, et al: ACC/AHA 2007 guidelines for the management of patients with unstable angina/non-ST-elevation myocardial infarction. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines for the Management of Patients with Unstable Angina/non ST-elevation Myocardial Infarction). Circulation 116:e148–e304, 2007
19. Hazinski MF, Samson R, Schexnayder S (eds): Handbook of Emergency Cardiovascular Care for Healthcare Providers. Dallas, TX: American Heart Association, 2010
20. 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care: Part 10—Acute coronary syndromes: 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 122(suppl 3):S787–S817, 2010
21. Poole-Wilson PA, Opie LH: Acute and chronic heart failure: Positive inotropes, vasodilators and digoxin. In Opie LH, Gersh BJ (eds): Drugs for the Heart. Philadelphia, PA: Elsevier Saunders, 2009, pp 160–197
22. Opie LH, Pfeffer MA: Inhibitors of angiotensin-converting enzyme (ACE), angiotensin-II receptors (ARBs), aldosterone and renin. In Opie LH, Gersh BJ (eds): Drugs for the Heart. Philadelphia, PA: Elsevier Saunders, 2009, pp 112–159
23. Jong P, Yusuf S, Rousseau MF, et al: Effect of enalapril on 12-year survival and life expectancy in patients with left ventricular systolic dysfunction: A follow-up study. Lancet 361:1843–1848, 2003
24. The CONSENSUS Trial Study Group: Effects of enalapril on mortality in severe congestive heart failure. Results of the Cooperative North Scandinavian Enalapril Survival Study (CONSENSUS). N Engl J Med 316:1429–1435, 1987
25. The SOLVD Investigators: Effect of enalapril on survival in patients with reduced left ventricular ejection fractions and congestive heart failure. N Engl J Med 325:293–302, 1991
26. The SOLVD Investigators: Effect of enalapril on mortality and the development of heart failure in asymptomatic patients with reduced left ventricular ejection fractions. N Engl J Med 327:685–691, 1992
27. Pfeffer MA, Braunwald E, Moye LA, et al: Effect of captopril on mortality and morbidity in patients with left ventricular dysfunction after myocardial infarction: Results of the survival and ventricular enlargement trial. The SAVE Investigators. N Engl J Med 327:669–677, 1992
28. Cleland JG, Erhardt L, Murray G, et al: Effect of ramipril on morbidity and mode of death among survivors of acute myocardial infarction with clinical evidence of heart failure: A report from the AIRE Study Investigators. Eur Heart J 18:41–51, 1997
29. Cohn JN, Johnson G, Ziesche S, et al: A comparison of enalapril with hydralazine-isosorbide dinitrate in the treatment of chronic congestive heart failure. N Engl J Med 325:303–310, 1991
30. Grundy SM, Cleeman JI, Merz CN, et al; for the Coordinating Committee of the National Cholesterol Education Program: Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III guidelines. Circulation 110: 227–239, 2004
31. Nissen SE, Nicholls SJ, Sipahi I, et al; for the ASTEROID Investigators: Effect of very high-intensity statin therapy on regression of coronary atherosclerosis: The ASTEROID trial. JAMA 295:1556–1565, 2006
32. Grundy SM, Cleeman JI, Bairey CN, et al: Implications of recent clinical trials for the National Cholesterol Education program Adult Treatment Panel III Guidelines. Circulation 110:227–239, 2004

Percutaneous Coronary Interventions and Percutaneous Balloon Valvuloplasty

1. American Heart Association: Heart Disease and Stroke Statistics—2010 Update. Dallas, TX: American Heart Association, 2010
2. Antoniucci D (ed): Primary Angioplasty. Rome, Italy: Taylor & Francis Publishing, 2004
3. Serruys PW, Unger F, Sousa JE, et al; for the Arterial Revascularization Therapies Study Group: Comparison of coronary artery bypass grafting and stenting for the treatment of multivessel disease. N Engl J Med 344:1117–1124, 2001
4. Society of Thoracic Surgeons: National Adult Cardiac Surgical Database Report 2000–2001. Chicago, IL: Author, 2004
5. Detre K, et al: New approaches to coronary interventions. J Am Coll Cardiol 35:1122–1129, 2000
6. Legrand VM, Serruys PW, Unger F, et al; for the Arterial Revascularization Therapy Study (ARTS) Investigators: Three-year outcome after coronary stenting versus bypass surgery for the treatment of multivessel disease. Circulation 109:1114–1120, 2004

7. Serruys PW, Unger F, Sousa JE, et al: Five-year outcomes after coronary stenting versus bypass surgery for the treatment of multivessel disease: The final analysis of the Arterial Revascularization Therapies Study (ARTS) randomized trial. J Am Coll Cardiol 46(4):575–581, 2005
8. Bentivoglio LG, Holubkov R, Kelsey SF, et al: Short and long term outcome of percutaneous transluminal coronary angioplasty in unstable versus stable angina pectoris: A report of the 1985/1986 NHLBI PTCA registry. Catheter Cardiovasc Diagn 23:227–238, 1991
9. O'Keefe JH Jr, Rutherford BD, McConahay DR, et al: Multi-vessel coronary angioplasty from 1980 to 1989: Procedural results and long-term outcome. J Am Coll Cardiol 16:1097–1102, 1990
10. Dorros G, Iyer SS, Hall P, et al: Percutaneous coronary angioplasty in 1001 multi-vessel coronary disease patients: An analysis of different patient subsets. J Interv Cardiol 4:71–80, 1991
11. Hannan EL, Racz MJ, Walford G, et al: Long-term outcomes of coronary artery bypass grafting versus stent implantation. N Engl J Med 352(21):2174–2183, 2005
12. Alderman EL, Bourassa MG, Cohen LS, et al; for the CASS Investigators: Ten-year follow-up of survival and myocardial infarction in the randomized coronary artery surgery study. Circulation 82:1629–1646, 1990
13. U.S. Food and Drug Administration and Center for Devices and Radiological Health: Cypher sirolimus-eluting coronary stent on RAPTOR over-the-wire delivery system or RAPTORRAIL rapid exchange delivery system. Rockville, MD: Author, 2003
14. Ellis S, Stone GW, Popma JJ, et al: Relationship between angiographic late loss and target lesion revascularization after coronary stent implantation: Analysis from the TAXUS IV Study. J Am Coll Cardiol 45(8):1206–1200, 2005
15. Colombo A, Drzewiecki J, Banning A, et al: Randomized study to assess the effectiveness of slow-and moderate-release polymer-based paclitaxel-eluting stents for coronary artery lesions. Circulation 108:788–794, 2003
16. Mallik S, Krumholz HM, Lin ZQ, et al: Patients with depressive symptoms have lower health status benefits after coronary artery bypass surgery. Circulation 111(3):250–253, 2005
17. Meyer J, Merx W, Schmitz H, et al: Percutaneous transluminal coronary angioplasty immediately after intracoronary streptolysis of transmural myocardial infarction. Circulation 66:905–913, 1982
18. Wiviott SD, Antman EM, Gibson CM, et al: TRITON-TIMI 38 Investigators (2006). Evaluation of prasugrel compared with clopidogrel in patients with acute coronary syndromes: Design and rational for the Trial to assess Improvement in Therapeutic Outcomes by optimizing platelet Inhibition with prasugrel. Thrombolysis In Myocardial Infarction 38 (TRITON-TIMI 38). Am Heart J 152(4):627–635, 2006
19. ACC/AHA (2009): 2009 Focused Updates: ACC/AHA guidelines for the Management of Patients With ST Elevation Myocardial Infarction (Updating the 2004 Guideline and 2007 Focused Update) and ACC/AHA/SCAI Guidelines on Percutaneous Coronary Intervention (Updating the 2005 Guideline and 2007 Focused Update): A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation 120: 2271–2306, 2009
20. Lehman R, Spyridopoulos I, Kremer J, et al: Favorable long-term survival in patients undergoing stent PCI of unprotected left main coronary artery compared to predicted short-term prognosis of CABG estimated by EuroSCORE: clinical determinants of long-term outcome. J Interv Cardiol 22(4):311–319, 2009
21. Gruberg L, Mintz GS, Mehran R, et al: The prognostic implications of further renal function deterioration within 48 hours of interventional coronary procedures in patients with pre-existent chronic renal insufficiency. J Am Coll Cardiol 36(5):1542–1548, 2000
22. Rihal CS, Textor SC, Grill DE, et al: Incidence and prognostic importance of acute renal failure after percutaneous coronary intervention. Circulation 105:2259, 2002
23. Grimes CL, Bonow RO, Casey DE, et al: Prevention of premature discontinuation of dual antiplatelet therapy in patients with coronary artery stents: A science advisory from the American Heart Association, American College of Cardiology, Society for Cardiovascular Angiography and Interventions, American College of Surgeons, and American Dental Association, with representation from the American College of Physicians. Circulation 115:813–818, 2007. doi: 10.1161/CIRCULATIONAHA.106.180944
24. Steinhubl SR, Berger PB, Mann JT; for the Clopidogrel for Reduction of Events During Observation (CREDO) Investigators: Early and sustained dual oral antiplatelet therapy following percutaneous coronary intervention: A randomized controlled trial. JAMA 288:2411–2420, 2002
25. Aversano T, Aversano LT, Passamani E, et al: Thrombolytic therapy versus primary percutaneous coronary intervention for myocardial infarction in patients presenting to hospitals without on-site cardiac surgery: A randomized controlled trial. JAMA 287:1943–1951, 2002
26. Braunwald E, Antman EM, Beasley JW, et al: ACC/AHA 2002 guideline update for the management of patients with unstable angina and non-ST segment elevation myocardial infarction: Summary article. Circulation 106:1893–1900, 2002
27. Goodkind J, Coombs VJ, Golobic RA: Excimer laser angioplasty. Heart Lung 22:26–35, 1993
28. Antoniucci D, Valenti R, Migliorini A, et al: Comparison of rheolytic thrombectomy before direct infarct artery stenting versus direct stenting alone in patients undergoing percutaneous coronary intervention for acute myocardial infarction. Am J Cardiol 93:1033–1035, 2004
29. Ho PC, Leung CY: Rheolytic thrombectomy with distal filter embolic protection as adjunctive therapies to high-risk saphenous vein graft intervention. Catheter Cardiovasc Interv 61:202–205, 2004
30. Waksman R, Robinson KA, Crocker IR, et al: Intracoronary radiation before stent implantation inhibits neointima formation in stented porcine coronary arteries. Circulation 92:1383–1386, 1995
31. American Diabetes Association: Consensus Statement. Peripheral arterial disease in people with diabetes. Diabetes Care 26:3333–3341, 2003

Intra-Aortic Balloon Pump Counterpulsation and Mechanical Circulatory Support

1. Antman EM, Smith SC, Alpert JS, et al: ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction—executive summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to revise the 1999 guidelines for the management of patients with acute myocardial infarction). Circulation 110:588–636, 2004
2. Sjauw KD, Engstrom AE, Vis MM, et al: A systematic review and meta-analysis of intra-aortic balloon pump therapy in ST-elevation myocardial infarction: Should we change the guidelines? Eur Heart J 30(4): 459–468, 2009
3. Parissis H, Leotsinidis M, Akbar MT, et al: The need for intra aortic balloon pump support following open heart surgery: Risk analysis and outcome. J Cardiothorac Surg 5:20, 2010
4. Onorati F, Santarpino G, Tangredi G, et al: Intra-aortic balloon pump induced pulsatile perfusion reduces endothelial activation and inflammatory response following cardiopulmonary bypass. Eur J Cardiothorac Surg 35(6):1012–1019, discussion 1019, 2009
5. Onorati F, Santarpino G, Rubino AS, et al: Body perfusion during adult cardiopulmonary bypass is improved by pulsatile flow with intra-aortic balloon pump. Int J Artif Organs 32(1):50–61, 2009
6. Schreuder JJ, Castiglioni A, Donelli A, et al: Automatic intraaortic balloon pump timing using an intrabeat dicrotic notch prediction algorithm. Ann Thorac Surg 79(3):1017–1022, discussion 1022, 2005
7. Stahl MA, Richards NM: Update on ventricular assist device technology. AACN Adv Crit Care 20(1):26–34, quiz 35–26, 2009
8. Andrew Rosenberg RT: Perioperative management for patients receiving ventricular assist devices and mechanical circulatory support: A systems-oriented approach. Contemp Crit Care 7(12):1–12, 2010
9. Delgado DH, Rao V, Ross HJ, et al: Mechanical circulatory assistance: State of art. Circulation 106(16):2046–2050, 2002
10. Amir O, Bracey AW, Smart FW, et al: A successful anticoagulation protocol for the first HeartMate II implantation in the United States. Tex Heart Inst J 32(3):399–401, 2005
11. Matthews JC, Koelling TM, Pagani FD, et al: The right ventricular failure risk score a pre-operative tool for assessing the risk of right ventricular failure in left ventricular assist device candidates. J Am Coll Cardiol 51(22):2163–2172, 2008

12. Potapov EV, Stepanenko A, Dandel M, et al: Tricuspid incompetence and geometry of the right ventricle as predictors of right ventricular function after implantation of a left ventricular assist device. J Heart Lung Transplant 27(12):1275–1281, 2008
13. Oz MC, Rose EA, Slater J, et al: Malignant ventricular rhythms are well tolerated in patients receiving long-term ventricular assist devices. J Am Coll Cardiol 24:1688–1691, 1994

Management of Dysrhythmias

1. Zipes DP, Camm AJ, Smith SC, et al: ACC/AHA/ESC 2006 Guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. J Am Coll Cardiol 48(5):e247–e346, 2006
2. 2005 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Part 5: Electrical therapies automated external defibrillators, defibrillation, cardioversion, and pacing. Circulation 112:IV-35–IV-46, 2005
3. Fuster V, Ryden LE, Cannom D, et al: ACC/AHA/ESC 2006 Guidelines for the management of patients with atrial fibrillation: Executive summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Revise the 2001 Guidelines for the Management of Patients with Atrial Fibrillation): J Am Coll Cardiol 48(4):854–906, 2006
4. Haines DE: Biophysics of radiofrequency lesion formation. In Zipes DP (ed): Cardiac Electrophysiology: From Cell to Bedside, 4th ed. New York, NY: WB Saunders, 2006:1018–1027
5. Scheinman M, Calkins H, Gillette P, et al; for the North American Society of Pacing and Electrophysiology: NASPE policy statement on catheter ablation: Personnel, policy, and therapeutic recommendations. Pacing Clin Electrophysiol 26(3):789–799, 2003
6. Calkins H, Brugada J, Packer DL, et al: HRS/EHRA/ECAS expert consensus statement on catheter and surgical ablation of atrial fibrillation: Recommendations for personnel, policy, procedures and follow-up. Heart Rhythm 4(6):816–861, 2007
7. Sawhney NS, Anousheh R, Chen WC, et al: Diagnosis and management of typical atrial flutter. Cardiol Clin 27(1):55–67, viii, 2009
8. Saxon LA, DiMarco T, Prystowsky EN, et al: Expert consensus statement: Resynchronization therapy for heart failure (2005). Available at: http://www.hrsonline.org/Policy/ClinicalGuidelines
9. Epstein AE, DiMarco JP, Ellenbogen KA, et al: ACC/AHA/HRS 2008 guidelines for device-based therapy of cardiac rhythm abnormalities: Executive summary. A report of the American College of Cardiology/American Heart Association Task Force on practice guidelines (ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices). J Am Coll Cardiol, 51:1–62, 2008, doi:10.1016/j.jacc.2008.02.032 (Published online 15 May 2008)
10. Bernstein AD, Daubert JC, Fletcher RD, et al: The revised NASPE/BPEG generic code for antibradycardia, adaptiverate, and multisite pacing. North American Society of Pacing and Electrophysiology/British Pacing and Electrophysiology Group. Pacing Clin Electrophysiol 25(2):260–264, 2002. Available at: http://www.hrsonline.org
11. Hauser RG, Hayes DL, Kallinen LM, et al: Clinical experience with pacemaker pulse generators and transvenous leads: An 8-year prospective multicenter study. Heart Rhythm ;4(2):154–160, 2007
12. Biffi M, Moschini C, Bertini M, et al: Phrenic stimulation: A challenge for cardiac resynchronization therapy. Circ Arrhythm Electrophysiol 2(4):402–410, 2009
13. Sudden Cardiac Arrest Key facts. Available at: http://www.heartrhythmfoundation.org/facts/scd.asp
14. Bernstein AD, Camm AJ, Fisher JD, et al: North American Society of Pacing and Electrophysiology policy statement. The NASPE/BPEG defibrillator code. Pacing Clin Electrophysiol 16(9):1776–1780, 1993

Cardiopulmonary Resuscitation

1. American Heart Association: 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 122:S676–S684, 2010
2. SOS-KANTO Study Group: Cardiopulmonary resuscitation by bystanders with chest compression only (SOS-KANTO): An observational study. Lancet 369(9565):920–926, 2007
3. Mair M: Monophasic and biphasic defibrillators: The evolving technology of cardiac defibrillation. Am J Nurs 103(8):58–60, 2003
4. Powers C, Martin K: When seconds count, use an AED. Am J Nurs 102(Suppl):8–10, 2002
5. Nolan JP, Morley PT, Vander Hoek TL, et al: Therapeutic hypothermia after cardiac arrest: An advisory statement by the Advanced Life Support Task Force of the International Liaison Committee on Resuscitation. Circulation 108:118–121, 2003
6. The Hypothermia after Cardiac Arrest Study Group: Mild therapeutic hypothermia to improve neurologic outcome after cardiac arrest. N Engl J Med 346(8):549–556, 2002
7. Bernard SA, Gray TW, Buist MD, et al: Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. N Engl J Med 346(8):557–563, 2002
8. Merchant RM, Becker LB, Abella BS, et al: Cost-effectiveness of therapeutic hypothermia after cardiac arrest. Circ: Cardiovasc Qual Outcomes 2:421–428, 2009
9. Tucker T: Family presence during resuscitation. Crit Care Nurs Clin North Am 14:177–185, 2002

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

19 Common Cardiovascular Disorders

Clifford C. Pyne and Sue Apple

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Differentiate between pericarditic and ischemic chest pain.
2. Explain the long-term effects of endocarditis on the heart valves.
3. Discuss key differences in the clinical management of dilated and hypertrophic cardiomyopathy.
4. Describe key differences in clinical presentation between arterial and venous peripheral vascular disease.
5. Compare and contrast the clinical findings of chronic aortic aneurysm with those of acute aortic dissection.
6. Construct a plan of care for the patient in hypertensive crisis covering the first hour of treatment.

The first intensive and coronary care units were developed in the mid-1960s to treat patients with acute myocardial infarction (MI). Since those early days, critical care nurses have expanded their knowledge base to include care of patients with a wide spectrum of cardiovascular diseases. In addition to acute MI, these disorders include inflammation and infections of the heart muscle, pericardium, and valves; dilation and/or thickening of the ventricular walls; and diseases involving the aorta and peripheral vascular system. This chapter reviews several common cardiovascular disorders, including pericarditis, myocarditis, endocarditis, cardiomyopathies, peripheral vascular disease, aortic diseases, and hypertensive crisis.

▲ Infection and Inflammation of the Heart

Infectious and inflammatory diseases of the heart have multiple etiologies, making diagnosis and treatment a clinical challenge. Patients may present with acute pain mimicking MI, or may seek medical attention because of fatigue and vague flu-like symptoms that fail to resolve over a period of weeks. Because of the permanent damage these diseases can cause to structures of the heart, patients often face serious long-term cardiac disability.

Pericarditis

The pericardium surrounds the external surface of the heart and the roots of the great vessels. It is composed of two layers: an outer tough fibrous pericardium and an inner serous layer.[1,2] The serous pericardium has two layers: the parietal and the visceral. The parietal layer lines the internal surface of the fibrous membrane. The parietal pericardium extends to the great vessels, where it then folds over on itself to form the inner visceral layer, also known as the epicardium (Fig. 19-1). From 10 to 50 mL of clear serous fluid lies between these layers and acts as a lubricant. The pericardium helps restrain the heart and isolate it from infections in the surrounding structures.[3,4]

Pericarditis is inflammation of the pericardium. Acute pericarditis is pericarditis that lasts no longer than 1 or 2 weeks.[4,5] Inflammation often involves the adjoining diaphragm. Pericarditis can be a primary disease or occur secondarily as the result of some other disorder, such as acute MI or renal failure.[5,6] The etiology of pericarditis varies. However, in almost 90% of patients diagnosed with acute pericarditis, the exact cause is unknown (idiopathic).[4,5] Causes of pericarditis are listed in Box 19-1. Dressler's syndrome refers to the development of pericarditis, malaise, fever, and elevated white blood cell count appearing weeks to months after a MI. This syndrome is believed to be the result of an autoimmune reaction that occurs after the infarction.[3] Infectious pericarditis remains a problem in the immunocompromised patient.[7]

Repeated episodes of pericarditis can lead to the formation of adhesions between the layers of the pericardium or between the pericardium and adjacent structures, resulting in constrictive pericarditis.[8] In constrictive pericarditis, the primary problem is failure of the heart to fill during diastole because of its inability to expand. Unless the diseased pericardium is removed surgically, diastolic filling continues to be impaired, eventually leading to a decrease in cardiac output and systemic signs of heart failure. Even with successful surgical removal of the diseased pericardium, the long-term survival rate is poor.[4,8]

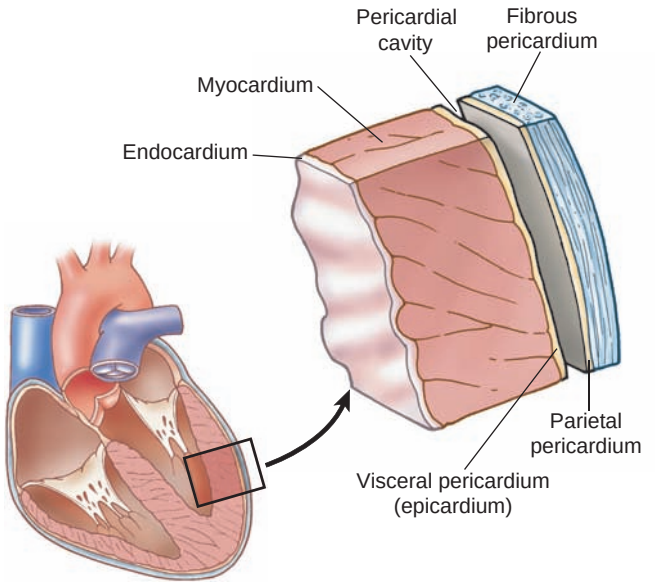


FIGURE 19-1 ▲ Layers of the heart, showing the visceral pericardium, pericardial cavity, parietal pericardium, fibrous pericardium, myocardium, and endocardium. (From Porth CM: Pathophysiology, Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 459.)

BOX 19-1 Causes of Pericarditis

- Idiopathic (usually presumed to be viral)
- Infectious
- Bacterial
- Tuberculosis
- Autoimmune or inflammatory
- Systemic lupus erythematosus
- Drugs
- Vaccinations
- Neoplasms
- Radiation therapy
- Following device implantation, such as an implantable defibrillator
- Acute myocardial infarction
- Trauma to the chest wall or myocardium, including cardiopulmonary surgery
- Chronic renal failure requiring dialysis

Assessment

Important clues to the diagnosis of pericarditis can be obtained from the history and physical examination. The primary symptom in acute pericarditis is chest pain.[4,6] The pain tends to be pleuritic in nature and classically is made worse by breathing deeply or lying supine. Because of pain from breathing, patients frequently complain of dyspnea. Relief is often obtained by sitting up, leaning forward, and taking shallow breaths. The chest pain of pericarditis may be difficult to distinguish from ischemic chest pain.[5] Differential vdiagnoses of chest pain are summarized in Table 19-1. One clue in the differentiation is that ischemic chest pain is not relieved by a change in the patient's position.

There may also be general symptoms of an infection, such as a low-grade fever, tachycardia, or malaise. The presence of a pericardial friction rub confirms the diagnosis; however, absence of a rub does not rule out pericarditis. The classic friction rub produces a rasping or scraping, high-pitched sound that varies with the cardiac cycle. The rub may wax and wane and may even transiently disappear during the course of the illness. It is best heard with the diaphragm of the stethoscope placed over the lower to middle left sternal edge.[3,6]

There are no specific guidelines for evaluating or managing acute pericarditis. The electrocardiogram (ECG) is the most important test in establishing the diagnosis.[4] It classically shows diffuse ST-segment elevation with an upward concavity and PR-segment depression (Fig. 19-2). This contrasts with the ECG seen in acute myocardial injury, which typically shows upward convexity in leads facing the infarction zone (Fig. 19-3).[4–6] The chest radiograph may not be helpful. Although the echocardiogram is usually normal in acute pericarditis, it is indicated in patients with suspected pericardial disease.[9]

Laboratory tests include complete blood count, cardiac enzyme levels (which may be elevated if the inflammation extends to the myocardium), rheumatoid factors, and antinuclear antibody titers. Blood cultures may be indicated if there is evidence of infection. Viral studies may be obtained if the rest of the diagnostic workup is negative.

Table 19-1 Differential Diagnosis of Chest Pain

Diagnosis	Onset of Pain	Quality of Pain	Relieved by
Angina pectoris	Sudden, after heavy meal or exertion	Crushing Squeezing Choking	Rest, nitrates
Acute myocardial infarction	Varies, may be associated with feeling of doom	Similar to angina, but more severe	No relief with rest
Pericarditis	Varies, may be preceded by "flu-like" symptoms for several days to weeks	Pleuritic Sharp, stabbing	Sitting up Shallow breathing NSAIDs
Acute aortic dissection	Sudden, may be associated with syncope Intense from the onset	Ripping Tearing Worst pain in patient's life	No relief

NSAIDs, nonsteroidal anti-inflammatory drugs.

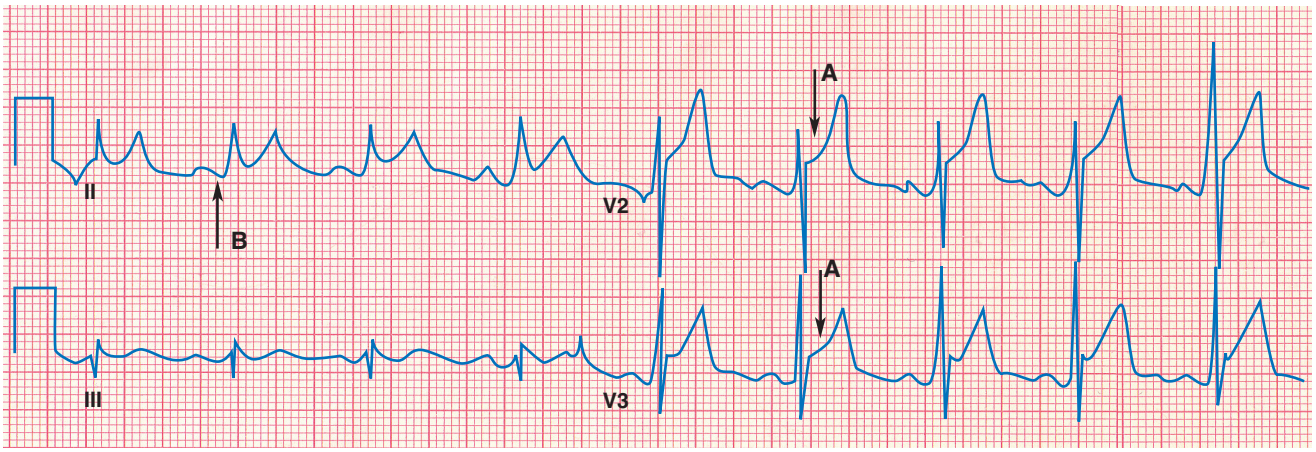


FIGURE 19-2 ▲ The 12-lead electrocardiogram in acute pericarditis. Note the diffuse upward concavity ST changes (*A*) and the PR-segment depression (*B*).

Management

Treatment goals for the patient with pericarditis are to relieve symptoms, eliminate any possible causative agents, and monitor for complications, such as constrictive pericarditis or pericardial effusions that could lead to cardiac tamponade.[3,6] Symptom relief includes the use of nonsteroidal anti-inflammatory drugs (NSAIDs), such as aspirin or ibuprofen. Steroids may be indicated in resistant cases in which infectious causes have been excluded. Anticoagulants should be avoided in the patient recovering from MI. Most episodes of pericarditis abate over 2 to 6 weeks. Rarely do patients experience recurrent episodes.

Myocarditis

Myocarditis is an inflammation of the myocardium.[3,10] Primary myocarditis is believed to be related to an acute viral infection or an autoimmune response to the infection. Secondary myocarditis is inflammation related to a specific organism. Potential causes of both types, which can occur in any age group, are listed in Box 19-2. The prevalence is unknown because the clinical presentation is so varied and often subacute.[11] Myocarditis can be a devastating illness that evolves into a chronic, progressive disease with a poor prognosis. The disorder may result in dysrhythmias, congestive heart failure, or death.[10] It is also recognized as a cause of sudden death in young athletes.[12]

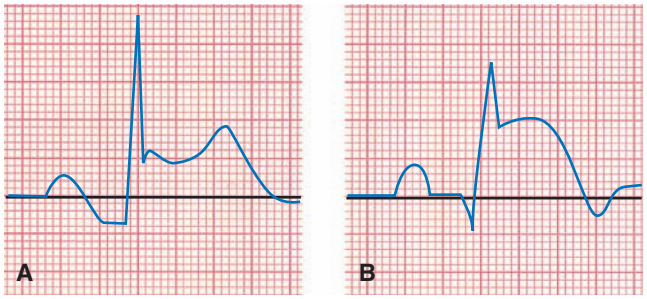


FIGURE 19-3 ▲ ST-segment changes seen in (**A**) acute pericarditis and (**B**) myocardial infarction.

Assessment

The clinical presentation of myocarditis is variable. With viral myocarditis, typically, there is a delay before the onset of cardiac symptoms, such as congestive heart failure or dysrhythmias.[3,10] The presence of symptoms, such as fatigue, dyspnea, palpitations, and precordial discomfort, accompanied by a slight rise in serum enzyme levels and nonspecific ST-T wave changes on the ECG suggests a diagnosis of myocarditis. Definitive diagnosis requires a positive endomyocardial biopsy.[10,11] However, lack of a positive biopsy does not rule out myocarditis. Current research focuses on finding a more reliable and safe method of diagnosing this complex disease.[10]

Management

Management of myocarditis depends on the etiology and clinical presentation; however, treatment is largely supportive.[3] Although myocarditis evokes a severe inflammatory response, treatment with corticosteroids or immunosuppressive agents has not been effective in changing the clinical course.[10,11] Some

BOX 19-2 Potential Causes of Myocarditis

Viruses
- Coxsackie virus
- Adenovirus
- Human immunodeficiency virus
- Influenza virus

Bacteria
- *Clostridium* species
- *Corynebacterium diphtheriae*
- Streptococci
- Spirochetes (Lyme's disease)

Fungi
- *Aspergillus* species
- *Candida* species

Toxins
- Tricyclic antidepressants
- Phenothiazines

BOX 19-3 EXAMPLES OF NURSING DIAGNOSES

- Acute Pain related to inflammation of the myocardium
- Fatigue related to deteriorating myocardial function
- Ineffective Coping related to sudden onset of a critical disease
- Ineffective Family Coping related to sudden onset of a critical disease
- Powerlessness related to medical regimen
- Grieving related to loss of former lifestyle

episodes of myocarditis resolve without further sequelae. In other patients, a subacute disease develops with persistent laboratory findings of inflammation (eg, an increased white blood cell count or an elevated sedimentation rate). Athletes with myocarditis should withdraw from competitive sports for a period of at least 6 months following the onset of disease. Return to training and competition depends on normalization of cardiac function and absence of any significant clinical findings, such as dysrhythmias.

Box 19-3 lists nursing diagnoses for a patient with myocarditis. Many of the skills required by the nurse to care for the patient with myocarditis are similar to those needed in the care of the patient with heart failure. In addition, the nurse must be prepared to help the patient and family deal with the unexpected reality of a potentially lethal disease that often has no cure and may require heart transplantation.[3]

Endocarditis

Endocarditis is an infection of the endocardial surface of the heart, including the valves, caused by bacterial, viral, or fungal agents.[13,14] Infectious endocarditis (IE) is a serious illness associated with considerable morbidity and mortality. The incidence of IE varies with the specific population under study, but overall, the incidence appears to be increasing.[3,13] Children with congenital heart disease, a group known to be at risk for IE, have increasingly higher survival rates, and this may contribute to the rise of IE in the pediatric population.[15] Adults at risk for IE include those with mitral valve prolapse or rheumatic heart disease, those who use illicit intravenous drugs, and patients with prosthetic valves or long-term indwelling devices (Box 19-4).[3,13,16,17] Common infectious organisms include streptococci, enterococci, and *Staphylococcus aureus*.

BOX 19-4 Risk Factors for Endocarditis

Native Valve Endocarditis
- Mitral valve prolapse
- Congenital heart disease
- Rheumatic heart disease
- Degenerative valve disease (such as aortic stenosis)
- Age greater than 60 years
- Intravenous drug abuse

Prosthetic Valve Endocarditis

Early (Within 60 Days of Surgery)
- Nosocomial infections
- Indwelling catheters
- Endotracheal tubes

Late (After 60 Days)
- Dental, genitourinary, or gastrointestinal manipulations

The development of IE is a complex process that requires the occurrence of several critical elements.[3,13,17] First, there must be endothelial damage that exposes the basement membrane of the valve to turbulent blood flow. Next, this exposure, especially in patients in a hypercoagulable state, must lead to the development of a platelet and fibrin clot on the valve leaflet. These clots, or vegetations, must be exposed to bacteria by way of the bloodstream, such as occurs after dental manipulations or urological procedures. Finally, bacterial proliferation must take place. Bacteria proliferate on these vegetations for two reasons: (1) the turbulent blood flow across the valves helps concentrate the numbers of bacteria near the vegetation and (2) the vegetation itself covers the bacteria with layers of platelets and fibrin, protecting the bacterial colony from the body's natural defense mechanisms. The infected vegetation interferes with normal valve function and eventually damages the valve structure. These incompetent valves eventually lead to severe heart failure. Particles from the infected vegetation or severely damaged valve can break loose and cause peripheral emboli.[3,13,14,17]

Assessment

Symptoms of endocarditis usually occur within 2 weeks of the precipitating bacteremia and are related to four underlying processes: bacteremia or fungemia, valvulitis, immunologic response, and peripheral emboli (Box 19-5).[13,16] Nonspecific complaints, such as general malaise, anorexia, fatigue, weight loss, and night sweats, are common. Because symptoms are nonspecific, a careful history focusing on risk factors for IE and a physical examination are needed to alert the nurse to the potential diagnosis of endocarditis.[3] Fever and a new or changed heart murmur are present in almost all patients.[13] The nurse should suspect IE in any patient with these clinical findings.[3,14]

Definitive diagnosis of IE includes persistent bacteremia caused by typical IE pathogens and evidence of myocardial involvement such as echocardiographic visualization of a vegetation or new or worsening murmur (Duke criteria).[18,19] Blood is usually drawn for three separate sets of cultures; meticulous site preparation is necessary to avoid contamination.[3]

BOX 19-5 Clinical Features of Endocarditis

- Fever
- Heart murmurs
- Splenomegaly
- Petechiae
  - Splinter hemorrhages
  - Osler's nodes (small, raised, tender nodules that occur on the fingers or toes)
  - Janeway's lesions (small erythematous or hemorrhagic lesions on the palms or soles)
- Musculoskeletal complaints
- Systemic or pulmonary emboli
- Neurological manifestations
  - Headache
  - Mycotic aneurysms

Management

Rapid diagnosis of IE, initiation of appropriate treatment, and early identification of complications are the keys to good patient outcomes.[3,16] Antibiotic therapy is based on the results and the clinical setting (ie, native valve vs. prosthetic valve IE). Recommended therapies have been revised to account for a dramatic increase in drug resistance among common IE organisms.[16] Treatment should not be delayed while waiting for identification of the specific organism but should begin as soon as blood culture specimens are drawn. Immediate surgical intervention is indicated in the presence of severe congestive heart failure secondary to valve dysfunction, uncontrolled infections, and prosthetic valve dysfunction or dehiscence.

Cure of IE is difficult and requires complete eradication of the bacterial colony from the vegetation. This usually involves a prolonged course of antibiotic therapy.[3,16]

▲ Cardiomyopathies

The cardiomyopathies are diseases of the heart muscle that cause cardiac dysfunction resulting in heart failure, dysrhythmias, or sudden death.[20–22] Since 1995, the cardiomyopathies have been separated into distinct categories: dilated, hypertrophic, restrictive, arrhythmogenic right ventricular cardiomyopathy, and unclassified.[23] However, advances in molecular genetics and improvements in diagnostic imaging reveal that the cardiomyopathies are more heterogeneous, which has led to the proposal of a new definition and classification scheme (Box 19-6).[20] See Spotlight on Genetics 19-1 for information about familial restrictive cardiomyopathy.

BOX 19-6 Cardiomyopathies and Their Classification

Definition

"Cardiomyopathies are a heterogeneous group of diseases of the myocardium associated with mechanical and/or electrical dysfunction that usually (but not invariably) exhibit inappropriate ventricular hypertrophy or dilatation and are due to a variety of causes that frequently are genetic. Cardiomyopathies either are confined to the heart or are part of generalized systemic disorders, often leading to cardiovascular death or progressive heart failure–related disability."[20]

Classification

Primary cardiomyopathies are solely or predominantly confined to heart muscle. Primary cardiomyopathies are classified according to etiology and include genetic (such as hypertrophic cardiomyopathy), mixed genetic and nongenetic (such as dilated cardiomyopathy), and acquired (such as inflammatory or peripartum cardiomyopathy).

Secondary cardiomyopathies have myocardial involvement as part of systemic disorders (such as amyloidosis or diabetes).

Adapted from Maron BJ, Towbin JA, Thiene G, et al: Contemporary definitions and classification of the cardiomyopathies: An American Heart Association scientific statement from the Council on Clinical Cardiology, Heart Failure and Transplantation Committee; Quality of Care and Outcomes Research and Functional Genomics and Translational Biology Interdisciplinary Working Groups; and Council on Epidemiology and Prevention. Circulation 113:1807–1816, 2006.

SPOTLIGHT ON GENETICS 19-1

FAMILIAL RESTRICTIVE CARDIOMYOPATHY

- The least common of the cardiomyopathies, the heart muscle is stiff and cannot fully relax after each contraction.
- Is caused by mutations in the *TNNI3* gene, which helps regulate contraction and relaxation of the heart muscle.
- *TNNI3* gene mutations associated with familial restrictive cardiomyopathy result in the production of a defective troponin I-cardiac isoform protein. The altered protein disrupts the function of the troponin protein complex and does not allow the heart muscle to fully relax
- Genetic testing for the *TNNI3* gene-**r**elated familial restrictive cardiomyopathy is available

Genetic Home Reference-http://ghr.nlm.nih.gov—Accessed July 14, 2011

Sen-Chowdhry S, Syrris P, McKenna WJ. Genetics of restrictive cardiomyopathy. Heart Fail Clin 6(2):179–186, 2010.

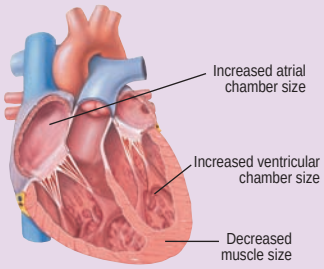
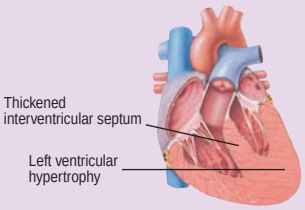
This section focuses on the most common types of primary cardiomyopathies in Western countries: dilated and hypertrophic cardiomyopathies (Table 19-2).

Exactly how cardiomyopathy develops is not completely understood. Current theories under investigation suggest that ischemic, immune, mechanical, and neurohormonal effects on the pericardium, myocardium, and endothelium lead to structural changes that result in functional changes. Structural changes at the cellular level include replacement of contractile and elastic muscle cells with fibrotic elements, which leads to stiffness of the ventricles and smooth muscle layers in the arteries. In hypertrophic cardiomyopathy (HCM), the heart muscle becomes thickened, with increased mass and poor relaxation. In dilated cardiomyopathy (DCM), the ventricular chamber dilates, thins, and changes from a normally elliptical shape to a less efficient spherical shape, reducing contractility and impairing emptying. Both stiffness and spherical remodeling may occur in the same heart, leading to a compromised cardiac output from impaired relaxation and impaired emptying. Stiffening of arteries seen in aging, atherosclerosis, and arteriosclerosis decreases stroke volume and exacerbates the ventricular wall stress by overfilling the ventricle. The heart attempts to maintain cardiac output in the face of a decreased stroke volume by increasing heart rate, which decreases relaxation time and impairs filling. This endless spiral of dysfunction is manifested by the progressive nature of heart failure.

The resulting decrease in cardiac output leads to activation of the renin–angiotensin–aldosterone system and the release of catecholamines. As previously described, these neurohormones were meant to respond to temporary decreases in blood pressure, such as hemorrhage, but in cardiomyopathy, the problem is chronic. Consequently, the neurohormonal effects, which were intended to be temporary, become permanent and become part of the problem instead of the solution to a decreased cardiac output.

The persistence of these neurohormones is hypothesized to be the mechanism by which the ventricle remodels from an elliptical shape to spherical, further decreasing its pumping efficiency. The realignment of the muscle fibers has been attributed to long-term exposure to aldosterone. Furthermore, long-term exposure to catecholamines leads to downregulation of β-adrenergic receptors and contributes to decreased contractility.

Table 19-2 Primary Cardiomyopathies

Cardiomyopathy	Pathology	Clinical Manifestations	Management
Dilated cardiomyopathy (DCM)  	Systolic dysfunction Chamber dilation with normal left ventricular wall thickness	• Congestive heart failure • Fatigue, weakness • Dysrhythmias • Systemic or pulmonary emboli	• Identify and eliminate potential causes such as alcohol • Symptomatic treatment • Manage heart failure, dysrhythmias • Biventricular pacing or implantable cardioverter defibrillator (ICD) in selected patients • Genetic testing • Family screening to identify asymptomatic members with DCM
Hypertrophic (HCM)  	Diastolic dysfunction Marked hypertrophy of left ventricle, occasionally also of right ventricle, and usually (but not always) disproportionate hypertrophy of septum	• Dyspnea • Angina • Fatigue • Syncope • Palpitations • Dysrhythmias • Congestive heart failure • Sudden death	• Symptomatic treatment • Medications • ICD • Septal wall ablation or surgery in select patients • Volume reduction surgery • Genetic testing • Family screening to identify asymptomatic members with HCM

Images adapted from the Anatomical Chart Company: Atlas of Pathophysiology. Springhouse, PA: Springhouse, 2010, p 45.

Dilated Cardiomyopathy

DCM is characterized by increased myocardial cavity size in the presence of normal or reduced left ventricular wall thickness and impaired systolic function.[20,21] The heart gradually assumes a globular shape accompanied by ventricular chamber dilation.[22] A decrease in contractility may occur for many reasons, including ischemia, alcohol abuse, endocrine disorders, pregnancy, viral infections, and valvular disease. The result of the decrease in contractility (ejection fraction <40%) is an increase in end-systolic volume. Over time, the ventricle dilates to accommodate the increased intraventricular volumes (preload). The increased preload in a normal heart would lead to an increase in stroke volume, but in the dilated heart, the increased volume leads to a decreasing stroke volume. As ventricular dilation progresses, mitral and tricuspid insufficiency develop as the valve leaflets are stretched and separated. Dysrhythmias, such as ventricular tachycardia, as well as conduction defects commonly occur.

DCM is the third most common cause of heart failure, the most common cause of heart failure in the young, and the most frequent cause of heart transplantation.[20] It occurs most frequently in middle-aged men, and 20% to 35% of cases are familial.[20,22] In most cases, the specific cause is unknown. The etiology of DCM is various, including familial and genetic factors, viral infections (ie, past episodes of viral myocarditis), immunological defects, and exposure to toxins.[20,21,24] Many researchers believe that alcohol is the most prevalent toxic cause of DCM.[21] DCM can be further divided into two types: ischemic and nonischemic.

Ischemic Cardiomyopathy

Ischemic cardiomyopathy is the result of oxygen levels that are inadequate to meet the metabolic demands of the myocardial cells. It occurs when there is obstruction in the coronary arteries and may be acute or chronic. Oxygen is essential to the function of cells. It is necessary for the metabolism of nutritional substrates and the formation of adenosine triphosphate (ATP), which powers all intracellular processes. When oxygen is inadequate, ATP becomes insufficient, and the calcium, sodium, and potassium pumps fail, leading to interruptions in both the mechanical and electrical function of the cells. The net result is a decrease in contractility and dysrhythmia. If oxygen is restored to the muscle cells, function returns and the dysrhythmia disappears.

If the ischemia is severe or persists, the muscle tissue dies, causing an MI. Dead muscle cannot regenerate and is replaced with scar tissue. The larger the scar, the greater the dysfunction. The decrease in muscle mass leads to decreased energy for pumping blood and therefore decreased cardiac output. The goal in treating unstable angina and acute MI is preservation of muscle mass to prevent systolic dysfunction.

If an MI is small, the damage may be insufficient to cause heart failure because there is still enough muscle to meet the body's demands for oxygen at rest and with exercise. The ejection fraction may still be within the normal range, although it may be decreased somewhat owing to the myocardial damage. However, repeated damage from subsequent infarctions or persistent ischemia in other areas of the heart muscle may exhaust the reserve function. "Hibernating" myocardium is an area of myocardial cells that are not dead (MI) but lack sufficient oxygen and nutrient substrates to contract. Once a patient's condition is stable after an MI, it is important to identify any viable myocardium that may be hibernating because of reversible ischemia. If perfusion can be restored to this viable but underperforming myocardium, ventricular function can be improved.

If an MI is very large, or critical structures such as the chordae tendineae are involved, then the consequences may be life-threatening. Damage or rupture of the chordae may

lead to acute, severe mitral regurgitation and profound heart failure. The loss of ventricular pumping function that results from a massive MI or smaller repeated MIs may produce such an acute loss of pump function that all the body's compensatory mechanisms are not effectively able to overcome the deficit in cardiac output.

This condition represents cardiogenic shock, in which cardiac output is severely inadequate and the left ventricle empties poorly (see Chapter 54). Consequently, left ventricular end-diastolic pressure increases, pulmonary artery pressures increase, and pulmonary edema results. End-organ damage caused by inadequate oxygen begins to occur depending on the function of the organ. The skin becomes cool, perhaps clammy and pale. The respiratory rate increases to supply as much oxygen as possible to the blood being pumped because the pulmonary edema severely decreases the effective area for gas transport. The pulmonary edema makes the lungs heavy and less compliant and reduces the effective tidal volume. Increases in respiratory rate are necessary to maintain minute volume. In addition, the tissues that are not adequately supplied with oxygen begin to produce lactic acid, leading to metabolic acidosis. The short-term compensation for metabolic acidosis is an increase in minute volume, or hyperpnea. The patient complains of feeling short of breath even at rest and may not be able to breathe in any recumbent position.

The hierarchy of protection in times of inadequate perfusion preserves most of the cardiac output for the brain, heart, and kidneys. Autoregulation mechanisms are present in all these organs to preserve pressure gradients and blood flow even when blood pressure and flow are compromised in other areas such as the skin, muscle, and gut. Indications that the brain is inadequately perfused are confusion, disorientation, somnolence, and agitation. Early indications of inadequate renal flow are an increase in blood urea nitrogen (BUN) and creatinine. Early on, the normal 10:1 to 20:1 ratio of BUN to creatinine increases to greater than 20:1; this signals the onset of prerenal azotemia. If perfusion is restored to the kidney at this time, the BUN and creatinine levels return to normal, as does kidney function. If the poor perfusion is profound or prolonged, the kidneys become damaged, and the BUN and creatinine continue to increase, although the ratio returns to normal. This ischemic damage to the kidneys is known as acute tubular necrosis and may be reversible.

If cardiogenic shock persists uncorrected for an extended period, the damage cannot be reversed, and the patient will die. Even if the patient is treated appropriately, further damage may occur in areas where the oxygen demand is lower than that of the brain and kidneys. Prolonged episodes of low cardiac output may lead to ileus, bowel infarction, liver failure, and increased risk for pneumonia and skin breakdown.

Patients who survive the initial episode of acute heart failure may recover completely if an intervention such as angioplasty or coronary artery bypass restores perfusion to the heart muscle and the damage to the remaining muscle is not severe. Chronic heart failure eventually develops in many patients and is characterized by the same symptoms as acute heart failure, but usually at a lower intensity; the body has had time to compensate for the decreased cardiac output. Usually, chronic heart failure does not have the intense limitations associated with acute heart failure. Patients often modify their activity to match the limited reserve of cardiac output available.

Nonischemic Cardiomyopathy

Nonischemic cardiomyopathy results from several causes. A large number of people have idiopathic DCM. For some as yet unknown reason, their hearts dilate, remodel, and become ineffective pumps. Others have myocarditis, often due to viral infection of the myocardium, hypothyroidism or hyperthyroidism, valvular disease, human immunodeficiency virus (HIV), or hemochromatosis. In addition, myocarditis may be bacterial or idiopathic. Nonischemic cardiomyopathy may also result from pregnancy, heavy alcohol use, hypertension, and tachycardia. Heart failure that results from hypothyroidism or hyperthyroidism, hemochromatosis, valvular disease, and tachycardia is reversible and disappears when these problems are corrected.

Nonischemic cardiomyopathy, like ischemic cardiomyopathy, may be acute or chronic. Patients with chronic disease are often quite limited in their ability to carry out everyday activities. The mechanism by which the dilation is triggered and progresses is not well understood. DCM, whether ischemic or nonischemic, produces symptoms after all the compensatory mechanisms have been exhausted.

Consequently, unless the onset of symptoms is acute, pathological changes may be quite advanced before activity is sufficiently limited and the patient seeks medical care. However, myocarditis frequently has an acute onset. The patient feels fine and is free of symptoms before fatigue and dyspnea on exertion, or, occasionally, pulmonary edema, suddenly develop. Dysfunction results from inflammation of the heart muscle. Metabolic function of inflamed muscle cells is impaired; the cells do not contract properly, leading to decreased cardiac output. Severity of the condition ranges from cardiogenic shock to mild limitation of activity. Once the initial acute phase passes, the patient has a low ejection fraction, with varying levels of physical limitation of activity and shortness of breath, or chronic heart failure.

Alcoholism, hypertension, and idiopathic etiologic factors are nonischemic conditions that may lead to DCM over longer periods—months to years as opposed to days to weeks with acute onset. As the ventricle begins to dilate, compensatory mechanisms, including the previously described catecholamines and other neurohormonal factors, begin to work. The proposed mechanism by which the ventricle remodels from the normal, efficient elliptical dimensions to a thin-walled, inefficient spherical shape involves constant exposure of the myocardium to these neurohormones. The natural progression is from dilation without symptoms to compensated heart failure, to uncompensated heart failure, and to refractory heart failure. Patients most often present when their heart failure is no longer compensated and symptoms interfere with normal daily activities. At this point, medication may relieve all or most symptoms. However, the structural changes that occur are progressive, and, even with medication, symptoms worsen over time. Medication can be adjusted to treat the worsening symptoms, but eventually, the medications will not be enough, and the patient dies. Usually mortality is due to worsening of the cardiac output, leading to system failure or sudden death from ventricular dysrhythmia. Before the stage of refractory heart failure is reached, much can be done to control the patient's symptoms, improve activity tolerance, control the progression of the disease, and improve quality of life.

ASSESSMENT. The natural history of DCM is not well defined. Some patients remain asymptomatic or have minimal clinical findings. Symptoms usually develop gradually and are typically related to left ventricular heart failure. The presence of right-sided heart failure is associated with poor prognosis.[3,21] Laboratory tests include screening for potentially reversible causes, including HIV. The echocardiogram is needed to differentiate the primary abnormality and determine the ejection fraction. Cardiac catheterization may be needed to rule out coronary artery disease.[3,21,24]

MANAGEMENT. Treatment goals include identifying and eliminating potential causes of DCM. Patients and their families should be questioned carefully about alcohol consumption because myocardial damage related to ingesting alcohol is reversible if detected early and the patient abstains from further drinking.[3,21,24] Clinical management is focused on control of heart failure and other problems such as dysrhythmias or intracoronary thrombus. Biventricular pacing may be helpful in medically refractory patients with severely symptomatic heart failure and a prolonged QRS on the ECG, dilated left ventricle, and poor ejection fraction.[25] Implantable cardioverter defibrillators (ICDs) may also be indicated in select patients to prevent sudden death associated with lethal dysrhythmias.[25] Only heart transplantation and some medical therapies have been shown to prolong life.[21]

Hypertrophic Cardiomyopathy

HCM is distinguished by a hypertrophied, nondilated left ventricle that is not related to any obvious cause, such as hypertension or aortic valve stenosis.[20,21] The most characteristic feature of HCM is diastolic dysfunction. The heart can contract but cannot relax and remains abnormally stiff in diastole. In a few patients, septal wall hypertrophy occurs, leading to a left ventricular outflow tract obstruction during systole.[3,21,26]

HCM is probably the most frequently occurring cardiomyopathy in the United States. It appears to be a common autosomal dominant genetic malformation; indeed, it is probably the most common genetic cardiovascular disorder, affecting approximately 1 in 500 of the population.[20,22,26]

Sudden death is a catastrophic outcome of HCM, usually from a ventricular dysrhythmia, in asymptomatic or mildly symptomatic people of any age group. In the United States, HCM is a leading cause of sudden death in competitive athletes as well as in people participating in recreational sports.[10,27] The risk for sudden death is constant; mortality is higher in younger patients.[3] Early identification of patients at risk for HCM (and therefore, sudden death) is imperative. However, there is no agreement on the best method to identify people at high risk at this time.[21,26]

Assessment

Many patients with HCM are asymptomatic or have only mild complaints.[3,21,24] The condition is often found unexpectedly during investigation of heart murmurs or family screening. The most common symptom is dyspnea, which may be exacerbated with exertion. Presyncope and syncope also frequently occur. Left ventricular hypertrophy (LVH) present on the echocardiogram confirms the diagnosis. Borderline LVH may be a normal finding in competitive athletes.[20]

Management

The goals of management include controlling symptoms, preventing complications, and reducing the risk for sudden death.[3,21] Genetic screening and counseling are also indicated.[26] Most symptomatic patients can be medically managed. ICDs are indicated in patients who have survived an episode of sudden death or have documented potentially lethal ventricular dysrhythmias.[25] In patients with symptoms resulting from septal hypertrophy, percutaneous ablation with ethanol or surgery to remove a portion of the septum may be necessary.[3,21]

Psychosocial concerns are important as patients and families try to cope with this debilitating and potentially fatal illness. They must deal with feelings of uncertainty and loss of control as well as the financial impact of a serious chronic illness.

▲ Peripheral Vascular Disease

Peripheral vascular disease includes a group of distinct disorders involving the arteries, veins, and lymphatic vessels of the peripheral circulation—the noncardiac diseases that affect the circulation as a whole.[28,29] The next section focuses on peripheral arterial and venous disease.

Peripheral Arterial Disease

Peripheral arterial disease (PAD) refers to processes that obstruct the blood supply of the lower or upper extremities.[28,29,30] The incidence of PAD depends on the population studied and the method used to establish the diagnosis. In general, symptomatic PAD is a disease of the elderly found more commonly in men aged 70 years and older.[28,29] Although the incidence of PAD increases steadily with age, the disease is more likely to occur in patients of any age with risk factors for atherosclerosis, such as smoking or diabetes. Other risk factors for PAD include hypertension, lipid disorders, family history, postmenopausal state, and hyperhomocysteinemia.[28,29] With the aging of the US population, management of PAD is a major focus not only of prevention and cure but also of maintenance of quality of life and independence (Box 19-7).

Atherosclerosis is the most common cause of PAD. The disease develops in major bifurcations and areas of acute angulations (Fig. 19-4). In people with diabetes, there is greater involvement of the smaller and more distal vessels. Upper extremity involvement is less common than lower extremity involvement.[28]

BOX 19–7 CONSIDERATIONS FOR THE OLDER PATIENT

Peripheral Arterial Disease

- Management of peripheral arterial disease (PAD) in older adults is often more complicated because of the presence of comorbidities, polypharmacy, financial concerns, physical and cognitive limitations, inadequate social support or isolation, and depression and anxiety.
- The incidence of symptomatic PAD increases with age, directly affecting quality of life.
- Conservative management (eg, smoking cessation, walking, foot care) can reduce symptoms and significantly improve quality of life in people of any age.

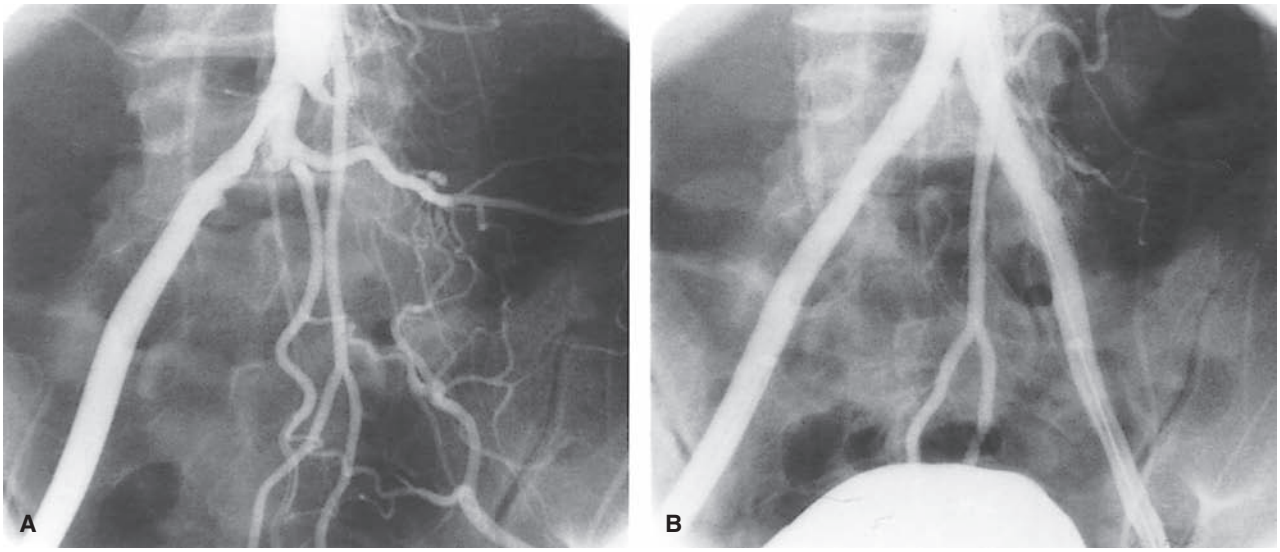


FIGURE 19-4 ▲ **A:** A baseline angiogram demonstrating a total occlusion of the left iliac artery. In addition, there is a significant stenosis of the right common iliac artery and occlusion of the internal iliac arteries. **B:** The final result following angioplasty and stenting of the right and left common iliac arteries with Palmaz stents. (Reprinted from Laird JR, Lansky AJ: Percutaneous transluminal angioplasty for the treatment of peripheral vascular disease. In Apple S, Lindsay J Jr (eds): Principles and Practice of Interventional Cardiology. Philadelphia, PA: Lippincott Williams & Wilkins, 2000, p 196, with permission.)

Thromboangiitis obliterans, or Buerger's disease, is a severe, chronic inflammatory disease affecting the intermediate and small arteries of the extremities. It may also involve adjacent veins and nerves. The etiology is unknown, but it is associated with heavy smoking, especially in young people. The chronic inflammatory process is often followed by thrombosis, with vascular lesions and fibrous obliteration of the vessel.[29] See Evidence-Based Practice Highlight 19-1.

Assessment

Clinical signs of PAD reflect the blood's inability to circulate freely to the extremity. Symptoms depend on the extent of the disease and the presence of collateral circulation. The classic symptom of PAD is intermittent claudication, experienced as a cramping, burning, or aching pain in the legs or buttocks that is relieved with rest.[28–31] Symptoms do not correlate with the extent of the disease. If the PAD is extensive and multilevel, the patient may present with "rest pain," that is, a sensation of burning or numbness in the foot or toes. Patients also experience trophic changes, such as hair loss on the extremities, thickening of the nails, and drying of the skin. Acute arterial obstruction, such as occurs with an embolism, results in the sudden onset of extreme pain and other signs of acute arterial obstruction (Box 19-8).[28–30]

Practice guidelines should be incorporated in the evaluation of the patient at risk for PAD.[28] This includes a careful vascular examination of the extremities and assessment of all peripheral pulses, including the measurement of segmental pressures in the legs and the ankle/brachial index (ABI). The ABI is the ratio of ankle to brachial systolic blood pressure. A normal ABI should be 1.0 or greater. Patients with critical limb ischemia may have an ABI of less than 0.518 (Fig. 19-5).[28,29]

EVIDENCE-BASED PRACTICE HIGHLIGHT 19-1

Heart Failure in Adults With Peripheral Arterial Disease

△ Risk Factors for Heart Failure in the Adult

- Atherosclerotic coronary artery disease and or myocardial infarction or ischemia
- Presence of pressure overload including hypertension or obstructive valvular disease
- Genetic or familial disorders, infiltrative disorders
- Advanced age
- Toxins (ie, alcohol)

△ Initial Evaluation of Patients

- Evaluation of exercise tolerance including walking
- Comprehensive physical evaluation including lungs, heart, and vascular systems
- Personal and family history including family history of heart failure or sudden death. History of drug or alcohol use

Excerpted from Jessup M, Abraham WT, Casey DE, et al: ACCF/AHA Guidelines for the Diagnosis and Heart and Lung Transplantation Guidelines Developed in Collaboration With the International Society for Cardiology Foundation/American Heart Association Task Force on Practice Management of Heart Failure in Adults: A Report of the American College of Foundation/American Heart Association Task Force on Practice Guidelines Developed in Collaboration With the International Society for Heart and Lung Transplantation. J Am Coll Cardiol 53:1343–1382, 2009.

BOX 19-8 Clinical Features of Vascular Obstruction

Acute Arterial Occlusion
- Pain
- Pulselessness
- Pallor
- Paresthesia
- Paralysis

Deep Venous Thrombosis
- Pain in calf with dorsiflexion of foot (Homans' sign)
- Pain when standing
- Inflammation
- Swelling
- Tenderness
- Redness, soreness

Treadmill exercise testing can provide an objective measurement of the patient's walking ability as well as an evaluation of possible coronary artery disease. Noninvasive imaging, such as magnetic resonance or computed tomography (CT), may be required to evaluate the extent of disease fully. Angiography is usually limited to revascularization procedures (see Fig. 19-4) or presurgical evaluation.[28,29]

Management

PAD is associated with an increased risk for atherosclerotic adverse events; the mortality rate is high in symptomatic patients.[29,30] Therefore, treatment goals include modifying or eliminating risk factors (especially smoking), improving leg symptoms, and maintaining limb viability. Risk factor modification incorporates national guidelines; these include immediate smoking cessation as well as aggressive treatment of hypertension, diabetes, and lipid disorders, with medication, if necessary. Other pharmacological agents include antiplatelets (aspirin or clopidogrel [Plavix]) to reduce the risk for MI and stroke and cilostazol (Pletal) to increase walking distance. In patients with claudication, exercise improves overall walking ability. Peripheral interventional procedures, such as balloon angioplasty, are successful in restoring circulation in many cases. Surgical bypass may be required when severe or diffuse arterial obstruction is present.[28–30]

Venous Disease

Phlebitis is inflammation of the vessel wall occurring as the result of direct injury to the vein or as a complication of varicose veins. It can lead to the formation of a thrombus, a solid

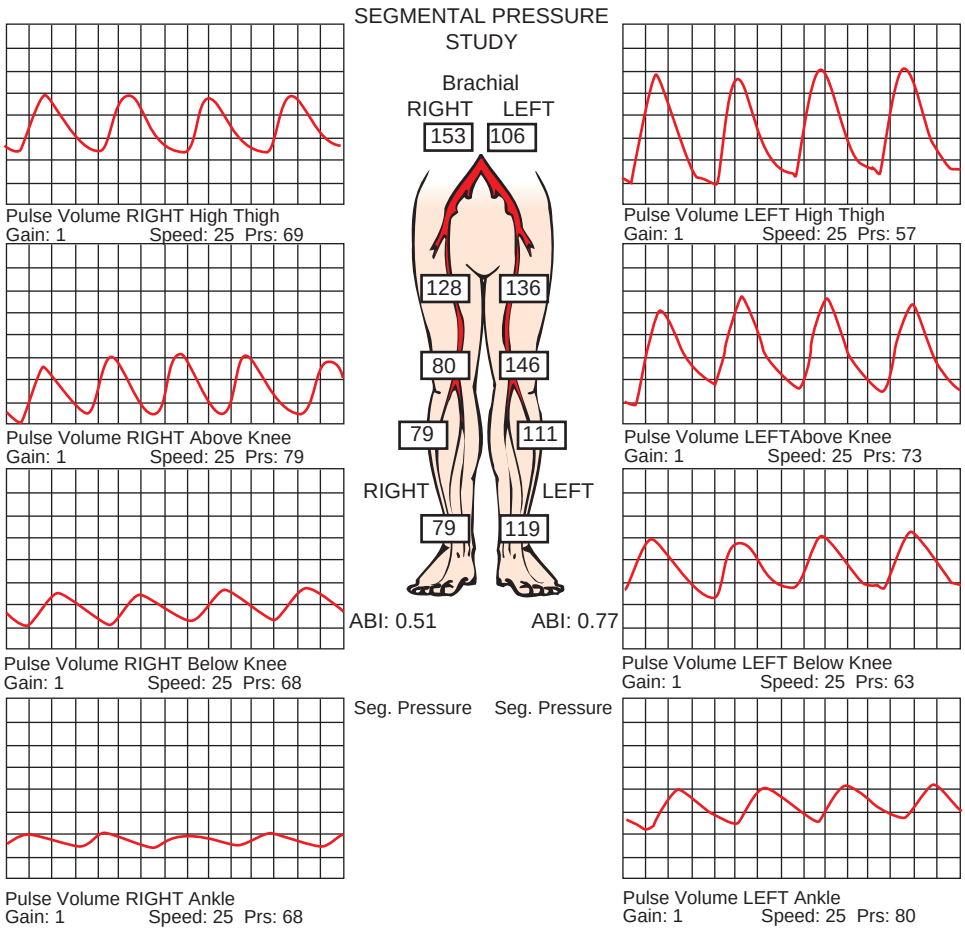


FIGURE 19-5 ▲ Segmental pressures and ankle/brachial indices indicating bilateral lower extremity occlusive disease with more severe involvement of the right lower extremity. There is also a probable significant stenosis of the left subclavian artery, which explains the difference between the right and left brachial pressures. Prs, pressures. (Reprinted from Saucedo JF, Laird JR: Peripheral vascular disease. In Apple S, Lindsay J Jr [eds]: Principles and Practice of Interventional Cardiology. Philadelphia, PA: Lippincott Williams & Wilkins, 2000, p 47, with permission.)

obstruction within the vein that can break loose and form a venous thromboembolism (VTE).[32,33] Factors that predispose a patient to thrombus formation are vessel wall injury, stasis of blood, and increased blood coagulability (Virchow's triad).[32,34] These three conditions have been recognized as causative factors in the development of thrombophlebitis since 1846.

An estimated 100,000 to 300,000 cases of VTE occur in the United States each year.[32,34] This incidence increases with aging.[34,35] More than half of the cases of VTE involve deep vein thrombosis (DVT),[34] and others include pulmonary embolism. Because VTE is associated with significant morbidity and mortality, it is important for the nurse to be familiar with risk factors for VTE as well as current recommendations for treatment. (See Chapter 26 for more information concerning pulmonary embolism.[33,36])

Assessment

DVT is characterized by pain, swelling, tenderness, and increased temperature over the affected area (see Box 19-8). The patient also exhibits a positive Homans' sign (pain in the calf with passive dorsiflexion of the foot).[32] However, these clinical findings are not specific for DVT. Accurate diagnosis usually requires diagnostic testing such as compression ultrasonography.[34]

Management

The focus of care for the patient with VTE is to relieve symptoms, increase blood flow, and prevent complications. Patients with DVT are at high risk for pulmonary embolism. Treatment strategies include anticoagulant therapy to prevent the formation of emboli, followed by long-term warfarin (Coumadin) use to prevent recurrence. Specific therapy depends on the patient's history and clinical setting.[33] Bleeding is the most common complication of therapy. Patient teaching includes safe administration of home anticoagulants as well as behaviors to decrease the recurrence of DVT.[32] Prevention of VTE is discussed in Evidence-Based Practice Highlight 19-2.

▲ Aortic Disease

The aorta is the longest and strongest artery in the body.[37] However, over time, congenital, degenerative, hemodynamic, and mechanical factors stress this elastic vessel. The result is dilation of the aortic wall, leaving the patient at risk for aortic dissection or rupture.[38]

Aortic Aneurysm

Aortic aneurysms are defined as a localized dilation of the aorta to a size greater than 1.5 times its normal diameter.[37] Aneurysms are classified according to their shape, morphology, and location (Fig. 19-6). Fusiform aneurysms, the more common type, are diffuse dilations of the entire circumference of the artery. Saccular aneurysms are localized balloon-shaped outpouchings. Aneurysms may be thoracic or abdominal; rarely, they are both.

True aneurysms involve the entire vessel wall and are classified as fusiform or saccular. False aneurysms are not actually aneurysms but are formed when blood leaks through the wall of the aorta and is contained by the surrounding tissues (a contained rupture).[37]

Abdominal Aortic Aneurysm

Abdominal aortic aneurysms (AAAs), which are more common than thoracic aortic aneurysms, occur more frequently in men. Smoking is the leading risk factor for AAAs, followed closely by age, hypertension, lipid disorders, and atherosclerosis.[37] Atherosclerosis is probably a major cause of AAAs, but other factors, such as genetic and environmental influences, almost certainly contribute to their development.[39] The major risk from AAAs is rupture, which is associated with a high rate of mortality.

ASSESSMENT. Most patients with AAAs are asymptomatic; they are typically identified during health screening for another problem. Abdominal or back pain is the most common complaint. Worsening of symptoms is usually related to expansion or rupture of the aneurysm.

Detection of AAAs by physical examination is difficult, especially in obese patients. The abdomen is examined for the presence of bruits or masses, and peripheral pulses are carefully evaluated. Abdominal ultrasonography is the most practical method of confirming the diagnosis.[37–39]

MANAGEMENT. Management of AAAs includes control of hypertension and elimination of risk factors, such as smoking. The patient should be followed with serial noninvasive tests, such as ultrasonography. Treatment of aneurysms involves surgical repair, which is usually indicated for AAAs larger than 5.5 cm (Box 19-9).[40]

In addition to surgery, AAAs may be repaired by a minimally invasive approach using an endovascular graft. This approach involves placement of a graft through the femoral artery. The graft is then anchored to the wall of the aorta by means of self-expanding or balloon-expanded stents. Endovascular repair has become the treatment of choice for high-risk patients with AAAs.[41]

Thoracic Aortic Aneurysm

Thoracic aortic aneurysms occur relatively infrequently and are classified by the involved segment of the aorta (root, ascending, arch, or descending). The location is important because the etiology, natural history, and treatment differ for each segment.[37,39] Most ascending thoracic aortic aneurysms are due to cystic medial degeneration. Ascending thoracic aortic aneurysms are also associated with connective tissue disorders, genetic disorders, bicuspid aortic valve, infections, inflammatory diseases, chronic aortic dissection, and trauma.[37–39]

ASSESSMENT. Like most patients with AAAs, most patients with thoracic aortic aneurysms are asymptomatic at the time of diagnosis. Symptoms are related to the size and location of the aneurysm; these include aortic insufficiency and signs of pericardial tamponade if the aneurysm involves the aortic root.[37] Rupture or acute dissection of a thoracic aneurysm can be fatal. Fewer than half of patients with rupture survive until they are hospitalized, and by 24 hours, mortality is almost 80%.[37]

MANAGEMENT. For most ascending thoracic aortic aneurysms, surgical repair is indicated at a diameter of

EVIDENCE-BASED PRACTICE HIGHLIGHT 19-2
Venous Thromboembolism Prevention

Expected Practice

- Assess all patients upon admission to the ICU for risk factors of venous thromboembolism (VTE) and anticipate orders for VTE prophylaxis based on risk assessment. [Level D]
- Clinical eligibility and regimens for VTE prophylaxis include:
  - Moderate-risk patients (medically ill and postoperative patients): low dose unfractionated heparin, low-molecular-weight heparin (LMWH), or fondaparinux [Level B]
  - High-risk patients (major trauma, spinal cord injury, or orthopedic surgery): LMWH, fondaparinux, or oral vitamin K antagonist [Level B]
  - Patients with high risk for bleeding: mechanical prophylaxis including graduated compression stockings and/or intermittent pneumatic compression devices [Level B]
  - Mechanical prophylaxis may also be anticipated in conjunction with anticoagulant-based prophylaxis regimens.
- Review daily—with the physician and during multidisciplinary rounds—each patient's current VTE risk factors including clinical status, necessity for central venous catheter (CVC), current status of VTE prophylaxis, risk for bleeding, and response to treatment. [Level E]
- Maximize patient mobility whenever possible and take measures to reduce the amount of time the patient is immobile because of the effects of treatment (eg, pain, sedation, neuromuscular blockade, mechanical ventilation). [Level E]
- Ensure that mechanical prophylaxis devices are fitted properly and in use at all times except when being removed for cleaning and/or inspection of skin. [Level E]

Supporting Evidence

- Multiple medical and surgical risk factors leading to VTE formation have been identified.[1] Iatrogenic risk factors for VTE include immobilization, sedation/neuromuscular blockade, CVCs, surgery, sepsis, mechanical ventilation, vasopressor administration, heart failure, stroke, malignancy, previous VTE, and renal dialysis; a vast majority of patients in critical care units have 1 or more major risk factors.[1–4] In five prospective studies, the rate of VTE in patients in critical care not receiving prophylaxis ranged from 13% to 31%.[5–8] Because signs and symptoms of VTE are frequently silent and can lead to fatal pulmonary embolism, multiple professional organizations recommend VTE prophylaxis for at-risk patients.
- Randomized trials indicate that both low dose unfractionated heparin and LMWH are efficacious in preventing VTE in moderate-risk critical care patients.[5–8,19] For patients at higher risk, such as those who have major trauma or have had orthopedic surgery, LMWH has been shown to provide superior protection over low dose unfractionated heparin.[1] Direct thrombin inhibitors can be used in place of LMWH or unfractionated heparin for patients with documented of suspected heparin-induced thrombocytopenia.[1,21–22] Numerous studies suggest that aspirin alone is not an efficacious means of VTE prophylaxis for any patient group.[23–27]
- Although examined less rigorously than anticoagulant-based methods, mechanical methods of prophylaxis (including graduated compression stockings, intermittent compression devices, and venous foot pumps) have been shown to reduce the risk of VTE.[28–38] One study involving non–lower extremity trauma patients compared the efficacy of intermittent pneumatic compression devices and venous foot pumps. VTE rates among the venous foot pump group were three times greater when compared with the rates of the intermittent pneumatic compression group. The researchers concluded that intermittent pneumatic compression devices provided superior prophylaxis in this patient population.[36]
- In general, mechanical prophylaxis is less efficacious when compared to anticoagulation-based therapy.[33–35,37–39] Reduction in risk of death or pulmonary embolism has not been attributed to mechanical methods of prophylaxis.[1] In one study involving below-the-knee graded stockings, 98% of commercially available stockings failed to produce an ideal pressure gradient and 54% were found to produce a dangerous reverse pressure gradient.[40] Mechanical prophylaxis methods are a desirable option because they do not pose bleeding concerns.[1] A combination of mechanical prophylaxis and chemoprophylaxis is thought to potentiate overall efficacy but this combination has not been tested in the critical care setting.[41]
- Written policies for VTE prophylaxis in conjunction with either preprinted or computerized ICU admission orders have been shown to increase compliance with prophylaxis measures.[42] One study found that implementation of a daily goals form, which included VTE prophylaxis in the ICU, resulted in a significant improvement in the percentage of residents and nurses who understood the patient's daily goals for care and decreased ICU length of stay by 1.1 days.[19,43,44]
- The presence of a CVC is an independent risk factor for upper extremity VTE in the general population.[45]
- Several studies involving a variety of patient populations with diagnostically confirmed VTE have identified immobility either as a comorbidity or independent risk factor.[46–48]
- Improperly fitted graduated compression stocking producing a reversed pressure gradient were associated with a statistically higher incidence of VTE compared with stockings that produced a proper gradient.[35] Studies evaluating compliance with intermittent pneumatic compression devices demonstrated rates of noncompliance ranging from 22% to 81% in at-risk patients.

AACN Evidence Leveling System

Level A Meta-analysis of quantitative studies or metasynthesis of qualitative studies with results that consistently support a specific action, intervention, or treatment.
Level B Well-designed, controlled studies with results that consistently support a specific action, intervention, or treatment.
Level C Qualitative studies, descriptive or correlational studies, integrative review, systematic reviews, or randomized controlled trials with inconsistent results.
Level D Peer-reviewed professional organizational standards with clinical studies to support recommendations.
Level E Multiple case reports, theory-based evidence from expert opinions, or peer-reviewed professional organizational standards without clinical studies to support recommendations.
Level M Manufacturer's recommendations only.

Excerpted from American Association of Critical-Care Nurses Practice Alert. Available online at http://aacn.org. All references cited in this alert are available with the associated resources related to this chapter. Visit: http://thepoint.lww.com

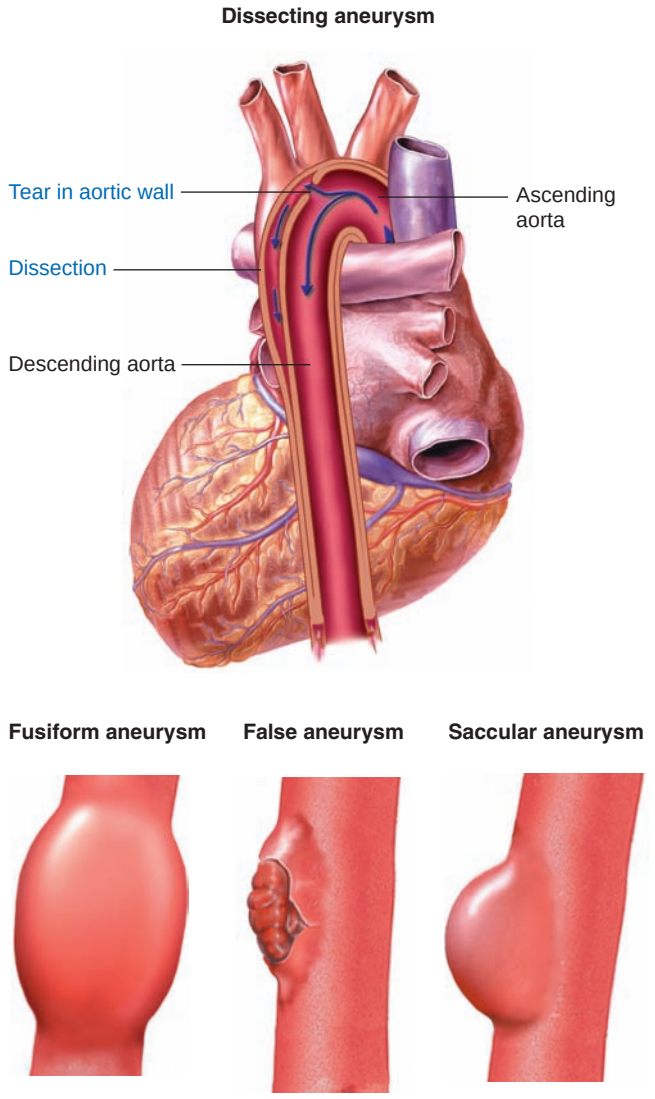


FIGURE 19-6 ▲ Types of aortic aneurysms. (Anatomical Chart Company: Atlas of Pathophysiology. Springhouse, PA: Springhouse, 2010, p 39.)

5.5 cm or more.[39] These indications vary according to the clinical situation and the existence of comorbidities. Repair of descending thoracic aneurysms is recommended when the diameter is 6 cm or more.[39]

Aortic Dissection

Acute aortic dissection is the most common and the most lethal process involving the aorta. Mortality rates are very high, approaching 1% per hour for this catastrophic event.[37] Death usually occurs from rupture of the aorta. The incidence is highest in men older than age 60 with a history of hypertension. Other risk factors include cystic medial degeneration, pregnancy, and trauma.[37,38]

Pathophysiology

Dissection involves a longitudinal separation of the medial layers of the aorta by a column of blood. The dissection begins at a tear in the aortic wall, usually at the proximal end of the dissection. Blood pumped through this tear creates a false channel, or lumen, that rapidly becomes larger than the true aortic lumen. Dissections are typically classified according to location, as illustrated in Figure 19-7.

BOX 19-9 General Indications for Surgical Repair of Aortic Aneurysms

Abdominal
- Diameter 5.5 cm of more (men)
- For women, 4.5 to 5.0 cm (due to greater incidence of rupture)
- Diameter 4.5 to 5.5 cm; clinical setting, patient preference

Ascending Thoracic
- Diameter 5.5 cm or more (5 cm in patients with Marfan's syndrome)
- Symptoms suggesting expansion or compression of surrounding structures

Other
- Rapidly expanding aneurysms (growth rate more than 0.5 cm over a 6-month period)
- Symptomatic aneurysm regardless of size

Assessment

More than 90% of patients present with sudden, intense chest pain. Frequently, the pain is described as "ripping" or "tearing" and may be accompanied by syncope (see Table 19-1). In most patients, the diagnosis can be determined with a careful history and physical examination. The clinician should

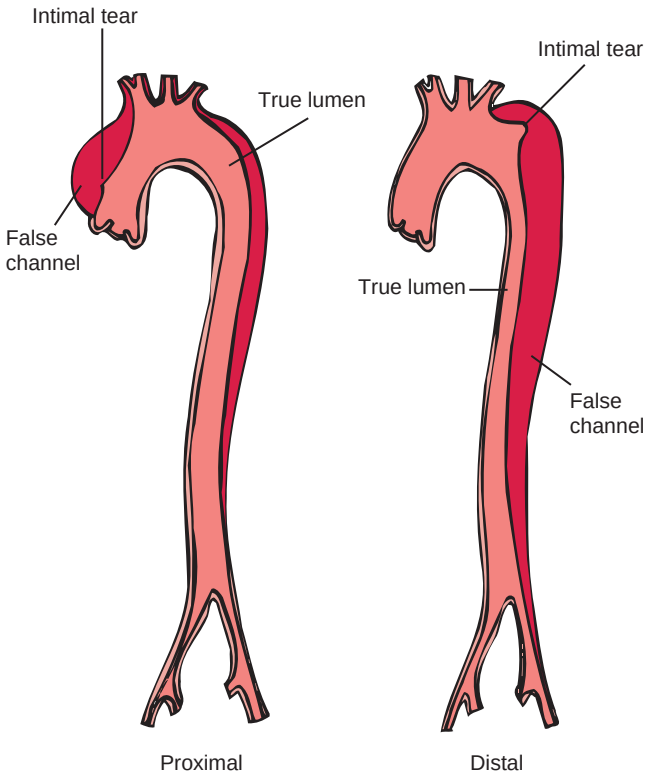


FIGURE 19-7 ▲ Two major patterns of aortic dissection. Blood pumps through a tear in the wall, creating a false channel or lumen. The false channel rapidly becomes larger than the true lumen.

look for the murmur of aortic regurgitation or alteration of the peripheral pulses in patients with known risk factors, such as hypertension. The chest radiograph may show a widened mediastinum. Cardiac ischemia may be present if the dissection involves the coronary arteries. Cardiac tamponade may be another complication of dissection involving the aortic root. Neurological deficits may occur if the aortic arch vessels are involved. Dissections involving the renal arteries result in elevated serum creatinine, decreased urine output, and severe hypertension that is difficult to manage. To confirm the diagnosis of acute aortic dissection, transesophageal echocardiography or contrast medium–enhanced CT may be ordered.[37–39]

Management

Survival of the acute phase depends on the location of the dissection, the severity of the complications, and the rapidity with which the diagnosis is confirmed. Clinical management focuses on controlling blood pressure and managing pain. Surgery is the treatment of choice when the dissection involves the ascending aorta.[37,38]

▲ Hypertensive Crisis

Hypertension affects approximately 50 million people in the United States and is a major controllable risk factor for the development of cardiovascular diseases.[42,43] Recognition of the extent of this risk led to the inclusion of a new category in the classification of hypertension, prehypertension, which includes individuals with a systolic blood pressure of 120 to 139 mm Hg or a diastolic blood pressure of 80 to 89 mm Hg (Table 19-3). People with prehypertension should be counseled to adopt healthy lifestyle modifications to reduce their risk for cardiovascular disease.

Patients with high blood pressure are at risk for experiencing a hypertensive crisis. A hypertensive crisis or emergency is defined as an acute elevation of blood pressure (>180/120 mm Hg) that is associated with acute or imminent target organ damage.[42] This rare but potentially fatal condition strikes about 1% to 2% of hypertensive patients, occurring more frequently in African American men and in elderly patients.

Pathophysiology

A hypertensive crisis is characterized by a marked rapid increase in blood pressure that initially leads to intense vasoconstriction as the body attempts to protect itself from the elevated pressure. If the blood pressure remains critically high, compensatory vasoconstriction fails, resulting in increased pressure and blood flow throughout the vascular system. In the cerebral circulation, this may quickly lead to hypertensive encephalopathy.[42,43] Hypertensive crisis is associated with a variety of clinical situations (Box 19-10).

Table 19-3 Classification of Blood Pressure for Adults

Blood Pressure Classification	Systolic (mm Hg)	Diastolic (mm Hg)
Normal	<120	and <80
Prehypertension	120–139	or 80–89
Stage 1 hypertension	140–159	or 90–99
Stage 2 hypertension	≥160	or ≥100

Adapted from the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7). May 2003. Available at: http://www.nhlbi.nih.gov/guidelines/hypertension.

BOX 19-10 Summary of Hypertensive Crisis

Causes
- Acute or chronic renal disease
- Exacerbation of chronic hypertension
- Sudden withdrawal of antihypertensive medications

Associated Clinical Situations
- Acute cerebrovascular syndrome
  - Acute stroke
  - Hypertensive encephalopathy
  - Acute cardiovascular syndromes
  - Myocardial infarction
  - Unstable angina
  - Pulmonary edema
- Aortic dissection
- Extensive burns
- Postoperative period
- Pheochromocytoma
- Eclampsia

Management
- Intravenous medications with continuous arterial pressure monitoring
- Goal is to reduce mean arterial blood pressure over 1 hour by no more than 25% while avoiding hypoperfusion

Assessment

Most patients who present with hypertensive crisis are critically ill and in need of immediate treatment. Clinical findings depend on the degree of vascular injury.[43] Signs of encephalopathy include headache, visual disturbances, confusion, nausea, and vomiting. Examination of the eyes may reveal cotton–wool exudates and hemorrhages, indicating damage to retinal nerves and rupture of retinal blood vessels; papilledema is diagnostic of increased intracranial pressure. Chest pain may represent acute coronary syndrome or aortic dissection. Depending on the damage to the kidneys, the patient may present with decreased urine output (oliguria) or azotemia (excess urea in the blood).[43,44]

Management

The goal is to reduce the mean blood pressure within 1 hour of starting treatment and to prevent or reverse target organ damage.[42,45,46] Several intravenous medications are indicated in treating hypertensive crises; the choice depends on availability and the clinical situation (Table 19-4). Constant monitoring is necessary to avoid lowering the blood pressure too quickly. This is best accomplished with an intra-arterial catheter.

Once the blood pressure has been stabilized, treatment goals depend on the etiology of the crisis. All patients require careful long-term management to control their blood pressure and prevent future episodes.

Table 19-4 Intravenous Medications in Hypertensive Emergencies*

Drug	Class	Onset of Action	Adverse Effects
Sodium nitroprusside	Vasodilator	Immediate	Hypotension, nausea, vomiting, muscle twitching, thiocyanate and cyanide toxicity, methemoglobinemia
Nitroglycerin	Vasodilator	1–2 min	Hypotension, reflex tachycardia, headache, tolerance with prolonged use
Labetalol	Adrenergic blocker	<5 min	Nausea, vomiting, bronchospasm, heart block
Fenoldopam	Vasodilator	<5 min	Reflex tachycardia, headache, nausea
Esmolol	Adrenergic blocker	Immediate	Hypotension, heart block
Nicardipine	Calcium channel blocker	5–6 min	Reflex tachycardia, headache, nausea, vomiting, flushing
Enalaprilat	Angiotensin-converting enzyme inhibitor	10–15 min	Hypotension, renal failure
Hydralazine	Vasodilator	15–30 min	Reflex tachycardia, headache, exacerbation of angina pectoris

*Choice of drug depends on the etiology of the hypertensive emergency and the clinical setting.

Adapted from Mansoor GA, Frishman WH: Comprehensive management of hypertensive emergencies and urgencies. Heart Dis 4:358, 2002; and Tuncel M, Ram VCS: Hypertensive emergencies: Etiology and management. Am J Cardiovasc Drugs 3(1):21–31, 2003.

▲ Clinical Applicability Challenges

CASE STUDY

Mr. S. is a 21-year-old Caucasian man who is being evaluated in the emergency room with a 2-day history of chest pain. The patient describes the chest pain as a sharp burning pain in the center of his chest that gets worse with deep inspiration and better when he changes position. He reports a history of a recent upper respiratory viral infection. He is physically active, does not smoke, and has no family history of premature heart disease. An ECG performed in the emergency room shows diffuse ST-segment elevations and PR-segment depression.

Physical examination reveals a normal S1 and S2 and no murmurs or gallops. There is a friction rub that is heard most prominently at the apex and varies with the cardiac cycle. Prior laboratory values obtained during the emergency room visit revealed potassium, 4.2 mEq/L; blood urea nitrogen, 20 mg/dL; creatinine, 1.0 mg/dL; brain natriuretic peptide, 50 pg/mL; troponin I, 0.10 ng/mL × 1 cycle; white blood cells, 9.0×10^3 mL; hemoglobin, 13.0 g/dL; and hematocrit, 43.3%. A C-reactive protein level was 10 mg/L. Results of a posteroanterior and lateral chest x-ray were unremarkable.

Mr. S. is admitted to your telemetry floor with a diagnosis of acute pericarditis for monitoring and cycling of his cardiac enzyme levels. Prior to your shift, an echocardiogram reveals an ejection fraction of 65% (normal 50% to 70%), normal valve structure and function, and normal myocardial wall motion. He was taking no medication prior to his admission. He received 30 mg of ketorolac (Toradol) IV while in the emergency room and was started on ibuprofen 800 mg by mouth every 8 hours.

During morning report, you learn that Mr. S. is very concerned about the diagnosis of pericarditis. He has questions regarding the long-term prognosis and the length of time he may be required to take medication and its impact on his ability to play sports. He does not care for the hospital food and wants to know when he can go home.

1. What are the priority medical problems for Mr. S.?
2. What are the priority nursing actions for Mr. S.?
3. What are some potential long-term problems facing Mr. S.?

References

1. Bond EF: Cardiac Anatomy and Physiology. In Woods SL, Sivarajan Froelicher ES, Motzer SA, et al (eds): Cardiac Nursing, 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2005, pp 3–48
2. Porth CM, Matfin G: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, pp 536–539
3. McNeill MM: Pericardial, myocardial, and endocardial disease. In Woods SL, Sivarajan Froelicher ES, Motzer SA, et al (eds): Cardiac Nursing, 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2005, pp 776–793
4. LeWinter MM: Pericardial diseases. In Libby P, Bonow RO, Mann DL, et al (eds): Braunwald's Heart Disease, 8th ed. Philadelphia, PA: Elsevier Saunders, 2008, pp 1829–1852
5. Lange RA, Hillis LD: Acute pericarditis. N Engl J Med 351(21):2195–2202, 2004
6. Carter T, Brooks CA: Pericarditis: Inflammation or infarction? J Cardiovasc Nurs 20(4):239–244, 2005
7. Maisch B, Ristic AD: Practical aspects of the management of pericardial disease. Heart 89:1096–1103, 2003
8. Wang A, Bashore TM: Undercover and overlooked. N Engl J Med 351(10):1014–1019, 2004
9. American College of Cardiology/American Heart Association/American Society of Echocardiography: 2003 Guideline Update for the Clinical Application of Echocardiography. Retrieved November 14, 2010, from http://www. cardiosource.org
10. Liu PP, Schultheiss H-P: Myocarditis. In Libby P, Bonow RO, Mann DL, et al (eds): Braunwald's Heart Disease, 8th ed. Philadelphia, PA: Elsevier Saunders, 2008, pp 1775–1791
11. Baughman KL: Diagnosis of myocarditis: Death of Dallas criteria. Circulation 113:593–595, 2006
12. Maron BJ, Ackerman MJ, Nishimura RA, et al: Task Force 4: HCM and other cardiomyopathies, mitral valve prolapse, myocarditis, and Marfan syndrome. J Am Coll Cardiol 45:1340–1345, 2005. Retrieved November 14, 2010, from http://content.onlinejacc.org/ cgi/content/full/45/8/1318
13. Karchmer AW: Infective endocarditis. In Libby P, Bonow RO, Mann DL, et al (eds): Braunwald's Heart Disease, 8th ed. Philadelphia, PA: Elsevier Saunders, 2008, pp 1713–1734
14. Fink AM: Endocarditis after valve replacement surgery. Am J Nurs 106:40–51, 2006
15. Ferrieri P, Gewitz MH, Gerber MA, et al: Unique features of infective endocarditis in childhood. Circulation 105:2115–2126, 2002
16. Nishmura RA, Carabello BA, Faxon DP, et al: ACC/AHA 2008 Guideline update on valvular heart disease: Focused update on infective endocarditis: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 52:676–685, 2008
17. Moreillon P, Que Y: Infective endocarditis. Lancet 363:139–149, 2004
18. Durak DT, Lukes AS, Bright DK, for the Duke Endocarditis Service: New criteria for diagnosis of infective endocarditis: Utilization of specific echocardiographic findings. Am J Med 96:200–209, 1994
19. Li JS, Sexton DJ, Mick N, et al: Proposed modifications to the Duke criteria for the diagnosis of infective endocarditis. Clin Infect Dis 30:633–638, 2000
20. Maron BJ, Towbin JA, Thiene G, et al: Contemporary definitions and classification of the cardiomyopathies: An American Heart Association scientific statement from the Council on Clinical Cardiology, Heart Failure and Transplantation Committee; Quality of Care and Outcomes Research and Functional Genomics and Translational Biology Interdisciplinary Working Groups; and Council on Epidemiology and Prevention. Circulation 113:1807–1816, 2006
21. Hare JM: The cardiomyopathies. In Libby P, Bonow RO, Mann DL, et al (eds): Braunwald's Heart Disease, 8th ed. Philadelphia, PA: Elsevier Saunders, 2008, pp 1739–1760
22. Hughes SE, McKenna WJ: New insights into the pathology of inherited cardiomyopathy. Heart 91:257–264, 2005
23. Richardson P, McKenna W, Bristow M, et al: Report of the 1995 World Health Organization/International Society and Federation of Cardiology Task Force on the definition and classification of cardiomyopathies. Circulation 93:841–842, 1996
24. Tarolli KA: Left ventricular systolic dysfunction and nonischemic cardiomyopathy. Crit Care Nurse 26:3–15, 2003
25. Epstein AE, DiMarco JP, Ellenbogen KA, et al: ACC/HA/HRS 2008 Guideline for Device-Based Therapy of Cardiac Rhythm Abnormalities: Executive Summary. Retrieved November 14, 2010, from http://www .content.onlinejacc.org
26. Ho CY, Seidman CE: A contemporary approach to hypertrophic cardiomyopathy. Circulation 113:858–862, 2006
27. Maron BJ, Chaitman BR, Ackerman MJ, et al: Recommendations for physical activity and recreational sports participation for young patients with genetic cardiovascular diseases. Circulation 109:2807–2816, 2004
28. Hirsch AT, Haskal ZJ, Hertzer NR, et al: ACC/AHA 2005 Practice guidelines for the management of patients with peripheral arterial disease (lower extremity, renal, mesenteric, and abdominal aortic): A collaborative report from the American Association for Vascular Surgery/Society for Vascular Surgery, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, Society of Interventional Radiology, and the ACC/AHA Task Force on Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Peripheral Arterial Disease). Circulation 113:e463–e654, 2006. Retrieved November 14, 2010 from http://www. americanheart.org
29. Creager MA, Libby P: Peripheral arterial diseases. In Libby P, Bonow RO, Mann DL, et al (eds): Braunwald's Heart Disease, 8th ed. Philadelphia, PA: Elsevier Saunders, 2008, pp 1491–1511
30. Hankey GJ, Norman PE, Eikelboom JW: Medical treatment of peripheral arterial disease. JAMA 295:547–553, 2006
31. Olson KWP, Treat-Jacobson D: Symptoms of peripheral arterial disease: A critical review. J Vasc Nurs 22:72–77, 2004
32. Munro N: Hematopoiesis, coagulation, and bleeding. In Woods SL, Sivarajan Froelicher ES, Motzer SA, et al (eds): Cardiac Nursing, 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2005, pp 150–172
33. Geerts WH, Pineo GF, Bergqvist D, et al: Prevention of venous thromboembolism: American College of Chest Physicians Evidenced-Based Clinical Practice Guidelines (8th Edition). Chest 133:381S–453S, 2008
34. Bates S, Ginsberg JS: Treatment of deep-vein thrombosis. N Engl J Med 351:268–277, 2004
35. Bockenstedt P: D-Dimer in venous thromboembolism. N Engl J Med 349:1203–1204, 2003
36. Sieggreen M: Venous disorders: Overview of current practice. J Vasc Nurs 23:33–35, 2005
37. Isselbacher EM: Diseases of the aorta. In Libby P, Bonow RO, Mann DL, et al (eds): Braunwald's Heart Disease, 8th ed. Philadelphia, PA: Elsevier Saunders, 2008, pp 1457–1487
38. Elefteriades JA, Olin JW, Halperin JL: Diseases of the aorta. In Fuster V, O'Rourke RA, Walsh R, et al, (eds): Hurst's The Heart, 12th ed. New York, NY: McGraw-Hill, 2007, pp 2305–2328
39. Isselbacher EM: Thoracic and abdominal aortic aneurysms. Circulation 111:816–828, 2005
40. Brewster DC, Cronenwett JL, Hallett JW Jr, et al: Guidelines for the treatment of abdominal aortic aneurysms: Report of a subcommittee of the Joint Council of the American Association for Vascular Surgery and Society for Vascular Surgery. J Vasc Surg 37:1106–1117, 2003
41. Katzen BT, Dake MD, MacLean AA, et al: Endovascular repair of abdominal and thoracic aortic aneurysms. Circulation 112:1663–1675, 2005
42. Seventh Report of the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7). Retrieved November 14, 2010, from http://www.nhlbi.nih.gov/guidelines/hypertension
43. Kaplan NM, Victor RG: Systemic hypertension: Mechanism and diagnosis. In Libby P, Bonow RO, Mann DL, et al (eds): Braunwald's Heart Disease, 8th ed. Philadelphia, PA: Elsevier Saunders, 2008, pp 1027–1046
44. Flanigan JS, Vitberg D: Hypertensive emergency and severe hypertension: What to treat, who to treat, and how to treat. Med Clin North Am 90:439–451, 2006
45. Kaplan NM: Systemic hypertension: Therapy. In Libby P, Bonow RO, Mann DL, et al (eds): Braunwald's Heart Disease, 8th ed. Philadelphia, PA: Elsevier Saunders, 2008, pp 1049–1068
46. Cunningham S: Hypertension. In Woods SL, Sivarajan Froelicher ES, Motzer SA, et al (eds): Cardiac Nursing, 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2005, pp 856–896

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

20 Heart Failure

Kay Blum

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Define heart failure.
2. Describe the classification systems used to define heart failure.
3. Explain the physiological basis for the clinical manifestations of heart failure.
4. Describe expected clinical assessment findings for patients with heart failure.
5. Explain the standard pharmacological therapies for chronic heart failure and acute exacerbation of chronic heart failure, and their rationale.
6. Describe the nonpharmacological therapies for management of heart failure.
7. Define expected outcomes for therapeutic management of patients with heart failure.
8. Formulate a teaching plan for patients and families regarding heart failure.

Approximately 5.8 million Americans live with heart failure. About 550,000 new people receive the diagnosis of heart failure each year. The incidence of heart failure approaches 10 per 1,000 population after age 65 years, and 75% of patients have antecedent hypertension. Incidence and prevalence statistics indicate that heart failure is a common occurrence in certain patient populations, most notably elderly patients and patients with a history of hypertension, myocardial infarction (MI), or both. Although other cardiovascular mortality and morbidity statistics have decreased, the incidence of new-onset heart failure has continued to increase.[1,2] An estimated 1,106,000 patients were discharged with heart failure in 2006.

Heart failure is a common diagnosis in the intensive care unit (ICU) because the onset is sudden. An acute MI or an acute exacerbation of chronic heart failure is often life threatening. Sudden cardiac death occurs six to nine times as often in patients with heart failure as it does in the general population. Overall, the 5-year mortality rate for heart failure is 59% for men and 45% for women. Hospitalization is associated with high financial costs; for 2010, the direct and indirect costs were estimated to be $39.2 billion.[1,3] In addition, the physical and emotional burdens of inpatient care are great both for patients and their families.

Management of patients with heart failure requires a collaborative effort on the part of physicians, nurses, pharmacologists, dietitians, and other allied health professionals. The care of patients with heart failure extends across all parts of the health care system. Patients with heart failure may be located in home care, ambulatory care, acute care, critical care, and rehabilitation facilities. As patients take charge of their own disease management, the home serves as a critical location. As emphasis on self-care increases, more and more disease management programs are partnering with patients at home to prevent hospitalization.

▲ Definition

Heart failure is a clinical syndrome characterized by shortness of breath, dyspnea on exertion (DOE), paroxysmal nocturnal dyspnea (PND), orthopnea, and peripheral or pulmonary edema. Not all patients have all these clinical indicators. Heart failure is a general term used to describe the general clinical syndrome regardless of the kind of heart failure or the etiology that produces the symptoms. Congestive heart failure is so named because the interruption in circulation related to failure of the heart to function normally leads to congestion in the vascular beds of the lungs and peripheral tissues, resulting in respiratory symptoms and peripheral edema. The revised guidelines recently published by a joint American College of Cardiology (ACC) and American Heart Association (AHA) task force use the preferred term *heart failure* rather than congestive heart failure because patients with chronic heart failure rarely demonstrate the rales and alveolar edema associated with congestion.[2] For this reason, it

is important to look at the way heart failure is classified because the pathophysiology and etiology are keys to appropriate management.

▲ Classification

Heart failure is more difficult to understand when signs and symptoms are common to more than one type of failure and when types of heart failure are used interchangeably. Several categories are used to describe and classify heart failure. Using these categories to organize information about heart failure and for discussion of any individual patient case makes diagnosis, management, and outcome evaluation clearer.

Acute Versus Chronic

The terms *acute* and *chronic* describe both the onset of symptoms of heart failure and the intensity of symptoms. Heart failure of acute onset refers to the sudden appearance of symptoms, usually over days or hours. Acute symptoms have progressed to a point at which immediate or emergency intervention is necessary to save the patient's life. Heart failure of chronic onset refers to the development of symptoms over months to years. Chronic symptoms represent the baseline condition, the limitations the patient lives with on a daily basis. If the cause of the acute onset or the acute symptoms is not reversible, then the heart failure may become chronic. For example, a patient who has an acute MI with severe damage to the left ventricle has acute heart failure with pulmonary edema, causing lasting damage to the left ventricle. As a result, the patient has poor contractility (and therefore DOE) after the MI has resolved. The patient's acute onset of heart failure has left him or her with chronic symptoms. Chronic heart failure does not disappear when the symptoms are controlled or absent. People with heart failure demonstrate various levels of compensation, meaning that they have enough reserve to "compensate" for the loss of function and they appear to be asymptomatic, usually at rest. The lack of symptoms, or compensation, should not be mistaken for absence of disease. Like most chronic conditions, heart failure is characterized by relatively stable periods interrupted by episodes of acute decompensation. Acute decompensation is often life threatening and frequently requires critical care. A common cause of acute decompensation is inadequate treatment for chronic heart failure. The following discussion will focus on evidence-based care for chronic heart failure and then diagnosis and management of acute decompensated heart failure (ADHF).

Left-Sided Heart Failure Versus Right-Sided Heart Failure

Left-Sided Heart Failure

Left-sided heart failure refers to failure of the left ventricle to fill or empty properly. This leads to increased pressures inside the ventricle and congestion in the pulmonary vascular system. Left-sided heart failure may be further classified into systolic and diastolic dysfunction.

SYSTOLIC DYSFUNCTION (HEART FAILURE WITH REDUCED LEFT VENTRICULAR FUNCTION). Systolic dysfunction is defined as an ejection fraction of less than 40% and is caused by a decrease in contractility. Left ventricular function is estimated by ejection fraction, or the percentage of the left ventricular end-diastolic volume (LVEDV) that is ejected from the ventricle in one cycle. If the LVEDV is 100 mL and the stroke volume is 60 mL, the ejection fraction is 60%. Normal ejection fraction is 50% to 70%. The ventricle does not empty adequately because of poor pumping, and the result is decreased cardiac output.

DIASTOLIC DYSFUNCTION (HEART FAILURE WITH PRESERVED LEFT VENTRICULAR FUNCTION). Diastolic dysfunction is less well defined and more difficult to measure. Diastolic dysfunction is caused by impaired relaxation and filling. Left ventricular filling, a complex process that occurs during diastole, is a combination of passive filling and atrial contraction. Pumping is normal or even increased, with an ejection fraction as high as 80% at times. If the ventricle is stiff and poorly compliant (due to aging, uncontrolled hypertension, or volume overload), relaxation is slow or incomplete. If the heart rate is fast, diastole is short, or if the patient has atrial fibrillation, there is no organized atrial contraction. These mechanisms all reduce filling of the ventricle and contribute to diastolic dysfunction, thereby decreasing cardiac output.

Right-Sided Heart Failure

Right-sided heart failure refers to failure of the right ventricle to pump adequately. The most common cause of right-sided heart failure is left-sided heart failure, but right-sided heart failure can exist in the presence of a perfectly normal left ventricle and does not lead to left-sided heart failure. Right-sided heart failure can also result from pulmonary disease and primary pulmonary artery hypertension (cor pulmonale). Pulmonary embolus is a common cause of acute right-sided heart failure.

Classification Systems

New York Heart Association Functional Classification

The New York Heart Association (NYHA) Functional Classification is a measure of how much the symptoms of heart failure limit the activities of patients (Box 20-1). Although ejection fraction is used to define left ventricular

BOX 20-1 New York Heart Association (NYHA) Functional Classification of Heart Failure

Class I: No limitation of physical activity. Ordinary physical activity does not cause undue fatigue or dyspnea.
Class II: Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue or dyspnea.
Class III: Marked limitation of physical activity without symptoms. Symptoms are present even at rest. If any physical activity is undertaken, symptoms are increased.
Class IV: Unable to carry on any physical activity without symptoms. Symptoms are present even at rest. If any physical activity is undertaken, symptoms are increased.

BOX 20-2 American College of Cardiology (ACC)/American Heart Association (AHA) Guidelines for Stages of Heart Failure*

A Patients at high risk for heart failure because of the presence of conditions that are strongly associated with the development of heart failure. Such patients have no identified structural or functional abnormalities of the pericardium, myocardium, or cardiac valves and have never shown signs or symptoms of heart failure.
B Patients who have structural heart disease that is strongly associated with the development of heart failure but who have never shown signs or symptoms of heart failure.
C Patients who have current or prior symptoms of heart failure associated with underlying structural heart disease.
D Patients with advanced structural heart disease and marked symptoms of heart failure at rest despite maximal medical therapy and who require specialized interventions.

*New York Heart Association classification is applicable only to stages C and D.

function, ejection fraction is poorly correlated with the patient's functional capacity or prognosis.[4]

American College of Cardiology/American Heart Association Guidelines

The ACC/AHA Guidelines outline four stages of heart failure that are useful for organizing the prevention, diagnosis, management, and prognosis for patients with heart failure[2] (Box 20-2). These stages are not meant to replace the NYHA functional classification but rather to augment it. Only stages C and D are applicable to the NYHA functional classification system. See also Evidence-Based Practice Highlight 20-1.

▲ Factors That Determine Cardiac Output

The underlying result of all types of heart failure is insufficient cardiac output. That is, the volume of blood pumped by the heart in 1 minute is inadequate. Some patients may have a normal cardiac output at rest, but they do not have the reserve function to increase cardiac output to meet the increased demands of exercise, hypoxemia, or anemia. Therefore, it is important to understand the physiological basis of cardiac output and review the mechanisms of compensation of decreased cardiac output. (See Chapter 16 for a review of cardiovascular physiology.)

Oxygen Demand

The required cardiac output is determined by the body's metabolic demand for oxygen. At rest, the body needs sufficient oxygen to burn calories to support cellular function, as measured by basal metabolic rate. Oxygen delivery to the tissues depends on arterial oxygen content (CaO_2) and cardiac output. CaO_2, a combination of arterial oxygen saturation (SaO_2) and hemoglobin (Hgb), is constant in healthy people. Any factor that increases metabolic demand for oxygen, such as exercise, fever, hyperthyroidism, or trauma, increases cardiac output. If CaO_2 is decreased, as it is in hypoxemia or anemia, then cardiac output increases to ensure sufficient oxygen to meet the metabolic demand. Exercise or fever in a patient with anemia puts a tremendous burden on the heart to supply sufficient oxygen to meet the metabolic demands.

A person with a healthy heart has sufficient reserve to meet this increased metabolic demand and increase cardiac output. At best, a patient with myocardial ischemia, cardiomyopathy, valvular disease, dysrhythmia, or lung disease

EVIDENCE-BASED PRACTICE HIGHLIGHT 20-1

Management of Heart Failure

It is recommended that heart failure disease management programs include the following components, based on patient characteristics and needs (strength of evidence = B):
- Comprehensive education and counseling individualized to patient needs
- Promotion of self-care, including self-adjustment of diuretic therapy in appropriate patients (or with family member or care giver assistance)
- Emphasis on behavioral strategies to increase adherence
- Vigilant follow-up after hospital discharge or after periods of instability
- Optimization of medical therapy
- Increased access to providers
- Early attention to signs and symptoms of fluid overload
- Assistance with social and financial concerns

It is recommended that heart failure disease management include integration and coordination of care between the physician and heart failure care specialists and with other agencies, such as home health and cardiac rehabilitation (strength of evidence = C).

It is recommended that patients in a heart failure disease management program be followed until they or a family member or care giver demonstrates independence in following the prescribed treatment plan, adequate or improved adherence to treatment guidelines, improved functional capacity, and symptom stability. Higher-risk patients with more advanced heart failure may need to be followed permanently. Patients who experience increasing episodes of exacerbation or who demonstrate instability after discharge from a program should be referred again to the service (strength of evidence = B).

Note: Rating scheme for the strength of the evidence: A, randomized, controlled, clinical trials (may be assigned based on results of a single trial); B, cohort and case-control studies (post hoc, subgroup analysis, and meta-analysis; prospective observational studies or registries); C, expert opinion (observational studies—epidemiological findings; safety reporting from large-scale use in practice).

Excerpted from Heart Failure Society of America 2010 Guidelines, Executive Summary, Section 8, Disease management in heart failure. J Card Fail 16(6)492–494, 2010.

may not be able to meet the metabolic demand for oxygen associated with exercise. At worst, the patient with one or more of these problems may not be able to meet the basal metabolic demand for oxygen and becomes symptomatic, even at rest.

Mechanical Factors and Heart Rate

Cardiac output equals stroke volume multiplied by heart rate.

This relationship between stroke volume, heart rate, and cardiac output is critical to understanding why heart failure may be present long before it produces the symptoms that cause patients to seek help. Preload, afterload, and the contractile force of the left ventricle determine stroke volume (see Chapter 16). These three components are in a constant and dynamic relationship. A decrease in one or more components is compensated by an increase in the others designed to maintain a constant stroke volume at rest. Catecholamines and other neurohormones contribute to the complex balance that preserves stroke volume over a wide range of supply and demand for oxygen at the tissue level. Relatively minor increases in stroke volume are possible because of neurohormonal regulation of reserve fluid volume stored in the liver and venous system until needed. The largest increase in cardiac output comes, not from increased stroke volume, but through increases in heart rate. Cardiac reserve is the ability to significantly increase oxygen delivery in response to increased demand. Reserve is meant to meet demand that exceeds that of rest. Patients with heart failure need their reserve just to function at rest. When that reserve is exhausted, they have symptoms even at rest. The increase in catecholamines increases the risk of dysrhythmias such as ventricular tachycardia and sudden death.

Heart Rate

As stated earlier, cardiac output equals stroke volume multiplied by heart rate. Therefore, just doubling the heart rate doubles cardiac output without changing stroke volume. The immediate response to a decrease in stroke volume, a decrease in arterial oxygen content, or an increase in metabolic demand is an increase in heart rate. However, at a certain point, increasing the heart rate can actually decrease the stroke volume and, therefore, cardiac output as well. Because the ventricle fills during diastole, preload becomes compromised at higher heart rates because of the shortened diastolic filling time. A decrease in preload compromises contractility.

The physiological role of heart rate in the regulation of cardiac output involves more than just the absolute rate. Cardiac rhythm is important. As previously stated, rapid tachycardia can compromise stroke volume. Any rhythm that does not include a rhythmic atrial contraction, such as atrial fibrillation and flutter, junctional rhythms, ventricular rhythms, and ventricular pacing, can compromise filling and therefore stroke volume and cardiac output. A heart rate that is too slow, such as that which occurs in third-degree atrioventricular (AV) block or sick sinus syndrome, may compromise cardiac output, not by decreasing stroke volume, but by decreasing overall cardiac output.

Neurohormonal Mechanisms

Metabolic demand for oxygen is the primary factor in the regulation of cardiac output, and the mechanical relationships between loading and contractility provide a means to regulate it. Neurohormones are the messengers that initiate, coordinate, and mediate the complex processes that meet the dynamic need for cardiac output[5–7] (Fig. 20-1).

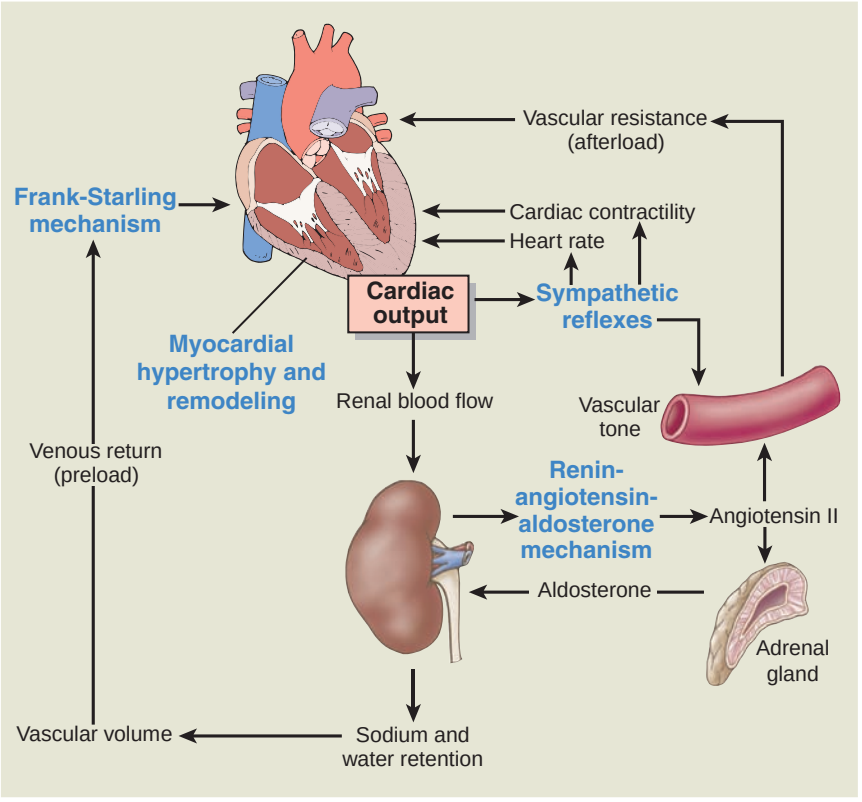


FIGURE 20-1 ▲ Compensatory mechanisms in heart failure. The Frank-Starling mechanism, sympathetic reflexes, renin–angiotensin–aldosterone mechanism, and myocardial hypertrophy work to maintain cardiac output in the failing heart. (From Porth CM: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 613.)

Catecholamines

Catecholamines are released from the adrenal medulla as part of the primitive "fight or flight" response to any stressor. Stressors can be physiological or psychological. Epinephrine and norepinephrine as well as cortical hormones, such as cortisol and aldosterone, are released.

Epinephrine and norepinephrine are the key catecholamines involved in the regulation of the cardiovascular system. The heart and blood vessels contain α- and β-adrenergic receptors that bind with these hormones to support cardiac output and blood pressure. Norepinephrine has almost exclusively α-adrenergic properties that increase vascular resistance and therefore blood pressure. Epinephrine has both α- and β-adrenergic properties. β-Agonist effects include increased heart rate, increased contractility, and vasodilation. The net effect of epinephrine is increased cardiac output; it increases stroke volume by increasing contractility and decreasing afterload. The increases in heart rate and stroke volume together produce a greater increase in cardiac output than either would alone.

Renin–Angiotensin–Aldosterone System

One of the most important mechanisms of blood pressure control in relation to heart failure is the renin–angiotensin–aldosterone system. Fluids, such as blood, flow down pressure gradients (ie, from higher pressure to lower pressure). Consequently, pressure in the aorta is higher than pressures distal to it, including the arteriolar and capillary levels. Arterial blood pressure is critical to the delivery of blood (and therefore oxygen) to the cells to support cellular function. Several mechanisms maintain normal blood pressure across variable body fluid volumes, in different positions (sitting or standing vs. supine), and with cardiac output demands.

Renin is an enzyme produced in the kidney in response to even small decreases in blood pressure. Renin has a direct effect on the kidney, causing increased reabsorption of salt and water. Much of the renin travels to the lung to act enzymatically on angiotensinogen to form angiotensin I. In the presence of angiotensin-converting enzyme (ACE) in the lung, angiotensin I is converted to angiotensin II.

A powerful vasoconstrictor, angiotensin II increases arterial resistance quickly and profoundly, providing immediate support for blood pressure and maintaining perfusion in the short term until a longer-term strategy can be implemented. Although angiotensin II has a much more modest effect on venous resistance, it does increase venous resistance and therefore venous return. Angiotensin II also stimulates the adrenal cortex to release aldosterone. Aldosterone then acts on the kidney to increase salt reabsorption in the distal tubule, and this salt increases water reabsorption in the kidney, resulting in increased circulating volume. Increased circulating volume is the longer-term strategy. The renin–angiotensin–aldosterone system initiates a process that assumes any decrease in blood pressure is a volume loss (eg, hemorrhage), and the long-term strategy is to replace that loss.

▲ Pathophysiology

The physiological principles discussed in the previous section form the basis for understanding the patient's signs, symptoms, responses, and compensation for the disease process as well as the basis for management strategies. Heart failure has many causes (Table 20-1).

Table 20-1 Causes of Heart Failure

Impaired Cardiac Function	Excess Work Demands
Myocardial Disease	**Increased Pressure Work**
Cardiomyopathies Myocarditis Coronary insufficiency Myocardial infarction	Systemic hypertension Pulmonary hypertension Coarctation of the aorta
Valvular Heart Disease	**Increased Volume Work**
Stenotic valvular disease Regurgitant valvular disease	Arteriovenous shunt Excessive administration of intravenous fluids
Congenital Heart Defects	**Increased Perfusion Work**
	Thyrotoxicosis Anemia
Constrictive Pericarditis	

From Porth CM: Pathophysiology: Concepts of Altered Health States, 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2005, p 608.

Cardiomyopathy

The distinguishing pathophysiological factor in heart failure is a cardiomyopathy, but cardiomyopathy is not synonymous with heart failure.[2] Literally, cardiomyopathy is a progressive pathological process in the heart muscle. Cardiomyopathy may be congenital or acquired. Hypertrophic, nonobstructive cardiomyopathy and dilated cardiomyopathy are the two most common forms. Hypertrophic cardiomyopathy is an increase in muscle mass in the ventricle resulting in a measurable increase in the thickness of the ventricular wall. Hypertrophy is a response to a prolonged increase in resistance (afterload). Dilated cardiomyopathy is an increase in the size of the ventricular chamber without an increase in wall size and is a response to decreased contractility. For a more detailed discussion of cardiomyopathies, see Chapter 19; see also Figure 20-2.

Dysrhythmia

Heart failure is commonly associated with dysrhythmias, both atrial and ventricular. The structural and metabolic changes that occur in heart failure frequently lead to dysrhythmia, and the dysrhythmia itself may lead to heart failure.

Atrial Dysrhythmias

Atrial tachycardias may cause heart failure in two ways. First, the shortened diastole leads to decreased filling and may cause or aggravate diastolic dysfunction, resulting in decreased cardiac output and the symptoms of heart failure. When the tachycardia is caused by atrial fibrillation, the loss of atrial kick increases the impact of the atrial dysrhythmia on left ventricular dysfunction. In one study, systolic dysfunction developed in 11% of patients with atrial fibrillation, and 6% of the patients died.[7]

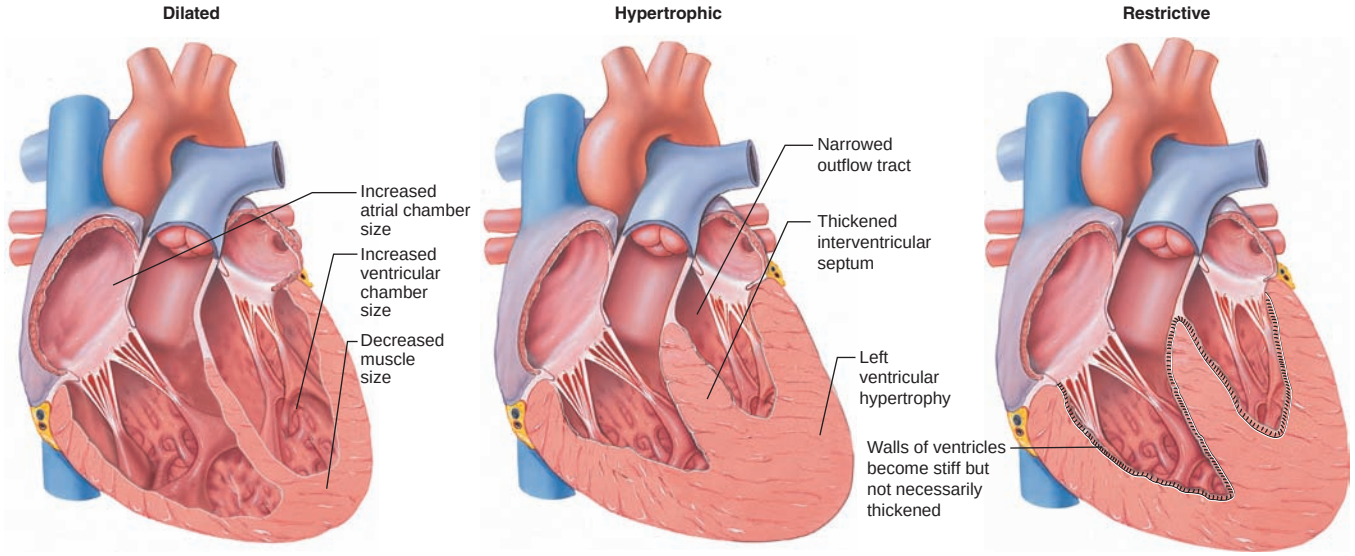


FIGURE 20-2 ▲ Patterns of ventricular hypertrophy and remodeling in various forms of cardiomyopathy. (Anatomical Chart Company: Atlas of Pathophysiology. Springhouse, PA: Springhouse, 2010, p. 45.)

Atrial fibrillation is a significant problem in patients with heart failure. The most common sustained dysrhythmia, atrial fibrillation, affects 2.2 million Americans. The median age for atrial fibrillation is 75 years; it affects 8.8% of Americans older than 80 years. The risk for stroke is increased five times in patients who have this dysrhythmia.[8] The incidence of both atrial fibrillation and heart failure increases with age, increasing the likelihood that patients with heart failure will also have atrial fibrillation at some time.

Ventricular Dysrhythmias

Ventricular dysrhythmias, in particular premature ventricular beats and nonsustained ventricular tachycardia (NSVT), are common in patients with dilated cardiomyopathy, whether ischemic or nonischemic. Prior to the common use of implantable cardioverter–defibrillators (ICDs), sudden death from ventricular dysrhythmia or bradycardia accounted for 30% to 40% of deaths associated with heart failure.[9–11] Since the widespread use of ICDs, the death rate from sudden cardiac death has decreased to 12.7%.[10] The presence of premature ventricular beats or even NSVT has not been shown to reliably predict risk for sudden death for any particular patient. However, the presence of these dysrhythmias does appear to reliably reflect a globally impaired myocardium.

Several mechanisms play a role in the development of ventricular dysrhythmias. The low ejection fraction leads to stretch of the myocardial fibers, thus increasing excitability. Excitability is also affected by the presence of increased catecholamines; increased sympathetic tone; and, on occasion, antiarrhythmic drugs. Activation of the renin–angiotensin–aldosterone system contributes to the overall environment that generates dysrhythmia. Ischemia leads to failure of the sodium–potassium pump, and the loss of potassium from the cell increases the risk for premature ventricular beats. Scar tissue from previous infarctions and surgery can stimulate dysrhythmia. Electrolyte shifts involving potassium, calcium, and magnesium are often associated with prolonged or aggressive diuretic use. Lung disease such as emphysema or chronic bronchitis is often comorbid with heart failure, and the lung disease may lead to hypoxemia, which contributes to the genesis of ventricular dysrhythmias. The traditional sources of ventricular dysrhythmia that occur in patients without heart failure, such as reentry, enhanced automaticity, and delayed after-potentials, may also be involved.

Acute Decompensated Heart Failure

Chronic disease is characterized by variable periods of relative stability or compensation interrupted by periods of exacerbation or decompensation. Patients with chronic heart failure may live from day to day with no symptoms of heart failure or well-controlled symptoms. However, chronic heart failure may become acutely worse, resulting in an increase in symptoms and limitations associated with left ventricular dysfunction. Several factors may lead to an exacerbation or ADHF.[12] Current therapy for ADHF is not guideline driven, and there is poor agreement on the appropriate course. There is agreement that most of the therapies available to treat ADHF, such as inotropes and loop diuretics, hasten the course of heart failure by destroying myocytes and stimulating the renin–angiotensin system. Unfortunately, they are often the only tools available. Consequently, one of the fastest growing areas of clinical research is directed toward understanding ADHF and developing better therapies to treat it based on accumulated evidence.[13]

Alcohol, anemia, hypoxemia, hypertension, ischemia, and worsening left ventricular function may trigger acute decompensation. Any factor that increases oxygen demand, and therefore demand for increased cardiac output beyond the ability of the ventricle to function (eg, hypertension, tachycardia, anemia, exercise), causes an exacerbation. Similarly, any factor that depresses the function of the already compromised ventricle leads to exacerbation (eg, alcohol, drugs that exert a negative inotropic effect, such as calcium channel

blockers and β-blockers). As the ventricle is called on to work harder, it works less efficiently, and the left ventricular end-diastolic pressure increases, leading to increased pulmonary artery pressures. The increased pulmonary artery pressures, in turn, lead to orthopnea, possibly pulmonary edema, elevated venous pressures, liver congestion, lower extremity edema, and PND. Patients may also present with lower blood pressures, more rapid heart rates, and prerenal azotemia. Potentially, the acute decompensation is reversible if treated quickly and aggressively.

▲ Assessment

Heart failure has long been defined by the presence of pulmonary edema characterized by bibasilar rales or crackles. At one time, the absence of crackles ruled out heart failure. However, chronic heart failure is a persistent, not episodic, condition, and it rarely includes pulmonary edema and crackles even in early decompensation. History, physical examination, diagnostic procedures, and hemodynamic evaluation all contribute to diagnosing heart failure, perhaps determining its cause, and evaluating the success of therapy.

History

The symptoms of heart failure are nonspecific (ie, they are common to many disease processes). The health history is used to put the symptoms into a context that may lead to their interpretation as heart failure and not pulmonary disease, deconditioning, or other conditions that produce shortness of breath, DOE, fatigue, and swelling of the lower extremities. History alone does not confirm the diagnosis, but it helps determine follow-up examinations and diagnostic tests that may be appropriate.

Onset

The basic question is, "When did the symptoms start?" The answer to this question helps categorize the condition as acute or chronic. Most patients indicate an acute onset of 2 weeks or fewer if this is their first visit for their symptoms. If they are asked additional questions about their activity tolerance for the past year or so, patients with chronic heart failure note a gradual slowing of activity to match the amount of energy available or to control symptoms. The recent identification of symptoms indicates that the patient is now aware of them or they have become unbearable. Acuity is important because reversible ischemia is a potentially life-threatening etiology that may present acutely. When identified and treated, chronic heart failure can be avoided, and perhaps a patient's life may be saved.

Duration

It is important to know whether the symptoms are persistent and independent of activity or come and go with activity, change of position, food ingestion, or other events. This helps differentiate between heart failure and other conditions that can cause the same symptoms. Heart failure symptoms typically worsen with activity and improve with rest. Cough and shortness of breath may increase when lying down and improve with sitting up. Hiatal hernia and gastric reflux may produce shortness of breath, chest pain, and cough but typically occur after eating and more often in the evening. Lung disease or sleep apnea may also cause the shortness of breath that occurs at rest or awakens the patient at night, characteristic of heart failure.

Severity

Severity of symptoms is important to determine because it is the basis for establishing functional class (see Box 20-1, p. 392). Severity of symptoms is also an important standard for evaluating the success of therapy. A major goal of therapy is symptomatic improvement or, if possible, elimination of symptoms. The evaluation of severity requires that patients be asked certain questions about their symptoms (Table 20-2).

Comorbid Diseases

Many patients with heart failure have comorbid disorders that contribute to or aggravate their heart failure. The most common of these diseases are coronary artery disease (CAD), hypertension, diabetes mellitus, chronic obstructive pulmonary disease (COPD), and chronic renal insufficiency. Worsening of one or more comorbid diseases may lead to an exacerbation of stable chronic heart failure. In the case of CAD, hypertension, and diabetes, heart failure may be the long-term result of complications of these disease processes. Identification and tight control of these comorbid diseases contribute to the control and treatment of the symptoms of heart failure.

Medications

It is very important to obtain a complete list of the patient's medications and dosages. The list should include prescription and nonprescription medications. In cases of new-onset heart failure, even old medications may contribute to the severity of symptoms. For example, patients who have been treated with a calcium channel blocker for hypertension and now present with a decreased ejection fraction and heart failure may improve when the medication is changed and does not depress myocardial function. Other medications may contribute to heart failure. Patients taking over-the-counter medications, such as nonsteroidal anti-inflammatory drugs (NSAIDs), may present with worsening heart failure and renal function because of the effect of the NSAIDs on renal blood flow. NSAIDs block the effect of prostaglandins, which the body secretes to maintain renal blood flow in the context of decreased cardiac output. Cold medicines with systemic decongestants can lead to increased blood pressure that precipitates worsening symptoms of heart failure.

Psychosocial Factors

Noncardiac factors may also affect patients with heart failure. Because many affected patients are elderly, they may have problems remembering to fill prescriptions or take medications. Financial hardships may force some patients to choose between buying medication and buying food. Transportation may depend on friends or family who may be unreliable. Housekeeping may be difficult or impossible because of

Table 20-2 Assessment of Severity of Heart Failure

Symptom	Measure(s)	Questions
Orthopnea	Number of pillows patient sleeps on regularly	How many pillows do you sleep on at night? If more than one, is it for comfort or because you cannot breathe with one or two?
Dyspnea on exertion	Number of blocks patient can walk without stopping to rest or catch breath Number of flights of stairs patient can climb without stopping to rest or catch breath Number of times patient must rest while doing activities of daily living such as toileting or minor housework	How many blocks and flights of stairs can you walk without stopping to rest or catch your breath? Do you stop because you cannot go further or because you want to avoid getting short of breath? For patients who are limited by peripheral vascular disease or orthopedic problems: Do you stop because you cannot breathe or because of pain? Which comes first?
Paroxysmal nocturnal dyspnea	Average number of times per night or week	After you go to bed, do you ever have to sit up suddenly to catch your breath? How much time passes before you can breathe normally? Do you need to do anything besides sit up to relieve the shortness of breath?
Dizziness or lightheadedness	Presence or absence (of real concern when symptom occurs when the patient is standing and persists or occurs with activity)	Do you ever become dizzy or lightheaded? What are you doing when this occurs?
Chest pain or pressure*	Presence or absence	Do you have chest pain or pressure? Do you become short of breath with the chest pain or pressure? Which comes first, the pain or the shortness of breath?†

*Chest pain should be fully investigated to determine whether active ischemia is present. This is especially true in patients who are presenting for the first time for evaluation of symptoms of heart failure. Once ischemia has been ruled out, patients may still have chest pain, and it should be evaluated by using these assessment questions.

†Chest pain that comes after shortness of breath is often caused by the heart failure.

fatigue and shortness of breath. Patients living on the second or third floor of buildings without elevators may become isolated and lonely. Depression is not uncommon; the exact incidence is not known. Ongoing family dysfunction and family members who depend on the patient for care and financial support (eg, grandchildren, dependent adult children, spouses or partners) add a burden to the patient's management. Illiteracy is still prevalent; even patients who can read may not comprehend medication instructions correctly. Some patients may skip diuretic doses when visiting places where they are uncertain about access to bathroom facilities; they may not take the diuretic when they return home.

Although many of these factors are significant, they may not be obvious until the patient has visited the same health care facility many times. Early case management and skillful discharge planning depend on recognizing these problems before they lead to repeated hospitalizations and increased mortality.

Substance Abuse

Alcohol and drug (eg, cocaine) use is also important because it may contribute to the development and progression of heart failure. If alcohol use is the cause of cardiomyopathy, abstinence may lead to complete reversal. Patients who have substance abuse problems often forget to buy or take medication. They may be homeless, which increases the likelihood that they will not return to the health care facility for regular follow-up.

Physical Examination

The physical findings in heart failure differ depending on whether the patient has (1) acute or chronic heart failure or (2) systolic or diastolic dysfunction. When the physiological changes of left ventricular dysfunction occur over a long period, the body adapts and compensates. Consequently, many of the findings on physical examination are normal, despite moderate to severe disease. However, when the problem occurs acutely, there is no time for compensation or adaptation, and the symptoms and consequences are severe. Patients who have chronic heart failure from systolic dysfunction and who have abnormal findings, have them persistently. Patients with diastolic dysfunction may have abnormal findings only during an exacerbation.

One or more of the following findings characterizes acute decompensation. The patient may be volume overloaded by 5 to 50 pounds over dry weight; dry weight is the patient's weight when he or she is euvolemic. Patient self-monitoring is often geared to maintenance of dry weight. In many cases, maintaining dry weight within 1 to 2 pounds can prevent decompensation. A second finding, often, is renal insufficiency characterized by an increase in both blood urea nitrogen (BUN) and creatinine levels, with a ratio of BUN to creatinine of greater than 20:1. The third finding is decreased cardiac output manifested by increased DOE and decreased exercise tolerance in general, often described as "fatigue."

Patients may also complain of increased orthopnea, PND, or both. Some patients have all of the findings, and it is not unusual for patients to be short of breath at rest (NYHA class IV) or demonstrate Cheyne-Stokes respirations.

General Findings

Patients with acute heart failure or acute decompensation of chronic heart failure appear ill; they are often breathing rapidly, looking anxious, and either sitting up straight or leaning forward and resting their arms on a table or their knees. Patients with stable, chronic heart failure may be quite comfortable but may have evidence of cachexia, muscle wasting, and thin skin.

Vital Signs

Patients with systolic dysfunction may have quite low, but asymptomatic, blood pressures (systolic, 80 to 99 mm Hg; diastolic, 40 to 49 mm Hg). Heart rates may be rapid (90 beats/min or more), or lower at rest. Patients with diastolic dysfunction may or may not be hypertensive.

Serial weights are very important in following fluid status. Daily weights, when performed properly on a calibrated scale, are more accurate estimates of fluid status than intake and output. Daily weights can be used to evaluate fluid status because 1 liter of water weighs 1 kg. Overnight fluctuations in weight are always related to water retention or diuresis.

Neck

Jugular venous pressure is an estimate of right heart filling pressures. When either the total-body fluid volume or the right atrial pressure increases, the jugular venous pressure increases, and the vein dilates. Jugular venous pressure is estimated by identifying the internal jugular vein and measuring the height of the pulse from the level of the clavicle in centimeters. The patient's head is elevated to 45 degrees. It is important not to use the external jugular vein, which often appears distended and prominent in patients with normal volume and pressure.

Lungs

It is necessary to determine the respiratory rate and observe the depth of respiration as well as the respiratory rhythm. It is not unusual for patients with severe NYHA class IV heart failure to have a Cheyne-Stokes respiratory pattern.[14] The heart failure may be chronic and persistently rated as class IV heart failure or may represent an acute exacerbation.

Results of chest auscultation may be completely normal. Because patients with increased pulmonary artery pressures have increased lymph drainage over time, fluid does not collect in the alveoli. Rales or crackles are sounds made by air bubbling through water in the alveoli, and if no water is present, the sounds are not audible. When pressures increase suddenly, water is forced into the alveoli by increased hydrostatic pressure. Consequently, in acute heart failure and acute decompensation, in which pulmonary edema is common, bibasilar crackles occur. Unilateral crackles or nondependent crackles are indicative of a pulmonary process, not heart failure. Pulmonary edema can cause wheezing that may be difficult to distinguish from reactive airway disease, such as asthma.

Heart

Progressions from left-sided heart failure to left-sided and right-sided heart failure or chronic elevations of pulmonary artery pressure often result in a visible, palpable right ventricular or pulmonary artery pulsation at the left sternal border. The point of maximal impulse may be extremely displaced. In advanced heart failure, the maximal impulse may be at the posterior axillary line and at the fifth or sixth intercostal space.

Figure 20-3 shows the areas of cardiac auscultation that are examined in a patient with heart failure. The first (S_1) and second (S_2) heart sounds are expected. The sudden appearance of a third heart sound (S_3) is a warning of impending or worsening heart failure. In chronic heart failure, S_3 is a common and chronic finding. A fourth heart sound (S_4) is common in patients with long-standing hypertension and is not considered ominous. However, in severe heart failure, all four heart sounds may be heard; this is known as a summation gallop.

When valvular disease is the cause of heart failure, a heart murmur associated with the diseased valve is heard. In patients with dilated cardiomyopathy, a mitral regurgitation murmur is commonly heard. This holosystolic murmur is best heard at the left sternal border or, in patients with very large hearts, at the apex. The mitral valve is usually structurally intact. The dilation of the left ventricle in chronic heart failure dilates the mitral annulus and prevents the close approximation of the valve leaflets. Consequently, blood regurgitates back across the mitral valve into the left atrium with each systole.

When a mitral regurgitation murmur develops acutely, as when there is damage to the papillary muscles that open and close the mitral valve, severe, acute heart failure results. The sudden appearance of a mitral regurgitation murmur in a patient with MI is a warning of impending heart failure. The disappearance of this murmur in a patient with severe systolic dysfunction suggests a worsening of the heart failure; the ventricle cannot pump enough to generate the turbulence necessary to make the sound of the murmur.

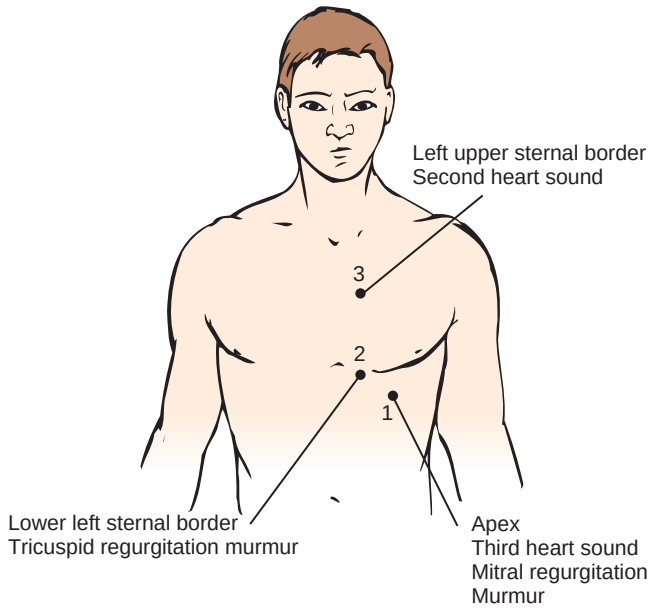


FIGURE 20-3 ▲ Cardiac auscultation in the patient with heart failure.

Tricuspid regurgitation develops in patients with right-sided heart failure alone or from left-sided heart failure for the same reasons as mitral regurgitation. This murmur is also a holosystolic murmur and is heard at the right sternal border. It may increase with inspiration. When both mitral regurgitation and tricuspid regurgitation murmurs are present, it may be impossible to distinguish between them.

Abdomen

It is necessary to palpate and percuss the abdomen to identify any ascites and the lower liver edge. High right atrial pressures that are translated into high venous pressures characterize right-sided heart failure, and the liver, which becomes a reservoir for the increased venous volume, increases in size (hepatomegaly) when congested. Once the liver becomes engorged, pressure increases in the portal vein and in the capillaries of the intestines. When the lymphatic system can no longer drain sufficient fluid to relieve the pressure, ascites develops. Ascites is the transudation or third spacing of fluid, and sometimes protein, into the abdominal cavity. In the absence of hepatomegaly and ascites, a congested liver may conceal significant fluid. Eliciting hepatojugular reflux may identify this concealed fluid. To assess hepatojugular reflux, it is necessary to observe the internal jugular vein while pressing on the liver. When the height of the pulse increases or the vein engorges, hepatojugular reflux is positive.

Extremities

The lower extremities are inspected for edema. The edema associated with heart failure is bilateral, dependent, and pitting. Unilateral or nonpitting edema is not related specifically to heart failure, and other causes, such as arterial insufficiency, myxedema, or lymphedema, should be suspected.

In the ambulatory patient, the edema can be assessed by pressing the skin over the tibia. Pitting here is referred to as pretibial edema. The edema is usually graduated and worse in the ankles than at the calf and is greater than at the thigh if the edema is present that high. In patients who are confined to bed, the edema is dependent posteriorly, and pretibial edema may be absent even in frank fluid overload. The patient must be assessed for pitting edema on the backs of the legs, the buttocks, and back. Occasionally, an ambulatory patient is so volume overloaded that presacral edema develops. To assess presacral edema and pitting, press the skin over the sacrum against the bone.

There are several schemes for describing the severity of pitting edema. None is superior to another; consistency is the most important factor. It is less important whether a series of pluses on a scale from 0 for no edema to 4+ for severe edema is based on the depth of the pit or the height of the edema on the lower extremity. When in doubt about the scale, a clear description of the depth of the pit and the level of the edema communicates the condition more effectively than a subjective number. A clear description allows for better continuity between clinicians and a better estimate of improvement. See Table 51-6 on page 1162 for the Pitting Edema Scale.

Long-standing venous stasis and the consequent edema produce skin color and texture changes. The skin becomes leathery and discolored and may be hard to assess. These changes always indicate that the edema is chronic and not acute. Acute increases in the chronic edema may also be hard to assess. Pressing the skin firmly to the side of the tibia instead of directly over it may be of some help. Figure 20-4 shows the physical assessment findings of a patient with ACC/AHA class D chronic heart failure.

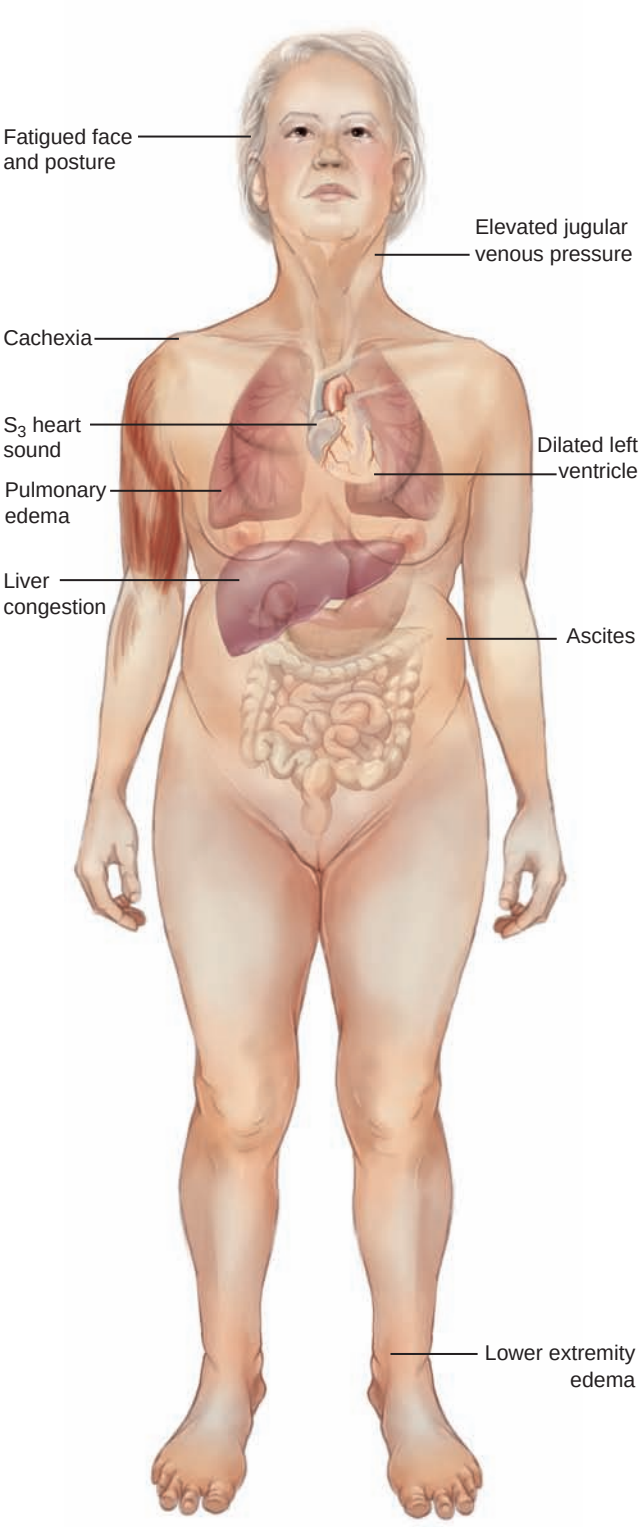


FIGURE 20-4 ▲ Physical examination findings for the person with ACC/AHA class D chronic heart failure.

Laboratory Studies

Laboratory studies are used to rule out some reversible causes of systolic dysfunction and to monitor the effects of management strategies. On initial evaluation of a patient presenting with new-onset heart failure, a battery of baseline laboratory studies is ordered (Table 20-3).

Patients receiving anticoagulation therapy with warfarin are also monitored regularly, using the international normalized ratio to adjust the dose. Before the initiation of amiodarone, patients have thyroid function and liver function tests performed to obtain baseline values, along with pulmonary function tests that include DLCO (diffusion capacity). These tests are repeated at least yearly and when any complications occur.

Brain natriuretic peptide (BNP) is a naturally occurring substance secreted by the ventricles when overfilled. It is elevated in proportion to increases in end-diastolic pressure, and levels may be greater than 1,000 pg/mL. Because the BNP level is also correlated with pulmonary artery occlusion pressure (PAOP), it is sometimes used as a marker of heart failure. Recent approval of laboratory assays for BNP and pro-BNP facilitate the use of BNP in the evaluation of patients with symptoms of heart failure. Patients with BNP levels greater than 80 pg/mL show evidence of elevated PAOP, confirming heart failure decompensation as the source of worsening symptoms.[15,16]

Although the relationship between BNP level and heart failure is clear, the appropriate use of BNP levels in heart failure management is less clear. One important use of BNP levels has been proposed: to distinguish between pulmonary-related and heart failure–related causes of dyspnea in the emergency department.[15] Many patients have both heart failure and lung disease, and the existence of a test that may distinguish between the two conditions as a cause of acute respiratory problems is a real advantage for individualizing and targeting treatment. In addition, BNP has been proposed as a marker for adequacy of treatment and for acute progression of heart failure, but the reliability of BNP for this use has not been established.[16]

Diagnostic Studies

Diagnostic studies are used to establish baseline values, identify potentially reversible etiologies, evaluate the effectiveness of treatment, and assess changes in condition. Several invasive and noninvasive tests are performed routinely when heart failure is suspected. Some tests are performed initially, when the symptoms of heart failure are first identified; some on a regular basis; and others only if indicated.

Electrocardiography

The electrocardiogram (ECG) is used to assess rate and rhythm, and it is also useful in diagnosing dysrhythmias, conduction defects, and MI. In addition, an ECG is often used to identify atrial enlargement and ventricular hypertrophy. However, in such cases, an echocardiogram is more helpful because it can quantify these structural changes.

ECGs are useful in identifying atrial fibrillation and ventricular dysrhythmias common in patients with heart failure. Sudden exacerbation of heart failure symptoms often results from new-onset atrial fibrillation, especially when it is associated with a rapid ventricular response. An ECG can also distinguish frequent premature ventricular beats, which are common in acute and chronic heart failure. Episodes of

Table 20-3 Laboratory Studies Used in the Baseline Evaluation of New-Onset Heart Failure

Laboratory Study	Significance	When Performed
Complete blood count	Used to identify any anemia or infection	Yearly if no specific indication With any exacerbation
Iron studies	Anemia workup Used to rule out hemochromatosis	As needed to evaluate any treatment for iron-deficiency anemia
Thyroid function tests (thyroid-stimulating hormone [TSH] and free thyroxine [T4])	Used to rule out hyperthyroidism or hypothyroidism as a cause of heart failure	No follow-up unless indicated before initiation of amiodarone
Electrolytes	Used to assess the effects of diuresis, in particular on potassium level Hyponatremia is common	With changes in diuretic dose, aggressive diuresis, and titration of drugs that affect potassium (angiotensin-converting enzyme [ACE] inhibitors, angiotensin receptor blockers, spironolactone)
Blood urea nitrogen (BUN) and creatinine levels	Used to assess renal function; BUN:creatinine ratio distinguishes between prerenal azotemia and kidney disease	With increased edema or an exacerbation With titration of ACE inhibitors
Liver function tests, especially albumin, bilirubin, and alkaline phosphatase (AP)	Bilirubin and AP are often elevated in liver congestion caused by heart failure Low albumin makes peripheral edema more difficult to reduce	With any exacerbation Before initiation of lipid-lowering drugs or amiodarone
HIV	Used to rule out HIV/AIDS as etiologic factor	As indicated by history or change in status
Lipid panel	Used to assess risk for coronary artery disease and nutritional status	Yearly or more often as indicated to evaluate treatment

asymptomatic NSVT often occur in patients who are monitored in ICUs, in telemetry units, or with Holter monitors. These asymptomatic dysrhythmias are usually not treated, and their prognostic importance is unclear. In contrast, symptomatic ventricular tachycardia, even if it is nonsustained, requires evaluation and usually results in placement of an implantable cardioverter–defibrillator.

Conduction defects are also common in patients with heart failure. A left bundle branch block is the most common conduction defect in patients with systolic dysfunction and may make interpretation of the ECG very difficult. New anterior ischemia or infarct may be impossible to identify because of this block. Bundle branch blocks and AV blocks require a 12-lead ECG for diagnosis.

ECGs are also useful in diagnosing ischemia, MI, and prior MI that may explain new-onset heart failure. For patients who do not present with typical chest pain (such as those with diabetes mellitus and women), the ECG may show a prior MI that was never diagnosed. New-onset heart failure may be the first indication of MI. An ECG is completed as part of the workup for new-onset heart failure and then repeated as necessary for any new symptoms that may reflect new ischemia or a rhythm change. In addition, ECGs are performed on inpatients who experience chest pain to rule out ischemia as the source of the pain. For further discussion of the 12-lead ECG, see Chapter 21.

Echocardiography

Echocardiography uses the reflection of sound waves off cardiac structures to recreate a two-dimensional representation of the heart chambers, walls, valves, and large vessels, such as the aorta, pulmonary artery, and vena cava. This technique provides information about both the structure and function of the heart and is used to measure ejection fraction, evaluate valve structure and competence, and describe wall motion abnormalities. The addition of Doppler ultrasonography to the traditional echocardiogram allows for the evaluation of volume and direction of blood flow through the vessels and the heart. The reliability of echocardiography is greatly influenced by the competence of the echocardiographic technician and the cardiologist who interprets the echocardiograph. Echocardiography is of limited use in patients who are obese, have very large breasts, or have an increased anteroposterior chest diameter and air trapping (eg, patients with COPD).

Transesophageal echocardiography may be performed in addition to the transthoracic echocardiography previously described. The limitations of the transthoracic procedure can be remedied by the use of the transesophageal procedure; however, the risks are increased because the transponder must be passed down the esophagus, and conscious sedation is often required. The ability to assess the mitral valve and to identify transmural blood clots is greatly improved when transesophageal echocardiography is used.

Radionuclide Ventriculography

A radionuclide ventriculogram or multigated acquisition (MUGA) scan is a precise means of calculating ejection fraction using a radioactive isotope. A MUGA scan is currently the gold standard for calculating ejection fraction because it is not based on the subjective analysis of the person who "reads" it. A MUGA scan can describe abnormal wall motion, dilation, and wall thickness in addition to ejection fraction. Valve function and flow direction cannot be evaluated by MUGA scan.

Chest Radiography

Chest radiography is useful in screening the patient with shortness of breath or DOE. It allows the clinician to rule out infection or pneumonia, COPD, or a mass as the cause of the patient's symptoms. Chest radiography may also help identify pulmonary edema and chronic congestion. However, because changes in the patient's condition and fluid status may not be apparent on a chest radiograph for several days, this procedure is not helpful in evaluating therapy.

Exercise Testing

The details of exercise testing for ischemia are described in Chapter 17 and will not be discussed here. Patients with chronic heart failure, particularly NYHA class III and IV, may not be able to walk long enough or fast enough to document ischemia even when it exists and is the cause of worsening symptoms, dysrhythmia, or acute decompensation. Consequently, pharmacologic stress testing may be more helpful in identifying ischemia.

Cardiopulmonary exercise testing is also used to determine whether DOE is more related to cardiovascular causes (ventricular dysfunction), pulmonary causes (COPD, restrictive lung disease), or deconditioning. Such testing is performed when a precise measure of activity limitation is needed or when a patient is being evaluated for heart transplantation. The patient exercises on a treadmill or exercise bicycle while a 12-lead ECG is obtained and blood pressure is measured in response to graded exercise. In addition, all the patient's expired gases are collected and carbon dioxide is measured. This measures oxygen consumption, cardiac index, and anaerobic threshold.

Hemodynamics

The basics of hemodynamic monitoring are also discussed in Chapter 17. The application of hemodynamic monitoring in the assessment and management of acute heart failure and acute decompensation of chronic heart failure is discussed here. It may be necessary to obtain more sensitive information about fluid status, cardiac function, and symptom causation to guide evaluation and therapy. For most patients with acute heart failure or acute decompensation of chronic heart failure, the problem is obvious based on history and physical examination. The problem is a combination of decreased cardiac output and increased left ventricular end-diastolic pressure related to volume overload added to poor contractility. Precise quantification of the low cardiac output or the estimation of left ventricular end-diastolic pressure by PAOP does not change the basic assessments made on physical examination and does not affect management.

Indications for Hemodynamic Monitoring

The decision to use aggressive diuresis or inotropes is not based on any specific numerical values for PAOP or cardiac output. Pulmonary artery catheters are common in critical

care units today, but they are expensive and not without risk. The potential benefit of more specific, guided management must be weighed against the risk associated with pulmonary artery catheter placement. Results of the Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness (ESCAPE) trial demonstrated no difference in change in symptoms, days alive and days out of the hospital during the first 6 months, length of stay in the hospital, deaths in hospital, or 30-day mortality. Adverse events were more common with pulmonary artery catheters.[17]

Three types of patients with heart failure may have indications for hemodynamic monitoring in the management of their condition. The first type is the patient who is empirically treated with inotropes and intravenous (IV) diuretics but has not responded appropriately by diuresis and improved symptoms. The second type of patient has both COPD and heart failure. At times, only pulmonary artery pressure measurements can differentiate the source of the current decompensation. BNP testing in this scenario may rule out heart failure when the result is less than 80 pg/mL, but elevations can be caused by either left-sided heart failure or right-sided heart failure associated with pulmonary embolus or exacerbation of COPD. The third type of patient continues to have congestion associated with peripheral edema or ascites and has renal function values indicating worsening azotemia. This patient may benefit from a clearer definition of fluid balance; without the aid of a pulmonary artery catheter, it may be impossible to determine fluid status.

In summary, a pulmonary artery catheter is indicated in the following situations:

- The patient does not respond to empirical therapy for heart failure.
- Differentiation between pulmonary and cardiac causes of respiratory distress is necessary.
- Complex fluid status needs to be evaluated.

These categories are not mutually exclusive, and there is much overlap. They are discussed separately here, for clarity.

INADEQUATE RESPONSE TO EMPIRICAL THERAPY FOR HEART FAILURE. Respiratory distress, volume overload, and renal insufficiency are common indicators of acute heart failure or acute decompensation of chronic heart failure. Typically, the patient needs inotropic support and IV diuresis to resolve the problem. These therapies are usually started empirically, and the patient's improvement is monitored as a basis for titration of dose. In most patients, improvement follows rapidly, and after 2 to 3 days of therapy, the inotrope is gradually discontinued, and the patient is restarted on oral therapy in preparation for discharge.

CARDIAC VERSUS PULMONARY CAUSE OF RESPIRATORY DISTRESS. In the minority of patients who do not respond to empiric therapy, a pulmonary artery catheter may be helpful in identifying any additional factors that have contributed to the persistence of symptoms, especially cardiac and pulmonary causes. It may be particularly difficult to differentiate the cause of worsening DOE, orthopnea, and PND in patients with both pulmonary disease and known heart failure. In COPD and in exacerbations of heart failure, results of history and physical examination are often identical. Pulmonary artery pressures, PAOP, and cardiac output or cardiac index can be very useful in distinguishing COPD from acute heart failure and therefore targeting therapy decisions based on the correct diagnosis. In patients with a predominantly pulmonary cause of their respiratory symptoms, pulmonary artery systolic and diastolic pressures are elevated, but PAOP, cardiac output, and cardiac index are normal. In patients with a primarily cardiac cause, pulmonary artery systolic and diastolic pressures are also elevated, but the PAOP is elevated and the cardiac output or cardiac index is decreased.

COMPLEX FLUID STATUS. Patients may respond initially to IV diuresis with or without inotropes. After this initial diuresis, they begin to have decreased urine output associated with increasing BUN and creatinine levels in the presence of persistent peripheral edema. They are typically referred to as intravascularly dry.

The strategy for dealing with this problem is unclear. Insertion of a pulmonary artery catheter may determine whether high pulmonary artery pressures are the cause and whether those pulmonary artery pressures are elevated because of an elevated left ventricular end-diastolic pressure. The readings can then be evaluated in light of the patient's serum albumin level and any comorbid diseases, such as primary liver failure, sepsis, or vascular insufficiency. Newer hypotheses about the relationship between cardiorenal syndrome and renal venous congestion may better explain this phenomenon.[18]

Pulse Oximetry

Pulse oximetry is a frequently used monitoring device in patients with heart failure. Unfortunately, routine intermittent monitoring is of little value. At best, it gives irrelevant information, and at worst, it enables a false sense of security over the patient's oxygen delivery status (Box 20-3). The results of pulse oximetry should be normal. Decreased estimates of oxygen saturation are usually not the result of heart failure unless the patient has severe pulmonary edema.

A low pulse oximetry reading in patients with heart failure and no pulmonary edema suggests that pulmonary disease is complicating the heart failure. Hypoxemia rarely occurs in the absence of comorbid pulmonary disease. Even patients with Cheyne-Stokes respirations associated with an acute decompensation may have blood oxygen saturations greater than 95%. The pulse oximetry reading is only half of the information needed to assess oxygenation accurately. The oxygen saturation is meaningless unless the hemoglobin level is known as well. Even normal arterial oxygen content in a patient with decreased cardiac output and no reserve may lead to tissue hypoxia. If the arterial oxygen content is decreased, as it is in patients with low hemoglobin (patients are rarely transfused unless the hemoglobin is <10 g/dL), cardiac output may not be able to increase enough to compensate in the patient with heart failure. Pulse oximetry may be of some value when used continuously in an ICU for patients with acute pulmonary edema. Particularly in patients with ischemic cardiomyopathy and MI, continuous monitoring may alert the nursing staff to impending ischemia or adverse effects of analgesia or conscious sedation.

BOX 20-3 Pulse Oximetry

Pulse oximetry (SpO_2) estimates arterial oxygen saturation (SaO_2) or the percentage of hemoglobin (Hgb) saturated with oxygen. Oxygen saturation and hemoglobin are the two major components of arterial oxygen content (CaO_2). The dissolved oxygen in the arterial blood (PaO_2) contributes only a tiny portion of the arterial oxygen content. Arterial oxygen content multiplied by cardiac output (CO) equals tissue oxygen delivery (DO_2). If arterial oxygen content is decreased for any reason, cardiac output (mostly heart rate) increases to compensate. This is why patients with anemia or hypoxemia are tachycardic. As long as cardiac output can increase to compensate for a decreased CaO_2, tissues have sufficient oxygen to carry out their functions and the patient is asymptomatic. When a patient cannot increase cardiac output, as in heart failure, then even modest decreases in CaO_2 produce symptoms and increase the likelihood of an exacerbation or death.

$$(SaO_2 \times Hgb \times 1.34) + (PaO_2 \times 0.0031) = CaO_2$$

$$CaO_2 \times CO \times 10 = DO_2$$

Most nurses would be concerned about a patient with a pulse oximetry reading of 85%, but not one with 98%. The following examples demonstrate that the patient with normal hemoglobin and a pulse oximetry reading of 85% has more oxygen in the blood and a better oxygen delivery than a person with a 98% saturation and a hemoglobin of 10. The patients in all these examples have a normal cardiac output at rest but cannot increase cardiac output in response to decreasing arterial oxygen content.

A patient with normal blood gases and a 5-L cardiac output would have a calculated oxygen delivery of 1,000 mL O_2/min:

$$(SaO_2 \times Hgb \times 1.34) + (PaO_2 \times 0.0031) = CaO_2$$

$$(0.98 \times 15 \times 1.34) + (90 \times 0.0031) =$$

$$19.7 + 0.3 = 20 \text{ mL } O_2/\text{min}$$

$$CaO_2 \times CO \times 10 = DO_2$$

$$20 \text{ mL } O_2/\text{min} \times 5{,}000 \text{ mL} \times 10 = 1{,}000 \text{ mL } O_2/\text{min}$$

Suppose a patient has a low SaO_2 and normal hemoglobin:

$$(SaO_2 \times Hgb \times 1.34) + (PaO_2 \times 0.0031) = CaO_2$$

$$(0.85 \times 15 \times 1.34) + (60 \times 0.0031) =$$

$$17.085 + 0.186 = 17.271 \text{ mL } O_2/\text{min}$$

$$CaO_2 \times CO \times 10 = DO_2$$

$$17.271 \text{mL } O_2/\text{min} \times 5{,}000 \text{ mL} \times 10 = 863.55 \text{ mL } O_2/\text{min}$$

Suppose a patient has a normal SaO_2 and low hemoglobin:

$$(SaO_2 \times Hgb \times 1.34) + (PaO_2 \times 0.0031) = CaO_2$$

$$(0.98 \times 10 \times 1.34) + (98 \times 0.0031) =$$

$$13.132 + 0.3 = 13.44 \text{ mL } O_2/\text{min}$$

$$CaO_2 \times CO \times 10 = DO_2$$

$$13.44 \text{ mL } O_2/\text{min} \times 5{,}000 \text{ mL} \times 10 = 672 \text{ mL } O_2/\text{min}$$

Suppose a patient has low SaO_2 and low hemoglobin:

$$(SaO_2 \times Hgb \times 1.34) + (PaO_2 \times 0.0031) = CaO_2$$

$$(0.85 \times 10 \times 1.34) + (60 \times 0.0031) =$$

$$11.39 + 0.186 = 11.58 \text{ mL } O_2/\text{min}$$

$$CaO_2 \times CO \times 10 = DO_2$$

$$11.58 \text{ mL } O_2/\text{min} \times 5{,}000 \text{ mL} \times 10 = 579 \text{ mL } O_2/\text{min}$$

▲ Management of Acute Decompensation of Heart Failure

Acute decompensation of heart failure is an acute worsening of chronic heart failure and may occur for many reasons. Left ventricular function may deteriorate; heart failure is a progressive disease. If function deteriorates beyond the patient's ability to compensate, then symptoms worsen. Although heart function may be stable, the development of other problems, such as pneumonia, anemia, dysrhythmia, hypertension, or trauma, may tax the ability of the compromised heart to increase cardiac output to meet the increased metabolic demand. Dietary lapses, medication disruption, or lack of vigilance on the part of the patient regarding progressive water weight gain may all contribute to decompensation. If possible, it is important to identify the cause of a decompensation so that a long-term strategy to control the underlying problem can be implemented. However, in the intervening period, an acute decompensation must be treated aggressively, often to save the life of a patient.

The main concerns for the care of patients with acute decompensation of chronic heart failure are the same as in any patient with any life-threatening condition. They start with the basic priorities: airway, breathing, and circulation. Once these issues are addressed, etiologic factors and long-term strategies can become the focus of care.

Airway and Breathing

For most patients with acute symptoms of heart failure, airway patency is not a problem. Likewise, oxygenation is not usually compromised unless pulmonary edema is severe or a comorbid pulmonary disease is present. However, when the acute onset of heart failure or the acute exacerbation is accompanied by profound pulmonary edema, such as in MI or flash pulmonary edema, the airway may become compromised. With severe pulmonary edema, surfactant may be washed out of the alveoli, decreasing lung compliance and making ventilation difficult. In patients who also have COPD or restrictive lung disease, the compromise in compliance may make normal minute ventilation difficult if not impossible. An indication

that normal minute ventilation is not being maintained is increased partial pressure of arterial carbon dioxide ($PaCO_2$) associated with increased work of breathing and respiratory acidosis. For example, a patient may initially do well but tire as the increased work of ventilating wet lungs is prolonged.

Intubation

The usual indications for endotracheal intubation in patients with heart failure are the same as for patients in respiratory distress. Intubation and assisted ventilation are indicated if patients are unable to maintain oxygenation or ventilation. Patients who have pulmonary edema and a persistent oxygen saturation level of less than 90% on 100% oxygen should be intubated and supported until they can obtain oxygen on their own. If the increased work of breathing is leading to fatigue of the respiratory muscles and the $PaCO_2$ is rising in association with a falling pH, intubation is indicated even if the patient is able to breathe unaided. The intubation may not be required for more than 12 to 24 hours, but it may be better to protect the airway than to try to intubate a patient after respiratory arrest. See Chapter 25 for more information about the care of the patient receiving mechanical ventilation.

Diuresis

Once the airway is protected, attention is directed toward reducing pulmonary edema. In most cases, aggressive IV diuresis is indicated. The presence of bilateral crackles on physical examination is not always an indication of total-body volume excess. Evaluation of crackles, along with peripheral edema, liver congestion or ascites, and renal function, allows for a better assessment of fluid status than crackles alone. If volume overload in the patient is determined, then IV diuretics facilitate the rapid excretion of excess fluid and the patient quickly feels better.

Aggressive diuresis usually starts with the patient's oral dose of loop diuretic in IV form. An adequate diuretic response is about 1 liter of urine within 2 hours of the IV dose. If urine output is less than 1 liter, the dose is doubled until a maximum dose is reached (for furosemide, a 400-mg single dose) or until the 1 liter urine output goal is met. If the IV loop diuretic is not sufficient to produce this level of diuresis, a thiazide, such as metolazone, may be given orally along with the loop diuretic.[19] The desired weight loss is 1 to 2 kg/d until the patient's dry weight is reached. Initial weight loss may be greater. Careful monitoring of potassium and magnesium is indicated. If the creatinine level begins to rise in response to the diuresis, then the ACE inhibitor should be held until after the diuresis is complete.[20]

Circulation

Once the airway is protected and breathing is adequate to maintain oxygen and carbon dioxide levels, the circulation of blood to perfuse cells and supply oxygen for cellular function becomes the priority. Two indicators are used to determine the adequacy of perfusion. The first indicator is function of organ systems. Inadequate perfusion affects the brain, leading to confusion and change in level of consciousness; the kidneys, leading to increased BUN and creatinine levels; and the gastrointestinal system, leading to ileus and liver failure. The second indicator is metabolic acidosis. If perfusion is severely inadequate or prolonged past the capacity of the body to buffer the lactic acid produced, the level of sodium bicarbonate decreases, as does the pH, producing metabolic acidosis. Metabolic acidosis is a system-wide measure of inadequate oxygen to meet the metabolic demands of tissues.

Hypotension alone is not sufficient to diagnose hypoperfusion in patients with heart failure because many such patients are chronically hypotensive. Hypotension associated with hypoperfusion should be treated in a way that increases flow without increasing afterload. The problem is decreased cardiac output caused by decreased contractility. Whether the patient has acute heart failure associated with cardiogenic shock or acute decompensation of chronic heart failure, the goal of treatment should be to increase cardiac output. Several interventions increase cardiac output.

The normal physiological response to decreased cardiac output is vasoconstriction and increased afterload. In patients with heart failure, afterload may be increased without a dramatic increase in blood pressure, and it is not safe to assume that a low blood pressure means a decreased afterload. Decreasing afterload increases stroke volume, and even in patients with low blood pressures, the increase in stroke volume and perfusion more than compensates for the low blood pressure.

One system for classifying patient symptoms and severity of congestion was developed by Nohria et al.[21] This 2 × 2 table can be used as a foundation for guiding therapy. Figure 20-5 is a representation of this original work.

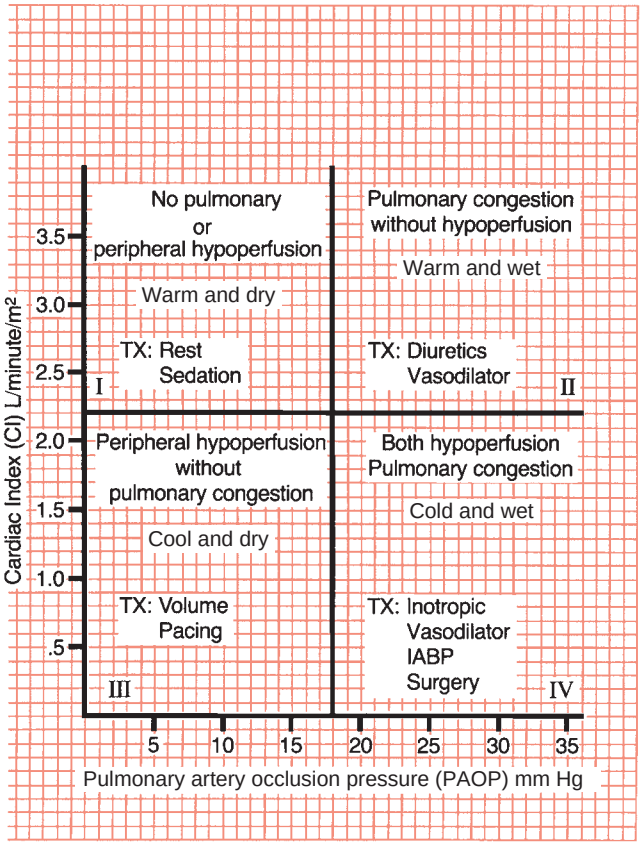


FIGURE 20-5 ▲ Forrester subsets: clinical states and therapy. IAPV, intra-aortic balloon pumping. (Woods SL, Froelicher S, Motzer, SA, et al: Cardiac Nursing, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 584.)

Optimize Hemodynamics

One way to increase cardiac output is to optimize preload.[21,22] If a patient is dehydrated or has fluid overload, contractility is compromised. Both the "warm and wet" and "cold and wet" categories benefit from diuresis. The "cold and wet" category may be impossible to diurese without inodilator or mechanical support. The "cold and dry" category usually requires careful rehydration.[23]

Decreased preload is usually related to iatrogenic overdiuresis. However, patients who are on stable doses of diuretics may become dehydrated if they become hyperglycemic or experience vomiting and diarrhea while continuing to take the prescribed diuretic dose. Careful fluid repletion usually corrects this problem and improves cardiac output. The symptomatic hypotension and increased BUN and creatinine that are the hallmarks of decreased preload should quickly return to baseline levels.

More commonly, increased preload or congestion is a problem; patients are total-body volume overloaded. The combination of fluid overload and decreased contractility leads to cardiopulmonary congestion with increased pulmonary artery pressures and overfilling of the heart. When the heart is overfilled, it becomes stiff and does not empty or fill well. The result is compromised stroke volume and sometimes localized ischemia. The ischemia further worsens contractility. Patients may present with classic angina even if they have no documented CAD. Diuresis with IV loop diuretics often restores the pressure–volume dynamics that optimize stroke volume. For patients who do not respond to diuresis, increasing contractility may decrease preload.

Persistent pulmonary artery pressure elevations lead to jugular venous congestion, liver congestion, ascites, and lower extremity edema. Newer theories of cardiorenal syndromes suggest that this congestion extends to the renal venous system raising efferent pressures sufficiently to impair afferent flow and decreasing glomerular filtration rate (GFR). The decreased GFR leads to the increase in BUN, creatinine, and BUN/creatinine ratio, resulting in poor response to diuresis and persistent symptoms despite an adequate cardiac output at rest.

Increase Contractility

To increase cardiac output, it may be necessary to increase contractility and decrease afterload. Drugs that directly increase contractility are called inotropes. All inotropes increase myocardial oxygen consumption. To be useful in patients with heart failure, there must be greater improvement in oxygen delivery than in oxygen consumption. For this reason, inotropes, such as epinephrine and isoproterenol, are not used.

The following are indications for using inotropes:

- Low cardiac output and high PAOP, especially with symptomatic hypotension
- High PAOP with poor response to diuretics in volume-overloaded patients
- Severe right-sided heart failure that is the direct result of left ventricular failure
- Symptoms of heart failure at rest despite excellent maintenance therapy.

Dopamine is also an excellent inotrope at mid-level doses. However, because dopamine is also a vasoconstrictor, especially at higher doses, it increases afterload in patients with heart failure and decreases stroke volume or, at the very least, does not increase it. Although there are no data to support its use, so-called renal-dose dopamine has been used frequently in patients with heart failure.[27] The use of renal-dose dopamine has been based on the knowledge that the effects of dopamine are dose related. At low doses of 1 to 3 mcg/kg/min, the hypothesized main effect of dopamine is stimulation of dopaminergic receptors that dilate renal and splanchnic circulations. Higher doses have inotropic and vasoconstrictor activity. There is no evidence supporting the routine administration of dopamine to improve diuresis or prevent acute renal failure

Drugs called inodilators are used to stimulate β-adrenergic receptors located in the heart and blood vessels to increase contractility and cause vasodilation.[27] The two inodilators most commonly used in ICUs are dobutamine and milrinone. Although these drugs have different pharmacological mechanisms, they both increase stimulation of β-adrenergic receptors. Because they stimulate β-adrenergic receptors, they are also chronotropic (ie, they increase heart rate), and they must be used carefully and titrated slowly in patients with tachycardia or ventricular dysrhythmia.

The effect of inotropes and inodilators can be measured when a pulmonary artery catheter is in place. As the drugs are titrated to optimum doses, cardiac output increases, and the PAOP decreases. Urine output should increase, and BUN and creatinine levels should return to baseline levels. Any organ function that was compromised because of inadequate perfusion should improve.

Vasodilation

Sometimes an inodilator alone is not sufficient to decrease afterload adequately. In patients with cardiogenic shock or patients who have an exacerbation related to hypertensive emergency, the afterload is the primary limiting factor. Decreasing and controlling the blood pressure or decreasing the workload of the damaged myocardium requires immediate treatment, and vasodilation with parenteral medications is necessary to maintain life or limit end-organ damage. Nitroprusside has the most rapid onset with the shortest half-life of any of these medications. It provides for rapid, efficient decrease in blood pressure, and the effect is limited to minutes if the medication is stopped because of an exaggerated response. Nitroprusside must be given as a continuous drip and requires reliable monitoring of blood pressure in a setting where emergency resuscitation is available.

Nesiritide, a BNP, has been approved as a vasodilator for treatment of acute decompensation of chronic heart failure.[22,24,25] It is unclear whether this vasodilator has any advantages over nitroprusside or nitroglycerin. Although nesiritide was originally marketed as superior to other vasodilators, questions were raised about increased risk of renal dysfunction and mortality. Much controversy still exists as to the appropriate use of nesiritide.[25]

For intermittent blood pressure control, IV or oral hydralazine provides vasodilation with a decrease in afterload, without any negative inotropic effects. Sublingual nifedipine should never be used to control blood pressure.[2] IV nitroglycerin is valuable in decreasing preload and in treating angina associated with hypertensive emergency, but it is not a good afterload reducer or antihypertensive.

Intra-aortic balloon counterpulsation has proved very successful in reducing afterload in cardiogenic shock by augmenting perfusion pressure and decreasing the workload of the left ventricle. Intra-aortic balloon counterpulsation is often critical

to survival in patients with acute MI who suffer acute left ventricular failure. Intra-aortic balloon counterpulsation is used for a limited time for support of the patient until a revascularization procedure can restore oxygenation and function or until the stunned myocardium has recovered somewhat (in a patient who cannot be revascularized). For a more detailed discussion of intra-aortic balloon counterpulsation, see Chapter 18.

Improvements in design and increased experience with left ventricular assist devices (LVADs) as well as FDA approval and Medicare reimbursement have led to increased use of these devices in treating intractable heart failure.[26] Where once LVADs were only approved as a bridge to transplant for those patients who were already on a waiting list for transplant, they are now used as destination therapy. That is, for patients who are not eligible for or do not desire transplant, LVADs are now used to treat heart failure. Devices include battery-operated functions that allow the patient to go home, socialize within the community, and to be an active participant in daily life.

Heart Rate

Heart rate and rhythm must be optimized for adequate cardiac output. If the heart rate is too slow, such as in sick sinus syndrome, second- or third-degree AV block, or sinus bradycardia, stroke volume cannot be increased adequately to compensate, resulting in an exacerbation. A heart rate that is too slow or too fast can compromise filling and, in patients with ischemia, can contribute directly to decreased contractility. A fast rate may be a compensation for a decreased stroke volume and usually responds to increasing stroke volume.

The administration of β-adrenergic inotropes may improve heart rate along with the inotropic effect and greatly improve cardiac output. However, the reason for the bradycardia must be identified and treated if the improvement is to be sustained. In many cases, problems with bradycardia result from ischemic damage to the conduction system. In this situation, a permanent pacemaker resolves the problem. If the bradycardia is the result of active ongoing ischemia, a temporary pacemaker along with treatment of the ischemia is indicated. (For a more detailed discussion of cardiac pacemakers, see Chapter 18.) If the bradycardia is the result of medication, the medication should be withheld or discontinued until the indication for the medication can be reevaluated. In this situation, β-blockers may be withheld for 24 to 36 hours but should not be discontinued suddenly. If the bradycardia is the result of β-blockers, temporary pacing may be required while the drug dosage is titrated down.

Sinus tachycardia is usually the result of decreased stroke volume and therefore cardiac output. Treatment of the tachycardia without increasing stroke volume leads to worsening end-organ perfusion. Sinus tachycardia usually resolves if the underlying decrease in stroke volume is corrected.

When the tachycardia is caused by atrial flutter or atrial fibrillation with rapid ventricular response, the heart rate is the cause of the problem, and it is necessary to control this directly. If the patient is unconscious secondary to the heart rhythm, direct-current countershock cardioversion is indicated. Otherwise, mechanical methods such as the Valsalva maneuver or carotid massage may be helpful. If medication is required to slow the rhythm, amiodarone is the least dangerous medication to use in systolic dysfunction. Calcium channel blockers, such as verapamil and diltiazem, are powerful negative inotropes and may aggravate the low–cardiac-output state. In many cases, the tachycardia is associated with ischemia or hypertensive crisis, and treatment of the underlying problem also treats the tachycardia.

BOX 20–4 EXAMPLES OF NURSING DIAGNOSES

- Decreased Cardiac Output related to altered preload
- Decreased Cardiac Output related to altered contractility
- Decreased Cardiac Output related to altered heart rate
- Activity Intolerance related to decreased cardiac output and deconditioning

After the patient is stabilized and cardiac output has been supported by inodilators or vasodilators, any uncontrolled comorbid diseases that may have triggered or worsened the exacerbation must be treated. Anemia with a hemoglobin level of less than 10 g/dL should usually be treated with transfusion, although this recommendation has recently been questioned. Transfusion is more clearly indicated in an anemic individual who is ischemic with diffuse CAD who is not a candidate for PCI and who is symptomatic or unstable, although this remains a decision based more on art than on science.[2] Pneumonia or other infection should be diagnosed and treated with the appropriate antibiotics. Blood glucose levels should be controlled using insulin if necessary. Examples of nursing diagnoses for heart failure are shown in Box 20-4.

Effective management of chronic heart failure is a key to the prevention of ADHF. Not all ADHF can be prevented because, even when medical regimens are optimal, patients are excellent at self-care, and providers have frequent and regular contact with patients and their care givers; events outside of their control can lead to decompensation. There should be a concerted effort to adequately reduce congestion, prescribe guideline-based medication regimens and educate patients and their care givers prior to discharge. It is critical then to understand guideline-based management of chronic heart failure.

▲ Management of Chronic Heart Failure

Heart failure is not a true disease but rather a manifestation of disease. Management is based on the same therapeutic principles that apply to any disease. The cause of disease should be identified and then treated. If an etiologic factor cannot be identified or cannot be treated, then its manifestations should be treated. Often, the cause of heart failure is not identified, and even when it is, it may not be reversible. Reversible causes of heart failure have been discussed previously and are not addressed here. Isolated right-sided heart failure (cor pulmonale) also is not addressed here.

Heart failure due to diastolic dysfunction is a complex and poorly defined entity. Few studies of investigational medications or therapies have included patients with diastolic dysfunction, and consequently, there is little in the way of evidence-based therapy. In general, treatment strategies are directed toward controlling blood pressure, fluid volume, and heart rate and rhythm. There is no consensus as to how this control should be established and maintained.

Chronic heart failure secondary to dilated cardiomyopathy and systolic dysfunction is better defined. This section discusses the current evidence-based guidelines for managing chronic heart failure and acute decompensation. When appropriate, management of acute heart failure is distinguished from management of acute decompensation; the use of IV inotropes, diuresis, and afterload reduction is similar in both conditions.

Pharmacological Treatment

The ACC and the AHA have published a consensus of evidence-based guidelines for the pharmacological management of heart failure[2] (Table 20-4). These guidelines present the most current recommendations based on available clinical trials for the medical management of heart failure. For example, heart failure in older patients has particular management implications (Box 20-5).

Table 20-4 Medications Used in the Treatment of Heart Failure

Drug	Action	Starting Dose	Target Dose	Indications, Contraindications, Adverse Effects
Chronic Heart Failure				
ACE inhibitors Lisinopril Enalapril Captopril	Block renin–angiotensin–aldosterone system, decrease symptoms and mortality Block conversion of angiotensin I to angiotensin II for afterload reduction	Lisinopril: 2.5–5 mg daily Enalapril: 2.5–5 mg twice daily Captopril: 6.25–12.5 mg twice daily	Lisinopril: 20–40 mg daily Enalapril: 10–20 mg twice daily Captopril: 50 mg twice daily	May cause angioedema, hyperkalemia, increased creatinine, symptomatic hypotension
Hydralazine	Pure vasodilator Used to decrease afterload	10–25 mg PO every 6–8 h	75 mg PO every 6 h or 100 mg PO every 8 h	May cause tachycardia Used for intolerance of ACE inhibitors, for additional blood pressure control, or for afterload reduction in severe mitral regurgitation or atrial insufficiency
Nitrates Isosorbide dinitrate Isosorbide mononitrate	Decrease preload, relieve angina, decrease orthopnea	Isosorbide dinitrate: 10 mg every 6 h (hold midnight dose) Isosorbide mononitrate: 30 mg daily	Isosorbide dinitrate: up to 40 mg every 6 h (hold midnight dose) Isosorbide mononitrate: up to 120 mg daily	Dose limited by symptoms such as headache or hypotension Use least dose that relieves symptoms
Digoxin	Oral inotrope Blocks neurohormonal bombardment of heart	0.125–0.25 mg PO daily	Same	Limited by renal excretion; smaller doses used when creatinine is >1.3 mg/dL Dose should be decreased in patients receiving amiodarone
Diuretics Furosemide Metolazone	Control fluid volume	Furosemide: 20–40 mg (in patient who has never been on diuretics) Metolazone: 2.5–5 mg daily	Up to 320 mg twice daily if necessary to control fluid Metolazone 10 mg daily if necessary in addition to furosemide	Diuretic dosage requirements are higher during aggressive diuresis than during maintenance Combination of furosemide and metolazone is very powerful, and loss of potassium, magnesium, and calcium can be dramatic, increasing risk for dysrhythmia
Spironolactone	Blocks effects of aldosterone and protects potassium	25 mg daily	25 mg daily	May cause hyperkalemia, so potassium should be monitored regularly May cause gynecomastia in men
β-Blockers Metoprolol SR Carvedilol Bisoprolol	Improve symptoms, increase exercise tolerance, decrease hospitalizations and mortality	Metoprolol SR: 12.5 mg daily Carvedilol: 3.125 mg twice daily Bisoprolol: 1.25 mg daily	Metoprolol SR: 100–200 mg daily Carvedilol: 25–50 mg twice daily Bisoprolol: 10 mg daily	May precipitate exacerbation during initiation and titration Monitor weight and heart rate carefully; do not stop drugs suddenly Benefit is long term and may not be evident for up to 3 mo

(continued on page 409)

Table 20-4 Medications Used in the Treatment of Heart Failure (continued)

Drug	Action	Starting Dose	Target Dose	Indications, Contraindications, Adverse Effects
Acute Heart Failure and Acute Exacerbation of Chronic Heart Failure				
Inodilators Dobutamine Milrinone	Increase contractility, decrease afterload and therefore increase cardiac output Increased forward flow decreases left ventricular end-diastolic pressure	Dobutamine: 2–5 mcg/kg/min Milrinone: 0.2–0.3 mcg/kg/min (with or without loading dose)	Dobutamine: 5–15 mcg/kg/min Milrinone: 0.375–0.7 mcg/kg/min	Use smallest dose that produces desired hemodynamic effect May cause tachycardia and ventricular dysrhythmias Can be given effectively to patients who are receiving β-blockers
Dopamine	May increase renal perfusion and improve diuresis	1–3 mcg/kg/min	1–3 mcg/kg/min	The higher the dose, the more likely dopamine is to increase afterload Do *not* give through a peripheral line
Nitroprusside	Used for afterload reduction and blood pressure control	0.5 mcg/kg/min	Up to 1.5 mcg/kg/min	High doses or prolonged administration is associated with increased cyanide levels and should be avoided
Nesiritide	Used for afterload reduction	2 mcg/kg/min bolus with 0.01 mcg/kg/min infusion	Increase by 0.005 mcg/kg/min to maximum of 0.3 mcg/kg/min	Use caution if systolic blood pressure <90 mm Hg
Hydralazine	Used for afterload reduction and blood pressure control	5–10 mg IV every 4 h PRN	5–10 mg IV every 4 h PRN	May cause tachycardia

BOX 20-5 CONSIDERATIONS FOR THE OLDER PATIENT

Heart Failure

Most patients with heart failure are elderly, and many fit the category of "old old." They have a variety of limitations and comorbid diseases that may or may not relate to heart failure, as well as a remarkable resiliency and adaptability not found in younger patients. Therefore, it is critical to evaluate their limitations and strengths on an individual basis. It is important to treat the comorbid diseases aggressively according to patients' wishes and to include them in the planning and treatment decisions at all levels.

It is also critical to assess fall risk, activity level, visual acuity, manual dexterity, cognitive ability, and memory when administering, evaluating, or teaching about any medication. For some older patients, the assistance of a family member or friend is critical to successful medication adherence. Financial considerations are also important because many older patients are on Medicare and have a limited drug plan to pay for expensive medications. Having to choose between medication and food is no choice.

Angiotensin-Converting Enzyme Inhibitors

ACE inhibitors are the mainstay of standard therapy for heart failure today; they represent one third of the classic three-drug combination used. The Studies of Left Ventricular Dysfunction (SOLVD) and Cooperative North Scandinavian Enalapril Survival Study (CONSENSUS) trials demonstrated improvement in mortality as well as symptom management and exercise tolerance in even the sickest of patients with heart failure.[2,27] ACE inhibitors are typically started at low doses and titrated to target doses established in clinical trials. Studies have shown that ACE inhibitors were being underprescribed for appropriate patients, and the Assessment of Treatment With Lisinopril and Survival (ATLAS) trial found that being on the medication alone was not sufficient and that target doses used in the clinical trials were necessary to achieve the optimum results.[28]

ACE inhibitors work by blocking the renin–angiotensin–aldosterone system, resulting in vasodilation and antagonism of aldosterone and decreasing afterload and sodium

reabsorption. Blockage of the long-term effects of myocardial cell exposure to the renin–angiotensin–aldosterone system is hypothesized to be the mechanism by which ACE inhibitors decrease mortality and limit the progression of remodeling.[29]

ACE inhibitors do have some side effects. Because some patients are allergic to ACE inhibitors, they experience angioedema, a potentially fatal reaction that involves edema of the mouth, pharynx, and larynx. There is no way to predict which patients will have this reaction, but when it presents, it is critical that the medication be stopped and a notation be made in the patient record so that it is not prescribed again. Patients should also be educated about the names of potential ACE inhibitors and why they should not be taken.

A troubling but not dangerous cough develops in some patients who take ACE inhibitors. Typically, after starting ACE inhibitors, patients may complain of a persistent, dry, nonproductive cough. The cough is not related to patient position or time of day, and it subsides when the ACE inhibitor is discontinued.

Hyperkalemia develops in some patients who take ACE inhibitors. Like the cough, the hyperkalemia resolves when the drug is discontinued. Serum potassium levels greater than 6.0 mEq/L are potentially dysrhythmogenic and should be treated with exchange resin. Patients with serum creatinine levels greater than 1.5 mcg/dL are often denied ACE inhibitors in the mistaken belief that they will develop renal failure if they are given ACE inhibitors. In fact, patients with elevated creatinine levels are no more likely to develop an increased creatinine level than patients with a normal creatinine level when the ACE inhibitors were started.[29,30] The two types of patients most likely to show increased creatinine levels when started on ACE inhibitors are patients with renal artery stenosis and those with hypovolemia.

Patients receiving ACE inhibitors also have decreased blood pressure. It is not uncommon to see asymptomatic systolic pressures of 80 to 99 mm Hg, and diastolic pressures may be 40 to 59 mm Hg. These low pressures are not symptomatic because perfusion of the brain and kidneys is not compromised as it might be in a patient with normal systolic function. The increased flow or stroke volume more than compensates for the decrease in resistance, and the tissues actually receive more blood and therefore oxygen than they would at a higher resistance and pressure. It is unnecessary to withhold or decrease ACE inhibitors for asymptomatic hypotension.

For patients who truly cannot tolerate ACE inhibitors, other options are available. The use of hydralazine and nitrates preceded the studies on ACE inhibitors, and these drugs offer similar reductions in mortality. Hydralazine must be taken three or four times daily, and many patients have trouble complying with a multidose medication regimen. Long-acting nitrates are used in conjunction with the hydralazine. Once-a-day preparations, such as isosorbide mononitrate or a nitroglycerin patch may be used if compliance is a problem. Isosorbide dinitrate can be used if a rest period of at least 6 to 8 hours is taken.

Another option for patients who cannot take ACE inhibitors because of cough is an angiotensin II receptor blocker. Losartan, valsartan, and candesartan were studied in the treatment of heart failure. Early results suggest that these agents are effective in patients who are not taking ACE inhibitors. Valsartan and candesartan have FDA indications for heart failure.

Digoxin

Cardiac glycosides have been used for centuries in the empirical management of heart failure. However, until recently, no objective evidence indicated that digitalis preparations made any actual difference in managing heart failure. Beginning in 1993, the Prospective Randomized Study of Ventricular Failure and the Efficacy of Digoxin (PROVED) trial and, more recently, the Randomized Assessment of Digoxin on Inhibitors of Angiotensin-Converting Enzyme (RADIANCE) and Digitalis Investigation Group (DIG) trials provided evidence that digoxin is of value in heart failure treatment.[31] Although none of the studies has shown that digoxin affects mortality, they all have consistently shown that digoxin leads to improvement in symptom management and exercise tolerance as well as decreased hospitalizations for heart failure.

Digoxin should be given in daily doses of 0.125 mg. Lower doses are used in patients who have renal insufficiency or also take amiodarone. Digoxin is safe and has few, if any, adverse effects as long as the blood levels remain less than 2.0 ng/mL. No studies have identified a therapeutic level for digoxin in heart failure or guidelines for interpreting drug levels.[2] The traditional therapeutic levels given in studies of atrial fibrillation may be excessively high; lower levels (ie, 1.0 ng/mL) may be equally beneficial and safer.

Diuretics

Since furosemide became available in the 1960s, diuretics have become a mainstay of heart failure management. Edema, a common finding in patients with heart failure, is the result of volume expansion in response to neurohormonally mediated salt and water retention. In certain conditions (eg, ascites, pleural effusions), "third spacing" of fluids is a common result of excess volume and increased hydrostatic pressure. Edema worsens when patients are unwilling or unable to reduce sodium in their diets. Patients who have advanced heart failure are frequently malnourished and may have low serum albumin levels with a consequent decrease in osmotic gradients to pull fluids back into the circulation. Patients who are symptomatic from volume overload feel dramatically better when diuresis reestablishes their dry weight. Drugs such as ACE inhibitors and β-blockers work best in euvolemic patients.

Loop diuretics, such as furosemide, are standard therapy for diuresis in patients with heart failure.[2] More expensive loop diuretics are available but have not been shown to be superior to furosemide. Loop diuretics are threshold drugs, and the threshold varies from patient to patient. This

means that the appropriate dosage must be determined by the patient's response. In a patient who requires furosemide in oral doses of 200 mg to maintain dry weight, 100 mg twice daily is not sufficient. Doses in excess of 200 mg daily may be necessary. When patients are receiving oral doses of 240 mg or more, yet continue to have edema or have increased edema, diuretic resistance must be considered. Loop diuretics should not be abandoned; however, a brief course of IV diuretic or the addition of a thiazide, such as metolazone, until the edema is controlled may be required.

The combination of loop and thiazide diuretics works more efficiently than either type of diuretic alone. However, this drug combination should be reserved for refractory edema, and when the edema resolves, an appropriate dose of loop diuretic should be determined and continued.

As heart failure progresses or when decompensations occur, dose adjustments are necessary. Patients should be taught to weigh themselves daily and record their weights. Increases of 2 pounds or more overnight or of 5 pounds or more in a week are water weight, which can be controlled with additional doses of diuretic (1 liter [1.06 quarts] of water weighs 1 kg [2.2 pounds]). Some patients can manage their fluid balance with a sliding-scale diuretic, much like patients with diabetes mellitus manage their blood glucose level with sliding-scale insulin.

Spironolactone

Spironolactone is a weak diuretic with potassium-sparing properties. It is not used specifically for its diuretic activity. The Randomized Aldactone Evaluation Study (RALES) trial[32] studied mortality in patients with NYHA class III or IV heart failure who took spironolactone as well as ACE inhibitors, digoxin, and diuretics. The results were a 30% reduction in mortality in patients who received only 25 mg/day of spironolactone. The reasons for the decreased mortality are unclear, but the hypothetical mechanism is that spironolactone blocks aldosterone and its damaging effects on heart muscle. Of theoretical concern is the addition of another potassium-sparing drug to the regimen of patients who are already taking an ACE inhibitor, which also spares potassium. However, few patients had serum potassium levels high enough to discontinue the spironolactone. Many of those patients tolerated every-other-day administration of spironolactone well, with excellent results.

β-Blockers

Intuitively, β-blockers, with their negative inotropic properties, ought to be the least likely intervention to benefit patients with systolic dysfunction. For many years, the prevailing standard of care specifically excluded β-blockers for patients with ineffective heart pumps. During the past 30 years, both small studies and large, multicenter, international, randomized, placebo-controlled studies challenged this idea. Meta-analysis of the smaller studies and primary analysis of the recent studies documented a 34% improvement in mortality in NYHA class II and III heart failure. Other long-term benefits of β-blockers include improved exercise tolerance, better symptom control, fewer hospitalizations, and improved ejection fraction.

Short-term use of β-blockers makes heart failure worse. Consequently, β-blockers should be used as a long-term strategy that is begun only when patients are stable using optimum background therapy with ACE inhibitors, digoxin, and diuretics. β-Blockers should not be started when a patient is in the midst of an decompensation. The specific drug used should be started at a very small dose and gradually increased to the target range. The initiation and titration of β-blockers are beyond the scope of this text but are outlined in detail elsewhere.[33]

Under no circumstances should β-blockers be stopped suddenly. The rebound tachycardia can be fatal, especially in patients with coronary insufficiency. Patients who come into the hospital because of a decompensation of heart failure who are on β-blockers should continue taking the β-blocker. If a temporal relationship exists between titration of the β-blocker dose and the onset of the exacerbation, the dose should be reduced to the last well-tolerated dose. Patients who are taking β-blockers may receive inotropes without discontinuing the β-blocker and may respond well because of the upregulation of β-adrenergic receptors.

Calcium Channel Blockers

First-generation calcium channel blockers, such as diltiazem, verapamil, and nifedipine, should be avoided in patients with systolic dysfunction. These drugs exert a strong negative inotropic effect without the long-term benefits of β-blockers. Second-generation calcium channel blockers, such as amlodipine or felodipine, have been used in patients with heart failure because they are vasodilators with minimal negative inotropic effects. They are most commonly used to control blood pressure in patients who are on target doses of ACE inhibitors but who continue to have blood pressure levels that exceed the recommendations of the Seventh Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure (JNC7).[34] (The JNC7 recommends that blood pressure in patients with heart failure be <130/80 mm Hg.)

Nitrates

Nitrates are venodilators, and their primary effect is to decrease preload. As coronary vasodilators, they are used to treat angina. In very high doses, they may lower blood pressure, but they are not first-line drugs for the treatment of hypertension. When given to patients who are volume depleted or have right ventricular infarctions, nitrates may lead to abrupt hypotension, which is the result of inadequate preload to maintain stroke volume and cardiac output.

Nitrates are used in heart failure to help alleviate the symptoms of orthopnea and DOE.[35] Often, when patients

lie down, the increased venous return (preload) leads to increased pulmonary artery pressure because the volume is too great for the weakened left ventricle. This sudden increase in preload and pulmonary artery pressure causes the sensation of dyspnea. Sitting up reduces the preload and relieves the symptoms. Nitrates decrease preload and mediate the volume of blood presented to the left ventricle, thus helping to control dyspnea. For this reason, nitrates may be used for patients who do not have angina specifically for the management of orthopnea and DOE.

Nonpharmacological Treatment

Role of the Patient

Several strategies can be used to manage symptoms and prevent hospitalization of patients with heart failure.[36–38] The participation and commitment of the patient is necessary for success.

Sodium restriction is critical. Patients often believe that if they no longer use a salt shaker, they have eliminated all excess salt from their diet, and they may be surprised to learn that canned soup and canned vegetables are extremely rich in salt. Education about the natural salt content of foods and the salt that is added as part of food processing is essential. Patients must be taught to read labels and shop for foods that provide optimum nutrition with minimal salt.

Alcohol use should be stopped. As noted previously, alcohol is a powerful cardiac depressant. Many patients have read that a glass of wine or a drink each day decreases the risk for CAD. Although this may be true, the studies were performed in patients who did not have systolic dysfunction. It is important to clarify this fact and explain to the patient the adverse effects of alcohol.

Exercise should be encouraged. Patients with heart failure have limited stamina, and the goal is to increase stamina with low-level exercise over a longer period of time instead of intense exercise for short periods of time. Obviously, some patients with heart failure start at a higher level of functioning and have a better exercise tolerance than patients with advanced heart failure. Exercise for patients with heart failure is not the same as that for development of cardiovascular fitness, and heart rate is not a good indicator of exercise efficacy.

Patients with heart failure should be encouraged to maintain their level of activity. Walking is by far the best recommended exercise. Neither speed nor distance is important. Patients should aim for 15 to 20 minutes each day without stopping to rest or "catch their breath" at whatever pace they are able to manage. Some patients need to take many rests before they begin to exercise, and it may be quite a while before they can exercise for this length of time even at low levels. Weight-lifting is not recommended because this activity increases afterload and may worsen symptoms.

The most important thing patients can do to stay out of the hospital and control symptoms is to take their medication. The second most important activity is to measure their weight every day. An overnight weight change of more than 3 pounds is due to water weight. If patients take and record their weight every day, modest fluid accumulations of 1 quart or less can be identified. Diuresis can be initiated before patients experience so much fluid overload that hospitalization for IV diuresis is necessary.

Fluid restrictions are punishing, and there is no evidence that water restriction has any value in the absence of significant hyponatremia. Likewise, there is no physiological basis for decreasing or controlling edema by fluid restriction, or any evidence that restricting fluids is effective.[38] The problem for patients with heart failure is the retention of sodium, which "holds on" to water. Restricting sodium does decrease or control edema, as discussed in the section on diuretics.

Implantable Cardioverter–Defibrillator

In dilated cardiomyopathy, the incidence of sudden death from ventricular tachycardia or ventricular fibrillation is very high. Asymptomatic ventricular tachycardia is common, but its prognostic impact is unknown. For patients who have syncopal episodes or survive sudden death, an implantable cardioverter–defibrillator is usually indicated. An implantable cardioverter–defibrillator interrupts life-threatening dysrhythmias. If this device fires frequently or symptomatic NSVT occurs, amiodarone may be added to the regimen for rhythm control. See Chapter 18 for more information about the implantable cardioverter–defibrillator.

Biventricular Pacing

In with heart failure and intraventricular conduction delays (QRS duration more than 130 milliseconds) that lead to dysynchronous activation of the left and right ventricles, biventricular pacing or cardiac resynchronization improves cardiac output and therefore symptoms and exercise tolerance.[39,40] Pacing both ventricles of the spherically dilated heart reproduces the bottom-to-top contraction of a normal ventricle that is lost with myocardial remodeling and bundle branch block.

Patient Education

Many times, severe exacerbations requiring hospitalization can be avoided. If a weight gain of 2 to 3 pounds can be treated with intermittent extra doses of diuretic, then 15- and 20-pound weight gains that require hospitalization will not occur. Helping patients control both their heart failure and their comorbid diseases empowers them instead of victimizes them and gives them a sense of control that also helps to limit hospitalization. There have been many reports of improved quality of life, decreased hospitalizations, and decreased cost of care for patients in disease management programs.[41–43]

Home care provides many opportunities for disease management. As the home care nurse enters the patient's environment, the opportunities for teaching become evident.

Even in situations in which the number of visits after a hospital stay is limited, such as with patients covered by Medicare, there are many opportunities for the home care nurse not only to assess but also to intervene.

Discharge planning begins with the first day of hospitalization. A program of education, referral, and follow-up is initiated with the goal of preventing further hospitalization. Patient teaching is necessary, but it is not sufficient alone to allow patients to be effective partners in their care. Close follow-up, coaching, and skill development allow patients to effectively use the information they have been taught. (Box 20-6). Clearly, patients must be on target levels of standard medications to reap the benefits of the clinical studies in heart failure. However, patients must collaborate with health care providers to maximize this benefit (Box 20-7).

BOX 20-6 TEACHING GUIDE *Living With Heart Failure*

Medications
- Take all medications as instructed. If you cannot afford them, please let your provider know so that you can be put in touch with someone to help.
- Do not stop taking medication because you feel better. These are lifetime medications in most instances. Some of the medications will need to be adjusted over time, but your health care provider will discuss the changes with you.
- You may be taking several drugs. These medications do not interfere with each other, and they are given together so that they can work together to do more than any one or two of them can do alone.
- Do not let your medication supply run out because stopping some medications suddenly can cause serious problems.
- Take your medications about the same time every day.
- If you are going out for a few hours and will not have easy access to a bathroom when you need it, hold off on your diuretic until you return home. Do not skip a day's dose of diuretic because this could lead to serious water accumulations and worsening of your heart failure.

Diet
- Restrict your salt intake by removing the salt shaker from the table and the food preparation area. Do not add salt to any food you are cooking or any food on your plate.
- Avoid foods that have a high salt content naturally or because of the way they are preserved. Foods such as canned soup, canned vegetables, canned meats, foods frozen in sauces, cold cuts, sauerkraut, dill pickles, cheese, and processed foods of any kind are loaded with salt. Seasonings such as garlic and onion salt, Old Bay, and monosodium glutamate are the same as salt. Avoid salt substitutes because they are made with potassium; in combination with the medications you are taking, they can lead to potassium excesses. Avoid fast food such as hamburgers, French fries, fried chicken, and tacos.
- Seasonings such as pepper, Mrs. Dash, onion and garlic powder, herbs, seeds, and spices are acceptable.
- Fresh or frozen vegetables (frozen without sauces), fresh lean meats and poultry, and fish (not fried) are all good choices.

Daily Weights
- Weigh yourself every day at about the same time and record the value.
- The best time to weigh yourself is in the morning when you first get up and after you go to the bathroom.
- Weigh yourself without clothes if possible.
- Record your weight and the date in a daily diary. Bring this diary with you to the office when you visit your health care provider.
- Call if your weight goes up more than 2 pounds overnight and does not go back to baseline the next day, or if you gain more than 3 pounds in a week.

Activity
- Stay as active as possible.
- The stronger your skeletal muscles are, the easier it is for your heart.
- Do not use heart rate as a measure of adequacy of exercise effort.
- If you get tired or short of breath, stop and rest, and then try again. The goal is 15 to 20 minutes of continuous activity each day.
- There are no speed or distance goals, and walking at whatever pace you can accomplish is a good choice. Homemaking and gardening are good choices as well. Choose an activity that you enjoy.
- Shortness of breath is uncomfortable but not dangerous. It is an indication that you are nearing the end of your exercise tolerance for this period, but once your breathing normalizes, you can go again. If you stop before you get short of breath out of fear, you will not be able to increase your activity tolerance.
- If you have any questions about how much exercise you can tolerate, discuss it with your health care provider. That person is the best advisor for you because you are well known to him.
- Do not lift weights unless your health care provider has specifically said it is an acceptable activity for you.

Call Your Health Care Provider If
- Your weight increases or decreases suddenly.
- You begin waking up at night short of breath and need to sit up to breathe.
- You start needing more pillows at night to breathe when you lie down or you are unable to lie down.
- You become short of breath at rest.
- You cannot walk up stairs that you used to climb regularly because now it makes you too short of breath or tired.
- Your feet and legs start to swell.
- You faint or feel as though you are going to faint.
- You become dizzy and weak when you stand.

BOX 20-7 COLLABORATIVE CARE GUIDE for the Patient With Acute Decompensation of Chronic Heart Failure

Outcomes	Interventions
Oxygenation/Ventilation	
There will be adequate oxygen to meet the metabolic demands of the tissue. Minimum arterial oxygen content evidenced by: 1. Hemoglobin (Hgb) = 10 g/dL or more 2. SpO_2 = 90% or more	• Consider the transfusion of red blood cells if Hgb is 9.0 g/dL or less. • Supplemental oxygen to maintain SpO_2 at >90% • Consider intubation and mechanical ventilation if patient develops respiratory acidosis or cannot maintain oxygen saturation on 100% oxygen by mask. • Consider primary pulmonary problem as cause of hypoxemia and check brain natriuretic peptide level.
The patient's symptom of dyspnea will be managed. 1. Patient denies dyspnea at rest 2. Patient reports increased activity before feeling sufficient dyspnea to limit activity 3. NYHA class equal to or better than baseline before decompensation	• Elevate head of bed or allow patient to select upright position that best relieves dyspnea. • Apply damp washcloth to patient's face. • Use a fan or other means to create air movement across the patient's face. • Encourage the patient to ambulate as soon and as much as possible once dyspnea at rest is relieved.
Circulation/Perfusion	
Cardiac output will be maximized. Optimum cardiac output evidenced by: 1. Cardiac index > 2.0 2. SvO_2 > 50% 3. Urine output > 30 mL/h 4. Baseline level of consciousness and orientation	• Optimize preload with diuresis, fluid administration, or vasodilation with agent such as nitroglycerin, nitroprusside, or nesiritide. • Increase contractility with inotrope such as milrinone or dobutamine. • Decrease afterload with diuresis and vasodilation.
Hypotension will be asymptomatic, and the patient's blood pressure is at baseline.	• Determine the patient's baseline blood pressure; systolic pressure may be <90 mm Hg. • If blood pressure is less than baseline, assess for orthostatic decreases in blood pressure and increases in heart rate that would suggest dehydration. • Continue to give angiotensin-converting enzyme inhibitors and other afterload reducers if hypotension is asymptomatic. • If patient is symptomatic on standing, keep on bed rest until orthostasis resolves. • If patient is orthostatic, symptomatic, and BUN and creatinine levels are elevated, hold diuretics and consider giving intravenous normal saline solution.
Fluids/Electrolytes	
Euvolemia will be achieved. Euvolemia evidenced by: 1. Absence of peripheral edema 2. Absence of ascites 3. Documented dry weight 4. Baseline BUN and creatinine 5. Moist mucous membranes	• Administer loop diuretic sufficient to produce 1 liter of urine output within 2 h of administration. • Obtain daily weights. • Strive for a weight loss of 1–2 kg/d until dry weight is achieved. • Monitor electrolytes at least daily. • Replenish potassium, magnesium, and calcium as needed. • Measure serum albumin. • If inadequate response to loop diuretics, add metolazone or inotropes as above. • Report new or worsened rales to physician.
Teaching/Discharge Planning	
Rehospitalization will be prevented.	• Assess the patient's understanding of medication regimen. • Assess the patient's reading ability before giving written instructions. • Include a family member in the discussions if the patient has trouble reading, seeing, or remembering. • Consider a means of preparing medications so that the patient has to open only one container each day. • Teach the patient the importance of daily weights to follow fluid balance. • Have the patient weigh himself each day and record. Call physician if weight is 3–5 pounds over baseline. • Have the patient or family member repeat early signs and symptoms of worsening heart failure and when to call physician for them. • Teach the patient about foods that have a high sodium content. • Encourage the patient to abstain from alcohol. • Encourage the patient to walk and stay as active as possible. • Consider case management referral or social work referral if the patient has multiple admissions or problems obtaining medications.

▲ Clinical Applicability Challenges

CASE STUDY

A.J. is a 64-year-old farmer who had a septal myocardial infarction (MI) 4 years ago and has had chronic heart failure since then. His ejection fraction is 36%, and on his current medical regimen, he is considered in a New York Heart Association class II. In his compensated state, he is able to continue farming, care for his cows and chickens, and meet his responsibilities without problem, although he has slowed his pace since the MI. In the interim, he has developed type 2 diabetes which is well controlled with glypizide 5 mg twice a day by mouth. His most recent HbgA1c was 5.8%. Over the last 3 weeks, he has begun to get fatigued more easily with the same amount of effort. He notes that his ankles are swollen and have not gone down over night as they usually do and that his abdomen has increased in size to the point he cannot fasten his trousers. Last night he was unable to lie down in bed because he could not breathe; he slept in his recliner. His wife insisted that he see his cardiologist today. A.J.'s ECG demonstrated a new inferior MI and a left bundle branch block with a QRS duration of 162 milliseconds. He is admitted to the Coronary Care Unit for a diagnostic evaluation including cardiac catheterization and treatment for his acute decompensated heart failure (ADHF). A.J. does not weigh himself regularly at home and he was surprised to find his weight had increased 43 pounds since his last physician visit 4 weeks ago. His physical examination was significant for a new mitral regurgitation murmur (II/VI), an S3, jugular venous distention to his earlobe, and ascites. He did not have crackles or decreased breath sounds.

Vital signs are blood pressure 88/46, pulse rate 76, respiratory rate 28, and temperature 98.8°F.

The cardiac catheterization demonstrated a mid-right coronary artery occlusion of 95%, which was treated with a drug-eluting stent. His cardiac index after the stent placement was 3.1 L/min/m^2. Furthermore, his cardiologist ordered a new echocardiogram that demonstrated a decrease in his ejection fraction to 26% with ventricular dysynchrony. He will also receive an implantable cardioverter–defibrillator with a biventricular pacemaker during this hospitalization.

His significant laboratory values on admission were:

Glucose 186 mg/dL
Na 128 mg/dL
K 5.8 mEq/L
BUN 68 mg/dL
Creatinine 2.1 mg/dL
t bilirubin 3.8 mg/dL
albumin 3.8 g/dL
alkaline phosphatase 160 units/L

1. Silent MIs are common in patients with diabetes. What effect did having a new MI have on the increase in symptoms and decompensation A.J. experienced?

2. A.J. has ventricular dysynchrony. How will having a biventricular pacemaker improve his symptoms?

3. What specific things can A.J. do to decrease the likelihood that he will be surprised by a large weight gain associated with ADHF?

References

1. Heart Disease and Stroke Statistics—2010 Update: A report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation 121:e46–e215, 2010
2. 2009 Writing Group to review new evidence and update the 2005 Guideline for the Management of Patients with Chronic Heart Failure Writing on Behalf of the 2005 Heart Failure Writing Committee; Jessup M, Abraham WT, Casey DE, Feldman AM, et al: 2009 focused update: ACCF/AHA Guidelines for the Diagnosis and Management of Heart Failure in Adults: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines: Developed in collaboration with the International Society for Heart and Lung Transplantation. Circulation 119: 1977–2016, 2009
3. Whellan DJ, Greiner M A, Schulman KA, et al: Costs of inpatient care among Medicare beneficiaries with heart failure, 2001 to 2004. Circ Cardiovasc Qual Outcomes 3:33–40, 2010
4. New York Heart Association: Diseases of the Heart and Blood Vessels: Nomenclature and Criteria for Diagnosis, 6th ed. Boston, MA: Little, Brown, 1964
5. Harding SE: The failing cardiomyocyte. Heart Fail Clin 1(2):171–181, 2005
6. Packer M: Evolution of the neurohormonal hypothesis to explain the progression of chronic heart failure. Eur Heart J 16(Suppl f): 4–6, 1995
7. Tang WHW, Francis GS: Neurohormonal upregulation in heart failure. Heart Fail Clin 1(1):1–9, 2005
8. Miyasaka Y, Barnes ME, Gersh BJ, et al: Incidence and mortality risk of congestive heart failure in atrial fibrillation patients: A community-based study over two decades. Eur Heart J 27(8):936–941, 2006
9. Boriani G, Diemberger I, Martignani C, et al: The epidemiological burden of atrial fibrillation: A challenge for clinicians and health care systems. Eur Heart J 27(8):893–894, 2006
10. Stockburger M, Krebs A, Nitardy A, et al: Survival and appropriate device interventions in recipients of cardioverter defibrillators implanted for the primary versus secondary prevention of sudden cardiac death. Pacing Clin Electrophysiol 32(Suppl 1):S16–S20, 2009
11. Daniel MB, Nelson CL, Anstrom KJ, et al; for the SCD-HeFT Investigators: Cost-effectiveness of defibrillator therapy or amiodarone in chronic stable heart failure: Results from the sudden cardiac death in heart failure trial (SCD-HeFT). Circulation 114(2):135–142, 2006

12. Nieminen MS, Harjola VP: Definition and epidemiology of acute heart failure syndromes. Am J Cardiol 96(6A);5G–10G, 2005
13. Gheorghiade M, Zannad F, Sopko G, et al: Acute heart failure syndromes: Current state and framework for future research. Circulation 112(25):3958–3968, 2005
14. Offer A, Reisfeld D, Sberro H, et al: Implications of Cheyne-Stokes breathing in advanced systolic heart failure. Clin Cardiol 33(3):E8–E12, 2010
15. Maisel A: B-type natriuretic peptide levels: A potential "white count" for congestive heart failure. J Card Fail 7:183–193, 2001
16. Wright GA, Struthers AD: Natriuretic peptides as a prognostic marker and therapeutic target in heart failure. Heart 92(2):149–151, 2006
17. Stevenson LW; the ESCAPE investigators and ESCAE Study Coordinators: Evaluation study of congestive heart failure and pulmonary artery catheterization effectiveness: The ESCAPE trial. JAMA 294(13): 1625–1633, 2005
18. Gheorghiade M, Follath F, Ponikowski P, et al: Assessing and grading congestion in acute heart failure: A scientific statement from the acute heart failure committee of the heart failure association of the European Society of Cardiology and endorsed by the European Society of Intensive Care Medicine. Eur J Heart Fail 12:423–433, 2010
19. Gupta S, Neyses L: Diuretic usage in heart failure: A continuing conundrum in 2005. Eur Heart J 26(7):644–649, 2005
20. Eshaghian S, Horwich TB, Fonarow GC: Relation of loop diuretic dose to mortality in advanced heart failure. Am J Cardiol 97:1759–1764, 2006
21. Nohria A, Tsang SW, Fang JC, et al: Clinical assessment identifies hemodynamic profiles that predict outcomes in patients admitted with heart failure. J Am Coll Cardiol 41(10):1797–1804, 2003
22. Joseph SM, Cedars AM, Ewald GA, et al: Acute decompensated heart failure: Contemporary medical management. Tex Heart Inst J 36(6): 510–520, 2009
23. Mehra MR: Optimizing outcomes in the patient with acute decompensated heart failure. Am Heart J 151(3):571–579, 2006
24. Moe GW: B-type natriuretic peptide in heart failure. Curr Opin Cardiol 5(4):385–391, 2006
25. Noviasky JA: Controversy and conflict in the treatment of acute decompensated heart failure: Limited role for nesiritide. Pharmacotherapy 27(5):626–632, 2007
26. Hernandez A, Shea AM, Milano CA, et al: Long-term outcomes and costs of ventricular assist devices among Medicare beneficiaries. JAMA 300(20):2398–2406, 2008
27. Garg R, Yusuf S: Overview of randomized trials of angiotensin-converting enzyme inhibitors on mortality and morbidity in patients with heart failure. Collaborative Group on ACE Inhibitor Trials. JAMA 273(18):1450–1456, 1995
28. Nicklas JM, Cohn JN, Pitt B: What does ATLAS really tell us about "high" dose angiotensin-converting enzyme inhibition in heart failure? J Card Fail 6(2):165–168, 2000
29. Arnlov J, Ramachandran SV: Neurohormonal activation in populations susceptible to heart failure. Heart Fail Clin 1(1):11–23, 2005
30. Solomon SD, Rice MM, Jablonski KA, et al; for the Prevention of Events With ACE Inhibition (PEACE) Investigators: Renal function and effectiveness of angiotensin-converting enzyme inhibitor therapy in patients with stable coronary disease in the prevention of events with ACE inhibition (PEACE) trial. Circulation 114(1):16–31, 2006
31. The Digitalis Investigation Group: The effect of digoxin on mortality and morbidity in patients with heart failure. N Engl J Med 336(8): 525–533, 1977
32. Pitt B, Zannad F, Remme WJ, et al; for the Randomized Aldactone Evaluation Study Investigators: The effect of spironolactone on morbidity and mortality in patients with severe heart failure. N Engl J Med 341(10):709–717, 1999
33. Clelend JGF, Huan L, Windram J: Are there clinically important differences between beta-blockers in heart failure? Heart Fail Clin 1(1):57–66, 2005
34. Chobanian AV, Bakris GL, Black HR, et al: National High Blood Pressure Education Coordinating Committee: The seventh report of the joint national committee for prevention, detection, evaluation, and treatment of high blood pressure. The JNC-7 Report. JAMA 289(19):2560–2571, 2003
35. Bitar F, Akhter MW, Khan S, et al: Survey of the use of organic nitrates for the treatment of chronic congestive heart failure in the United States. Am J Cardiol 94:1465–1468, 2004
36. Bean MK, Gibson D, Flattery M, et al: Psychosocial factors, quality of life and psychological distress: Ethnic differences in patients with heart failure. Prog Cardiovasc Nursing 24:131–140, 2009
37. Riegel B, Moser DK, Anker SD, et al: State of the science: Promoting self-care in persons with heart failure. A statement from the American Heart Association. Circulation 120:1141–1163, 2009
38. Heart Failure Society of America: 2010 Comprehensive heart failure practice guideline executive summary. J Cardiac Fail 16(6):475–539, 2010
39. Tarcho JA: Biventricular pacing. N Engl J Med 355(3):288–294, 2006
40. McAlister FA, Ezekowitz J, Dryden DM, et al: Cardiac resynchronization therapy and implantable cardiac defibrillators in left ventricular systolic dysfunction. Evidence report/technology assessment No 152 (Prepared by the University of Alberta Evidence-Based Practice Center under Contract No 290-02-0023). AHRQ Publication No 07-E009. Rockville, MD: Agency for Healthcare Research and Quality, June 2007.
41. Rich MW: Heart failure disease management: A critical review. J Card Fail 5(1):64–75, 1999
42. Inglis SC, Clark RA, Cleland JGF, et al: Structured telephone support or telemonitoring programs for patients with chronic heart failure. Cochrane Database Syst Rev 4(8):CD007228, 2010. DOI: 10.1002/14651858.CD007228.pub3
43. Stewart S, Horowitz JD: Home-based intervention in congestive heart failure: Long-term implications on readmission and survival. Circulation 105:2861–2866, 2002

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

21 Acute Myocardial Infarction

Patricia Gonce Morton

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Explain the pathophysiology and risk factors for atherosclerosis.
2. Describe the classification, assessment, and management of patients with angina pectoris.
3. Compare and contrast the pathophysiological principles and assessment findings of a patient with angina pectoris versus a patient with a myocardial infarction (MI).
4. Discuss the diagnostic tests used for a patient with an MI.
5. Summarize principles of managing the patient with an MI in the early phase, intensive care phase, and intermediate care phase of management.
6. Describe the complications for a patient with an MI.
7. Explain the principles of cardiac rehabilitation and patient education.

Cardiovascular disease is a significant global health problem. Worldwide, an estimated 17.3 million people died from cardiovascular disease in 2008.[1] About 80% of these deaths occur in low- and middle-income countries.[1] In the United States, cardiovascular disease continues to be the leading cause of death for men and women.[2] About 2,200 Americans die each day from cardiovascular disease; this represents an average of one death every 39 seconds.[3]

Among the white population, 11.9% have cardiovascular disease; among blacks or African Americans, the rate is 11.2%; and among Latinos or Hispanics, 8.5% have cardiovascular disease. The overall death rate from cardiovascular disease is 251.2 per 100,000.[3] For white males the death rate is 294 per 100,000, and for black males the death rate is 405.9 per 100,000. For white females the overall death rate from cardiovascular disease is 205.7 per 100,000, and for black females the overall death rate from cardiovascular disease is 286.1 per 100,000.[3]

Of those who die from cardiovascular disease, the majority die as a result of coronary heart disease (myocardial infarction [MI] and angina pectoris).[3] About every 25 seconds, an American has a coronary event, and about every minute, a person dies from one.[3] Coronary heart disease caused about one in every six deaths in the United States in 2007.[3] Every year, about 785,000 Americans will have a new coronary attack and about 470,000 will have a recurrent attack.[3] An additional 195,000 silent first MIs occur each year.[3]

Overwhelming as the mortality and morbidity statistics appear, much progress has been made in the prevention, diagnosis, and management of cardiovascular disease. Since the Framingham Study of risk factors in 1951 and the development of coronary care units in the 1960s, the critical care nurse has played a major role in helping to reduce the mortality associated with heart disease. The critical care nurse uses advanced assessment skills, rapid decision making, and therapeutic interventions to treat the patient in the acute phase of cardiovascular disease. Patient education and psychological support provided by the nurse have enabled patients and their families to return home and maximize their health status.

▲ Atherosclerosis

Atherosclerosis is a major cause of cardiovascular disease. The term atherosclerosis comes from the Greek words *athere*, meaning "gruel" or "paste," and *sclerosis*, meaning "hardness."

Pathophysiological Principles

Atherosclerosis is a complex, insidious process, beginning long before symptoms occur. Although the process is not completely understood, scientific evidence suggests that it begins when the inner, protective layer of the artery (endothelium) is damaged. Three known causes of the damage include elevated levels of cholesterol and triglycerides in the blood, hypertension, and cigarette smoking.

Gradually, as fatty substances, cholesterol, cellular waste products, calcium, and fibrin pass through the vessel, they are deposited in the inner lining of an artery. As a result of the deposition of these materials, a lipid plaque with a fibrous

covering, also known as an atheroma, builds up, and blood flow in the artery becomes partially or completely blocked.

The injury to the vessel and the resulting accumulation of these substances in the inner lining of the artery cause white blood cells, smooth muscle cells, and platelets to aggregate at the site. As a result, a matrix of collagen and elastic fibers form, and the endothelium becomes much thicker. The core of the fibrous plaque can become necrotic, and hemorrhage and calcification may result. A thrombosis may also form, thus contributing even more to the blockage of the vessel lumen (Fig. 21-1). These fibrous plaques are most often found in the coronary, popliteal, and internal carotid arteries and in the abdominal aorta.

Because of the fibrous plaque, the amount of blood flow through the artery is reduced, resulting in decreased supply of oxygen to tissues. However, symptoms often do not occur until 75% or more of the blood supply to the area is occluded. The occurrence of symptoms may depend to an extent on the development of collateral circulation. Collateral vessels are small arteries that connect two larger arteries or different segments of the same artery. Under normal conditions, these collateral arteries carry very little of the blood flow. As the larger artery gradually occludes, pressure builds on the proximal side of the occlusion. As a result, flow is redirected through the collateral vessels, which enlarge and dilate over time (Fig. 21-2). Blood is then allowed to flow around an area of blockage through these alternate routes.

Scientific advances have highlighted the role of inflammation in the pathophysiological process of atherosclerosis. The classic signs and symptoms of inflammation include redness, pain, heat, and swelling. They indicate that the injured tissue is in the process of restoring homeostasis, which includes three phases: vasodilation and increased permeability of the blood vessels, emigration of phagocytes from the blood into the tissue, and tissue repair. This process of restoring homeostasis is meant to be protective, but in the setting of atherosclerosis, the process has been found to be destructive. The atherosclerotic plaque continues to develop, aided by inflammatory molecules, and a fibrous cap forms over the lipid core. As the cap matures, inflammatory substances weaken the cap and cause it to rupture. Once the cap is ruptured, the coagulation cascade is initiated, and a clot is formed, resulting in obstruction of blood flow in the vessel.

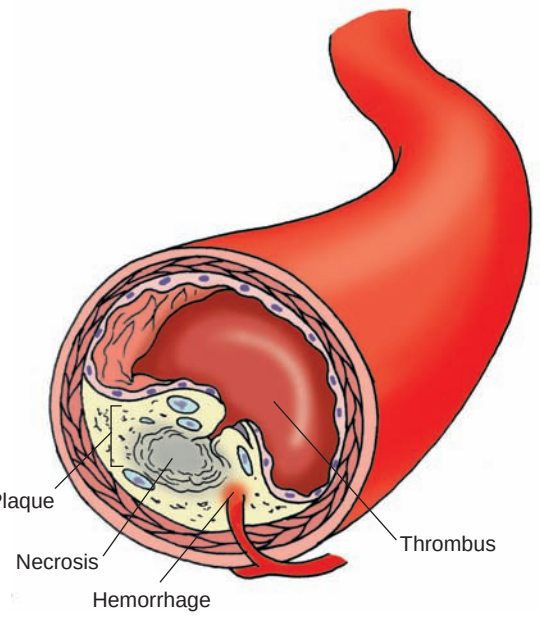


FIGURE 21-1 ▲ Thrombosis of an atherosclerotic plaque. It may partially or completely occlude the lumen of the vessel. (From Bullock BL: Pathophysiology: Adaptations and Alterations in Function, 4th ed. Philadelphia, PA: Lippincott-Raven, 1996.)

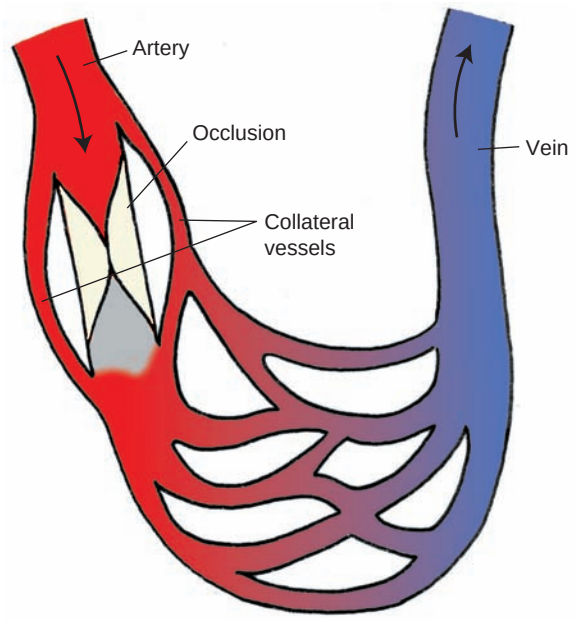


FIGURE 21-2 ▲ Collateral circulation can develop for slowly developing lesions to provide myocardial blood flow until the atherosclerosis progresses beyond the limits of collateral supply. (From Bullock BL: Pathophysiology: Adaptations and Alterations in Function, 4th ed. Philadelphia, PA: Lippincott-Raven, 1996.)

Markers of inflammation are now being used to assess the risk for atherosclerosis. C-reactive protein (CRP) is an acute-phase protein that increases during systemic inflammation. A newer, more sensitive CRP blood test called a highly sensitive C-reactive protein (hs-CRP) can be used to determine the risk for heart disease. High levels of hs-CRP have consistently predicted recurrent coronary events in patients with unstable angina and MI. An hs-CRP less than 1.0 mg/dL is associated with a low risk for developing cardiovascular disease, a value between 1.0 and 3.0 mg/dL with a moderate risk, and a value greater than 3.0 mg/dL with a high risk.[4]

Risk Factors

The cause of atherosclerosis is not clearly known. Through epidemiological studies, risk factors for the development of atherosclerosis have been identified. These risk factors are usually classified into two groups: major uncontrollable risk factors and major risk factors that can be modified, treated, or controlled. Major risk factors are those that have been shown through research to increase significantly the risk of cardiovascular disease. Other risk factors are known to be associated with an increased risk for cardiovascular disease, but their significance and prevalence are still under investigation. The more risk factors a patient has, the greater the chance of developing coronary heart disease.[5]

Major Uncontrollable Risk Factors

AGE. There is an increased incidence of all types of atherosclerotic disease with aging. More than 83% of people who die from coronary heart disease are 65 years of age and older. At older ages, women who have an MI are more likely than men to die from it within a few weeks.[5]

HEREDITY (INCLUDING RACE). The tendency to develop atherosclerosis seems to run in families, although the risk is presumed to be a combination of environmental and genetic influences. Even when other risk factors are controlled, the chance for developing coronary artery disease increases when there is a familial tendency. Most people with a strong family history of cardiovascular disease also have one or more additional risk factors. Rates of cardiovascular disease are higher in African Americans, Mexican Americans, Native Americans, Native Hawaiians, and some Asian Americans. High blood pressure, which is more severe in African Americans than Caucasians, contributes to a higher rate of heart disease. Higher rates of obesity and diabetes in Mexican Americans, American Indians, and Native Hawaiians help account for their higher rates of cardiovascular disease.[5]

GENDER. Men have a greater risk for coronary artery disease than woman, and men have MIs at a younger age. However, after menopause, the death rate from coronary disease rises in women, but it never reaches the risk level of men.[5]

Major Risk Factors That Can Be Modified, Treated, or Controlled

CIGARETTE SMOKING. A smoker's risk for an MI is two to four times that of a nonsmoker. Smoking is the biggest risk factor for sudden cardiac death. Cigarette smoking also acts with other risk factors to increase greatly the risk of coronary heart disease. Exposure to environmental smoke also increases the risk for heart disease in nonsmokers.[5]

HIGH BLOOD CHOLESTEROL. High cholesterol levels increase the risk for coronary artery disease. When other risk factors are present, the risk is even greater. Middle-aged adults with a total blood cholesterol level below 200 mg/dL have a relatively low risk for coronary artery disease. A total blood cholesterol level in the range of 200 to 239 mg/dL represents a moderate but increasing risk. When the level rises above 240 mg/dL, the risk for coronary artery disease is about double.[6]

Most cholesterol in the blood is carried in low-density lipoprotein (LDL), often called "bad" cholesterol. This type of cholesterol is deposited in the artery walls, and high blood levels of LDL increase the risk for coronary heart disease. An LDL level of less than 100 mg/dL is optimal. For patients classified as very high risk for MI, a therapeutic goal of less than 70 mg/dL may be recommended.[6]

High-density cholesterol (HDL) removes cholesterol from tissues and transports the excess cholesterol back to the liver, where it is metabolized. For this reason, HDL is often called "good" cholesterol. A low level of HDL (<40 mg/dL) is associated with a higher risk for coronary artery disease.

Triglyceride is the most common type of fat in the body, and the normal levels vary by age and sex. The rate of atherosclerotic development seems to be accelerated by a combination of high triglycerides with either high amounts of LDL or low amounts of HDL.

Patients at very high risk for MI are defined as those patients with cardiovascular disease with either multiple risk factors (especially diabetes) or severe or poorly controlled risk factors or metabolic syndrome (a group of risk factors associated with obesity including high triglycerides and low high-density lipids [HDL]). High-risk patients are those who have coronary heart disease or disease of the blood vessels of the brain or extremities, or diabetes, or multiple (two or more) risk factors resulting in a greater than 20% chance of having a heart attack within 10 years. Moderately high-risk patients are those with two or more risk factors for coronary heart disease with a 10% to 20% risk of heart attack within 10 years. Patients with lower/moderate risk of having an MI are those with two or more risk factors plus an under 10% risk of a heart attack in 10 years or those with zero to one risk factor.[6]

HYPERTENSION. Hypertension is a major risk factor that is termed the silent killer because it has no specific symptoms and no early warning signs. About 33.5% of U.S. adults over the age of 20 have hypertension.[3] The prevalence of hypertension is nearly equal in men and woman.[3]

In the United States, the prevalence of hypertension in blacks is among the highest in the world at 44%, and this value is increasing. Compared with whites, blacks develop hypertension earlier in life, with average blood pressures that are significantly higher.[2] When the risk factors of obesity, smoking, high cholesterol level, or diabetes occur with hypertension, the risk of heart disease or stroke increases several times.[5]

PHYSICAL INACTIVITY. A lack of physical activity plays a significant role in the development of heart disease. When lack of regular exercise is combined with overeating and obesity, high cholesterol can result and further increase the risk for heart disease. Regular moderate to vigorous physical activity helps prevent cardiovascular disease. Even moderate-intensity exercise helps prevent heart disease if done regularly and long-term.[5]

OBESITY AND OVERWEIGHT. Approximately 67% of the U.S. adult population is overweight, and about 33.7% is considered obese.[3] Obesity and excess weight are associated with an increased mortality rate from coronary artery disease and stroke. Excess weight is also linked with an increased incidence of hypertension, insulin resistance, diabetes, and dyslipidemia. Central obesity (intra-abdominal fat) appears to be a stronger predictor of cardiovascular disease than peripheral or subcutaneous obesity. The waist measurement and the body mass index (a measure of weight relative to height) are the recommended means to estimate a person's body fat. A higher risk for cardiovascular disease is found for women with a waist greater than 35 inch and for men with a waist greater than 40 inch.[3]

DIABETES MELLITUS. Diabetes mellitus is associated with a markedly increased risk for cardiovascular disease. This increased risk occurs even if the person maintains control of blood glucose levels. The risk of heart disease is even greater if blood glucose is poorly controlled. About 75% of people with diabetes die of some form of heart or blood vessel disease.[3,5]

Contributing Risk Factors

STRESS. A person's response to stress may be a contributing factor to cardiovascular disease. The behaviors that a person engages in when under stress (such as smoking and overeating) may contribute to the risk for cardiovascular disease.[3,5]

EXCESSIVE ALCOHOL INTAKE. The excessive intake of alcohol has been associated with hypertension, heart failure, and stroke. The risk for heart disease in moderate drinkers is lower than in nondrinkers. Moderate drinking means one drink for a woman per day and two drinks for a man per day. One drink is defined as 1.5 fluid ounces of 80-proof spirits, 1 fluid ounce of 100-proof spirits, 4 fluid ounces of wine, or 12 fluid ounces of beer.[5]

▲ Acute Coronary Syndrome

The term acute coronary syndrome (ACS) is used to describe patients with clinical symptoms compatible with acute myocardial ischemia. This term includes unstable angina and acute myocardial infarction (AMI). Unstable angina refers to unexpected chest pain or discomfort that usually occurs while at rest. Patients with MI are further classified into one of two groups: those with ST-segment elevation MI (STEMI) and those with non–ST-segment elevation MI (NSTEMI). The pathophysiologic origins and clinical presentations of unstable angina and NSTEMI are similar, but differ in severity. An NSTEMI is diagnosed when the ischemia is severe enough to cause myocardial damage and the release of a biomarker indicating myocardial necrosis into the circulation. However, on the electrocardiogram (ECG), ST segments do not elevate. For a patient with unstable angina, biomarkers are not detected in the circulation hours after the initial onset of ischemic pain. Unstable angina can present as rest angina usually lasting more than 20 minutes; new-onset (<2 months) severe angina; and a crescendo pattern of occurrence that increases in intensity, duration, frequency, or any combination of these factors. A patient with an STEMI shows the ST changes on the ECG and has detectable biomarkers in the circulation.[7] About two thirds of patients with an AMI have a non–ST-segment MI and about one third have an STEMI.[3]

▲ Angina Pectoris

The term angina comes from the Latin word meaning "to choke." Angina pectoris is the term used to describe chest pain or discomfort that result from coronary artery disease. The patient may describe the sensation as pressure, fullness, squeezing, heaviness, or pain.

Pathophysiological Principles

Angina pectoris is caused by transient, reversible myocardial ischemia precipitated by an imbalance between myocardial oxygen demand and myocardial oxygen supply. In most cases, angina pectoris is the result of a reduced oxygen supply. The most common cause of a reduced supply of oxygen is atherosclerotic narrowing of the coronary arteries. A nonocclusive thrombus develops on a disrupted atherosclerotic plaque, resulting in a reduction in myocardial perfusion. As blood flow to the myocardium decreases, autoregulation of coronary blood flow occurs as a compensatory mechanism. The smooth muscles of the arterioles relax, thus decreasing resistance to blood flow in the arteriolar bed. When this compensatory mechanism can no longer meet the metabolic demands, myocardial ischemia occurs, and the person feels pain.

A less common cause of unstable angina is dynamic obstruction resulting from intense focal spasm of a coronary artery. The spasm is caused by hypercontractility of vascular smooth muscle, endothelial dysfunction, or abnormal constriction of small resistance vessels. As a result of the spasm, perfusion to the myocardium is interrupted, thus reducing the supply of oxygen.

Arterial inflammation may be another cause of decreased oxygen supply that results in unstable angina. The inflammatory process may cause arterial narrowing, plaque destabilization, rupture, and thrombogenesis. More recent research has contributed to a better understanding of the role of inflammation in ACSs.

A marked increase in oxygen demand is another cause of unstable angina. Conditions such as fever, tachycardia, and thyrotoxicosis may result in an increased oxygen demand that is unable to be met, especially if the patient has underlying coronary artery disease.[8,9]

When the balance between oxygen supply and demand is not met, the myocardial tissue's need for oxygen and nutrients continues. The same work of pumping blood must be accomplished with less available energy and oxygen. The tissue that depends on the blood supply becomes ischemic as it functions with less oxygenated blood. Anaerobic metabolism can provide only 6% of the total energy needed. Glucose uptake by the cells is markedly increased as glycogen and adenosine triphosphate stores are depleted. Potassium rapidly moves out of the myocardial cells during ischemia. An acidotic cellular bath develops, further compromising cellular metabolism.

Classification of Angina

Many terms are used clinically to describe angina. Stable angina (also known as chronic stable angina, classic angina, or exertional angina) is a term used to describe paroxysmal substernal pain that is usually predictable. The pain occurs with physical exertion or emotional stress and is relieved by rest or nitroglycerin.[10]

Unstable angina, also called preinfarction angina or crescendo angina, refers to cardiac chest pain that usually occurs while at rest. The patient with unstable angina has more prolonged and severe chest discomfort than the person with stable angina. Unstable angina is a type of ACS and requires immediate treatment because the patient is at increased risk for AMI, cardiac dysrhythmias, or cardiac sudden death.[10]

Variant angina, also known as Prinzmetal's angina or vasospastic angina, is a form of unstable angina. Variant angina usually occurs at rest, most often between midnight and 8:00 AM. It does not usually occur after exertion or emotional stress. Variant angina is the result of coronary artery

BOX 21-1 Grading of Angina Pectoris by the Canadian Cardiovascular Society Classification System

Class I: Ordinary physical activity does not cause angina, such as walking and climbing stairs. Angina occurs with strenuous, rapid, or prolonged exertion at work or recreation.

Class II: Slight limitation of ordinary activity occurs. Angina occurs when walking or climbing stairs rapidly, walking uphill, walking or climbing stairs after meals, in cold, in wind, under emotional stress, or during the few hours after awakening. Angina occurs when walking more than two level blocks and climbing more than one flight of ordinary stairs at a normal pace and in normal conditions.

Class III: Ordinary physical activity is markedly limited. Angina occurs when walking one to two level blocks and climbing one flight of stairs in normal conditions and at a normal pace.

Class IV: Physical activity without discomfort is impossible; anginal symptoms may be present at rest.

From Campeau L: Grading of angina pectoris [letter]. Circulation 54:522–523, 1976; copyright 1976, American Heart Association, Inc, used with permission.

spasm. Most people who experience variant angina have severe coronary atherosclerosis of at least one major coronary artery, and the spasm occurs very near the area of blockage.[10]

The Canadian Cardiovascular Society also has proposed a classification system for grading for angina. Each stage of the four-class system is described in Box 21-1.

Assessment

History

The five most important factors that indicate a likelihood of ischemia from coronary artery disease are obtained rapidly during the health history. These factors include a description of the symptoms, information about a prior history of coronary artery disease, the patient's sex and age, and the number of risk factors present.[8,9]

The nurse uses the NOPQRST method of pain assessment when taking the patient's history. For a review of the assessment questions, see Box 17-1. After determining the patient's normal baseline, the nurse asks about the time of onset of the pain. The nurse determines causes (provocative) of the pain and any measures the patient has used to relieve the pain (palliative), such as rest or nitroglycerin. The pain of angina is often brought on by exertion or emotion. It may also occur after meals, exposure to cold, and at rest. Patients with angina often obtain relief from the pain with rest or by taking sublingual nitroglycerin. As the angina becomes more severe (unstable angina), the pain may occur at rest or be caused by less exertion and is no longer relieved with rest or sublingual nitroglycerin. The quality of anginal pain is frequently described as deep, poorly localized chest or arm discomfort. Patients often describe heaviness, squeezing, choking, or smothering sensations. When asked about region and radiation of the pain, patients report substernal, left-sided chest, or epigastric pain that may radiate to the left arm, neck, back, or jaw. The severity of the pain is evaluated by asking the patient to rate the pain on a scale of 0 to 10, with 10 being the worst pain they have experienced. Additional information is obtained related to time. The nurse asks how long the pain lasts, how frequently it occurs, and the time of day it occurs. Finally, the nurse asks about associated symptoms, such as dyspnea, nausea, vomiting, and diaphoresis. Box 21-2 summarizes the assessment findings for a patient with myocardial ischemia. Based on the information obtained, the angina may be classified as one of three principal presentations: rest angina, new-onset (<2 months) severe angina, and increasing angina (in intensity, duration, or frequency).[8,9]

The older patient, especially women, who experiences angina may have a different presentation because of changes in neuroreceptors. Considerations for the older patient are described in Box 21-3.

BOX 21-2 The NOPQRST Characteristics of Chest Pain Due to Myocardial Ischemia

N—Normal
- The patient's baseline before the onset of the pain

O—Onset
- The time when the pain/discomfort started

P—Precipitating and Palliative Factors

Precipitating
- Exercise
- Exercise after a large meal
- Exertion
- Walking on a cold or windy day
- Cold weather
- Stress or anxiety
- Anger
- Fear

Palliative
- Stop exercise.
- Sit down and rest.
- Use sublingual nitroglycerin; pain of myocardial infarction (MI) is often not relieved by sublingual nitroglycerin.

Q—Quality
- Heaviness
- Tightness
- Squeezing
- Choking
- Suffocating
- Vise-like

R—Region and Radiation
- Substernal with radiation to the back, left arm, neck, or jaw
- Upper chest
- Epigastric
- Left shoulder
- Intrascapular

S—Severity
- Pain rated on a scale of 0 to 10, with 10 being the worst pain ever experienced, often rated as 5 or above

T—Time
- Pain lasts from 30 seconds to 30 minutes.
- Pain can last longer than 30 minutes for unstable angina or MI.

BOX 21-3 CONSIDERATIONS FOR THE OLDER PATIENT

Acute Coronary Syndrome

Coronary artery disease is more common and more severe in the older patient. Older patients often present with special problems because of their numerous comorbidities, such as diminished β-sympathetic response, increased cardiac afterload due to decreased arterial compliance and arterial hypertension, cardiac hypertrophy, and ventricular diastolic dysfunction.[11]

The older patient is more likely to present with atypical symptoms such as dyspnea, confusion, weakness, or fainting rather than with typical substernal chest pain. Because of differences in amount and distribution of subcutaneous fat, the older person may develop anginal symptoms more quickly when exposed to cold. The older person should be taught to dress in warm clothing and to recognize feelings of weakness, shortness of breath, or fainting as possible indicators of angina.

Physical Examination

The physical examination helps determine the cause of the pain, detect comorbid conditions, and assess any hemodynamic consequences of the pain. When the vital signs are taken, the nurse should measure the blood pressure in both arms of the patient. If the physical examination is performed during an anginal episode, the patient may present with tachycardia and pulsus alternans. Pulsus alternans is a physical finding characterized by a regular alternation of the force of the arterial pulse. During the initial phase of an anginal episode, the patient may be hypertensive or hypotensive. The patient may exhibit pallor with cold, clammy skin. On further examination of the skin, the nurse may detect xanthomas, which are yellow nodules or plaques, especially on the skin. Xanthomas may be indications of hypercholesterolemia. Carotid or femoral bruits may be auscultated, indicating the possible presence of obstructive cardiovascular disease. The nurse may hear a paradoxical split of S_2 or auscultate an S_3 heart sound. Both sounds are indicators of left ventricular failure. An S_4 may be heard, which is suggestive of decreased left ventricular compliance. Deficits in peripheral pulses may indicate peripheral vascular disease.

Diagnostic Tests

A 12-lead ECG is a standard diagnostic test for patients with angina and should be obtained immediately in patients with chest discomfort. During the anginal episode, the ECG may show T-wave inversions and ST-segment depressions in the ECG leads associated with the anatomical region of myocardial ischemia (Fig. 21-3). Transient ST-segment changes (0.05 mV or more) that occur during a symptomatic episode while at rest and that resolve when the patient is asymptomatic are highly suggestive of severe coronary artery disease.[8,9] Ectopic beats may also be present during an anginal episode. The ECG should be compared with previous ECGs. Between anginal episodes, the ECG may appear normal. Ambulatory ECG monitoring may be used to assist in the diagnosis of angina, especially for patients with angina at rest. The standard 12-lead ECG is a limited diagnostic tool because it does not provide adequate information about the posterior, lateral, and apical walls of the heart.[7] A normal ECG does not exclude the possibility of ACS.[7]

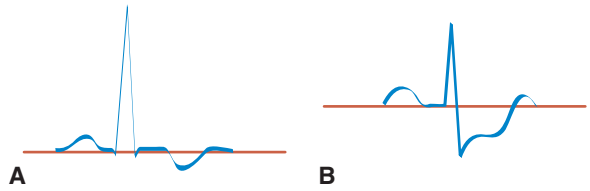


FIGURE 21-3 ▲ Inversion of T wave (**A**) and depression of ST segment (**B**). (From Bullock BL: Pathophysiology: Adaptations and Alterations in Function, 4th ed. Philadelphia, PA: Lippincott-Raven, 1996.)

Biochemical cardiac markers are useful in determining both the diagnosis and the prognosis of ACSs. For a more detailed discussion of cardiac markers, see Chapter 17. A cardiac-specific troponin (troponin T or troponin I) is the preferred marker to obtain in all patients who present with chest discomfort consistent with ACS. Troponin has replaced creatine kinase-MB (CK-MB) as the preferred biomarker for the diagnosis of myocardial necrosis.[7] However, troponin levels usually do not increase until at least 6 hours after the onset of symptoms.[7] For patients who present to the hospital within 6 hours of the onset of symptoms consistent with ACS, an early marker of cardiac injury, such as myoglobin, in conjunction with a late marker, such as troponin, may be obtained. If the patient has a negative cardiac marker within 6 hours of the onset of chest discomfort, another blood sample should be drawn in the 6- to 12-hour period after onset of chest discomfort.[8,9] Troponin levels remain elevated for between 5 and 14 days and therefore may be a useful biomarker for the patient who presents for evaluation several days after the onset of symptoms.[7] Additional blood tests include chemistry, complete blood count, and coagulation studies. A full lipid profile should be obtained within 24 hours of the onset of ACS.[6] Plasma markers of inflammation may also assist in the diagnosis of ACS. Elevated CRP levels detected by a high-sensitivity CRP test have been related to an increased risk of mortality.[7] A white blood cell count is another helpful marker of inflammation associated with higher mortality rates.[7]

Other diagnostic tests include exercise stress testing in which the ECG and blood pressure are monitored before, during, and after exercise. The exercise stress test is especially useful in risk stratification of patients. For patients who are unable to exercise, pharmacological stress testing may be done in which the medication increases myocardial oxygen demand while the patient remains inactive. Intravenous (IV) medications used for pharmacological stress testing include adenosine, dobutamine, and dipyridamole.

Cardiac imaging studies usually start with chest radiographs, although they have limited value in diagnosing coronary heart disease. Perfusion imaging can be used with exercise or pharmacological stress testing to detect perfusion defects. Positron emission tomography (PET) may be helpful in differentiating ischemic from infarcted myocardium. Echocardiography is performed to evaluate wall motion abnormalities and thickness, valvular function, and ejection fraction. Magnetic resonance imaging (MRI) and coronary computed tomographic angiography may be used to view structural cardiovascular abnormalities when other

diagnostic techniques (eg, the echocardiogram) are inconclusive or ambiguous.

Coronary angiography is an invasive diagnostic test that provides a definitive diagnosis of coronary artery disease. Results from coronary angiography are used to guide the decision whether to manage the patient medically or surgically. For further discussion of cardiovascular diagnostic tests, see Chapter 17.

Management

The goal of therapy for the patient with angina pectoris is to restore the balance between oxygen supply and oxygen demand. The nurse assesses the patient's vital signs and mental status frequently. The patient is placed on a cardiac monitor for ischemia and dysrhythmia detection. The patient is placed on bed rest until stabilized to minimize oxygen demands. Supplemental oxygen may be given to unstable patients to increase oxygen supply. A pulse oximeter and arterial blood gases are used to evaluate oxygenation status.

Pharmacological Therapy

Pharmacological therapy is an important component in managing patients with angina pectoris. The severity of symptoms, hemodynamic status of the patient, and medication history guide the drug regimen.

Nitroglycerin is a mainstay of therapy because it is a vasodilator that reduces myocardial oxygen demand by decreasing ventricular preload via vasodilation. Through vasodilation, nitroglycerin improves arterial and collateral flow to ischemic areas.[7] Nitroglycerin is used sublingually or as a spray for acute anginal attacks. If three sublingual tablets (0.4 mg) or spray taken 5 minutes apart (no more than three sprays in 15 minutes) does not relieve the pain of angina, IV nitroglycerin may be useful. IV nitroglycerin should be started at a rate of 10 mcg/min by continuous infusion and titrated up by 10 mcg/min every 3 to 5 minutes until some symptom or blood pressure response is noted. If signs and symptoms are relieved, there is no need to continue to increase the dose. However, if relief is not obtained, the dose can be increased until a blood pressure response is noted. A ceiling dose of 200 mcg/min is recommended. Once patients have been pain-free and have no other indications of ischemia for 12 to 24 hours, the IV nitroglycerin should be discontinued and replaced with oral or topical nitrates.[8,9]

Morphine sulfate is indicated for patients whose symptoms are not relieved after three serial sublingual nitroglycerin tablets or whose symptoms recur with adequate anti-ischemic therapy. Morphine is a potent analgesic and anxiolytic with hemodynamic benefits.[7] A dose of 1 to 5 mg IV is recommended to relieve symptoms and maintain comfort. The nurse carefully monitors the patient's respiratory rate and blood pressure, especially if the patient continues to receive IV nitroglycerin.[8,9]

β-Blockers may be used to decrease myocardial oxygen consumption by reducing myocardial contractility, sinus node rate, and atrioventricular (AV) node conduction velocity. The reduction in myocardial contractility reduces the work of the heart and decreases myocardial oxygen demand. The slowing of the heart rate helps increase the time for diastolic filling, thus improving blood flow to the coronary arteries. β-Blockers are started orally within the first 24 hours for patients with unstable angina and NSTEMI.[8,9]

Calcium channel blockers may be beneficial for the patient with unstable angina and NSTEMI. Calcium channel blockers decrease myocardial oxygen demand by decreasing afterload, contractility, and heart rate. Verapamil (Calan) and diltiazem (Cardizem) have been shown to have the greatest benefit. The nurse carefully monitors the patient for side effects, such as hypotension, worsening heart failure, bradycardia, and AV block. Calcium channel blockers can be administered to treat ischemia-related symptoms in patients unresponsive to or intolerant of nitrates and β-blockers.[8,9]

The combination of aspirin, an anticoagulant, and an additional antiplatelet drug is recommended for the patient with unstable angina or NSTEMI. Aspirin should be administered as soon as the diagnosis of unstable angina or NSTEMI is made or suspected, unless contraindicated. Clopidogrel (Plavix) is used if the patient is intolerant of asprin.[9] For additional antiplatelet therapy, thienopyridine (Ticlid) therapy is used and approved thienopyridine agents include clopidogrel and prasugrel (Effient).[9] Anticoagulant therapy also is recommended to modify the disease process and its consequences for the patient with unstable angina and NSTEMI. Anticoagulant drugs that are recommended include enoxaparin, unfractionated heparin, or fondaparinux (a factor XA inhibitor) and bivalirudin (direct thrombin inhibitor).[8,9]

For patients with pulmonary congestion or a left ventricular ejection fraction of 40% or less and in the absence of hypotension, an angiotensin-converting enzyme (ACE) inhibitor is administered orally within the first 24 hours. An angiotensin II receptor blocker is used for patients who cannot tolerate an ACE inhibitor.[8,9]

Invasive Therapy

Invasive therapy may be indicated for the management of patients with unstable angina. Intra-aortic balloon pump (IABP) support may be used in the critically ill patient to provide increased coronary artery perfusion and to decrease afterload. Percutaneous transluminal coronary angioplasty (PTCA) and stent placement may be used for treating patients with unstable angina. See Chapter 18 for a more detailed discussion of the IABP, PTCA, and stent placement. Coronary artery bypass grafting (CABG) is another invasive option for treatment. See Chapter 22 for a more detailed discussion of cardiac surgery.

Risk Factor Modification

Risk factor modification may help prevent an anginal episode or delay the worsening of existing angina. Patients should be encouraged to stop smoking, achieve or maintain optimal weight, and exercise daily. Diet and medications may be prescribed to control hypertension, diabetes, and hyperlipidemia. Patient education, including home care considerations, is essential for patients with angina pectoris. Patient education guidelines and home care considerations are described in Box 21-4.

BOX 21-4 TEACHING GUIDE Angina Pectoris

Activity and Exercise
- Participate in a daily program of exercise that does not precipitate pain.
- Alternate activity with periods of rest and moderate activity level as needed.

Diet
- Eat a well-balanced diet with an appropriate caloric intake.
- If obese, participate in a supervised weight-reduction program.
- Avoid activity immediately after meals.
- Restrict intake of caffeine because it can increase heart rate.
- Maintain a diet low in fat.

Smoking
- Participate in a smoking cessation program. Smoking can increase heart rate, blood pressure, and blood carbon monoxide levels.
- Avoid smoke-filled environments.

Cold Weather
- Avoid exposure to cold and windy weather. Exercise indoors when necessary.
- When outdoors, dress in warm clothing and cover mouth and nose with a scarf.
- Use a moderate pace when walking in cold weather.

Medications
- Carry sublingual nitroglycerin at all times.
- Keep the pills in a dark-colored glass bottle to protect them from sunlight.
- Do not place cotton in the bottle because the cotton will absorb the active ingredients of the medication.
- If pain occurs, place tablet under the tongue, stop activity, and wait for medication to dissolve. Take another tablet in 3 to 5 minutes if pain does not resolve.
- If pain continues, seek immediate care.
- Be aware of side effects of nitroglycerin, including headache, flushing, and dizziness.

▲ Myocardial Infarction

Prolonged ischemia caused by an imbalance between oxygen supply and oxygen demand causes MI. The prolonged ischemia causes irreversible cell damage and muscle death. Although multiple factors can contribute to the imbalance between oxygen supply and oxygen demand, the presence of a coronary artery thrombosis characterizes most MIs. In a classic investigation, DeWood et al[11] demonstrated that 87% of patients studied in the first 4 hours after onset of MI symptoms had a thrombotic occlusion. The incidence of thrombotic occlusion decreases to 65% at 12 to 24 hours.

MI can be determined from several different perspectives, including clinical, electrocardiographic, biochemical, imaging, and pathological. The European Society of Cardiology, the American College of Cardiology Foundation, the American Heart Association, and the World Heart Federation developed a joint consensus document for the redefinition of MI.[12] Their clinical classification of an AMI is shown in Box 21-5.

Pathophysiological Principles

Most patients who sustain an MI have coronary atherosclerosis. The thrombus formation occurs most often at the site of an atherosclerotic lesion, thus obstructing blood flow to the myocardial tissues. Plaque rupture is believed to be the triggering mechanism for the development of the thrombus in most patients with an MI. As mentioned previously, the role of inflammatory processes in the development of atherosclerotic plaque is an area of intense scientific investigation. Cardiovascular risk factors play a role in endothelial damage, resulting in endothelial dysfunction. The dysfunctioning endothelium contributes to the activation of the inflammatory response and the formation of atherosclerotic plaques. When the plaques rupture, a thrombus is formed at the site that can occlude blood flow, thus resulting in an MI. Figure 21-4 shows the atherosclerotic plaque in stable angina and in ACSs.

Irreversible damage to the myocardium can begin as early as 20 to 40 minutes after interruption of blood flow. However, the dynamic process of infarction may not be completed for several hours. Necrosis of tissue appears to occur in a sequential fashion. Reimer and associates demonstrated that cellular

BOX 21-5 Clinical Classification of Different Types of Myocardial Infarction

Type 1
Spontaneous MI related to ischemia due to a primary coronary event such as plaque erosion and/or rupture, fissuring, or dissection

Type 2
MI secondary to ischemia due to either increased oxygen demand or decreased supply (eg, coronary artery spasm, coronary embolism, anemia, dysrhythmias, hypertension, hypotension)

Type 3
Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggestive of myocardial ischemia, accompanied by presumably new ST-segment elevation, or new left bundle branch block, or evidence of fresh thrombus in a coronary artery by angiography and/or at autopsy, but death occurring before blood samples could be obtained, or at a time before the appearance of cardiac biomarkers in the blood

Type 4a
MI associated with percutaneous coronary intervention

Type 4b
MI associated with stent thrombosis as documented by angiography or at autopsy

Type 5
MI associated with coronary artery bypass grafting

From Thygesen K, Alpert JS, White HD; on behalf of the Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction: Universal definition of myocardial infarction. Circulation 116:2637, 2007.

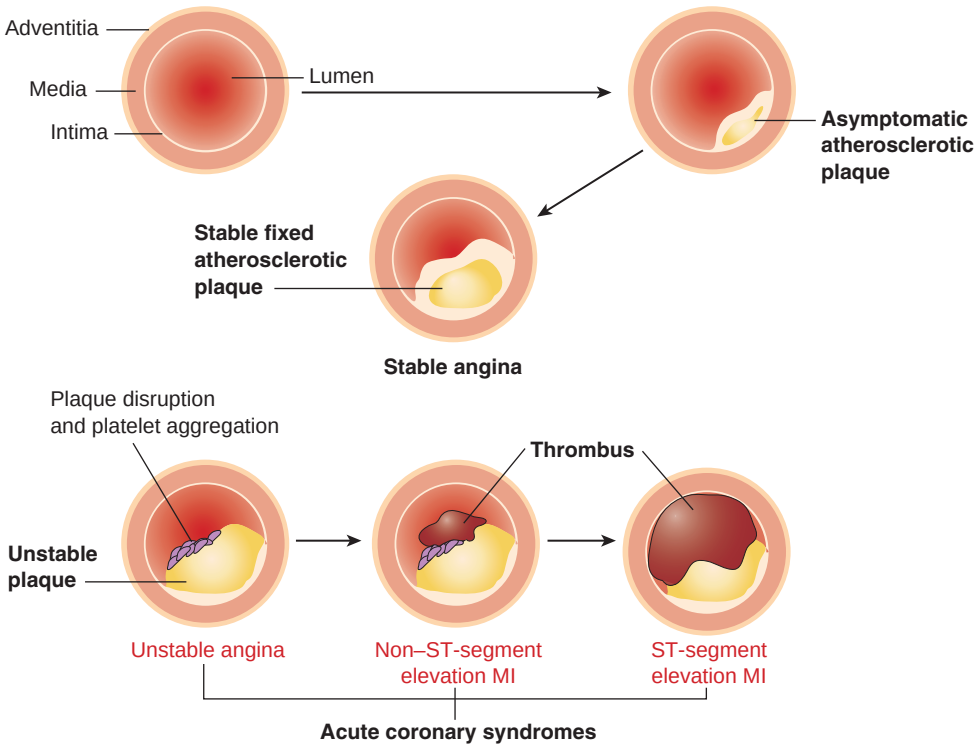


FIGURE 21-4 ▲ Atherosclerotic plaque. Stable fixed atherosclerotic plaque in stable angina and the unstable plaque with plaque disruption and platelet aggregation in the acute coronary syndromes (ACSs). (From Porth CM: Essentials of Pathophysiology, 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2011, p 453.)

death occurs first in the subendocardial layer and spreads like a "wave front" throughout the thickness of the wall of the heart.[13] Using dogs, they showed that the shorter the time between coronary occlusion and coronary reperfusion, the greater the amount of myocardial tissue that could be salvaged. Their classic work indicates that a substantial amount of myocardial tissue can be salvaged if flow is restored within 6 hours after the onset of coronary occlusion. For the clinician, this means time is muscle.

The cellular changes associated with an MI can be followed by the development of infarction extension (new myocardial necrosis), infarction expansion (a disproportionate thinning and dilation of the infarct zone), or ventricular remodeling (a disproportionate thinning and dilation of the ventricle).

Size of the Infarction

Several factors determine the size of the resulting MI. These factors include the extent, severity, and duration of the ischemic episode; the size of the vessel; the amount of collateral circulation; the status of the intrinsic fibrinolytic system; vascular tone; and the metabolic demands of the myocardium at the time of the event. MIs most often result in damage to the left ventricle, leading to an alteration in left ventricular function. Infarctions can also occur in the right ventricle or in both ventricles.

The term transmural infarction is used to imply an infarction process that has resulted in necrosis of the tissue in all the layers of the myocardium. Because the heart functions as a squeezing pump, systolic and diastolic efforts can be significantly altered when a segment of the heart muscle is necrotic and nonfunctional. If the area of the transmural infarction is small, the necrotic wall may be dyskinetic, a term meaning "difficulty in moving." If the damage to the myocardial tissue is more extensive, the myocardial muscle may become akinetic, meaning "without motion."

The normal myocardial muscle contracts with systole and relaxes with diastole. When normal motion is not possible because of infarction, diastolic filling and systolic pumping are altered. As a result, cardiac output is compromised. The larger the area of infarction, the greater is the impact on ventricular function.

Location of the Infarction

In addition to size, location of the infarction is an important determinant of ventricular function. MIs can be located in the anterior, septal, lateral, posterior, or inferior walls of the left ventricle. In more recent years, clinicians have acknowledged the presence and clinical significance of MIs occurring in the right ventricle.

Anterior Left Ventricle

Infarctions of the anterior wall of the left ventricle and the interventricular septum result from occlusion of the left anterior descending (LAD) coronary artery. The LAD coronary artery supplies oxygenated blood to the anterior wall of the left ventricle, the interventricular septum, and the ventricular conducting tissue. (See Chapter 16 for a more detailed discussion of coronary artery anatomy and physiology.) Anteroseptal wall MIs are the most frequent type of infarction and have the potential for causing a significant amount of left ventricular dysfunction. Patients with an anteroseptal MI are at high risk for heart failure, pulmonary edema, cardiogenic shock, and death because of an inadequate pump. Anteroseptal wall MIs are also associated with increased risk for intraventricular conduction disturbances, such as bundle branch blocks and fascicular blocks, which are also known as hemiblocks.

Lateral and Posterior Left Ventricle

Infarctions of the lateral and posterior walls of the left ventricle result from occlusion of the left circumflex vessel. In addition to supplying oxygenated blood to the lateral and posterior walls, the left circumflex vessel is the source of blood supply to the sinoatrial (SA) node in about 50% of the population and to the AV node in about 10% of the population. Infarctions of the lateral and posterior walls are less common than infarctions of the anteroseptal wall. Although muscle necrosis occurs with lateral and posterior wall MIs, the impact on left ventricular function is usually less than for patients with anteroseptal MI. Patients with a lateral or posterior wall MI are also at risk for dysrhythmias associated with dysfunction of the SA or AV nodes. Examples include sinus arrest, wandering atrial pacemaker, sinus pause, or junction rhythm.

Inferior Left Ventricle

Infarctions of the inferior wall result from occlusion of the right coronary artery. The right coronary artery supplies oxygenated blood to the inferior wall and the right ventricle. In addition, it is the source of blood supply to the SA node in about 50% of the population and the AV node in about 90% of the population. Infarctions of the inferior wall are less common than anteroseptal MIs but occur more frequently than MIs of the lateral or posterior walls. The potential impact on left ventricular function usually is less for a patient with an inferior wall MI than for a patient with an anteroseptal wall infarction. Because the right coronary artery supplies oxygenated blood to much of the conducting tissue, patients are at frequent risk for dysrhythmias related to altered function of the SA and AV nodes.

Right Ventricle

The right coronary artery provides the blood supply to the inferior wall and the right ventricle. Consequently, right coronary artery disease causing an inferior wall MI is likely to be associated with concomitant right ventricular infarction. Patients may experience significant hemodynamic compromise due to biventricular dysfunction. As a result, patients with a right ventricular infarction and hemodynamic abnormalities with a concurrent inferior wall MI have a significantly higher mortality rate (25% to 30%).[14] Dysrhythmias associated with right ventricular infarction involve dysfunction of the SA and AV nodes.

Type of Infarction

Patients with chest pain may present with or without ST-segment elevations on their ECG. In most patients with ST-segment elevation, a Q wave ultimately develops on the ECG, and the term Q-wave MI is used to describe the type of MI they experience. In a much smaller number of patients who present with ST-segment elevation, a Q wave does not develop, and the term non–Q-wave MI is used to classify these patients. Patients who present without ST-segment elevations are diagnosed with either unstable angina or an NSTEMI[14] (Fig. 21-5).

The ST segment is the portion of the ECG tracing from the end of the QRS complex to the beginning of the T wave. Normally, the ST segment is isoelectric, meaning it joins the QRS complex at the baseline. When the ST segment is elevated, the amount of elevation is measured in millimeters on the ECG paper. See Evidence-Based Highlight 21-1 for information about ST-segment monitoring.

A Q wave is a portion of the QRS complex on the ECG. Specifically, the Q wave is the initial downward deflection of the QRS complex. A Q wave is not present on the normal ECG. The presence of significant Q waves indicates an MI. For a review of ECG waveforms, see Chapter 17.

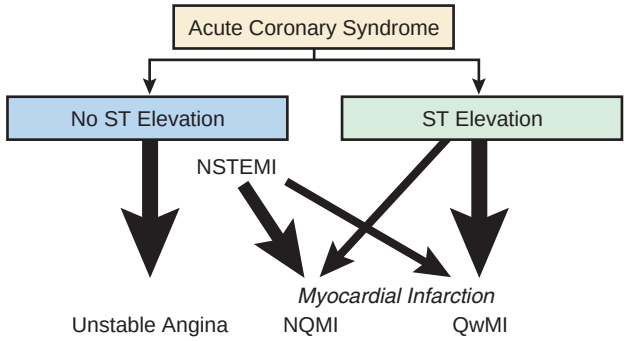


FIGURE 21-5 ▲ Acute coronary syndrome. Patients may present with or without ST-segment elevation on electrocardiography. Most patients with ST-segment elevation (*large arrows*) ultimately develop a Q-wave acute myocardial infarction (QwMI), whereas a minority (*small arrow*) develop a non–Q-wave AMI (NQMI). Patients who present without ST-segment elevation are experiencing either unstable angina or NSTEMI (non–ST-segment elevation MI). (Adapted from Committee to revise the 1999 guidelines for the management of patients with acute myocardial infarction. Circulation 110:e82–e293, 2004, p e88.)

Assessment

The nursing assessment of a patient with a probable MI must be organized and thorough. It is best to start with the history because this establishes rapport and provides valuable data. The history is followed by the physical examination and evaluation of diagnostic tests. Based on the data, a management plan is developed initially for the acute phase. Once the patient is stabilized, plans for cardiac rehabilitation are initiated.

History

The most common presenting complaint of a patient with an MI is the presence of chest discomfort or pain. Other assessment findings are similar to those described in Box 21-2 on page 421. Like patients with angina, patients with MI describe a heaviness, squeezing, choking, or smothering sensation. Patients often describe the sensation as "someone sitting on my chest." The substernal pain can radiate to the neck, left arm, back, or jaw. Unlike the pain of angina, the pain of an MI is often more prolonged and unrelieved by rest or sublingual nitroglycerin. For a review of the assessment questions, see Box 17-1 on page 207. Women and the elderly may present differently and often present with a primary complaint of shortness of breath.

Associated findings on history include nausea and vomiting, especially for the patient with an inferior wall MI. These gastrointestinal complaints are believed to be related to the severity of the pain and the resulting vagal stimulation. Patients may initially seek relief of the gastrointestinal

EVIDENCE-BASED PRACTICE HIGHLIGHT 21-1

ST-Segment Monitoring

△ Expected Practice

- If 12-lead ECG is available, continuous ST-segment monitoring should be performed using all 12 leads.
- If 12-lead ECG is unavailable, use the most appropriate leads for ST-segment monitoring based on the patient's needs and risk for ischemia and/or dysrhythmia.
  - For patients with acute coronary syndrome (ACS) and a known "ST fingerprint," obtained during ST-segment elevation myocardial infarction (STEMI) or percutaneous coronary intervention (PCI), use the lead(s) that best display(s) the patient's "ST fingerprint" when monitoring.
  - If the "ST fingerprint" is not known in ACS, use leads III and V_3.
  - For patients without definitive ACS, but are suspected of having or being ruled out for ACS, leads III and V_5 should be monitored.
  - In noncardiac patients undergoing surgical procedures or admitted to the ICU, lead V_5 is valuable for identifying demand-related ischemia, which appears to be more common in this group of patients.
- Properly prepare the patient's skin before attaching the ECG skin electrodes.
- Once proper lead placement has been determined, mark skin electrode placement with indelible ink. Do not alter the location of the skin electrodes during monitoring as this can created false-positive ST-segment changes.
- Evaluate ST segment with the patient in the supine position, set the ST alarm parameter 1 to 2 mm above and below the patient's baseline ST segment and measure ST-segment changes 60 ms beyond the J point of the ECG complex (See Fig. below).
- ST depression or elevation of 1 to 2 mm that lasts for at least 1 minute can be clinically significant and warrants further patient assessment.

△ Supporting Evidence

- ST-segment monitoring is useful for detecting silent ischemia.[2–4] ST-segment monitoring is more sensitive than patient's self-reporting of symptoms because 70% to 90% of episodes of myocardial ischemia detected with ECG are clinically silent.[2,4–8] It is important to point out that no randomized controlled trials have been conducted to determine whether the addition of ST-segment monitoring improves patient outcomes.[9] (Level V)
- Several studies have demonstrated that silent myocardial ischemia, as detected by continuous ST-segment monitoring, may occur during the process of weaning from mechanical ventilation.[10–15] The presence of ST-segment deviation prior to the initiation of the weaning process has been shown to increase the likelihood of weaning failure.[12–15] However, the effect of ST-segment monitoring on weaning outcomes is not known. The clinical utility of continuous ST-segment monitoring during weaning has not been studied. (Level IV)
- Research demonstrates that monitoring for ST-segment changes in multiple leads, preferably 12 leads, substantially improves the chance of identifying ischemic events.[4,16,17](Level V)
- If all 12 leads are not available in the bedside monitor, use the patient's "ST fingerprint" to select the best ECG lead(s), which show(s) maximal ST-segment deviation. An ST fingerprint is defined as the pattern of ST-segment elevation and/or depression unique to a particular patient based on the anatomic site of coronary occlusion. A fingerprint can be obtained during known ischemia (STEMI or during PCI).[4,9,18–22] (Level V)
- If only two leads are available for ST-segment monitoring, and an ST fingerprint is not available, leads III and V_3 are recommended for patients with acute coronary syndromes or suspected ACS.[4,9,19,23] (Level IV)
- In noncardiac patients undergoing surgical procedures[24] or admitted to the ICU,[26] lead V_5 is valuable for identifying demand-related ischemia, which appears to be more common in this group of patients. (Level IV)
- Failure to properly prep the skin before placing the electrodes may cause the monitoring alarms to sound erroneously. Preparation may include carefully clipping hair areas where electrodes are to be placed and/or cleaning the skin with alcohol to remove skin oils.[4,9,27, 28] (Level IV)
- Variability of electrode placement may occur during routine ECG. Expert consensus recommends marking the locations of the electrodes with indelible ink to assure that if electrodes are removed for any reason (leads V_2 and V_3 are typically removed during recording of echocardiograms), they can be replaced in their original locations. ECG information obtained from electrodes located close to the heart (precordial leads) is especially prone to waveform changes when the electrodes are relocated as little as 1 cm away from the original locations.[9,29] (Level II)
- Because a change in body position (right-, left-side lying) can alter the ST segment mimicking ischemia[30] when an ST alarm sounds and the patient is found in a side-lying position, the patient should be returned to the supine position. If the ST-segment deviation persists in the supine state, it should be considered indicative of myocardial ischemia.[3,14,9] If possible, obtain "positional 12-lead ECGs" with the patient assuming right and left side–lying positions at the initiation of ST monitoring. These positional ECGs can be used to identify false ST-segment changes. (Level IV)

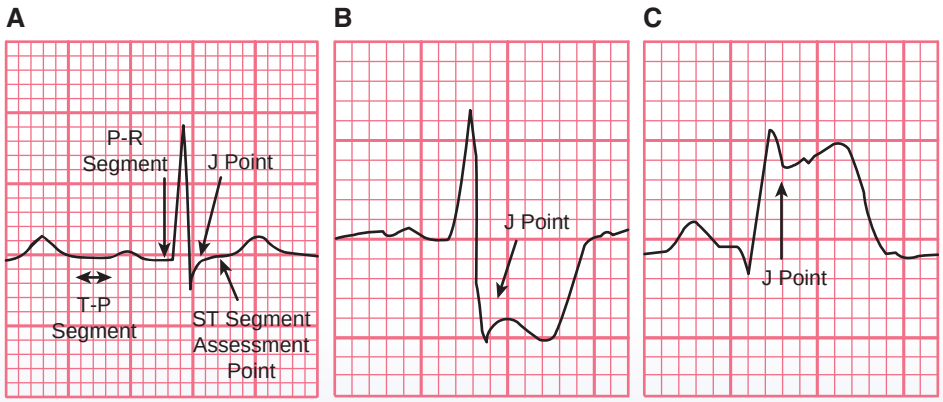


FIGURE ▲ A: A normal ECG complex shows T-P segment and P-R segment, which may be used as reference points to the isoelectric line. The ST-segment is measured at 0.06 seconds after the J point. This ST-segment is isoelectric. **B:** ECG complex show ST-segment depression of almost 5 mm. **C:** ST-segment elevation of approximately 4 mm is depicted. (Flanders SA. Continuous st-segment monitoring: Raising the bar. Crit Care Nurs Clin N Am. 2006;18(3):169–177.)

(continued on page 428)

EVIDENCE-BASED PRACTICE HIGHLIGHT 21-1 (continued)

ST-Segment Monitoring

- Set the ST-segment alarm parameter at 1 mm above and below the baseline ST segment in patients at high risk for ischemia and 2 mm in more stable patients.[9] (Level II)
- Measure ST-segment changes 60 ms beyond the J point of the ECG complex.[9] (Level II)
- ST depression or elevation of 1 to 2 mm that lasts for at least 1 minute can be clinically significant and warrants further patient assessment.[4,26,27] (Level II)
- Because most patients with coronary artery disease do not have perfectly isoelectric ST segments,[31] it is important to set alarm parameters to 1 to 2 mm around the patient's baseline ST level. (Level IV)
- The goal of monitoring must be considered for each patient. For instance, in patients presenting for STEMI, the goal of ST monitoring is to observe rapid ST-segment recovery (back to isoelectric) within the first hour of treatment. Whereas, in patients presenting with ACS, the goal is to defect transient or recurrent ST-segment changes.[9] (Level II)
- ST-segment elevations greater than 1 mm above the isoelectric line are uncommon in the newborn. In neonates and infants, it is better to consider as the isoelectric line the TP segment instead of the PR segment. T waves are normally quite variable in the first week of life. After 1 week, the T wave is negative in lead V_1 and positive in V_5 to V_6.[32] (Level II)

AACN Evidence Leveling System

Level A Meta-analysis of quantitative studies or metasynthesis of qualitative studies with results that consistently support a specific action, intervention, or treatment

Level B Well-designed, controlled studies with results that consistently support a specific action, intervention, or treatment

Level C Qualitative studies, descriptive or correlational studies, integrative review, systematic reviews, or randomized controlled trials with inconsistent results

Level D Peer-reviewed professional organizational standards with clinical studies to support recommendations

Level E Multiple case reports, theory-based evidence from expert opinions, or peer-reviewed professional organizational standards without clinical studies to support recommendations

Level M Manufacturer's recommendations only

Excerpted from American Association of Critical-Care Nurses Practice Alert. Available at: http://aacn.org. All references cited in this alert are available with the associated resources related to this chapter. Visit: http://thepoint.lww.com

symptoms through antacids and other home remedies, thus delaying their decision to go the hospital. Additional complaints described during the history include diaphoresis, dyspnea, weakness, fatigue, anxiety, restlessness, confusion, shortness of breath, or a sense of impending death.

After the patient is stabilized, a more comprehensive history is obtained. Information about risk factors, previous cardiac illnesses and surgeries, and family history is important to acquire. This information will be useful in guiding patient education, cardiac rehabilitation, and care at home.

Physical Examination

On physical examination, patients usually appear restless, agitated, and in distress. They often assume a position to promote breathing and alleviate pain. The skin is cool and moist. Vital signs may reveal a low-grade fever, hypertension, and tachycardia from increased sympathetic tone or hypotension and bradycardia from increased vagal tone. The pulse may be irregular and faint.

The cardiovascular examination may reveal additional abnormalities. When the patient is placed in the left lateral decubitus position, abnormalities of the precordial pulsations can be felt. These abnormalities include a lack of a point of maximal impulse or the presence of diffuse contraction. On auscultation, the first heart sound may be diminished as a result of decreased contractility. A fourth heart sound is heard in almost all patients with MI as a result of decreased left ventricular compliance. A third heart sound may be detected due to left ventricular systolic dysfunction. Transient systolic murmurs may be heard because of papillary muscle dysfunction. After about 48 to 72 hours, many patients acquire a pericardial friction rub. Additional findings on physical examination, such as jugular venous distention, may be related to the development of complications, such as heart failure or pulmonary edema. Breathing may be labored and rapid, and fine crackles, coarse crackles, or rhonchi may be heard when auscultating the lungs. These sounds may indicate the presence of heart failure or pulmonary edema.

Patients with right ventricular infarctions may present with jugular venous distention as well as peripheral edema and elevated central venous pressure. Their lungs may be clear because the failing right ventricle has not provided adequate forward flow.

Diagnostic Tests

THE ELECTROCARDIOGRAM. When a coronary artery becomes about 70% occluded and oxygen demand exceeds oxygen supply, myocardial ischemia may result. If the ischemic state is not corrected, injury to the myocardium may occur. Eventually, if adequate blood flow to the myocardium is not restored, an MI may result. Ischemia and injury are reversible processes; however, infarction is not.

An ECG can be used to detect patterns of ischemia, injury, and infarction. When the heart muscle becomes ischemic, injured, or infarcted, depolarization and repolarization of the cardiac cells are altered, causing changes in the QRS complex, ST segment, and T wave in the ECG leads overlying the affected area of the heart. Table 21-1 shows location of the MI, the artery affected, findings from the ECG, and clinical implications.

Ischemia. Myocardial ischemia may be a transient finding on ECG, or ischemic patterns may be more prolonged due to the presence of ischemic tissue surrounding a region of infarcted tissue. On the ECG, myocardial ischemia results in T-wave inversion or ST-segment depression in the leads facing the ischemic area. The inverted T wave representative

Table 21-1 Location of Myocardial Infarction, Electrocardiographic (ECG) Findings, and Clinical Implications

Anatomical Location	Coronary Artery	ECG Evidence	Clinical Implications
Anteroseptal wall	Left anterior descending: Supplies blood to the anterior wall of left ventricle, the interventricular septum, and the ventricular conducting tissue	V_1 through V_4, Q waves and ST-segment elevations	Potential for significant hemodynamic compromise; heart failure, pulmonary edema, cardiogenic shock; intraventricular conduction disturbances
Lateral wall	Left circumflex: Supplies blood to the left lateral and left posterior walls and to the sinoatrial (SA) node in 45% of people and atrioventricular (AV) node in 10% of people	I, aVL, V_5, and V_6, Q waves and ST-segment elevations	Evaluation for posterior wall involvement; some hemodynamic changes; dysrhythmias caused by SA and AV node dysfunction
Posterior wall	Left circumflex: Supplies blood to the left lateral and left posterior walls and to the SA node in 45% of people and AV node in 10% of people	V_1 and V_2, tall upright R waves with ST-segment depression; Q waves and ST-segment elevation in V_7 through V_9	Evaluation for lateral wall involvement; some hemodynamic changes; dysrhythmias caused by SA and AV node dysfunction
Inferior wall	Right coronary artery: Supplies blood to the inferior wall of the left ventricle, the right ventricle, and the SA node in 55% of people and the AV node in 90% of people	Q waves and ST-segment elevation in II, III, aVF	Evaluation for right ventricular wall involvement; some hemodynamic changes; potential for significant dysrhythmias caused by SA and AV node dysfunction
Right ventricular wall	Right coronary artery: Supplies blood to the inferior wall of the left ventricle, the right ventricle, and the SA node in 55% of people and the AV node in 90% of people	Q waves and ST-segment elevations in right precordial chest leads (RV_1 through RV_6)	Evaluation for inferior wall involvement; some hemodynamic changes; potential for significant dysrhythmias caused by SA and AV node dysfunction

of ischemia is symmetrical, relatively narrow, and somewhat pointed. In contrast, asymmetrical inversion of the T wave usually does not indicate ischemia. Instead, it may signify ventricular hypertrophy or bundle branch block (Fig. 21-6). ST-segment depressions of 1 to 2 mm or more for a duration of 0.08 second may indicate myocardial ischemia. Ischemia also should be suspected when a flat or depressed ST segment makes a sharp angle when joining an upright T wave rather than merging smoothly and imperceptibly with the T wave (Fig. 21-7).

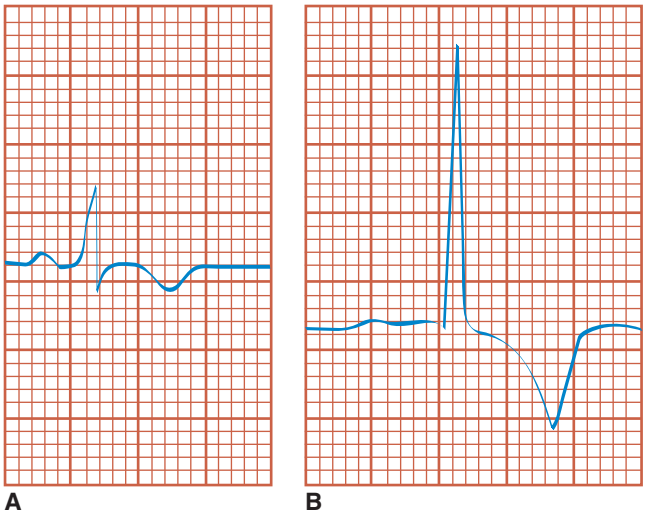


FIGURE 21-6 ▲ T-wave inversion seen with ischemia (**A**) versus T-wave inversion seen with left ventricular hypertrophy (**B**).

Injury. ECG patterns of myocardial injury indicate a state of cellular damage beyond ischemia. Like ischemia, myocardial injury is a reversible process if interventions are instituted rapidly. As described previously, the injury process begins in the subendocardial layer and moves throughout the thickness of the wall of the heart like a wave. If the injury process is not interrupted, it eventually results in a transmural MI.

On ECG, the hallmark of acute myocardial injury is the presence of ST-segment elevations. In the normal ECG, the ST segment should not be elevated more than 1 mm in the standard leads or more than 2 mm in the precordial leads. With an acute injury, the ST segments in the leads facing the injured area are elevated. The elevated ST segments also have a downward concave or coved shape and merge unnoticed with the T wave (Fig. 21-8).

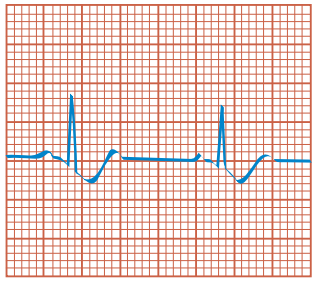


FIGURE 21-7 ▲ An ST-segment pattern consistent with myocardial ischemia. Notice how the ST segment forms a sharp angle when joining an upright T wave rather than merging smoothly and imperceptibly with the T wave.

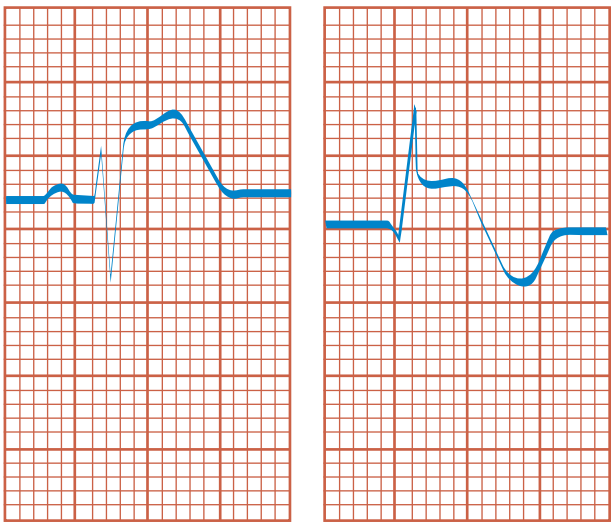


FIGURE 21-8 ▲ ST-segment pattern consistent with acute myocardial injury. **A:** ST-segment elevation without T-wave inversion. **B:** ST-segment elevation with T-wave inversion. The elevated ST segments have a downward concave or coved shape and merge unnoticed with the T wave.

Infarction. When myocardial injury persists, MI is the result. The pattern of the ECG indicative of an MI is seen on the ECG in stages and involves changes in the T wave, the ST segment, and the Q wave in the leads overlying the infarcted area. Figure 21-9 shows the evolution of the ECG in an MI. During the earliest stage of MI, known as the hyperacute phase, the T waves become tall and narrow. This configuration is referred to as hyperacute or peaked T waves. Within a few hours, these hyperacute T waves invert.

Next, the ST segments elevate, a pattern that usually lasts from several hours to several days. In addition to the ST-segment elevations in the leads of the ECG facing the injured heart, the leads facing away from the injured area may show ST-segment depression. This finding is known as reciprocal ST-segment changes. Reciprocal changes are most likely to be seen at the onset of infarction, but their presence on the ECG does not last long. Reciprocal ST-segment depressions may simply be a mirror image of the ST-segment elevations. However, others have suggested that reciprocal changes may reflect ischemia due to narrowing of another coronary artery in other areas of the heart.[15,16]

The last stage in the ECG evolution of an MI is the development of Q waves, the initial downward deflection of the QRS complex. Q waves represent the flow of electrical forces toward the septum. Small, narrow Q waves may be seen in the normal ECG in leads I, II, III, aVR, aVL, V_5, and V_6. Q waves compatible with an MI are usually 0.04 second or more in width or one fourth to one third the height of the R wave. Q waves indicative of infarction usually develop within several hours of the onset of the infarction, but in some patients, they may not appear until 24 to 48 hours after the infarction.

Within a few days after the MI, the elevated ST segments return to baseline. Persistent elevation of the ST segment may indicate the presence of a ventricular aneurysm. The T waves may remain inverted for several weeks, indicating areas of ischemia near the infarcted region. Eventually, the T waves should return to their upright configuration. The Q waves do not disappear and therefore always provide ECG evidence of a previous MI.

The ECG pattern can be used to distinguish acute MIs from "old" MIs. Abnormal Q waves accompanied by ST-segment elevations indicate an acute MI. Abnormal Q waves accompanied by a normal ST segment indicate a previous MI. How long ago the infarction occurred cannot be determined by the ECG. The pattern could signify an infarction that occurred 2 weeks or 20 years before.

The ECG is helpful not only in determining patterns of ischemia, injury, and infarction but also in revealing the anatomical region of the heart where the abnormality has occurred. ECG leads V_1 through V_4 show the anteroseptal wall of the left ventricle. The inferior wall is seen in leads II, III, and aVF. Leads I, aVL, V_5, and V_6 reveal the lateral wall of the left ventricle (see Fig. 21-10). The routine 12-lead ECG does not provide an adequate view of the right ventricle or of the posterior wall of the left ventricle. As a result, additional leads are needed to view these anatomical areas. To attain an accurate view of the right ventricle, right-sided chest leads are recorded by placing the six chest electrodes on the right side of the chest using landmarks analogous to those used on the left side (see Fig. 17-9, p. 224). These six right-sided views are examined for patterns of ischemia, injury, and infarction in the same way left-sided chest leads are evaluated.

Detection of posterior wall abnormalities is also difficult on the standard 12-lead ECG because none of the six chest leads provides an adequate view of the posterior wall. To detect posterior wall abnormalities, three of the precordial electrodes are placed posteriorly over the heart, a view

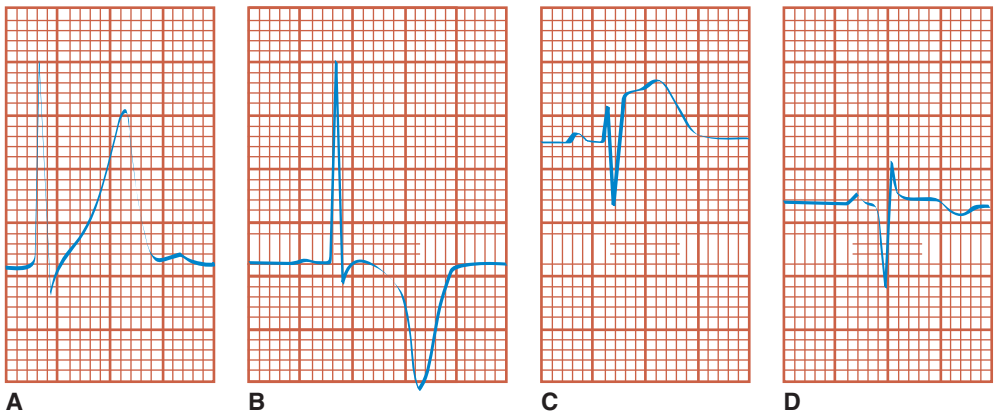


FIGURE 21-9 ▲ Evolution of the electrocardiogram (ECG) in a patient with MI. **A:** Tall peak T waves known as hyperacute T waves. **B:** Symmetrical T-wave inversions. **C:** ST-segment elevation. **D:** Development of the Q wave.

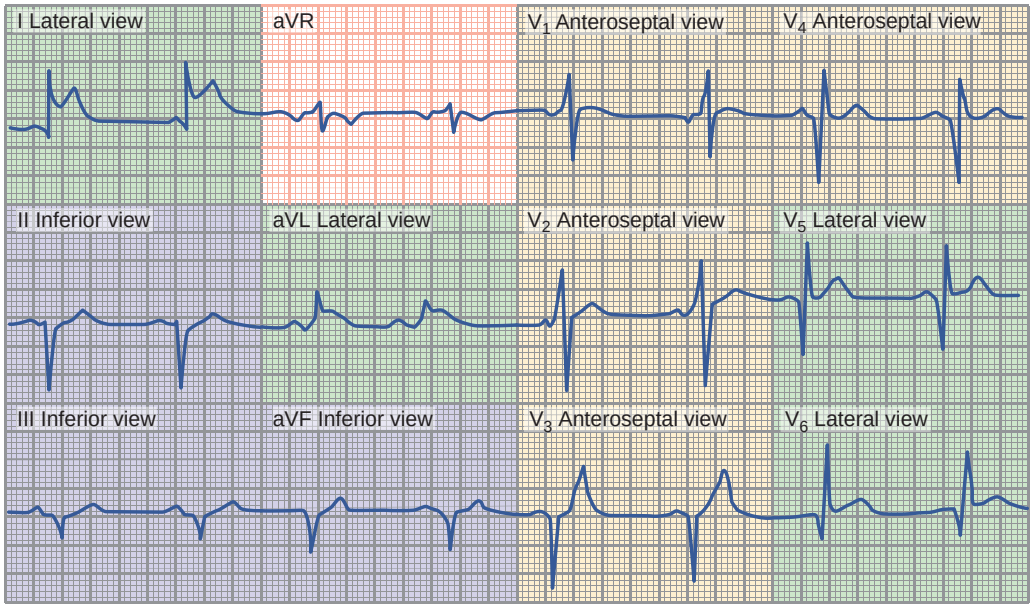


FIGURE 21-10 ▲ The 12-lead ECG: Correlation of lead with the view of the heart.

known as V_7, V_8, and V_9. V_7 is positioned at the posterior axillary line; V_8, at the posterior scapular line; and V_9, at the left border of the spine. All three posterior leads are positioned along the same horizontal line established by V_6. The recording is examined for evidence of ischemia, injury, or infarction using the same criteria as described previously. If posterior leads were not recorded, it may still be possible to detect posterior wall abnormalities. To do so, the principle of reciprocal change is used. When an infarction in the posterior wall is suspected, the leads anatomically opposite the posterior wall are examined. These include V_1 and V_2 because the anterior wall is anatomically opposite the posterior wall. If tall R waves with ST-segment depressions are noted in V_1 and V_2, the pattern is consistent with a posterior wall MI. Figures 21-11 through 21-14 show the 12-lead ECGs of patients with MIs.

LABORATORY TESTS. When myocardial cells are damaged by an infarction, biochemical markers are released into the bloodstream and can be detected by laboratory tests. The presence of abnormally high levels of biochemical markers, their distribution, and the time pattern for their appearance and disappearance make them very useful in the diagnosis of acute MI. For a more detailed discussion of laboratory tests, see Chapter 17.

Creatine Kinase. Creatine kinase (CK) is an enzyme found mainly in heart and skeletal muscles. When heart muscle is damaged, CK is released into the blood. The level of CK becomes abnormal within 6 to 8 hours after the onset of infarction, peaks within 12 to 28 hours, and returns to normal in 24 to 36 hours. The isoenzymes of CK are measured to determine whether the CK came from the heart (MB) or the skeletal muscle. Elevation of CK-MB offers a more definitive indication of myocardial cell damage than total CK alone. For the patient with an MI, CK-MB appears in the serum in 3 to 12 hours, peaks in 24 hours, and returns to normal levels in about 48 to 72 hours.[14]

New assay techniques to measure CK-MB based on monoclonal antibodies offer greater sensitivity and specificity than conventional means. In addition, the results can be available in 30 minutes, which provides a distinct advantage in the diagnosis of an MI, especially in the emergency department (ED).

Creatine Kinase Isoforms. When the myocardial cells release CK-MB, it is quickly transformed into two isoforms, also known as subforms. $CK\text{-}MB_1$ is the isoform found in the plasma, and $CK\text{-}MB_2$ is found in the tissues. In the normal person, these two isoforms are found in about equal amounts, resulting in a ratio of approximately 1. In the patient with an MI, the $CK\text{-}MB_2$ level rises, resulting in a $CK\text{-}MB_2/CK\text{-}MB_1$ ratio greater than 1. This ratio can be rapidly measured in the laboratory and provides an excellent diagnostic marker for acute MI. The $CK\text{-}MB_2$ to $CK\text{-}MB_1$ ratio has improved sensitivity and specificity for diagnosis of MI within the first 6 hours compared with conventional assays for CK-MB. Isoform $CK\text{-}MB_2$ is also a sensitive test for detecting an early extension of an MI during the first 24 hours.[14]

Myoglobin. Myoglobin is an oxygen-binding protein found in skeletal and cardiac muscle. Myoglobin's release from ischemic muscle occurs earlier than the release of CK. As a result, elevation of serum levels of myoglobin can be detected soon after the onset of symptoms. The myoglobin level can elevate within 1 to 4 hours of acute MI and peaks within 6 to 7 hours. Because myoglobin is also present in skeletal muscle, an elevated myoglobin level is not specific for the diagnosis of MI. Consequently, its diagnostic value in detecting an MI is limited. However, the early release of myoglobin makes it valuable in helping to detect MI.[14]

Troponin. Troponin is a contractile protein with two subforms (troponin T and troponin I) that are highly specific for cardiac muscle. Troponin levels are not detected in the healthy person, and skeletal muscle injury does not affect the level. Troponin has been found to be a sensitive marker

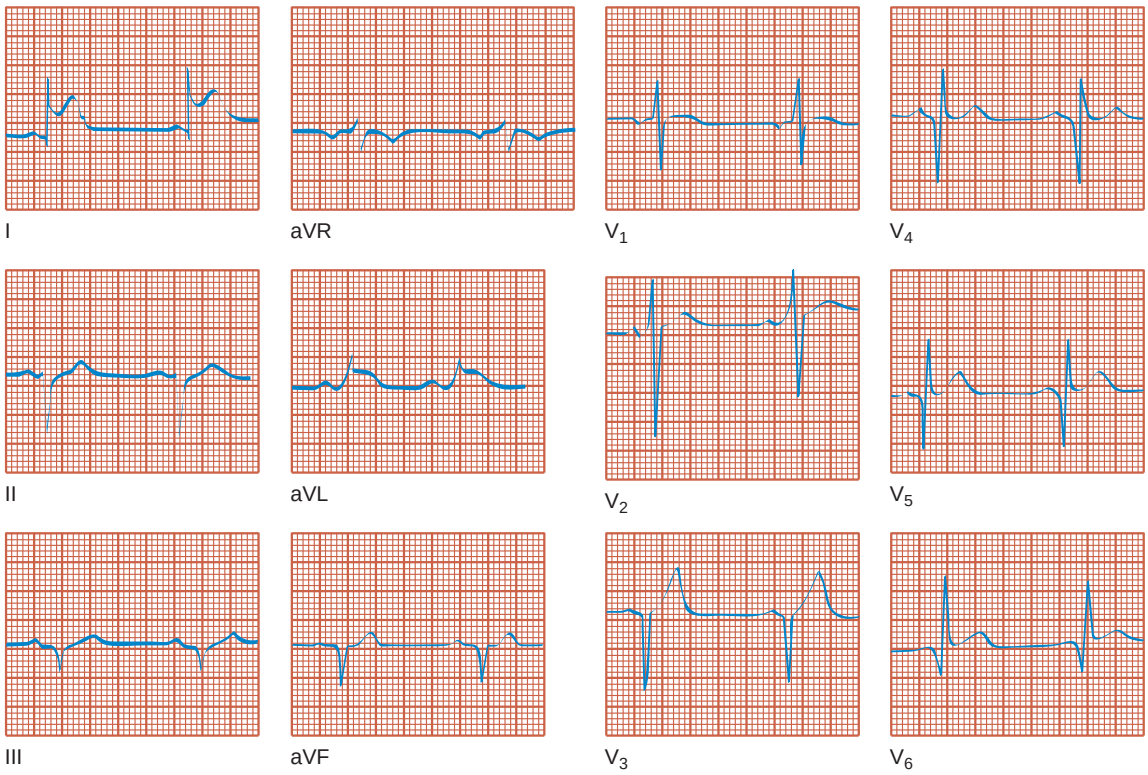


FIGURE 21-11 ▲ Twelve-lead ECG showing an acute lateral wall MI. ST-segment elevations can be seen in leads I, aVL, V5, and V6. Note also the deep Q waves in II, III, and aVF and normal ST segments, indicating a previous inferior wall MI.

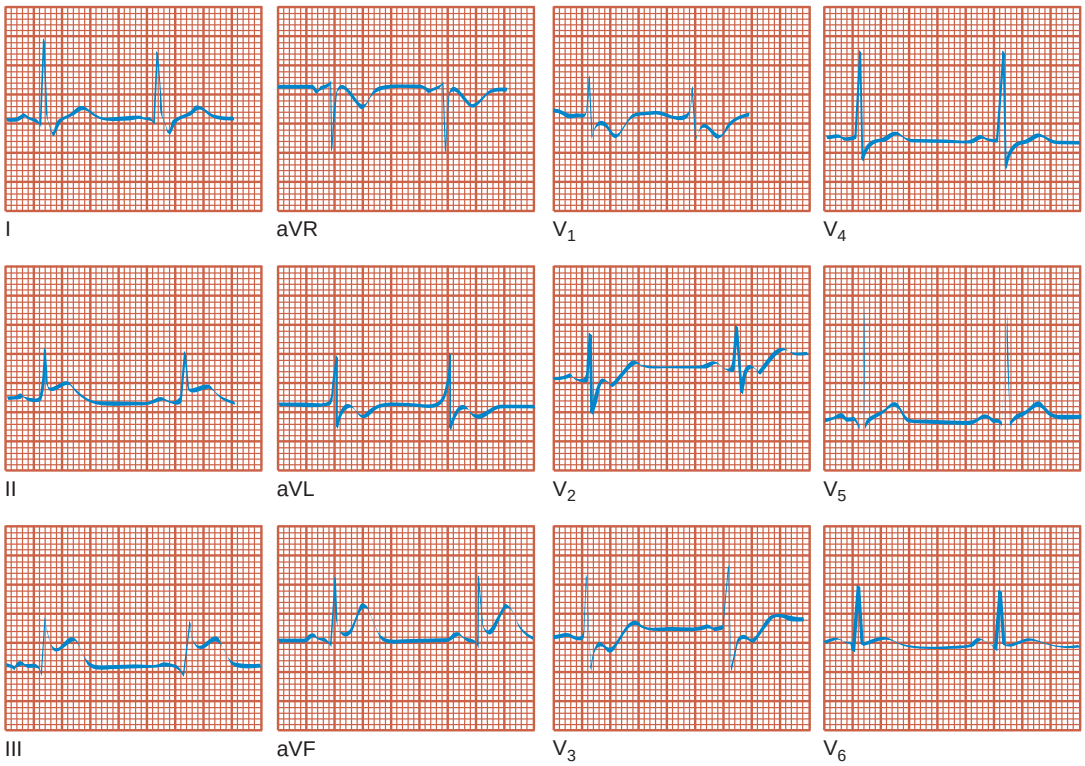


FIGURE 21-12 ▲ Twelve-lead ECG showing an acute inferior wall MI. Note the ST-segment elevations in II, III, and aVF. The posterior wall infarction is evidenced by a tall R wave, ST-segment depression, and inverted T wave in V1 and V2.

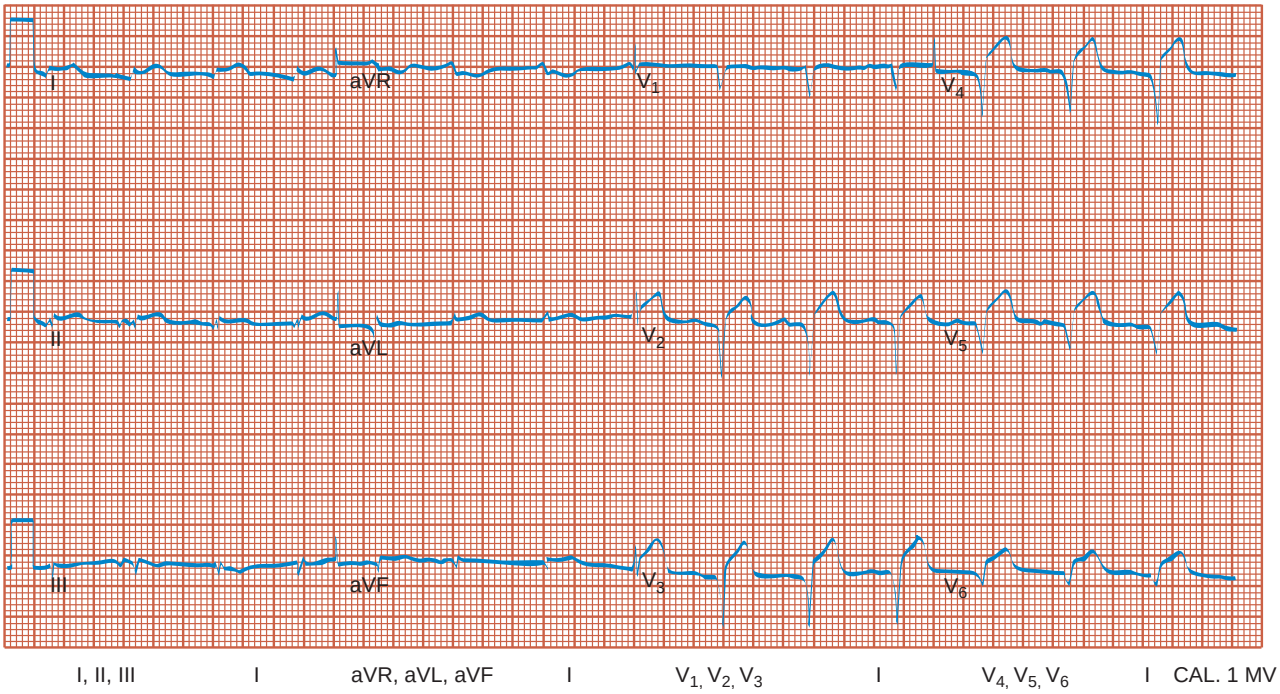


FIGURE 21-13 ▲ Twelve-lead ECG showing an acute anterior and lateral wall MI. Note the ST-segment elevations and Q waves in I, aVL, V5, and V6 (lateral), and V2, V3, and V4 (anterior).

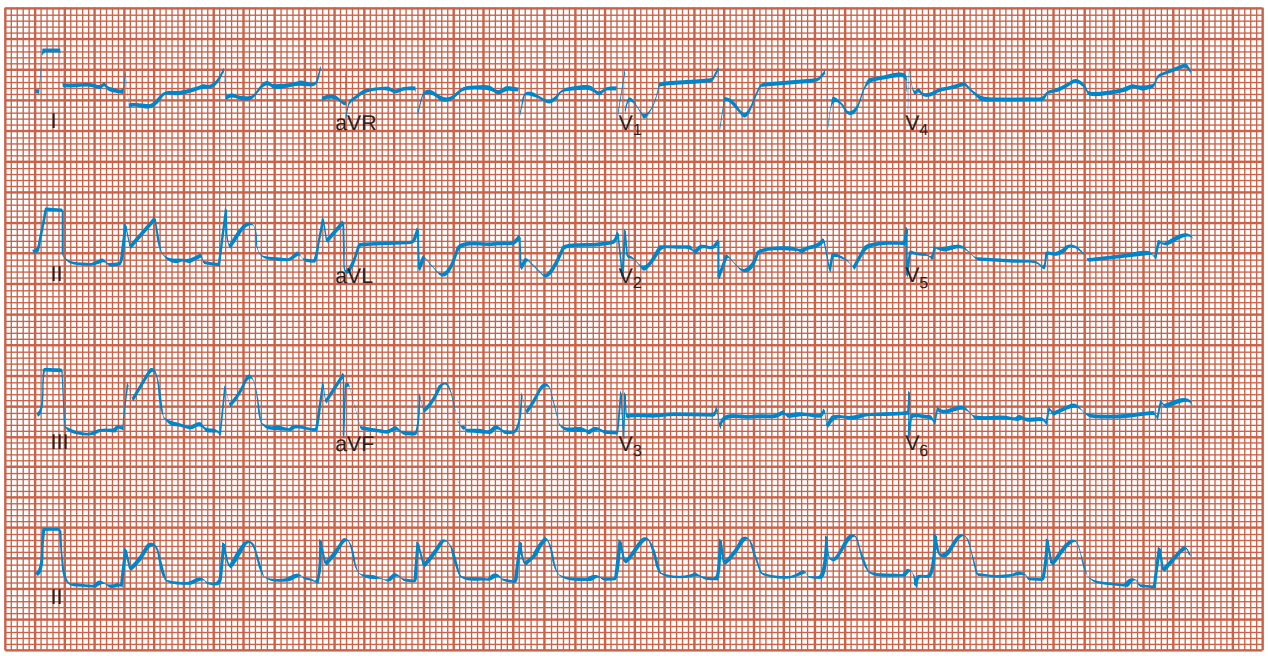


FIGURE 21-14 ▲ Twelve-lead ECG showing right ventricular infarction. The six chest leads have been positioned on the right side of the chest. Note the ST-segment elevation in RV4, RV5, and RV6. The ECG also shows elevated ST segments in the inferior leads (II, III, aVF). Patients with an inferior wall MI often also have an infarction in the right ventricle.

during the early hours after an MI. Troponin I levels rise in 3 to 12 hours, peak at 24 hours, and remain elevated for 5 to 10 days. Troponin T levels rise in 3 to 12 hours, peak in 12 hours to 2 days, and remain elevated for 5 to 14 days.[14] Because the cardiac troponins are highly sensitive and specific for MI, they are the preferred biomarker for diagnosing this coronary event.[14] Other laboratory tests are the same as described previously for the patient with suspected angina and include blood chemistry, complete blood count, coagulation tests, lipid panel, CRP, and white blood cell count.

Other Diagnostic Tests

Patients with an MI should have a chest radiograph. An echocardiogram may be done to detect structural abnormalities, such as valvular problems. Other tests may include radionuclide angiography, MRI, magnetic perfusion imaging, digital subtraction angiography, and single-photon emission computed tomography (SPECT) radionuclide imaging. For a more detailed discussion of these diagnostic tests, see Chapter 17.

Management

Early Management

When a patient with a possible MI arrives in the ED, the diagnosis and initial management of the patient must be rapid because the benefit of reperfusion therapy is greatest if therapy is initiated quickly. An initial evaluation of the patient should occur ideally within the first 10 minutes after arrival.[14] The patient's history and 12-lead ECG are the primary methods used to determine initially the diagnosis of MI. The ECG is examined for the presence of ST-segment elevations of 1 mm or greater in contiguous leads. This pattern provides evidence of thrombotic coronary arterial occlusion. The patient is placed on a continuous cardiac monitor with ST-segment monitoring capabilities.

If the initial screening suggests an MI, the interventions listed in Box 21-6 are initiated. The nurse checks the vital signs frequently, establishes IV access, and continuously assesses the patient's cardiac rhythm. Blood is drawn for serum cardiac markers, hematology, chemistry, and lipid profile. A chest radiograph and echocardiogram, obtained as soon as possible, are useful in ruling out an aortic dissection and acute pericarditis. During the initial evaluation, the patient and family may be anxious, necessitating brief and clear explanations of the interventions. Reassurance and support are essential components of the nurse's responsibilities.

FIBRINOLYTIC THERAPY. If the patient is diagnosed with an MI, fibrinolytic therapy may be used to establish reperfusion if there are no contraindications to its use. Fibrinolytic drugs lyse coronary thrombi by converting plasminogen to plasmin. This conversion causes the degradation of fibrin and fibrinogen, resulting in clot lysis. Box 21-7 lists contraindications to fibrinolytic therapy.[14] Fibrinolytic agents used for treating patients with STEMI include streptokinase, alteplase, reteplase, and tenecteplase.[17]

The goal is to complete the assessment of the patient and the administration of the fibrinolytic drug (if indicated) within 30 minutes of the patient's arrival to the ED. Fibrinolytic therapy provides maximal benefit if given within the first 3 hours after the onset of symptoms. There is a time-dependent decrease in efficacy of fibrinolytic therapy.[14]

For the patient receiving fibrinolytic therapy, two to three 18-gauge peripheral IV lines are usually started. One line is for the fibrinolytic agent, and one to two lines are for the administration of other drugs. Subclavian and jugular sites are avoided because they are noncompressible, and blood could be lost into the chest or neck. Some type of blood sampling device is also inserted so that peripheral venous punctures can be avoided.

The patient is closely monitored during and after the infusion of a fibrinolytic agent. The nurse assesses the patient for

BOX 21-6 Initial Management of the Patient With a Suspected Myocardial Infarction

Action: Administer aspirin, 160 to 325 mg chewed.
Rationale: Aspirin is used because it diminishes platelet aggregation. This effect is important because platelets are one of the main components in thrombus formation when a coronary plaque is disrupted. Aspirin has been shown to reduce mortality rates independently in patients with acute myocardial infarction (AMI). Patients diagnosed with an MI should be continued on aspirin indefinitely.
Action: After recording the initial 12-lead electrocardiogram (ECG), place the patient on a cardiac monitor and obtain serial ECGs.
Rationale: The 12-lead ECG is central in the decision pathway for the diagnosis and treatment of the patient. The patient is placed on a continuous cardiac monitor after the 12-lead ECG is recorded to detect dysrhythmias and to monitor ST-segment changes.
Action: Give oxygen by nasal cannula.
Rationale: Hypoxemia often occurs in patients with an MI because of pulmonary edema. If severe pulmonary edema is present and the patient is in respiratory distress, intubation may be necessary. A pulse oximeter is often applied, and, when time permits, an arterial blood gas may be drawn.
Action: Administer sublingual nitroglycerin (unless the systolic blood pressure is <90 mm Hg or the heart rate is <50 or >100 beats/min). Give 0.4 mg every 5 minutes for a total of three doses.
Rationale: Nitroglycerin helps to promote vasodilation but is relatively ineffective in relieving pain in the early stages of an MI. Intravenous nitroglycerin is recommended for patients with AMI with persistent pain, for control of hypertension, or for management of pulmonary congestion.
Action: Provide adequate analgesia with morphine sulfate.
Rationale: Morphine is the drug of choice to relieve the pain of an MI. The drug is given intravenously in small doses (2 to 4 mg) and can be repeated every 5 minutes until the pain is relieved. Close respiratory and blood pressure monitoring are indicated because morphine can depress respirations and cause hypotension.
Action: Administer β-blocker.
Rationale: During the first few hours after the onset of ST-segment elevation MI, β-blocking agents may diminish myocardial oxygen demand by reducing heart rate, systemic arterial pressure, and myocardial contractility.

From Antman, EM, Anbe DT, Armstrong PW, et al: ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to revise the 1999 guidelines for the management of patients with acute myocardial infarction). Circulation 110:e82–e293, 2004.

BOX 21-7 Contraindications and Cautions for Fibrinolysis in ST-Segment Elevation Myocardial Infarction*

Absolute Contraindications
- Any prior intracranial hemorrhage
- Known structural cerebral vascular lesion (eg, arteriovenous malformation)
- Known malignant intracranial neoplasm (primary or metastatic)
- Ischemic stroke within 3 months *except* acute ischemic stroke within 3 hours
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Significant closed-head or facial trauma within 3 months

Relative Contraindications
- History of chronic, severe, poorly controlled hypertension
- Severe uncontrolled hypertension on presentation (systolic blood pressure >180 mm Hg or diastolic blood pressure >110 mm Hg)†
- History of prior ischemic stroke greater than 3 months, dementia, or known intracranial pathology not covered in contraindications
- Traumatic or prolonged (>10 minutes) CPR or major surgery (<3 weeks)
- Recent (within 2 to 4 weeks) internal bleeding
- Noncompressible vascular punctures
- For streptokinase/anistreplase: prior exposure (more than 5 days ago) or prior allergic reaction to these agents
- Pregnancy
- Active peptic ulcer
- Current use of anticoagulants: the higher the INR, the higher the risk of bleeding

*Viewed as advisory for clinical decision making and may not be all inclusive or definitive.

†Could be an absolute contraindication in low-risk patients with myocardial infarction.

CPR, cardiopulmonary resuscitation; INR, international normalized ratio.

From Antman EM, Anbe DT, Armstrong PW, et al: ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to revise the 1999 guidelines for the management of patients with acute myocardial infarction). Circulation 110:e127, 2004, with permission.

resolution of chest pain, normalization of elevated ST segments, development of reperfusion dysrhythmias, any allergic reactions, evidence of bleeding, and the onset of hypotension. Commonly seen reperfusion dysrhythmias include an accelerated idioventricular rhythm, ventricular tachycardia, and AV heart block.

Evaluation of complications remains a key nursing intervention. The patient is closely monitored for evidence of reocclusion of the coronary artery. Indicators of reocclusion include chest pain, ST-segment elevation, and hemodynamic instability. Close observation for evidence of bleeding also is essential. The patient is carefully assessed for indications of subcutaneous or mucous membrane bleeding. The nurse also monitors the patient for signs of internal bleeding, including positive results of urine and stool for blood or altered levels of consciousness due to intracranial bleeding.

PERCUTANEOUS CORONARY INTERVENTION. Early reperfusion of myocardial tissue is essential to preserve myocardial function. In addition to pharmacological therapy, percutaneous coronary intervention (PCI) is an effective alternative to reestablish blood flow to ischemic myocardium. PTCA, an invasive procedure in which the infarction-related coronary artery is dilated with a balloon catheter, is the type of PCI used. Once the artery is opened by the balloon, a stent may be placed in the artery. PTCA is used for patients who present within 12 hours of the onset of symptoms, and it also can be performed in patients whose ischemic symptoms persist. This therapeutic intervention necessitates the availability of a cardiac catheterization laboratory and skilled personnel at all times.[14] A glycoprotein IIb/IIIa antagonist is often used for the MI patient who is undergoing a PCI. These drugs are potent and specific inhibitors of platelet aggregation.[7] (See Chapter 18 for a more detailed discussion of the PTCA procedure.)

Evaluation of patients for PTCA is similar to that of fibrinolytic therapy. The accessibility of the lesion in the coronary artery is an additional factor that must be considered. PTCA may be an excellent reperfusion alternative for patients ineligible for fibrinolytic therapy. The nurse must carefully monitor the patient after a PTCA for evidence of complications. These complications can include retroperitoneal or vascular hemorrhage, other evidence of bleeding, early acute reocclusion, and late restenosis. If the PCI is not successful, the patient may be evaluated for an emergent CABG procedure.

Intensive and Intermediate Care Management

The management goal for the patient in the intensive care unit and intermediate care unit continues to be maximizing cardiac output while carefully minimizing cardiac workload. To achieve this goal, the patient frequently has vital signs taken and continues on a cardiac monitor with ST-segment monitoring. The lead selected for monitoring should be based on the infarct location and underlying rhythm. Serial ECGs and serial evaluations of serum cardiac markers of infarction are recorded. Serum hematology and chemistry are monitored.

For the first 12 hours of hospitalization, patients who are hemodynamically stable and free of ischemic-type chest discomfort remain on bed rest with bedside commode privileges. Activity level increases gradually in hemodynamically stable patients. Careful attention is paid to maximal pain relief. Nitroglycerin is not an appropriate substitute for analgesics. A pulse oximeter is used to monitor oxygen saturation continuously and is a good indicator of early hypoxemia. When the oxygen saturation level is stable for more than 6 hours, the need for continuous oxygen therapy is reassessed.[14]

The patient is often not given anything by mouth until pain free. When pain free, the patient is given clear liquids and progressed to a heart-healthy diet as tolerated. Daily weights are recorded, and intake and output are measured to detect fluid retention. Stool softeners are administered so that the patient avoids a Valsalva maneuver. During a Valsalva maneuver, forced expiration against a closed glottis causes sudden and significant changes in systolic blood pressure and heart rate. These changes may influence regional endocardial repolarization and place the patient at risk for ventricular dysrhythmias. Nursing diagnoses for patients with acute MI are listed in Box 21-8.

PHARMACOLOGICAL THERAPY. Prophylactic antidysrhythmics during the first 24 hours of hospitalization are not recommended. However, easy access to atropine, lidocaine, amiodarone, transcutaneous pacing patches,

BOX 21–8 EXAMPLES OF NURSING DIAGNOSES

For the Patient With Acute Myocardial Infarction

- Acute Pain related to MI, angina
- Decreased Cardiac Output: Electrical factors affecting rate, rhythm, or conduction
- Decreased Cardiac Output: Mechanical factors related to preload, afterload, or left ventricular failure
- Deficient Knowledge related to illness and impact on patient's future
- Anxiety, Stress Overload related to fear of illness, death, and critical care environment
- Activity Intolerance related to decreased cardiac output or alterations in myocardial tissue perfusion
- Risk for Ineffective Cerebral Tissue Perfusion related to thrombolytic therapy impact

transvenous pacing wires, a defibrillator, and epinephrine is essential for management of dysrhythmias. Daily aspirin is continued on an indefinite basis. Clopidogrel is added to the aspirin regimen for patients with an STEMI and is continued for 14 days.[17]

ACE inhibitors are administered orally within the first 24 hours to patients with anterior wall MI, pulmonary congestion, or left ventricular ejection fraction less than 40%, in the absence of hypotension. ACE inhibitors help prevent ventricular remodeling (dilation) and preserve ejection fraction.[14]

During the first several days after STEMI, it is important to normalize the patient's blood glucose levels. An insulin infusion may be required to achieve this goal.[14]

Patients with a documented magnesium deficit should receive magnesium. Patients with torsades de pointes ventricular tachycardia with prolonged QT intervals also should be treated with magnesium.[14]

IV β-blocker therapy should be administered within the initial hours of the evolving infarction, followed by oral therapy provided there are no contraindications. β-Blockers are one of the few pharmacological agents that have been shown to reduce morbidity and mortality in the patient with an MI. They reduce oxygen demand by decreasing the heart rate and contractility. They also increase coronary artery filling by prolonging diastole. Calcium channel blockers may be given to patients in whom β-blocker therapy is ineffective or contraindicated.[14] The continuation of nitrate therapy beyond the first 24 to 48 hours may be useful for patients with recurrent angina or persistent heart failure.[14]

IV unfractionated heparin or low–molecular-weight heparin is used after STEMI in patients who are at high risk for systemic emboli. The risk is highest in patients with an anterior MI, atrial fibrillation, cardiogenic shock, or a previous embolus.[14]

HEMODYNAMIC MONITORING. Use of a pulmonary artery catheter for hemodynamic monitoring is indicated in the patient with MI who has severe or progressive congestive heart failure or pulmonary edema, cardiogenic shock, progressive hypotension, or suspected mechanical complications, such as ventricular septal defect, papillary muscle rupture, or pericardial tamponade.[14] The pulmonary artery occlusion pressure (PAOP) is closely followed for assessment of left ventricular filling pressures. A PAOP below 18 mm Hg may indicate volume depletion, whereas a PAOP greater than 18 mm Hg indicates pulmonary congestion or cardiogenic shock. Using the thermodilution technique, frequent measurements of cardiac output and cardiac index can be made to evaluate hemodynamic status further. In some situations, monitoring venous oxygen saturation may also be useful. For a more detailed discussion of hemodynamic monitoring, see Chapter 17.

Invasive arterial monitoring is indicated for patients with MI who have severe hypotension or for those receiving vasopressor or vasodilator drugs. The collaborative care guide for the patient with an MI (Box 21-9) provides further information about the care of these patients.

ADDITIONAL DIAGNOSTIC TESTING. At times, additional testing may be needed.

Computer Imaging Tests. One category of computer imaging test is radionuclide imaging and radionuclide angiography. Radionuclide studies provide information about the presence of coronary artery disease as well as the location and quantity of ischemic and infracted myocardium. A radioactive tracer is injected into the patient, and computer-generated images are created. Radionuclide tests include thallium tests, multiple-gated acquisition scans, and infarction scintigraphy.

Another type of computer imaging test is MRI. This diagnostic test uses strong magnets and low-energy radiofrequency signals to reveal structural and functional abnormalities of the heart and aorta. Coronary magnetic resonance angiography uses the principles of MRI in combination with a contrast medium to create images of vessel walls and the presence of any plaques.

Computed tomography (CT) is a type of computer imaging study that provides cross-sectional images of the chest, including the heart and aorta. CT angiography involves a CT scan of the heart after injection of a contrast medium. In addition to providing information about the structure of the heart, CT angiography offers information about the circulation of blood in the heart and coronary arteries. Electron beam CT is a type of ultrafast CT and is considered the gold standard for detecting and quantifying the amount of calcium in coronary plaques. Electron beam CT can note the formation of an atherosclerotic plaque before the development of significant stenotic lesions.[18]

Cardiac PET is another type of computed imaging test. PET combines CT imaging with radionuclide agents to detect coronary artery disease and injured but viable myocardial muscle. PET is a helpful test to determine myocardial muscle viability.

SPECT is a computed imaging test that involves the injection of a radionuclide agent followed by a series of computed graphics of the chest. SPECT is used to determine the extent and severity of blood flow abnormalities and coronary artery disease.[18]

A more detailed discussion of computed imaging tests can be found in Chapter 17.

Echocardiogram. An echocardiogram is a noninvasive ultrasonographic test involving the transmission of high-frequency sound waves into the heart. This commonly used diagnostic test helps determine ejection fraction, segmental wall motion, systolic and diastolic ventricular volumes, valve function, mural thrombi, pericardial fluid, intracardiac

BOX 21–9 COLLABORATIVE CARE GUIDE for the Patient With Myocardial Infarction

Outcomes	Interventions
Oxygenation/Ventilation	
Patient has arterial blood gases within normal limits and pulse oximeter value >90%.	• Assess respiratory rate, effort, and breath sounds every 2–4 h. • Obtain arterial blood gases per order or signs of respiratory distress. • Monitor arterial saturation by pulse oximeter. • Provide supplemental oxygen by nasal cannula or face mask for the first 6 h, then as needed. • Provide intubation and mechanical ventilation as necessary. (Refer to Box 25-16, pp. 538–539).
There is no evidence of pulmonary edema on chest x-ray and by clear breath sounds.	• Obtain chest x-ray daily. • Administer diuretics per order. • Monitor signs of fluid overload as described below.
There is no evidence of atelectasis.	• Encourage nonintubated patients to use incentive spirometer, cough, and deep breath every 4 h and PRN. • While on bed rest, turn patient side to side every 2 h.
Circulation/Perfusion	
Vital signs are within normal limits, including MAP > 70 mm Hg and cardiac index > 2.2 L/min/m^2.	• Monitor HR and BP every 1–2 h and PRN during acute failure phase. • Assist with pulmonary artery catheter insertion. • Monitor PAP and pulmonary artery occlusion pressure (PAOP), CVP, or right atrial pressure (RAP) every 1 h and cardiac output, SVR, and PVR every 6–12 h if pulmonary artery catheter is in place. • Maintain patent IV access. • Administer positive inotropic agents, and reduce afterload with vasodilating agents guided by hemodynamic parameters and physician orders. • Evaluate effect of medications on BP, HR, and hemodynamic parameters. • Prepare patient for intra-aortic balloon pump assist if necessary.
Patient has no evidence of heart failure due to decreased cardiac output.	• Restrict volume administration as indicated by PAOP or CVP values. • Assess for neck vein distention, pulmonary crackles, S_3 or S_4, peripheral edema, increased preload parameters, elevated a wave of CVP, RAP, or PAOP waveform. • Monitor 12-lead ECG daily and PRN.
Patient has no evidence of further myocardial dysfunction, such as altered ECG or cardiac enzymes.	• Monitor cardiac markers, magnesium, phosphorus, calcium, and potassium as ordered. • Monitor ECG for changes consistent with evolving MI. • Consider obtaining right precordial chest leads, 12-lead ECG, if inferior wall/right ventricle is involved. • Report and treat abnormalities per protocols or orders.
Dysrhythmias are controlled.	• Provide continuous ECG and ST-segment monitoring in the appropriate leads. • Document rhythm strips every shift. • Anticipate need for/administer pharmacological agents to control dysrhythmias.
After fibrinolytic therapy, patient will have relief of pain; no evidence of bleeding; no evidence of allergic reaction.	• Assess, monitor, and treat pain as described below. • Monitor signs of reperfusion, such as dysrhythmias, ST-segment return to baseline. • Monitor for signs of bleeding, including neurological, GI, and GU assessment. • Monitor PT, aPTT, ACT per protocol. • Have anticoagulant antidotes available. • Assess for itching, hives, sudden onset of hypotension, or tachycardia. • Administer hydrocortisone or diphenhydramine (Benadryl) per protocol.
There is no evidence of cardiogenic shock, cardiac valve dysfunction, or ventricular septal defect.	• Monitor ECG, heart sounds, hemodynamic parameters, level of consciousness, and breath sounds for changes. • Report and treat deleterious changes as indicated.
Fluids/Electrolytes	
Renal function is maintained as evidenced by urine output >30 mL/h and normal laboratory values.	• Monitor intake and output every 1–2 h. • Monitor blood urea nitrogen, creatinine, and electrolytes daily and PRN. Take daily weights. • Administer fluid volume and diuretics as ordered.

(continued on page 438)

BOX 21–9 COLLABORATIVE CARE GUIDE for the Patient With Myocardial Infarction (continued)

Outcomes	Interventions
Mobility/Safety	
Patient will comply with activity of daily living limitations.	• Provide clear explanation of limitations. • Provide bed rest with bed side commode privileges first 6 h. • Progress to chair for meals, bathing self, and bathroom privileges. Continually assess patient response to all activities.
Patient will not fall or accidentally harm self.	• Provide environment to prevent falls, bruising, or injury. • Use self-protective devices as indicated and per hospital policy.
Skin Integrity	
Patient has no evidence of skin breakdown.	• Turn side to side every 2 h while patient is on bed rest. • Evaluate skin for signs of pressure areas when turning. • Consider pressure relief/reduction mattress for high-risk patients. • Use Braden scale (see Chapter 51) to monitor risk for skin breakdown.
Nutrition	
Caloric and nutrient intake meets metabolic requirements per calculation (eg, basal energy expenditure).	• Provide appropriate diet: oral, parenteral, or enteral feeding. • Provide clear or full liquids during the first 24 h. • Restrict sodium, fat, cholesterol, fluid, and calories if indicated. • Consult dietitian or nutritional support services.
Patient has normal laboratory values reflective of nutritional status.	• Monitor albumin, prealbumin, transferrin, cholesterol, triglycerides, total protein.
Comfort/Pain Control	
Patient has relief of chest pain.	• Use visual analog scale to assess pain quantity.
There is no evidence of pain, such as increased HR, BP, RR, or agitation during activity or procedures.	• Assess quality, duration, and location of pain. • Administer IV morphine sulfate, and monitor pain and hemodynamic response. • Administer analgesics appropriately for chest pain and assess response. • Monitor physiological response to pain during procedures or after administration of pain medication. • Provide a calm, quiet environment.
Psychosocial	
Patient demonstrates decreased anxiety by calm demeanor and vital signs during, for example, procedures and discussions.	• Assess vital signs during treatments and interactions. • Provide explanations and stable reassurance in calm and caring manner. • Cautiously administer sedatives and monitor response.
Patient/family demonstrates understanding of MI and treatment plan by asking questions and participating in care.	• Consult social services and clergy as appropriate. • Assess coping mechanism history. • Allow free expression of feelings. • Encourage patient/family participation in care as soon as feasible. • Provide blocks of time for adequate rest and sleep.
Teaching/Discharge Planning	
Patient reports occurrence of chest pain or discomfort.	• Explain importance of reporting all episodes of chest pain. • Provide frequent explanations and information to family.
Family demonstrates appropriate coping during the critical phase of an acute MI.	• Encourage family to ask questions regarding treatment plan, patient response to therapy, prognosis, and so forth.
In preparation for discharge to home, patient understands activity levels, dietary restrictions, medication regimen, and what to do if pain recurs.	• Make appropriate referrals and consults early during hospitalization. • Initiate family education regarding heart-healthy diet, cardiac rehabilitation program, stress-reduction strategies, and management of chest pain after crisis phase has passed.

tumors, and aortic dissection.[14] Two-dimensional, Doppler, and transesophageal echocardiograms are the most frequently used types of echocardiograms for patients with an MI. (See Chapter 17 for further discussion of echocardiograms.)

Stress Test. Stress testing, also known as exercise electrocardiography, may be performed before discharge or within the first 3 weeks after discharge. The test is intended to assess the patient's functional capacity and ability to perform activities of daily living, to evaluate the efficacy of the patient's medical therapy, and to risk-stratify the patient based on the likelihood of a subsequent cardiac event. Stress testing may be combined with perfusion imaging to determine better the size of the infarction. (See Chapter 17 for further discussion of stress testing.)

Coronary Angiography. During the course of hospitalization, patients may be further evaluated by coronary angiography. Results of the angiography help the physician determine whether a PTCA or placement of a stent is indicated, or if the patient is a candidate for CABG. (A more detailed discussion of PTCA can be found in Chapter 18, and a more detailed discussion of CABG can be found in Chapter 22.)

Complications

The nurse closely monitors the patient with MI for evidence of complications. Numerous complications can occur, and a list of possible complications is provided in Box 21-10. Prompt recognition and management of complications are essential in reducing mortality and morbidity.

Hemodynamic Complications

Recurrent myocardial ischemia can occur in patients and is often transient. A recurrent MI is another possible complication. If the reinfarction occurs within the first 24 hours, it may be hard to diagnose because the cardiac serum markers have not yet returned to baseline. Early recognition and management are essential for both of these vascular complications. Efforts are made to lower myocardial oxygen demand and to relieve pain. Emergent PTCA or surgical revascularization may be considered.

Cardiogenic shock is the most serious myocardial complication of MI. It occurs because of the loss of contractile forces in the heart, resulting in left ventricular dysfunction. Cardiogenic shock is the most common cause of in-hospital death for patients with MI. (For a more detailed discussion of cardiogenic shock, see Chapter 54.)

Clinical manifestations of cardiogenic shock include a rapid, thready pulse; a narrow pulse pressure; dyspnea; tachypnea; inspiratory crackles; distended neck veins; chest pain; cool, moist skin; oliguria; and decreased mentation. Arterial blood gas analysis reveals a decreased PaO_2 and respiratory alkalosis. Hemodynamic findings include a systolic blood pressure less than 85 mm Hg, a mean arterial blood pressure less than 65 mm Hg, a cardiac index less than 2.2 L/min/m^2, and a PAOP greater than 18 mm Hg. Cardiac enzymes may show an additional rise or a delay in reaching peak values.

The goal of treatment for cardiogenic shock is to minimize myocardial workload and maximize myocardial oxygen delivery. Immediate actions must be taken to improve tissue perfusion and preserve viable myocardium. To improve oxygenation, supplemental oxygen is given to the patient and, if necessary, the patient may be intubated and placed on a mechanical ventilator. Efforts are aimed toward restoring blood pressure. This may require discontinuation of vasodilator drugs and drugs with negative inotropic effects. An IV dopamine drip may be initiated to improve the patient's blood pressure and improve myocardial contractility. Dobutamine may be used to improve contractility, especially in low cardiac output states. Nitroprusside, a vasodilator, may be used with a vasopressor to improve cardiac output by decreasing peripheral vascular resistance and reducing left ventricular preload. Treatment may also require the use of an IABP. This invasive device helps improve coronary artery perfusion and decrease left ventricular afterload. (For a more detailed discussion of IABP therapy, see Chapter 18.)

BOX 21-10 Complications of Acute Myocardial Infarction

Hemodynamic Complications
- Hypotension
- Pulmonary congestion
- Cardiogenic shock
- Right ventricular infarction
- Recurrent ischemia
- Recurrent infarction

Myocardial Complications
- Diastolic dysfunction
- Systolic dysfunction
- Congestive heart failure

Mechanical Complications
- Mitral valve regurgitation
- Left ventricular free wall rupture
- Ventricular septal rupture
- Left ventricular aneurysm

Pericardial Complications
- Pericarditis
- Dressler's syndrome
- Pericardial effusion

Thromboembolic Complications
- Mural thrombosis
- Systemic thromboembolism
- Deep venous thrombosis
- Pulmonary embolism

Dysrhythmia Complications
- Ventricular tachycardia
- Ventricular fibrillation
- Supraventricular tachydysrhythmias
- Bradydysrhythmias
- Atrioventricular block (first, second, or third degree)

From Antman EM, Anbe DT, Armstrong PW, et al: ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to revise the 1999 guidelines for the management of patients with acute myocardial infarction). Circulation 110:e82–e293, 2004.

Mechanical Complications

The most catastrophic mechanical complications of MI are intraventricular septal rupture and left ventricular free wall rupture. These clinical situations develop rapidly and result in almost immediate physiological deterioration.

VENTRICULAR SEPTAL WALL RUPTURE. The frequency of acute rupture of the intraventricular septum has declined with the use of reperfusion therapy. Ventricular septal rupture occurs in about 1% of patients with an STEMI and in about 4% of those patients who also have cardiogenic shock.[14] The greatest risk for ventricular septal wall rupture is within the first 24 hours and continues for up to 5 days.[14] The patient presents with a new, loud, holosystolic murmur associated with a thrill felt in the parasternal area. In addition, the patient has progressive dyspnea, tachycardia, and pulmonary congestion. Oxygen samples taken from the right atrium, right ventricle, and pulmonary artery show a higher PaO_2 in the right ventricle than in the right atrium because the oxygenated left ventricular blood is shunted to the right ventricle. This testing can be accomplished during pulmonary artery catheterization. Urgent cardiac catheterization and surgical correction are needed. The patient can be supported with fluid administration, inotropic support (dopamine and dobutamine), afterload reduction (nitroprusside), and IABP counterpulsation until emergency surgery is possible. Some fibrosis of the tissue is needed for suturing. Often it is impossible to maintain the patient medically until this occurs.

LEFT VENTRICULAR FREE WALL RUPTURE. Left ventricular free wall rupture occurs in about 1% to 6% of patients with STEMI.[14] Left ventricular free wall rupture is more likely to occur either within the first 24 hours after MI or 3 to 5 days after MI.[14] Early rupture is a result of the initial evolution of the infarction before significant collagen deposition. Late rupture is related to expansion of the infarction in the ventricular wall.[14] Left ventricular free wall rupture is more likely to occur in patients older than 70 years of age, women, hypertensive patients, and patients with their first MI. The patient experiences prolonged chest pain, dyspnea, sudden hypotension, jugular venous distention, tamponade, and ECG evidence of electrical–mechanical dissociation. This event occurs so suddenly and with such severity that lifesaving efforts are often futile.

PERICARDIAL COMPLICATION. Pericarditis is common after MI and can occur as soon as the first 3 days after infarction or as late as several weeks after infarction. The patient reports chest pain that may be confused with ischemic pain. The precordial pain of pericarditis intensifies with deep breathing, coughing, swallowing, and lying flat. The pain is lessened when the patient sits up and leans forward. The patient may have a fever, with a temperature usually less than 38.6°C, that lasts for several days. Often on auscultation, a friction rub can be heard along the left sternal border. Some friction rubs are transient; therefore, the absence of such a rub is not conclusive. Often the ECG shows concave upward ST-segment elevation on five or more leads. Anti-inflammatory agents, such as aspirin, indomethacin, and corticosteroids, are given in usual doses for 7 to 14 days.[14]

THROMBOEMBOLIC COMPLICATIONS. Thromboembolisms occur in fewer MI patients than previously because of the routine use of anticoagulants. Those patients with a deep venous thrombosis (DVT) may be predisposed because of the systemic inflammatory response associated with infarction, immobility, venous stasis, and reduced cardiac output. Pulmonary embolism is a risk for patients with DVT. After MI, patients are also at risk for systemic emboli that usually originate in the wall of the left ventricle. These emboli can occlude the cerebral, renal, mesenteric, or iliofemoral artery. Patients are systemically anticoagulated with unfractionated or low–molecular-weight heparin followed by warfarin (Coumadin) for 6 to 12 months.[14]

DYSRHYTHMIA COMPLICATIONS. Cardiac dysrhythmias and conduction disturbances often accompany acute MIs and can be life threatening. The causes of electrical complications are many and include myocardial ischemia, myocardial necrosis, altered autonomic tone, electrolyte imbalances, acid–base disturbances, and adverse drug effects.

Ventricular dysrhythmias that occur in the prehospital phase cause the majority of sudden cardiac deaths. Ischemic myocardium has a lower fibrillatory threshold, and few ventricular dysrhythmias are considered benign after an infarction. Patients may experience tachydysrhythmias or bradydysrhythmias during the hospital phase of treatment. Supraventricular rhythms may be the result of high left atrial pressures caused by left ventricular failure.

Conduction disturbances after MI can include those caused by SA node, AV node, or ventricular conducting tissue abnormalities. The right coronary artery supplies the SA node in about half of all patients, and the left circumflex coronary artery supplies the SA node in the other half. Because the right coronary artery is also the source of oxygenated blood for the inferior, right posterior, and right ventricular walls, patients with inferior, right posterior, or right ventricular wall MIs are at risk for conduction disturbances resulting from poor SA node functioning. Patients with lateral wall MIs also are at risk for SA nodal conduction disturbances because the left circumflex vessel supplies the lateral wall of the heart.

The right coronary artery also is the source of oxygenated blood for the AV node in about 90% of people. Therefore, patients with inferior, right posterior, or right ventricular wall infarctions due to right coronary artery occlusion are at risk for AV nodal conduction disturbances. First-degree heart block and Mobitz type I (Wenckebach) block may appear, but often are transient. These rhythm disturbances may progress to complete heart block and require pacing therapy.

The LAD coronary artery is the primary source of blood supply to the bundle of His and bundle branches. Therefore, patients with an anterior wall MI caused by an LAD occlusion are at risk for ventricular conduction defects. Conduction defects, such as right bundle branch block, left bundle branch block, anterior fascicular block, posterior fascicular block, bifascicular block, or trifascicular block, may occur.

For patients with MI, the nurse continuously monitors the cardiac rate and rhythm, assesses the apical and peripheral pulses, auscultates the heart, and monitors blood pressure and other indicators of hemodynamics, such as urine output and level of consciousness. The goals of therapy for cardiac dysrhythmias and conduction disturbances are to restore normal rate, rhythm, and AV synchrony and to maintain

Table 21-2 Patient Teaching: Goals After Acute Myocardial Infarction

	When Mastery of Content Is To Be Expected:		
Content	***Acute Phase***	***Before ICU Discharge***	***At Hospital Discharge***
Pathophysiology of heart disease	Can identify angina, using 0–10 pain scale for reference	Can initiate treatment of angina (rest, nitroglycerin, O_2 use)	Knowledgeable about medications, when to seek medical assistance
Environment of hospital	Understands procedures	Asks appropriate questions	Knowledgeable about disease process and therapy
Lifestyle modifications	Complies with activity limitations Complies with dietary limitations	Can state relationship between activity and cardiac workload Begins light activity States risk factors Selects appropriate meals	Can progress activity as tolerated Placement in cardiac rehabilitation program Can state dietary restrictions
Treatment of disease	Accepts medications as ordered	Can identify medications Can identify risk factors	Knowledgeable about medications, dose, timing, action, and side effects Plans for risk factor reduction Begins cardiac rehabilitation program
Emotional adaptation	Able to define support system	Begins to communicate about lifestyle changes Becomes involved with resolving emotions related to surviving a critical illness	Involves self and loved ones in plans for lifestyle changes Expresses feelings Participates in group recovery program

adequate cardiac output. To achieve these goals, pharmacological therapy may be indicated. Cardioversion may be used to treat patients with supraventricular dysrhythmias, such as atrial fibrillation or atrial flutter. Transcutaneous pacing may be indicated in an emergent situation for heart block dysrhythmias until a transvenous temporary pacemaker can be initiated. The patient may require permanent pacemaker implantation to maintain an adequate rate and rhythm. Some patients may require an implantable cardioverter–defibrillator to manage ventricular dysrhythmias. (For a more detailed discussion of pacemakers and implanted cardioverter–defibrillators, see Chapter 18.)

Cardiac Rehabilitation

Preparation for discharge must begin early in the patient's course of hospitalization. Patient and family education is an essential component of the process. A severely compromised, critically ill patient may lack the ability to process and retain new information but usually is motivated to learn after the life-threatening event. Guidelines for patient and family education after an acute MI are described in Table 21-2.

Cardiac rehabilitation is recommended for most patients after MI. Cardiac rehabilitation involves a combination of prescribed exercise, education, and counseling. The goals of cardiac rehabilitation are to limit the adverse physiological and psychological effects of heart disease, modify risk factors, reduce the risk for sudden death or reinfarction, control cardiac symptoms, stabilize or reverse the atherosclerotic process, and enhance the patient's psychosocial and vocational status.[14] Components of cardiac rehabilitation programs include exercise, smoking cessation, lipid management, weight control, blood pressure control, psychological interventions, and guidance for return to work. Cardiac rehabilitation programs have been shown to improve the patient's functional capacity and quality of life and to decrease emotional distress, risk for subsequent coronary events, and cardiovascular mortality. Although the benefits of cardiac rehabilitation are well known, fewer than one third of patients receive information or counseling about cardiac rehabilitation before being discharged from the hospital.[14]

Family members of patients with MI should be included in the educational process so that they can learn about heart disease and help the patient achieve the goals of rehabilitation. Family members also should be given the opportunity to learn cardiopulmonary resuscitation because most episodes of cardiac arrest in patients with MI occur within the first 18 months after discharge from the hospital.[14]

▲ Clinical Applicability Challenges

CASE STUDY

Mr. M., a 71-year-old white male, was brought to the emergency department by an ambulance at 10:30 AM. He described substernal chest pain with radiation to his left arm that began 1 hour ago. The pain is not relieved by rest or sublingual nitroglycerin. He describes the pain as dull and rates it an 8 on a scale of 10. He feels nauseated but has not vomited. Mr. M. has a history of smoking, obesity, diabetes, and elevated cholesterol levels. He has no known drug allergies.

On physical examination, he was awake, alert, oriented, and agitated. His skin was cool and diaphoretic. Blood pressure was 98/50 mm Hg; heart rate, 110 beats/min and regular; respiratory rate, 22 breaths/min on 2 L O_2 per nasal cannula; and temperature, 98°F (36.7°C). Cardiac examination revealed S_1, S_2, and an S_3. Mr. M. had no jugular venous distention. Peripheral pulses were present but thready, and there was 1+ pedal edema bilaterally. Auscultation of the lungs revealed bilateral basilar crackles. He had no evidence of cyanosis or clubbing. His abdominal examination showed positive bowel sounds in all quadrants. His abdomen was soft and nontender with no palpable masses.

The nurse immediately recorded a 12-lead ECG that showed a 4-mm ST-segment elevation in leads V_1 through V_4. Blood samples were drawn that revealed an elevated CK level positive for MB. His troponin level was also abnormal. Mr. M. was given an aspirin, and an intravenous (IV) line was started. His pain was treated with IV morphine sulfate. Mr. M. was diagnosed with an acute anteroseptal wall MI and was admitted to the coronary care unit. On day 2 after admission, Mr. M.'s blood pressure was 82/58 mm Hg. His pulse was 128 beats/min and thready and respirations were 28 breaths/min. On physical examination, he displayed inspiratory crackles, distended neck veins, and cool moist skin. His urine output declined to 30 mL/h.

1. Mr. M. was diagnosed with an anteroseptal wall myocardial infarction (MI). What coronary artery is most likely occluded and what potential complications are priorities for you to monitor?

2. On day 2, Mr. M. sustained significant physiologic changes as noted by the physical examination findings. Mr. M. is most likely having what complication post-MI?

3. What are the goals for treating Mr. M. during day 2 after admission?

References

1. Mendis S, Puska P, Norrving B: Global atlas on cardiovascular disease prevention and control. The World Health Organization in collaboration with the World Heart Federation and the World Stroke Organization, Geneva, Switzerland, 2011
2. Kochanek KD, Xu J, Murphy SL, et al: Deaths: Preliminary Data for 2009 National Vital Statistics Reports, 59(4) by Division of Vital Statistics U.S. Department Of Health And Human Services Centers for Disease Control and Prevention National Center for Health Statistics National Vital Statistics System, March 16, 2011
3. Roger VL, Go AS, Lloyd-Jones DM, et al; on behalf of the American Heart Association Statistics Committee and Stroke Statistics Subcommittee: Heart disease and stroke statistics—2011 update: A report from the American Heart Association. Circulation 123:459–463, 2011
4. Ridker, PM: C-reactive protein: A simple test to help predict heart attack and stroke. Circulation 108:e81–85, 2003
5. American Heart Association, Risk Factors and Coronary Heart Disease: AHA Scientific Position. Available at www.americanheart.org. Accessed June 27, 2011
6. National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) Executive Summary. National Cholesterol Education Program, National Heart, Lung, and Blood Institute National Institutes of Health Publication Number 01-3670, May 2001
7. Kumar A, Cannon C: Acute coronary syndromes: Diagnosis and management, Part 1. Mayo Clin Proc 84(10):917–938, 2009
8. Anderson JL, Adams CD, Antman EM, et al: ACC/AHA 2007 guidelines for the management of patients with unstable angina/non-ST-segment elevation myocardial infarction: Executive summary. A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines for the Management of Patients With Unstable Angina/Non-ST-Elevation Myocardial Infarction). Circulation 116:803–877, 2007
9. Wright RS, Anderson JL, Adams CD, et al: 2011 ACCF/AHA focused update of the guidelines for the management of patients with unstable angina/non-ST-elevation myocardial infarction (Updating the 2007 guidelines): A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation 123:2022–2060, 2011
10. What is Angina? National Heart Lung and Blood Institute People Science Health. Available at: http://wwwnhlbi.nih.gov/health-topics/topics/angina. Accessed December 15, 2011
11. DeWood MA, Spores J, Notske R, et al: Prevalence of total coronary occlusion during the early hours of transmural myocardial infarction. N Engl J Med 303:897–902, 1980
12. Thygesen K, Alpert JS, White HD; on behalf of the Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction: Universal definition of myocardial infarction. Circulation 116:2634–2653, 2007
13. Reimer KA, Lower JE, Rasmussen MM, et al: The wave front phenomenon of ischemic cell death. 1. Myocardial infarct size versus duration of coronary occlusion in dogs. Circulation 56:786–794, 1977

14. Antman EM, Anbe DT, Armstrong PW, et al: ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to revise the 1999 guidelines for the management of patients with acute myocardial infarction). Circulation 110:e82–e293, 2004
15. Grauer K: A Practical Guide to ECG Interpretation, 2nd ed. St. Louis, MO: Mosby-Year Book, 1998
16. Jong GP, Ma T, Chou P, et al: Reciprocal changes in 12-lead electrocardiography can predict left main coronary artery lesion in patients with acute myocardial infarction. Int Heart J 47(1):13–20, 2006
17. Kumar A, Cannon C: Acute coronary syndromes: Diagnosis and management, Part II. Mayo Clin Proc 84(11):1021–1036, 2009
18. Soine L, Hanrahan M: Nuclear and other imaging studies. In Woods SL, Froelicher ES, Motzer SA, et al: Cardiac Nursing, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, pp 319–325

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

22 Cardiac Surgery

Nancy Munro

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Discuss the indications for coronary artery bypass grafting surgery and valvular surgery.
2. Describe the nursing care of the patient before and after coronary artery bypass grafting surgery.
3. Compare and contrast the pathophysiological implications of stenosis and insufficiency in the mitral and aortic valves.
4. Explain nursing interventions used to prevent complications post cardiac surgery.
5. Discuss the nursing care of the patient before and after carotid endarterectomy.

Despite emphasis on modifying and eliminating risk factors, cardiovascular disease remains a leading cause of disability and death in the United States. Development of new treatments, such as thrombolytic and anticoagulation therapy, balloon and laser angioplasty, and coronary artery stenting, has improved medical management of cardiac disease. These nonsurgical approaches are discussed in Chapter 18. However, surgical intervention remains the treatment of choice in some patient populations with coronary artery disease (CAD) and valvular disease.

▲ Indications for Cardiac Surgery

Coronary Artery Disease

A discussion of the pathophysiology of coronary artery disease (CAD) is found in Chapter 21.

Coronary Artery Bypass Graft Surgery

In coronary artery bypass graft (CABG) surgery, native vessels or conduits are "harvested" during the initial phase of surgery and used to reroute or bypass blood flow past diseased areas of the coronary arteries. CABG surgery has become an acceptable treatment for CAD. Compared with medical treatment, CABG surgery has proved effective in relieving angina and improving exercise tolerance, and it prolongs life in patients with left main CAD and three-vessel disease with poor left ventricular function.[1]

Increased use of percutaneous transluminal coronary angioplasty and stenting has decreased the need for CABG surgery in many cases. Patients selected for such surgery today are older, have more advanced coronary disease, have more impaired left ventricular function, and, in many cases, have had previous CABG surgery. To decrease the mortality associated with bypass surgery, it is necessary to consider several factors: urgency of operation, age, previous heart surgery, sex, left ventricular ejection fraction, percentage stenosis of the left main coronary artery, and number of major coronary arteries with greater than 70% stenosis.[1]

Desired characteristics for a graft or conduit are (1) diameter similar to the coronary arteries, (2) no disease or vessel wall abnormalities, and (3) adequate length. Commonly used grafts include saphenous vein grafts and internal mammary artery grafts.

SAPHENOUS VEIN GRAFTS. Saphenous vein grafts are used to bypass the obstruction in the coronary artery by anastomosing one end of the vein to the aorta (proximal anastomosis) and the other end to the coronary artery just past the obstruction (distal anastomosis). Saphenous vein grafts may be simple, with an end-to-side anastomosis to the aorta and the coronary artery, or sequential (also called skip), with an end-to-side anastomosis to the aorta, a side-to-side anastomosis to one coronary artery, and an end-to-side anastomosis to another coronary artery (Fig. 22-1).

Although the saphenous vein can be taken from above or below the knee, a vein from below the knee is generally preferred because it is close in diameter to the size of the coronary artery. To remove the vein, an incision is made along the inner aspect of the leg. Alternatively, small incisions can be made in the area of the vein, and a flexible fiberoptic scope is inserted to visualize the vessel and remove it. The fiberoptic method of vein removal is associated with improved wound healing and reduced complications involving the incision site.[2]

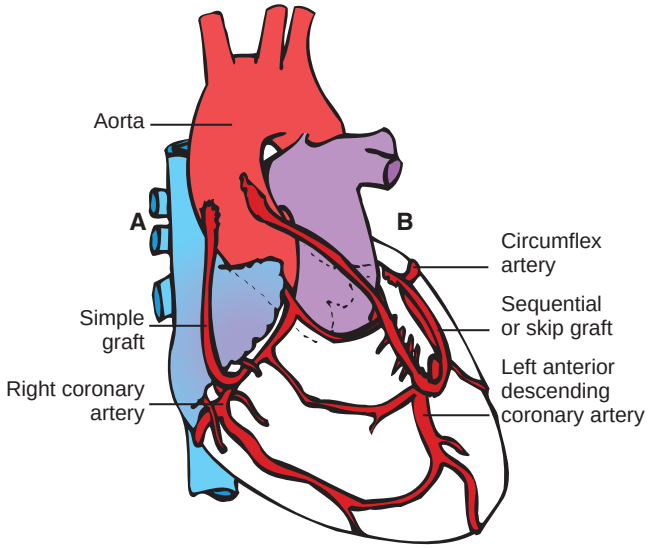


FIGURE 22-1 ▲ Aortocoronary bypass grafts using saphenous vein. **A:** Simple graft from aorta to right coronary artery. **B:** Sequential graft from aorta to left anterior descending coronary artery to diagonal or circumflex artery.

About 50% of saphenous vein grafts are occluded after 10 years. Three main processes account for saphenous vein failure: thrombosis, fibrointimal hyperplasia, and atherosclerosis. Thrombosis is most common in the first month but may occur as long as 1 year after surgery. Aspirin is the drug recommended for use postoperatively to prevent early saphenous vein graft closure and should be continued indefinitely.[1] To decrease the incidence of occlusion of the saphenous vein graft, grafts from other vessels are being used.[3]

INTERNAL MAMMARY ARTERY GRAFTS. The internal mammary artery is a preferred alternative to the saphenous vein for surgical revascularization. The internal mammary artery is used as a pedicle graft (ie, the proximal end remains attached to the subclavian artery) to bypass diseased coronary arteries. Both the left and the right internal mammary artery can be used. Because the left internal mammary artery is longer and larger than the right, it is usually used to bypass the left anterior descending coronary artery. The right internal mammary artery is anastomosed to the right coronary artery or the circumflex coronary artery. To isolate the internal mammary artery, the pleural space is entered, the internal mammary artery is dissected free from the chest wall, and the intercostal artery branches are cauterized. Compared with saphenous vein grafts, internal mammary artery grafts have superior graft patency rates; 90% were patent 10 years after surgery. In addition, internal mammary artery grafts exhibit less atherosclerosis over time, and they have been associated with lower long-term morbidity and improved long-term survival.[1]

OTHER GRAFTS. The search for other native vessels to serve as conduits continues as patients return for reoperation. The use of the radial artery has gained popularity; occlusion rates have lowered as harvesting techniques have improved. The radial artery, a thick, muscular artery, is prone to spasm with mechanical stimulation, and to prevent spasm, the artery is perfused with a calcium channel blocker solution during surgery and minimally stimulated. After the radial artery has been implanted, spasm has not been a major factor, and this conduit has good patency rates.[4] Initiation of nitroglycerin followed by oral nitrates (isosorbide mononitrate) postoperatively has helped decrease the occurrence of spasm; results have been better than with calcium channel blockers.[5]

Acceptable alternative conduits must have short- and long-term acceptable patency rates. The right gastroepiploic artery, which is harvested by extending the sternotomy incision toward the umbilicus and dissecting the artery off the greater curvature of the stomach, is used for coronary grafting. Patency rates have been acceptable, but long-term data are not available.[1] Homologous (nonnative) conduits using the saphenous vein, umbilical vein, or bovine internal mammary artery have resulted in poor patency rates and therefore are not recommended.[1] A comparison of common conduits used for revascularization is presented in Table 22-1.

Table 22-1 Common Conduits Used for Coronary Artery Bypass Grafting

Type of Graft	Advantages	Disadvantages
Internal mammary artery	• Vascular endothelium adapted to arterial pressure and high flow, resulting in decreased intimal hyperplasia and atherosclerosis • Improved long-term patency • Retains nerve innervation and therefore its ability to adapt diameter to blood flow • No leg incision • Diameter closer to coronary artery	• Dissection off the chest wall takes more time; long dissection time may increase risk for postoperative bleeding • Pleural chest tube needed because pleural space violated • Increased postoperative pain • Use of bilateral internal mammary arteries may increase risk for infection and sternal infection, especially in patients with diabetes
Saphenous vein	• Technically easier to harvest • Longer length (if able) may allow for several grafts	• Less long-term patency compared with internal mammary artery graft • Leg incision has tendency toward edema and infection; less common with fiberoptic approach
Radial artery	• Technically easier to harvest • Better patency rate compared with saphenous vein graft • Vascular endothelium adapted to arterial pressure and high flow, resulting in decreased intimal hyperplasia and atherosclerosis	• Tendency to spasm, although this can be treated medically • Preoperative assessment of ulnar artery's ability to supply alternative blood flow is important

Off-Pump Coronary Artery Bypass Graft Surgery

CABG surgery actually began as a surgical procedure performed on a beating heart because the cardiopulmonary bypass machine was not yet available. Once the cardiopulmonary bypass machine was perfected, "beating heart" surgery was used less often. This machine, also called a pump oxygenator, assumes the job of oxygenating the patient's blood and circulating it throughout the body. However, complications associated with cardiopulmonary bypass have led surgeons to reconsider performing CABG surgery "off pump" (OPCABG)—in other words, on beating hearts—in the hope of improving patient outcome.

Initially, surgeons wanted to avoid using the midline sternotomy incision and use the less invasive "mini" left and right thoracotomy approaches while performing CABG surgery, on or off bypass. This approach, which is known as minimally invasive direct coronary artery bypass grafting (MIDCABG), restricts the number of grafts that can be performed because the small incision does not allow access to the entire heart surface. Grafts to the left anterior descending artery are most frequent. Depending on where the "mini" incision is placed, grafts to the right coronary artery and the posterior descending artery can also be made.[6] Experience with MIDCABG has not been as successful as anticipated, but the technique is still used depending on the patient situation. The trend is toward using the median sternotomy incision but performing the grafting process off bypass.

In the 1990s, as neurological complications associated with the cardiopulmonary bypass machine, especially cognitive dysfunction, became more prominent, OPCABG surgery was reinstituted.[7] The initial results of OPCABG surgery have been promising but sometimes difficult to compare with the large data pool for on-pump CABG surgery. Length of stay in patients after OPCABG surgery has decreased compared with length of stay in patients who have had on-pump CABG surgery.[8] In addition, neurological dysfunction seems less after OPCABG surgery. However, Peel et al demonstrated that the stroke rate is similar in patients who undergo OPCABG compared with those who undergo on-pump CABG; after OPCABG, the symptoms of the stroke appear 48 to 72 hours after surgery, and after on-pump CABG, the symptoms of stroke occur immediately after surgery.[9] The explanation for this finding is that systemic inflammatory response syndrome (SIRS; discussed in detail in Chapter 54) causes diffuse microembolic events that take time to develop. The microembolization is the result of the inflammation of the endothelium, which activates the coagulation cascade.

The significance of these differences for patient care after OPCABG surgery is not well documented in the nursing literature because OPCABG surgery is a new procedure. Data from institutions with larger numbers of patients who have had this surgery seem to suggest that emphasis should be placed on anticoagulation interventions.[9] Traditional agents, such as heparin (weight-based protocols), aspirin, clopidogrel (Plavix), and low-molecular-weight heparin, are aggressively implemented to prevent platelet activation and suppress activation of the coagulation cascade. Nursing assessment focuses on the detection of embolic events in any body system and monitors for side effects of anticoagulation, such as gastrointestinal bleeding and heparin-induced thrombocytopenia. Because SIRS can develop in 48 to 72 hours, the critical care nurse must continually reassess the patient who has undergone OPCABG surgery and report any changes that may occur, especially with regard to neurological changes or electrocardiographic (ECG) ST-segment monitoring. If the thoracotomy approach is used, the increased need for pain medication may decrease patient compliance to cough and deep-breathe postoperatively. A high index of suspicion is the key nursing intervention after OPCABG surgery and may improve patient outcome.

Transmyocardial Laser Revascularization

Transmyocardial laser revascularization (TMR; TMLR) may be an option for patients who continue to have unstable angina that is refractory to interventions. Eligible patients usually have had prior CABG surgery, multiple cardiac interventions with maximal medication manipulation, or both. A laser probe is inserted into the wall of the left ventricle to create channels that encourage revascularization. The location and number of channels created depend on the patient's preoperative cardiac performance. Revascularization theoretically occurs through two mechanisms: (1) angiogenesis and (2) direct channel patency and endothelialization.[10] With angiogenesis, the formation of new blood vessels or the modeling of existing blood vessels increases collateral flow to the dysfunctional areas. Direct channel patency with endothelialization may result in direct perfusion to impaired walls of the left ventricle. Although the actual mechanisms are not clearly understood, the clinical outcomes are promising. In TMR, revascularization takes months to develop, which should be emphasized to the patient and family. In TMR, three types of lasers of different wavelengths are used: holmium:YAG, excimer, and carbon dioxide. All produce clean channels with minimal tissue trauma.[10]

Care of patients after TMR is similar to care after cardiovascular surgery, with some special concerns. The life of patients who have had TMR can become relatively normal, but careful observation is required. In TMR, patients have sustained direct myocardial insult that can result in a decrease in left ventricular function 48 to 72 hours after the surgery. Inotropic support using dobutamine or milrinone may be required for several days. Vigilant monitoring of fluid status is necessary because congestive heart failure tends to occur, although patients have higher filling pressures because the desired effects of TMR develop over time. Antiarrhythmic therapy may also be necessary because there may be irritable foci in the ventricle; amiodarone is frequently used. Angina may occur, but because the channel areas are denervated in TMR, the patient may be unable to perceive anginal pain. Continuous ST-segment monitoring is needed to detect any changes that occur. Nitrates are started as part of the medication regimen. Anticoagulation is initiated to prevent myocardial infarction (MI) and also to maintain the patency of the channels.[11]

Valvular Disease

Cardiac valves maintain the unidirectional flow of blood. If structural changes occur as a result of disease, this function is disrupted. Disease causes either valvular stenosis or insufficiency (regurgitation; Fig. 22-2). The stenotic valve

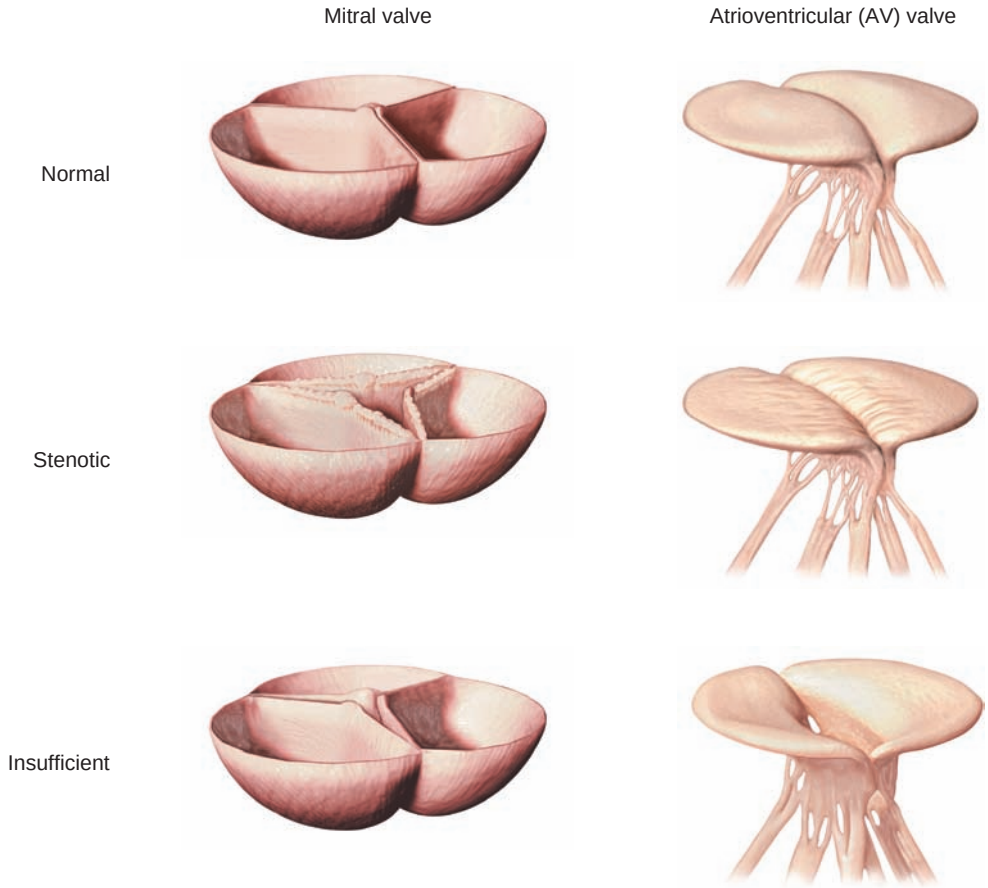


FIGURE 22-2 ▲ Normal and diseased heart valves. (From Anatomical Chart Company: Atlas of Pathophysiology. Springhouse, PA: Springhouse, 2010, pp 77.)

has a narrowed orifice that creates a partial obstruction to blood flow, resulting in increasing pressure behind the valve and decreasing forward blood flow. The insufficient valve is incompetent or leaky; blood flows backward, increasing the pressure and volume behind the valve. Stenosis and insufficiency can occur alone or in combination, in the same valve, or in more than one valve. Abnormalities can affect all four valves, but mitral and aortic abnormalities are more common and produce profound hemodynamic changes.

The diagnosis of valvular disease is suggested by the history, clinical signs and symptoms, physical examination, and auscultation of the characteristic murmur. Diagnosis is confirmed by echocardiography and catheterization of both sides of the heart, at which time valvular gradients are measured. To determine the gradient across the mitral valve, left atrial and left ventricular pressures are measured during diastole. A gradient of more than 15 to 20 mm Hg (ie, left atrial diastolic pressure is 15 to 20 mm Hg higher than left ventricular diastolic pressure) means that severe mitral stenosis exists. Valve area is also calculated during cardiac catheterization. The normal mitral valve area is 4 to 6 cm^2. An area less than 1.5 cm^2 signifies critical mitral stenosis, and surgery is indicated.

To determine the gradient across the aortic valve, the left ventricular and aortic root pressures are measured during systole. A gradient of more than 50 mm Hg is associated with clinically significant aortic stenosis. Normal aortic valve area is 2.6 to 3.5 cm^2. Hemodynamically, significant aortic stenosis occurs if the valve area is less than 1 cm^2. Valvular insufficiency is diagnosed by regurgitation of the contrast medium backward through the incompetent valve.

Pathophysiology

MITRAL STENOSIS. Mitral stenosis (Fig. 22-3A) occurs most frequently as a result of rheumatic heart disease. The disease process causes fusion of the commissures and fibrotic contraction of valve leaflets, commissures, and chordae tendineae. As forward flow from the left atrium to the left ventricle decreases, cardiac output falls, creating a decrease in systemic perfusion. Blood backed up behind the stenotic valve causes left atrial dilation and increased left atrial pressure. This pressure is reflected backward into the pulmonary circulation, and with prolonged high pressures, fluid moves from the pulmonary capillaries into the interstitial space and eventually the alveoli. Pulmonary hypertension develops, which can eventually lead to right-sided heart failure. As a result of this pathophysiology, patients with mitral stenosis present with fatigue, exertional dyspnea, orthopnea, and even pulmonary edema. Left atrial dilation causes atrial fibrillation in 40% to 50% of affected patients.

MITRAL INSUFFICIENCY. Mitral insufficiency (see Fig. 22-3B) can occur acutely or develop over a period of time. Chronic mitral insufficiency may result from rheumatic heart disease, degenerative changes associated with aging, or left ventricular dilation. The basic valve dysfunction is caused by

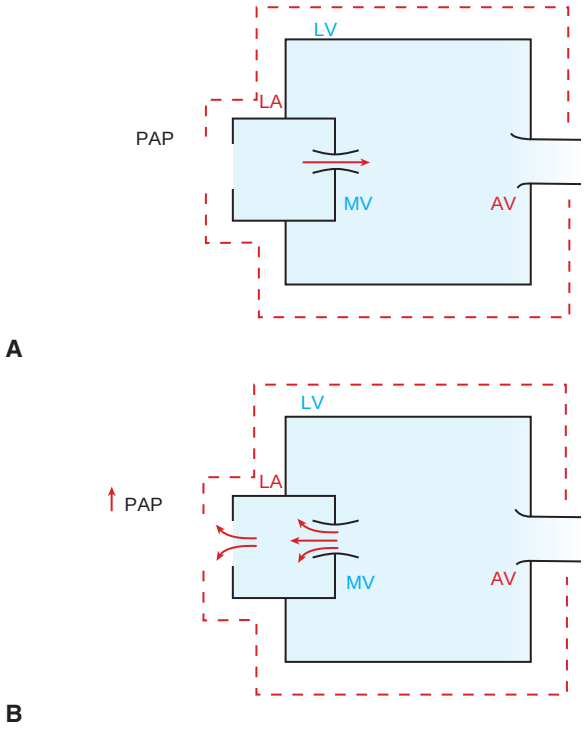


FIGURE 22-3 ▲ Mitral valve dysfunction. **A:** Mitral stenosis. **B:** Mitral insufficiency. AV, aortic valve; LA, left atrium; LV, left ventricle; MV, mitral valve; PAP, pulmonary artery pressure.

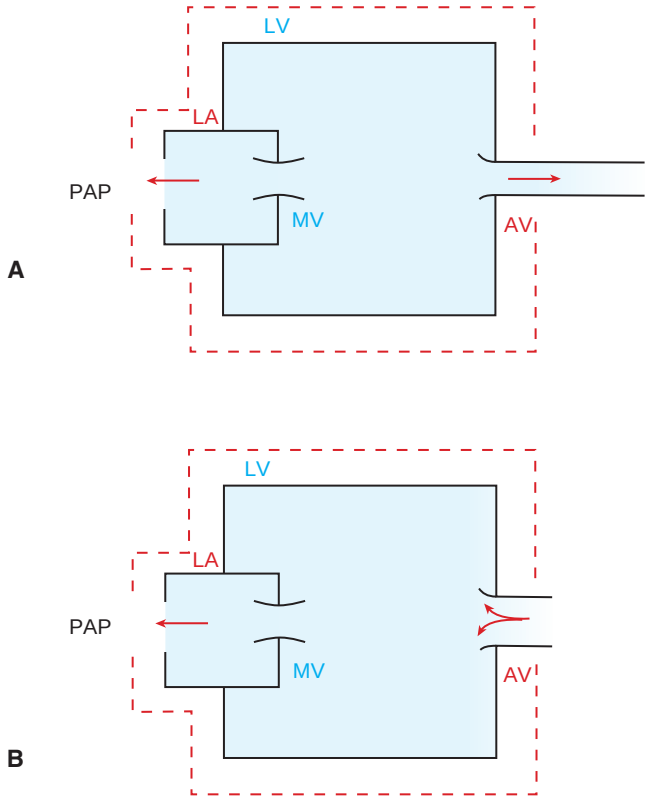


FIGURE 22-4 ▲ Aortic valve dysfunction. **A:** Aortic stenosis. **B:** Aortic insufficiency. AV, aortic valve; LA, left atrium; LV, left ventricle; MV, mitral valve; PAP, pulmonary artery pressure.

thickening or stretching of the leaflets, resulting in backward blood flow. During ventricular systole, some of the left ventricular blood regurgitates into the atrium rather than being ejected through the aortic valve. This regurgitation decreases the forward cardiac output. Left ventricular hypertrophy occurs in an attempt to improve the cardiac output, but the hypertrophy can actually worsen the regurgitation. Left ventricular volume overload causes left ventricular dilation. Regurgitant flow into the left atrium causes increased left atrial pressure and dilation. This volume overload can be reflected backward to the pulmonary circulation; however, pulmonary and right-sided heart symptoms usually do not develop until late in the disease process. As a result of this pathophysiology, patients with chronic mitral insufficiency commonly present with fatigue, palpitations, and sometimes shortness of breath.

Acute mitral insufficiency may result from endocarditis, chest trauma, or MI. Endocarditis erodes or perforates the valve leaflets or chordae. Trauma may rupture the chordae. MI may cause papillary muscle rupture, allowing blood to flow backward into the left atrium during ventricular systole. Because of the acute nature of the valve dysfunction, there is inadequate time for dilation or hypertrophy to compensate. In acute mitral insufficiency, cardiac output decreases dramatically, cascading into pulmonary edema and shock. The treatment of choice for acute mitral regurgitation with hemodynamic compromise is emergent mitral valve replacement.

AORTIC STENOSIS. Aortic stenosis may develop as a result of rheumatic fever, calcification of a congenital bicuspid valve, or calcific degeneration, especially in elderly patients. The resultant fusion of the commissures and fibrous contractures of the cusps leads to obstruction of left ventricular outflow. Forward cardiac output is diminished, and the left ventricle hypertrophies to maintain the cardiac output. As the stenosis worsens, compensation fails, and volume and pressure overload in the left ventricle causes left ventricular dilation. Increased left ventricular pressures are reflected backward through the left atrium and pulmonary vasculature (Fig. 22-4A).

Diminished cardiac output in the person with aortic stenosis may lead to two major problems—angina and syncope. Extreme left ventricular hypertrophy increases myocardial oxygen demand at the same time that cardiac output and coronary artery perfusion are decreased. Ischemic myocardium develops, which may lead to angina. Syncope occurs in the late stages of aortic stenosis, when the forward cardiac output cannot increase to meet the body's demands. As a person with severe aortic stenosis exercises, the blood vessels to the skeletal muscles dilate to increase the blood supply. The normal response to this increased demand is increased cardiac output. However, the person with aortic stenosis is unable to respond in such a way. The vasodilation without a concomitant increase in cardiac output results in insufficient cerebral perfusion and syncope. Patients with aortic stenosis also experience exertional dyspnea, orthopnea, and paroxysmal nocturnal dyspnea.

AORTIC INSUFFICIENCY. Aortic insufficiency, like mitral insufficiency, can occur acutely or develop over a period of time. Chronic aortic insufficiency is commonly caused by rheumatic fever and aneurysm of the ascending aorta. Rheumatic disease results in thickened and retracted valve cusps, whereas aortic aneurysm causes annular dilation. Both conditions prevent the edges of the valve leaflets

from approximating, allowing blood to regurgitate backward from the aorta into the left ventricle during ventricular diastole. Forward cardiac output decreases, and left ventricular volume and pressure increase. Left ventricular hypertrophy ensues. Eventually, the increase in left ventricular pressure is reflected backward into the left atrium and pulmonary circulation (see Fig. 22-4B). Patients with chronic aortic insufficiency present with fatigue, and they have a low diastolic blood pressure and a widened pulse pressure. The pulse may rise rapidly and collapse suddenly (water-hammer or Corrigan's pulse) because of the forceful ventricular contraction and subsequent diastolic regurgitation from the aortic root into the left ventricle. Angina may occur because aortic insufficiency creates an imbalance between left ventricular myocardial oxygen supply and demand. As left ventricular hypertrophy worsens, the oxygen demand increases, but regurgitant flow from the aortic root during diastole decreases coronary artery perfusion.

Acute aortic insufficiency may be caused by blunt chest trauma, ruptured ascending aortic aneurysm, or infective endocarditis. Left-sided heart failure and pulmonary edema develop rapidly in the patient with acute aortic insufficiency because compensatory left ventricular hypertrophy does not have time to develop. In response to the diminished cardiac output, systemic vascular resistance (SVR) increases to maintain the blood pressure. The elevated SVR increases the degree of regurgitation and worsens the situation.

Surgical Treatment

The goals of valvular surgery are to relieve symptoms and restore normal hemodynamics. Surgery is indicated before left ventricular function deteriorates significantly and the patient's activity becomes severely limited or before severe signs and symptoms, such as angina or syncope from aortic stenosis or pulmonary hypertension from mitral stenosis, develop. Percutaneous balloon valvuloplasty, a procedure that is indicated primarily for patients considered too high risk for surgery, is discussed in Chapter 18. Surgical intervention consists of either valve reconstruction or valve replacement. Because valve reconstruction is associated with decreased operative mortality and fewer thromboembolic and anticoagulation-related complications than valve replacement, valve reconstruction is gaining popularity.

VALVE RECONSTRUCTION. With the development of transesophageal echocardiography to assess the effectiveness of repair during surgery, the use of valve reconstruction is increasing. Most valve reconstruction procedures are performed on the mitral valve. Compared with mitral valve replacement, reconstruction eliminates the need for long-term anticoagulation, decreases the risks for thromboembolism and endocarditis, decreases the need for reoperation, and increases survival rates. However, for aortic valve disorders, most attempts at reconstruction have not been successful because of late insufficiency and restenosis.

A common reconstruction technique for mitral stenosis is commissurotomy. Although not indicated for patients with severe mitral stenosis, commissurotomy may be effective for patients with moderate stenosis with minimal calcification and regurgitation. During commissurotomy, the fused commissures are surgically divided. Calcified tissue is débrided and fused, and shortened chordae are incised. This procedure improves leaflet mobility and increases the mitral valve area, decreasing the degree of stenosis.

Another technique for treatment of mitral insufficiency is reconstruction. If annular dilation causes the regurgitation, annuloplasty can be performed using sutures or a prosthetic ring (eg, Carpentier-Edwards annuloplasty ring). The ring is sewn around the mitral annulus so that excess annular tissue is drawn up. Suturing and the ring reduce the circumference of the enlarged annulus so that the edges of the leaflets coapt, diminishing regurgitation. If the chordae tendineae are stretched or ruptured, surgical shortening or transposition of chordae to substitute for ruptured chordae can be effective. Redundant mitral leaflets are repaired by resecting a portion of the leaflet, and perforated valve leaflets can be reconstructed by patching. Such repairs are usually supported by an annuloplasty ring.

Reconstruction procedures are more likely to be successful if performed early in the course of the disease, before left ventricular function deteriorates and irreparable damage occurs. Anticoagulation is not usually needed after valve repair unless an annuloplasty ring is used. In such cases, anticoagulants are given for only 3 months until the ring is endothelialized. If reconstruction cannot be accomplished, valves are replaced.

VALVE REPLACEMENT. The first valve replacement was performed by Harken and Starr in 1960 with a caged ball prosthesis. Since then, many new valve designs have evolved. Valve replacement surgery is done through a median sternotomy incision, and cardiopulmonary bypass and myocardial preservation techniques (discussed in detail later in this chapter) are used. The mitral valve is approached through the left atrium. Rather than excising the native valve, the chordae and papillary muscles are preserved when the prosthetic valve is sutured in place. This technique helps maintain left ventricular function and ejection fraction. The aortic valve is approached through the ascending aorta. The native aortic valve is excised, the annulus is sized, and the prosthetic valve of correct size is sutured to the annulus. Once the surgery is completed, the patient is transferred to the intensive care unit (ICU).

Ideally, a prosthetic valve would be durable, last for a patient's life, and perform exactly like a normal human valve. The valve would have normal hemodynamics with unimpeded, nonturbulent blood flow through a central opening, no transvalvular gradient, and no regurgitation when closed. It would be nonthrombogenic and not damaging to blood components and acceptable to the patient in terms of noise and the need for anticoagulation. Unfortunately, no artificial valve currently meets these criteria, so research continues.

Choosing a Valve. Two major types of prosthetic valves are available—biological and mechanical. Mechanical valves are made entirely of synthetic materials, whereas biological valves combine synthetic materials with chemically treated biological tissues. When choosing an appropriate valve for a particular patient, it is necessary to compare the advantages and disadvantages of the various valve types. The advantages and disadvantages of prosthetic cardiac valves are listed in Box 22-1. Mechanical valves offer the benefits of good long-term durability but pose a significant risk for thromboembolism and require long-term anticoagulation. Biological valves decrease the risk for thromboembolism and can obviate the

BOX 22-1 Advantages and Disadvantages of Prosthetic Cardiac Valves

Mechanical Valves
- Good long-term durability
- Adequate hemodynamics
- High risk for thromboembolism; necessity for long-term anticoagulation
- Increased risk for bleeding complications

Biological Valves
- Poor long-term durability
- Better hemodynamics than mechanical valves (except in small sizes)
- No hemolysis
- Low incidence of thromboembolism; possibly no necessity for anticoagulation
- Fewer bleeding complications

need for long-term anticoagulation, but they are not as durable as mechanical valves. Biological valves studied at autopsy have shown structural deterioration beginning as early as 6 years after implantation, and their total useful lifetime is usually considered to be less than 10 years.

Patients with a long life expectancy may receive mechanical valves because they are particularly durable.

Biological Valves. Older patients may receive biological valves because less calcification and deterioration occur in older people, long-term durability is less important, and the risk for anticoagulation may increase with advancing age. Biological valves are indicated for patients who are unable to comply with an anticoagulation regimen, for those in whom a long-term anticoagulation regimen is contraindicated, and for women of childbearing age who plan to become pregnant (the anticoagulant warfarin crosses the placental barrier).

Mechanical Valves. Mechanical valves include the caged ball, tilting disk, and bileaflet designs (Fig. 22-5). The caged ball valve consists of a plastic or metal ball inside of a metal cage attached to a sewing ring. When pressure behind the valve increases, the ball is forced down into the cage, and blood flows around it. When pressure in front of the valve increases, the ball is forced upward against the sewing ring, preventing regurgitant flow. An example of the caged ball valve is the Starr-Edwards valve (see Fig. 22-5C, D).

Hemodynamically, the ball in the cage produces a central obstruction to blood flow, which can result in a small stenotic pressure gradient, and ventricular outflow may be partially obstructed because of the cage's size and high profile. Because of the thrombogenicity of the plastic and metal and the turbulent flow around the ball and through the cage, blood clots can form on or around the valve. Thromboembolism is a common problem, and chronic anticoagulant therapy is essential. Caged ball valves have good long-term durability.

The tilting disk valve is constructed of a disk held in place by struts attached to a sewing ring. When the pressure behind the valve increases, the disk tilts open approximately 60 to 80 degrees, allowing blood to flow around it. When the pressure in front of the valve increases, the disk tilts back flush with the sewing ring to close. Because of its semicentralized flow and lower profile, the tilting disk valve

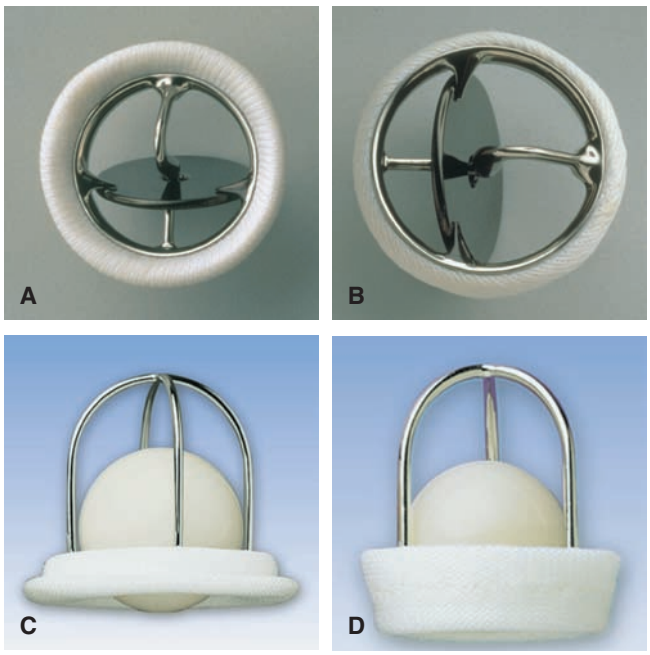


FIGURE 22-5 ▲ **A:** Medtronic Hall Easy-Fit, aortic model. **B:** Medtronic Hall Easy-Fit, mitral model. **C:** Starr-Edwards Silastic ball valve, aortic model. **D:** Starr-Edwards Silastic ball valve, mitral model. (A and B courtesy of Medtronic Heart Valves, Minneapolis, MN; C and D courtesy of Edwards Life Sciences, Irvine, CA.)

produces less obstruction to blood flow and has better hemodynamic characteristics than the caged ball valve. The tilting disk valve has good long-term durability, but the risk for thromboembolism requires long-term anticoagulant therapy. Examples of the tilting disk valve are the Medtronic-Hall and the omniscience valves.

The bileaflet tilting disk valve, which consists of two pyrolytic carbon semicircular disks or leaflets hinged to a sewing ring, is the newest type of mechanical valve (see Fig. 22-5A, B). When the pressure behind the valve increases, the leaflets open perpendicular to the sewing ring, and blood flows through the central opening with minimal obstruction. When pressure in front of the valve increases, the leaflets return to their flat position against the sewing ring, preventing insufficiency. The bileaflet tilting disk valve has good hemodynamic characteristics and durability, but it is thrombogenic and requires long-term anticoagulation. An example of the bileaflet tilting disk valve is the St. Jude Medical valve.

Biological Valves. Biological prostheses, or tissue valves, offer another alternative for valve replacement. The porcine heterograft is constructed of an excised pig aortic valve preserved in glutaraldehyde and mounted on a frame attached to a sewing ring. Examples of porcine valves are the Hancock and the Carpentier-Edwards valves. Biological prostheses provide good hemodynamics, except in smaller sizes, where obstruction to flow can occur and a gradient can develop. Their main advantage is a lower risk for thromboembolism compared with mechanical valves. Because most thromboembolic events occur during the first 3 months after implantation before the sewing ring is endothelialized, most patients with biological valves receive anticoagulants during that time only. However, the decision regarding anticoagulation must be based on the patient's condition. Patients in chronic

atrial fibrillation undergoing mitral valve replacement frequently receive long-term anticoagulation therapy even with a biological prosthesis because of stagnant blood flow in the atria, a condition that may lead to clot formation.

▲ Cardiac Surgery

With managed care, rising costs, and the demand for high-quality care, cardiac surgery has come under increased scrutiny. The unique challenge for the critical care nurse is to integrate theoretical knowledge, assessment skills, and problem-solving ability to provide optimal nursing care and maintain high-quality outcomes while decreasing resource consumption, yet always keeping the patient as the focus.

Preoperative Phase

Preoperative preparation for cardiac surgery includes physiological and psychological components. The physiological preparation is similar to that for any preoperative patient and includes history, physical examination, chest radiography, and an ECG. The history and physical examination are extremely important; they can provide information about previous neurological status, current medications, and any other coexisting conditions (eg, diabetes mellitus, pulmonary disease, renal disease). The chest radiograph can give the surgeon general information about aortic calcification, and the ECG provides baseline information about the patient's heart rhythm. Laboratory tests include complete blood count (CBC), electrolytes, prothrombin time (PT), partial thromboplastin time (PTT), blood urea nitrogen (BUN), and creatinine. Pulmonary function tests and arterial blood gas (ABG) analyses may be performed if a patient has underlying pulmonary problems. Cost is always a consideration; only necessary tests should be ordered.

Effective preoperative teaching, which reduces anxiety and physiological responses to stress before and after surgery, is an important aspect of psychological preparation. The surgical procedure and the intraoperative and postoperative experiences are explained. The patient usually is not in the ICU before surgery, and a tour of the ICU helps familiarize the patient and family with the specialized equipment and environment. The sight of a patient who is successfully recovering from cardiac surgery helps instill confidence and allay anxiety. Incorporating family members or significant others in the education process is pivotal in patient care. Specific teaching topics related to the patient's stay in the ICU are listed in Box 22-2.

Intraoperative Phase

Surgical Approach

The surgical approach most commonly used for myocardial revascularization and valve surgery is median sternotomy. The sternum is split with a sternal saw from the manubrium to below the xiphoid process, and the ribs are spread to expose the anterior mediastinum and pericardium. After the pericardium is opened and the heart and aorta are exposed, the patient is connected to the cardiopulmonary bypass machine.

BOX 22-2 TEACHING GUIDE *Preoperative Teaching About the ICU Experience for the Patient Undergoing Cardiac Surgery*

Equipment to Point Out
- Cardiac monitor
- Arterial line
- Thermodilution catheter
- IV lines and IV infusion pumps
- Endotracheal tube and ventilator
  - Suctioning
  - Explain how to communicate when intubated; unable to talk
  - Explain when extubation can be anticipated
- Foley catheter (increased sensation to urinate)
- Chest tubes (anticipated removal)
- Pacing wires
- Nasogastric tube
- Soft hand restraints

Incisions and Dressings to Expect
- Median sternotomy or other incision
- Leg incision (if saphenous vein is used)

Patient's Immediate Postoperative Appearance
- Skin yellow from use of Betadine solution in operating room
- Skin pale and cool to touch because of hypothermia during surgery
- Generalized "puffiness," especially noticeable in neck, face, and hands because of third spacing of fluid given during cardiopulmonary bypass

Awakening From Anesthesia
- Patient recovers in the intensive care unit (ICU); does not go to the postanesthesia care unit (PACU)
- Each patient recovers from anesthesia differently
- Patient may feel certain sensations
- Patient may hear certain noises
- Patient may be aware or able to hear but unable to respond

Discomfort
- Amount of discomfort to be expected
- When pain might be expected
- Relief mechanisms
  - Positioning/splinting
  - Medications
  - Patient-controlled analgesia (PCA) and the importance of early administration of pain medication

Postoperative Respiratory Care
- Turning
- Use of pillow to splint median sternotomy incision
- Effective coughing and deep breathing after extubation; have patient practice exercises before surgery
- Incentive spirometry
- Early mobilization

Miscellaneous
- Postoperative activity progression
- Hospital visiting policy in intensive care area
- Avoiding use of arms to protect stability of sternotomy

As myocardial revascularization has become increasingly sophisticated, new interventions to minimize the invasive nature of the surgery have been developed. The number of patients undergoing reoperations using CABG surgery has increased, and if the mediastinal approach is used in a reoperation, injury to old bypass grafts or embolization of debris resulting from manipulation of diseased grafts may cause problems.[12] A smaller lateral thoracotomy incision is often used to decrease the risks associated with reentry into the mediastinum. The choice of incision is based on the specific needs of each patient and the experience of the surgeon.

CARDIOPULMONARY BYPASS. Cardiac surgery as it is known today was made possible by the development and practical application of cardiopulmonary bypass procedure by Gibbon in 1953.[1] Because the heart must be still (not beating) and empty during the surgery, a cardiopulmonary bypass machine is used, unless OPCABG surgery is to be performed. Before the bypass is implemented, the tubing of the machine is primed with a balanced electrolyte solution. Blood can be used if indicated. The patient's deoxygenated venous blood is brought to the pump either through one cannula placed in the right atrial appendage or by two cannulas, one of which is placed directly in the inferior vena cava and the other directly in the superior vena cava. Another cannula is placed in the ascending aorta to return oxygenated blood to the patient's systemic circulation (Fig. 22-6). Heparin is administered throughout cardiopulmonary bypass to prevent massive extravascular coagulation as the blood circulates through the mechanical parts of the bypass system. During bypass, the patient's core body temperature is lowered to 28° to 32°C (82.4°F to 89.6°F) to decrease metabolism. This reduction in metabolic demands helps protect the major organ systems from possible ischemic injury and the adverse effects of nonpulsatile perfusion during cardiopulmonary bypass.

Oxygenated blood is filtered and returned to the patient's ascending aorta through the arterial cannula (see Fig. 22-6). Once extracorporeal circulation is established and systemic hypothermia is achieved, the aorta is cross-clamped just above the coronary arteries, and either crystalloid or blood cardioplegia solution is infused into the aortic root. After the aorta is cross-clamped, no blood circulates through the coronary arteries, so the myocardium becomes ischemic. Cold cardioplegia solution at 4°C (39.2°F) is infused into the aortic root under pressure. As it circulates through the coronary arteries, the high potassium concentration causes immediate asystole and relaxation, and the cold produces myocardial hypothermia. Asystole and hypothermia protect against myocardial ischemia by decreasing the metabolic needs of myocardial tissue. The cardioplegia solution provides a substrate for ongoing cellular metabolism and ensures appropriate pH and calcium ion levels for myocardial preservation.[13] The inclusion of blood or oxygenated crystalloid in the cardioplegia solution lessens myocardial ischemia by supplying oxygen. Cardioplegia solution may be infused into the aortic root continuously or intermittently every 15 to 30 minutes and whenever cardiac electrical activity recurs. This process varies, depending on the surgeon's preference.

Because perfusion of cardioplegia solution through occluded or diseased coronary arteries may not produce an even myocardial cooling, inadequately cooled areas risk ischemic damage. Therefore, hypothermia is also created topically by pouring iced normal saline slush solution over the heart into the pericardial well. Cardioplegia with concomitant topical hypothermia cools the heart evenly. Several disadvantages to cold cardioplegia have been identified, including postoperative myocardial depression, ventricular dysrhythmias, decreased cerebral blood flow, irreversible platelet dysfunction, and shifts of the oxygen–hemoglobin dissociation curve to the left so that blood delivers oxygen to the tissues less readily. A heart receiving cold crystalloid cardioplegia must have blood reintroduced into the coronary circulation (reperfusion). This reintroduction of oxygen may cause release of toxic substances that injure myocardial cells (reperfusion injury). To avoid these disadvantages, some cardiac surgeons use normothermic blood cardioplegia delivered at 37°C (98.6°F), which keeps the heart at a normal temperature. Patients who have undergone warm cardioplegia require less time on the ventilator and almost no rewarming technology in the ICU.

After surgery is completed, the heat exchanger rewarms the blood to return the patient's core temperature to 37°C (98.6°F) if hypothermic techniques were used. After air is vented from the heart chambers and the aortic root, the aortic cross-clamp is removed so that blood again perfuses the coronary arteries, warming the myocardium. As perfusion

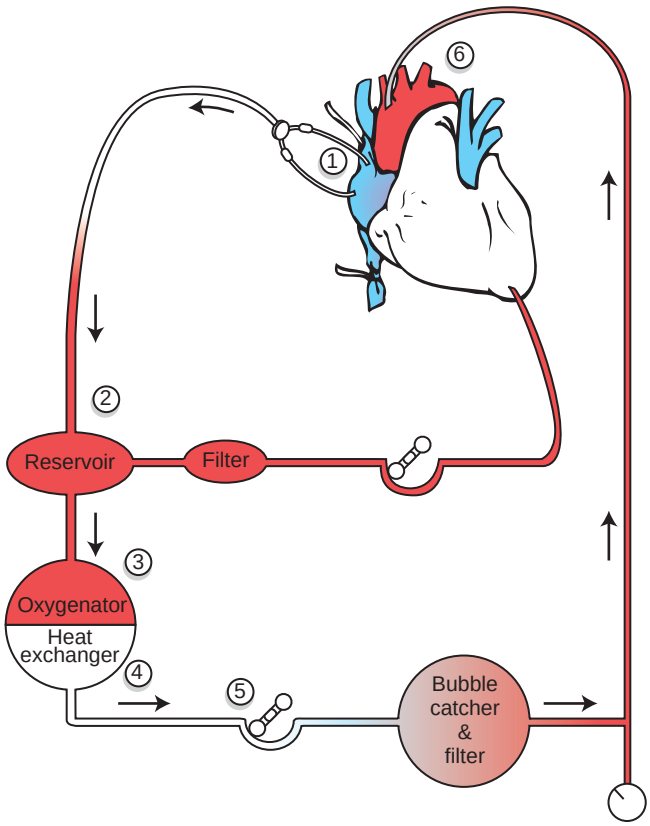


FIGURE 22-6 ▲ Blood flow through the circuit of the cardiopulmonary bypass machine: (**1**) Patient's deoxygenated blood enters the bypass circuit from the venous cannulas in the superior and inferior vena cavae. (**2**) The reservoir holds the blood temporarily. (**3**) The oxygenator removes carbon dioxide from and adds oxygen to the patient's blood. (**4**) The heat exchanger initially cools the blood and then rewarms the blood. (**5**) Roller pumps pump the blood through the circuit and back to the patient. (**6**) Oxygenated blood is returned to the ascending aorta by way of the aortic cannula.

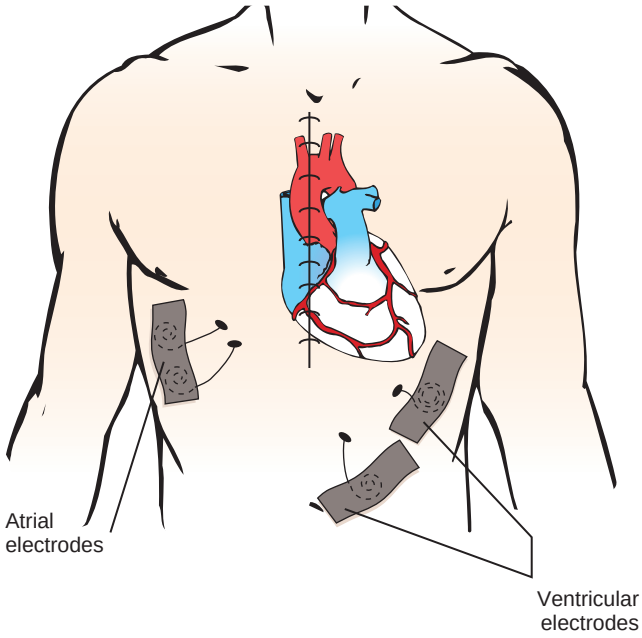


FIGURE 22-7 ▲ Temporary epicardial pacing wires: position of atrial and ventricular wires on chest wall.

and rewarming continue, a spontaneous cardiac rhythm may resume, ventricular fibrillation may develop (necessitating internal defibrillation), or pacing may be used to initiate a rhythm. After a reliable rhythm with a rate adequate to maintain the cardiac output and blood pressure is established, the patient is weaned from total cardiopulmonary bypass, and the cannulas are removed from the right atrium and aorta. Heparinization is reversed by the administration of protamine sulfate. If adequate cardiac output cannot be maintained during the weaning process, positive inotropic agents or intra-aortic balloon counterpulsation can be instituted (see Chapter 18 for more information about care of the patient on the intra-aortic balloon pump [IABP]).

COMPLETION OF SURGERY. If the need for postoperative cardiac pacing is anticipated, temporary pacing electrodes are placed on the epicardial surface of the heart and brought out through the chest wall on either side of the median sternotomy incision. Ventricular pacing electrodes are typically located to the left and atrial wires to the right of the sternum (Fig. 22-7). For more information about care of the patient with a pacemaker, see Chapter 18.

Chest tubes placed in the mediastinum and pericardial space for drainage are brought out through stab wounds just below the median sternotomy. If the pleural space has been entered, pleural tubes are also placed. Smaller, more flexible chest drains are being used instead of the stiff, larger-bore tubes to enhance drainage of the pleural and mediastinal space.[14] After adequate hemostasis is obtained, the edges of the sternum are approximated with stainless steel wires, the incision is closed, and dressings are applied.

Postoperative Phase

Patients are transported directly to the ICU, where they recover from anesthesia and usually remain for 24 hours after surgery. Patients arrive in the ICU with numerous lines and tubes (eg, endotracheal tube, hemodynamic monitoring lines). Immediate postoperative care involves cardiac monitoring and maintenance of oxygenation and hemodynamic stability, as described in Box 22-3. Because cardiopulmonary bypass produces abnormal blood interface and altered blood flow patterns, it has profound physiological effects (Table 22-2).

The postoperative course depends on the patient's preoperative condition. Factors that may increase mortality include age, sex, previous similar surgery (resurgery), preoperative occurrence of acute MI, and concomitant conditions such as diabetes mellitus, peripheral vascular disease, renal insufficiency, and chronic obstructive pulmonary disease (COPD).[1] Whether the surgery is elective or emergent may also influence outcome. Awareness of these conditions helps the critical care nurse anticipate problems. Accurate assessments, vigilant monitoring, and proper interventions are critical in stabilizing patients who have just undergone cardiac surgery. Box 22-4 lists some nursing diagnoses in cardiac surgery. Box 22-5 presents a collaborative care guide for the patient after such surgery. Certain patient populations present special problems. Box 22-6 on page 457 lists factors to consider in managing older cardiac patients.

BOX 22-3 Nursing Responsibilities in Caring for the Cardiac Surgery Patient in the Immediate Postoperative Period

Priority Interventions Performed by the Critical Care Team on Arrival

- Attach patient to bedside cardiac monitor and note rhythm.
- Attach pressure lines to bedside monitor (arterial and pulmonary artery); level and zero transducers and note pressure values and waveforms.
- Obtain cardiac output/index and note existing inotropic or vasoactive drips.
- After ventilator is connected to endotracheal tube, auscultate breath sounds bilaterally.
- Apply end-tidal carbon dioxide ($ETCO_2$) device to ventilator circuit and note waveform and value (best indicator of endotracheal tube placement).
- Apply pulse oximetry device to patient and note SpO_2 value and waveform.
- Check peripheral pulses and perfusion signs.
- Monitor chest tubes and character of drainage: amount, color, flow. Check for air leaks.
- Measure body temperature and initiate rewarming if temperature is less than 96.8°F (36°C).

Once the Patient Is Determined to Be Hemodynamically Stable

- Measure urine output and note characteristics.
- Obtain clinical data (within 30 minutes of arrival).
- Obtain chest radiograph.
- Obtain 12-lead ECG.
- Obtain routine blood work within 15 minutes of arrival; tests may include ABGs, potassium, glucose, PTT, and hemoglobin (varies with institution).
- Assess neurological status.
- Test pacemaker function by assessing capture and sensing.

Table 22-2 Effects of Cardiopulmonary Bypass

Effects	Clinical Implications
Increased Capillary Permeability	
Interface between blood and nonphysiological surfaces or bypass circuit leads to • Complement activation that increases capillary permeability • Platelet activation—platelets secrete vasoactive substances that increase capillary permeability • Release of other vasoactive substances that increase capillary permeability	Large amounts of fluid move from the intravascular to the interstitial space during and up to 6 h after cardiopulmonary bypass. Patient becomes edematous.
Hemodilution	
Solution used to prime extracorporeal circuit dilutes patient's blood. Secretion of vasopressin (antidiuretic hormone) is increased. Levels of renin–angiotensin–aldosterone are increased because of nonpulsatile renal perfusion. Total body water is increased.	Decreased blood viscosity improves capillary perfusion during nonpulsatile flow and hypothermia. Hgb and Hct decrease. Levels of coagulation factors are decreased because of dilution. Intravascular colloid osmotic pressure is decreased, contributing to movement of fluid from intravascular to interstitial spaces. Water is retained at collecting tubule of kidney. Aldosterone causes retention of sodium and water at renal tubule. Weight gain occurs.
Altered Coagulation	
Procoagulant effects: • Interface between blood and nonendothelial surfaces of bypass circuit activates intrinsic coagulation cascade. • Platelet damage activates intrinsic pathway. • Anticoagulant effects: • Interface between blood and nonendothelial surfaces of bypass circuit causes platelets to adhere to tubing and to clump; abnormal platelet function; activation of coagulation cascade, which depletes clotting factors; denaturation of plasma proteins, including coagulation factors. • Coagulation factors are decreased as a result of hemodilution.	Risk for microemboli is increased. Platelet count decreases by 50%–70% of baseline. Abnormal postoperative bleeding occurs. Possibility of bleeding diathesis exists.
Damage to Blood Cells	
Exposure of blood to nonendothelial surfaces causes mechanical trauma and shear stress. • Platelet damage occurs. • Red blood cell hemolysis occurs. • Leukocytes are damaged.	 Platelet count is decreased. Free hemoglobin and hemoglobinuria are increased. Hct is decreased. Immune response is diminished.
Microembolization	
Emboli form from tissue debris, air bubbles, platelet aggregation.	Microemboli to body organs (brain, lungs, kidney) are possible.
Increased Systemic Vascular Resistance (SVR)	
Catecholamine secretion is increased when cardiopulmonary bypass is initiated. Renin secretion is due to nonpulsatile flow to kidney. Hypothermia develops.	Hypertension is possible. Increased SVR may decrease cardiac output.

Prevention of Hypothermia

Whether the cardiac procedure is performed on or off the cardiopulmonary bypass system, hypothermia is a common side effect. During rewarming on cardiopulmonary bypass, the patient's core temperature is returned to 98.6°F (37°C). However, as this warmed blood begins to circulate to the periphery, heat transfer to the surrounding tissues again causes the core temperature to decline. Patients frequently enter the ICU with a temperature in the 95° to 96.8°F (35° to 36°C) range. OPCABG surgery causes hypothermia because of heat loss secondary to prolonged exposure to cool operating room temperatures. Hypothermia causes peripheral vasoconstriction

BOX 22-4 EXAMPLES OF NURSING DIAGNOSES

For Cardiac Surgery Patients

- Decreased Cardiac Output related to changes in ventricular preload, afterload, and contractility
- Decreased Cardiac Output related to cardiac dysrhythmias
- Ineffective Peripheral Tissue Perfusion related to cardiopulmonary bypass, decreased cardiac output, hypotension
- Ineffective Peripheral Tissue Perfusion related to microembolization secondary to the SIRS process
- Impaired Gas Exchange related to cardiopulmonary bypass, anesthesia, poor chest expansion, atelectasis, retained secretions
- Impaired Comfort related to endotracheal tube, surgical incision, chest tubes, rib spreading
- Anxiety related to fear of death, ICU environment
- Risk for Deficient Fluid Volume related to abnormal bleeding
- Risk for Infection related to surgical procedure, invasive lines, drainage tubes, hypoventilation, retained secretions

and a shift of the oxygen–hemoglobin dissociation curve to the left, which means that less oxygen is released from the hemoglobin to the tissues. Hypothermia can also impair coagulation because all enzyme systems in the body depend on a tight temperature range for optimal performance.[15]

The nurse assesses the patient's temperature on ICU admission using pulmonary artery or tympanic membrane temperature; if performed properly, both are considered accurate indicators of core temperature. Rectal temperature does not correlate with core temperature measurements until 8 hours after surgery, and bladder temperature differs significantly from core temperature with rapid cooling and rewarming. Increasing the room temperature and using radiant heat, blankets, or a warming blanket are effective techniques for increasing core temperature. Rewarming should occur slowly to prevent hemodynamic instability caused by rapid vasodilation.

It is important to prevent shivering, which occurs most often from 90 to 180 minutes after ICU admission, because shivering increases metabolic rate, oxygen consumption, carbon dioxide production, and myocardial workload. If left ventricular function is compromised, shivering should be managed with a neuromuscular blocker in combination with simultaneous sedation to avoid further cardiac compromise. In unusual situations, the patient may experience discomfort with shivering, which can be treated with meperidine (Demerol).

After rewarming, many patients experience an overshoot in body temperature. One etiological theory is that narcotics

BOX 22-5 COLLABORATIVE CARE GUIDE for the Patient After Cardiac Surgery

Outcomes	Interventions
Oxygenation/Ventilation	
Patient will have ABG values within normal limits and pulse oximeter value >92%.	• Obtain ABG levels per protocol. • Correlate pulse oximeter and end-tidal CO_2 with ABG results.
Pulmonary edema will be minimized on chest x-ray and demonstrated by improved breath sounds.	• Adjust ventilator settings after consulting with the respiratory therapist and physician. • Wean from mechanical ventilation per protocol using the expertise of respiratory therapy. • Extubate when patient is hemodynamically stable; able to protect airway. • Provide supplemental oxygen after extubation.
Atelectasis will improve.	• Encourage use of incentive spirometer, cough, and deep breath every 2–4 h after extubation.
Chest tubes will remain patent.	• Milk chest tubes if necessary to facilitate forward drainage movement.
Circulation/Perfusion	
Patient will maintain adequate clinical perfusion. Vital signs will be within normal limits, including MAP more than 70 mm Hg; cardiac index will be in a suitable range for the patient's left ventricular function.	• Monitor pulmonary artery pressure (PAP) and PAOP, CVP, cardiac output, SVR, and pulmonary vascular resistance (PVR) per protocol if pulmonary artery catheter is in place. • Monitor ECG, ST segments, and arterial blood pressure continuously. • Administer positive inotropic agents and reduce afterload with vasodilating agents guided by hemodynamic parameters and physician orders. • Regulate volume administration as indicated by PAOP or CVP values. • Evaluate effect of medications on BP, HR, and hemodynamic parameters. • Monitor and treat dysrhythmias per protocol and physician orders. • Anticipate need for temporary cardiac pacing; wires will be properly isolated for electrical safety. • Prepare patient for IABP assist if necessary. • Congestive heart failure from decreased cardiac output or perioperative MI will be minimized by collaborating with a physician. • Assess for neck vein distention, pulmonary crackles, S_3 or S_4, peripheral edema, increased preload parameters, elevated "a" wave of CVP, or PAOP waveform.
Patient will be euthermic.	• Monitor 12-lead ECG if ECG changes are observed. • Assess temperature every hour. • Warm patient 1°C/h by using warming blankets, lights, and fluid warmer.

(continued on page 456)

BOX 22-5 *COLLABORATIVE CARE GUIDE for the Patient After Cardiac Surgery (continued)*

Outcomes	Interventions
Hematological Issues	
Patient will have minimal bleeding and avoid cardiac tamponade.	• Chest tube drainage will be <200 mL/h. • Monitor for signs of cardiac tamponade (hypotension, pulsus paradoxus, tachycardia, PA pressure equalization). • Evaluate chest x-ray for widened mediastinum, consulting with a physician as needed. • Monitor PT, PTT, CBC per protocol. • Administer protamine, blood products, and other procoagulants per order or protocol. • Monitor vasoactive drug need and report marked increase of drugs to physician immediately because this change may indicate possible tamponade.
Fluids/Electrolytes	
Patient will maintain or improve preoperative renal function.	• Renal function will be maintained as evidenced by urine output of approximately 0.5 mL/kg/h. • Potassium will be replaced to maintain K^+ at more than 4.0 mEq/L. • Monitor intake and output every 1 to 2 h. • Monitor BUN, creatinine, electrolytes, magnesium, PO_4. • Record daily weights. • Administer fluid volume or diuretics as ordered.
Mobility/Skin Integrity	
Patient will maintain range of motion and muscle strength and will have intact skin integrity	• Turn patient side to side every 2 h while on bed rest and evaluate skin closely. • Mobilize out of bed after extubation. • Progress activity to chair for meals, bathroom privileges, increased distance walking, delegating to assistive personnel as indicated. • Monitor vital signs, respiratory effort during activity.
Incisions will heal without evidence of infection.	• Check stability of sternotomy incision daily, especially with diabetic patients. • Assess sternotomy and leg incision for redness, swelling, drainage. • Apply compression hose and elevate legs to reduce edema. • Caloric and nutrient intake meets metabolic requirements per calculation for long-term patients. • Monitor prealbumin for trends on long-term patients.
Comfort and Pain Control	
Patient will have relief of surgical pain. Patient will demonstrate no evidence of pain or anxiety such as increased heart rate, blood pressure, respiratory rate, or agitation during activity or procedures. Timely administration of pain medication will be a priority.	• Assess quality, duration, location of pain. Use visual analog scale to assess pain quantity. • Provide a calm environment. Provide for adequate periods of rest and sleep.
Teaching/Discharge Planning	
Patient and family will understand need for: Tests, procedures, treatments. Self-protective devices as indicated and per hospital policy. In preparation for discharge to home, patient will understand activity levels, dietary restrictions, medication regimen, incision care.	• Consult nutritional support services. • Make appropriate social work referrals early during hospitalization. • Initiate family education regarding heart-healthy diet, physical activity limitations (eg, lifting over 10–15 pounds and driving restrictions), stress reduction strategies, management of pain, incision care.

and anesthetics administered during surgery reset the hypothalamic regulatory center, altering peripheral blood flow and feedback.[16] A cold, constricted peripheral vascular bed may also be a factor in preventing heat dissipation. If the patient is bleeding after surgery, correction of temperature is imperative to aid in the return of normal coagulation enzyme function and clotting ability.

Monitoring for Systemic Inflammatory Response Syndrome

Any process, including surgery, initiates the SIRS. In recent years, research in critical care medicine has focused on SIRS because it may be the cause of many patient problems. An entire "body" inflammatory response may occur after CABG

BOX 22-6 CONSIDERATIONS FOR THE OLDER PATIENT: AFTER CARDIAC SURGERY

Physiological Changes

Cardiovascular System
- Increased stiffness of myocardial muscle
- Increased stiffness of peripheral vasculature and decreased ability to adjust to changes in blood volume
- Replacement of cells in conduction system with collagen and elastin
- Decreasing number of pacemaker cells in the sinoatrial (SA) and atrioventricular (AV) nodes
- Decreased cardiac responsiveness to β-adrenergic stimulation

Pulmonary System
- Breakdown of elastin and collagen that impairs elastic recoil of lung
- Thoracic cage less compliant
- Decreased expiratory muscle strength and mucociliary clearance

Renal System
- Progressive loss of cortical nephrons and decrease in corticomedullary concentration gradient
- Impaired renal concentrating ability
- Decreased clearance of medications excreted by the kidneys (may be reduced by up to 40% by 80 years of age)

Gastrointestinal System
- Decreased and more variable gastrointestinal absorption of medications
- Decline in liver function, resulting in decreased hepatic breakdown of medications

Musculoskeletal System
- Skeletal osteoporosis

Immune System
- Immune response may be decreased, especially if concomitant malnutrition and decrease in serum proteins

Neurological System
- Decline in neurotransmitters
- Increased risk for acute confusion

Response to Medications
- Decreased percentage of lean body tissue
- Increased percentage of body fat
- Decrease in body water

Clinical Effect
- Higher filling pressures (pulmonary artery diastolic [PAD] pressure and pulmonary artery occlusion pressure [PAOP])
- Decreased ability for vasoconstriction with position change, leading to orthostatic hypotension
- SA and AV node impairment
- Cardiac output maintained by increase in stroke volume
- Slowing of renal response to dehydration
- Decreased effectiveness of fluid conservation
- Toxic medication levels or abnormally prolonged duration of action
- Sensitive to drugs with narrow therapeutic range, such as digoxin
- More intense medication effect and longer duration of action for medications broken down in liver (eg, benzodiazepines)
- As a result of decreased body water, water-soluble medications concentrated in the bloodstream, resulting in higher serum drug levels
- Fat-soluble medications stored in fat; increase in fat tissue may result in slower therapeutic response and longer duration of action as drug is released slowly from fat

Patient Teaching
- Accommodate sensory deficits.
  - Ensure hearing aids are in and functional.
  - Speak loudly and face patient.
  - Use large print, easy-to-read materials.
- Teach one thing at a time and ensure that patient understands before moving on.
- Start with simple and progress to more complex information.
- Teach both patient and care giver.

Adapted from Dixon V: Effects of vascular surgery on the elderly vascular patient. J Vasc Nurs 17:86–88, 1999.

surgery that appears to be an infection. Symptoms and signs include fever, tachycardia, tachypnea, and an increased white blood cell count. To attempt to differentiate between SIRS and infection or sepsis, the American College of Chest Physicians convened a consensus conference in 1997.[17] The conference developed definitions differentiating the two conditions (Box 22-7), which are now used frequently by critical care experts.

SIRS is a natural defense mechanism that is initiated when tissue or vessels are injured. A vascular injury, the inflammatory response, and the coagulation cascade are interrelated. An event that disrupts the integrity of the endothelium, such as trauma from cutting the vessel or hypoxia in a few endothelial cells, triggers the process. Once the injury occurs, a local inflammatory reaction begins with the release of mediators called cytokines from "protector" cells (eg, lymphocytes, macrophages). These mediators signal other cells (eg, neutrophils, monocytes) to the injured area, which release other mediators. The endothelium then releases vasodilating mediators (eg, nitric oxide), which increase blood flow to the area, thereby increasing oxygen delivery. Counter-regulatory mediators cause vasoconstriction to balance vasodilatory actions. Platelets are attracted to the area to start coagulation. As a result of the endothelial damage, increased capillary permeability inevitably occurs.[18] This highly complex process is discussed in more detail in Chapter 54.

It was once believed that the cardiopulmonary bypass machine was the major trigger of SIRS; this prompted experts to reconsider the use of this machine. However, Vallely et al[7]

BOX 22-7 Definition of Systemic Inflammatory Response Syndrome (SIRS)

SIRS is defined by the presence of two or more of the following conditions:

Temperature	More than 100.4°F (38°C) or <96.8°F (36°C)
Heart rate	More than 90 beats/min
Respiratory rate	More than 20 breaths/min or $PaCO_2$ <32 mm Hg
White blood cell count	More than 12,000 cells/m^3, <4,000 cells/m^3, or more than 10% immature (bands) cells

Sepsis is a systemic response to documented infection and is determined by the same criteria as SIRS.

From Muckart D, Bhagwanjee S: American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference: Definitions of the systemic inflammatory response syndrome and allied disorders in relation to critically injured patients. Crit Care Med 25:1789–1795, 1997.

demonstrated that OPCABG surgery also initiates a SIRS response involving the release of different mediators from the on-pump procedure. Few interventions limit SIRS; the inflammatory process is so complex that it has been difficult to develop medications to counter all the numerous reactions. Steroids have been shown to decrease SIRS somewhat if given before surgery, but they should be used with caution, especially in patients with diabetes mellitus.[1] Nursing responsibilities focus on refining assessment skills to increase early detection of embolic events in any system, especially the nervous, cardiovascular, pulmonary, and renal systems. It is important to have a high degree of suspicion when assessing patients and providing postoperative care.

Controlling Pain

After cardiac surgery, the patient may experience pain resulting from the chest or leg incision, the chest tubes, rib spreading during surgery, and care activities. The ICU environment may accentuate the pain physiologically because of light and noise as well as psychologically because of separation and fear. Pain often stimulates the sympathetic nervous system, increasing heart rate and blood pressure, which can be detrimental to the patient's hemodynamic status. Discomfort can also result in diminished chest expansion, increased atelectasis, and retention of secretions.

Although pain perception varies from person to person, a median sternotomy incision is usually less painful than a thoracotomy incision, and most people report that the pain is most severe the first 3 to 4 days after surgery. Discomfort from the leg incision often worsens after the patient is ambulatory, especially if leg swelling occurs. Stretching of back and neck muscles as the ribs are spread and immobilization for several hours during surgery can cause back and neck discomfort. Patients who have internal mammary artery grafts may have increased pain because of increased stretching of the intercostal muscles and the incision into the parietal pleura, which is richly innervated.

Angina after CABG surgery may indicate graft failure; therefore, the nurse must be able to differentiate angina from incisional pain. Typical median sternotomy pain is localized, does not radiate, and can be sharp, dull, aching, or burning. It is often worse with deep breathing, coughing, or movement. Angina is usually precordial or substernal; not well localized; and frequently radiates to arms, neck, or jaw. It is often described as a pressure sensation and is not affected by respiration or movement.

One of the goals of nursing management is a thorough assessment of the patient's pain using a pain scale; administration of analgesics based on the reported pain intensity; provision of adequate pain relief as reported by the patient; and alleviation of factors that enhance pain perception, such as anxiety and fear. The common analgesic drugs used are morphine sulfate, fentanyl, and hydromorphone (Dilaudid), as needed. These drugs can be supplemented with nonsteroidal anti-inflammatory drugs (NSAIDs), such as ketorolac (Toradol), which decrease pain through a different mechanism. Caution should be used in administering NSAIDs to patients with compromised renal function. Patient-controlled analgesia (PCA) pumps are frequently used to allow the patient to control administration of pain medication. Interventions, such as intercostal nerve blocks and spinal analgesia, are less common. Regardless of the mechanism used, pain control is aggressively pursued to ensure comfort and rapid mobilization, which in turn can lessen complications. Alternative therapies, such as music therapy and guided imagery, may also be useful in controlling pain.

Preventing Cardiovascular Complications

Many cardiovascular complications can be anticipated and prevented, which can lead to decreased length of stay and better patient outcomes. Astute nursing observations and appropriate interventions can contribute to better outcomes.

VOLUME RESUSCITATION. Adequate intravascular volume to provide preload is a primary concern. Increased capillary permeability resulting from SIRS causes intravascular volume to shift into the interstitial spaces. To maintain optimal cardiac performance and blood pressure, proper volume resuscitation is imperative. A variety of fluids may be used, including normal saline, hetastarch, and hyperosmolar fluids (eg, 3% saline).[19] No fluid is definitively recommended. If the patient is bleeding, blood products should be the fluid of choice. If the patient's blood pressure is unresponsive to moderate infusion rates, usually 500 mL is infused using a pressure bag and a large-bore catheter. Hemodynamic parameters, including a low central venous pressure (CVP; <8 to 10 mm Hg), low pulmonary artery diastolic pressure, and low pulmonary artery occlusion pressure (PAOP; <14 to 18 mm Hg), in combination with a low cardiac index (<2.5 L/min/m^2), help guide interventions.[15] Caution should be exercised in using these numerical values as absolute goals.

The preoperative condition of the heart is important to consider. If the patient has had a recent MI or poor left ventricular performance, higher pressures may be required to maintain optimal cardiac work. The patient with a hypertrophied left ventricle, especially with valvular disease, is heavily dependent on volume resuscitation.

The effectiveness of all interventions must be assessed against the patient's response. Mottling of the extremities, especially the knees, and the character of the peripheral pulses (especially dorsalis pedis) are clinical indications of perfusion.[20] The combination of weak pulses and mottled knees may indicate hypoperfusion. Resolution of these clinical findings, as well as improved pressure values, signals the return of adequate perfusion. The astute critical care nurse continually monitors the appearance of the extremities and the pulses.

MONITORING FOR DYSRHYTHMIAS. Dysrhythmias are a major issue after CABG surgery. The hemodynamic response to a change in cardiac rhythm dictates the speed of the intervention in patients who have undergone CABG surgery, as in all critical care patients. In emergent situations, advanced cardiac life support (ACLS) algorithms are used. Knowledge of the patient's baseline rhythm is important. The types of dysrhythmias that may occur range from premature atrial contractions to ventricular fibrillation and asystole.

Sinus tachycardia is common and may result from many factors. Some of the more common causes are sympathomimetic drugs, SIRS, hypovolemia, fever, and pain. Prolonged periods of tachycardia may be harmful because of decreased coronary artery filling time. Sinus bradycardia may occur, but it is not anticipated because patients are in a sympathetically responsive state. In many cases, preoperative beta blockade may be the cause.

Causes of premature atrial contractions are usually electrolyte disturbances, ischemia or infarction, or hypoperfusion. Frequent premature atrial contractions may be a precursor to atrial fibrillation and occur very commonly, especially in patients with a previous history of pulmonary or valve disease in which the atria can be distended. The simple treatment for premature atrial contractions is repletion of potassium and magnesium. Maintenance of adequate potassium (range, 4 to 4.5 mEq/L) and IV infusion of 2 g magnesium may minimize premature atrial contractions.

Atrial fibrillation may occur after CABG surgery, and prevention is a high priority. Cardiac decompensation or cerebrovascular accidents are the major risks associated with atrial fibrillation. For new-onset atrial fibrillation, the goal is conversion to sinus rhythm using antiarrhythmics, especially amiodarone (Cordarone). The loading dose is 150 mg IV over 10 minutes followed by a 1 mg/min drip for 6 hours, then a 0.5 mg/min drip for 18 hours. Additional loading may be necessary. Control of the ventricular response is a goal and can be achieved using diltiazem (Cardizem), starting with a loading dose up to 0.75 mg/kg IV followed by a drip, which is usually started at 5 mg/h. Intravenous beta blockers including metoprolol are also used and administered in small boluses of 2.5 to 5 mg. Combination of antiarrhythmics may be used but should be monitored carefully. If atrial fibrillation persists or reoccurs for more than 24 hours, warfarin (Coumadin) anticoagulation for 4 weeks may be needed.[1] For chronic atrial fibrillation, conversion is not a goal (unless the patient has received anticoagulants) because of the risk for atrial thrombus and possible embolization. If emergent cardioversion is required in the immediate postoperative period and anticoagulation is not an option, echocardiography, either Doppler or transesophageal, may be performed to check for thrombus formation in the left atrium.

Heart block dysrhythmias occur in patients with valve surgery secondary to the edema at the surgical site, near the conduction system. Resolution of this rhythm is usually attained 48 to 72 hours after surgery once the edema decreases. Myocardial ischemia and infarction also cause the heart block. Patients who have had cardiac surgery have an advantage with the placement of epicardial pacing wires. Use of these wires allows better control of ventricular response compared with the use of drugs, such as atropine and isoproterenol (Isuprel). Atrial pacing is preferred if the atrioventricular (AV) node is intact because it allows optimal hemodynamics with an atrial contraction. If the AV node is not functioning properly, AV sequential pacing may be required. Ventricular pacing is the last choice. If pacing is required for more than 72 hours, permanent pacemaker placement should be considered, especially in patients who have had valve surgery. An in-depth discussion of pacing can be found in Chapter 18.

The occurrence of tachyarrhythmias may lead to emergent situations. If the patient is hemodynamically unstable in a fast rhythm, the first intervention is cardioversion, following ACLS guidelines. If ventricular dysrhythmias develop, electrical or pharmacological interventions are necessary. If premature ventricular contractions appear after surgery, 2 to 4 g IV of magnesium may help resolve this problem. The new ACLS guidelines de-emphasize the use of lidocaine and recommend amiodarone as the drug of choice, especially in patients with poor left ventricular function. If ventricular tachycardia deteriorates into ventricular fibrillation or other rhythms with no pulse, cardiopulmonary resuscitation should be started immediately; medical personnel should be prepared to open the chest at the bedside to determine and correct the cause of arrest.

IMPROVING CARDIAC CONTRACTILITY. Contractility may be depressed because of the exposure of the heart muscle to manipulation, temperature change, and possible hypoperfusion. The first step taken to improve performance is to ensure optimal volume resuscitation; it will quickly become clear if volume does not increase the cardiac output and index. The addition and titration of sympathomimetic drugs is a common part of the care of patients with decreased contractility. Various drugs, including epinephrine, dobutamine, and milrinone, may be used. Dopamine is another drug that can increase contractility but may cause unwanted tachycardia. The choice of drug varies with institution and health care provider.

As the drug of choice is added, the cause of the ventricular dysfunction should be pursued. Myocardial ischemia and infarction typically cause decreased cardiac function, but other factors may be sources of the problem. Stunned myocardium, the transient depression of left ventricular function from a temporary reduction of myocardial blood flow, may cause transient dysfunction; it is usually associated with normally functioning myocardium.[21] Hibernating myocardium is chronically impaired yet viable myocardial tissue, which results in left ventricular dysfunction at rest because of persistently hypoperfused myocardium or repeated stunning.[21] This state can lead to more chronic dysfunction. The state of the right ventricle should also be considered. If a patient had a right ventricular infarct preoperatively, surgery should be delayed up to 4 weeks to allow recovery of function.[1] If right ventricular dysfunction develops after CABG surgery, use of nitric oxide is one of the more effective interventions. Evaluation of cardiac work is not limited to cardiac output and cardiac index. Cardiac biomarkers can be used in the first 24 hours after CABG surgery. Troponin and creatine kinase muscle band are parameters that can be monitored to determine if the patient may have sustained myocardial damage.[1] In more complicated cases, sampling of mixed venous blood gases/mixed venous saturation (SvO_2) and the arterial–venous difference in oxygen may be useful. These values are indicators of oxygen transport and consumption and can help direct therapy. Although continuous SvO_2 monitoring may be used in patients with severe myocardial dysfunction, it is not a standard intervention; it is costly and has not been shown to make a difference in outcomes.

The IABP is a mechanical method used to improve coronary perfusion in any of the previously mentioned situations.[1] For a complete discussion of IABP, see Chapter 18. Mechanical factors may lead to depressed cardiac function. Cardiac tamponade, the most common such factor, may require surgical correction. This topic is discussed later in the chapter. Whatever the cause, time and support of function are usually the major factors that improve cardiac performance. However, protracted periods of time with mechanical or pharmacological support may be the signal to start to consider the placement of a ventricular assist device, usually as a bridge to heart transplantation.

CONTROLLING BLOOD PRESSURE. A reduction in SVR is another clinical maneuver that can increase cardiac performance. If the patient has an adequate blood pressure (mean arterial pressure [MAP] exceeding 70 mm Hg or

systolic blood pressure over 120 mm Hg) without pharmacological support (ie, alpha-agonist agents to increase blood pressure), afterload reduction should be started even if the patient is on inotropic support. Various agents, including nitroprusside, nitroglycerin, hydralazine, labetalol, and angiotensin-converting enzyme (ACE) inhibitors, such as captopril (preferred agent), can be used. The necessary speed of response dictates the choice of drug. For example, IV drugs, especially nitroprusside, rapidly cause a reduction in afterload. Other agents, such as hydralazine and ACE inhibitors, can then be used to augment this effect. ACE inhibitors should be used with caution in patients with impaired renal function because this drug category can exacerbate renal dysfunction. The previously named drugs also reduce blood pressure; in the immediate postoperative period, this is very important in maintaining the integrity of the grafts.

Preventing Pulmonary Complications

Postoperative pulmonary function depends on preoperative function. The degree of preoperative evaluation in preparation for surgery has changed in the current era of cost containment. If the patient has a significant pulmonary history (eg, COPD, pulmonary hypertension), baseline pulmonary function tests and ABG values can be very helpful in setting goals in the postoperative period. These tests may help predict how the patient will respond to mechanical ventilation.

The causes of pulmonary dysfunction after cardiac surgery can be attributed to changes that occur with the inflammatory response. Various triggers, such as surgical trauma and regional myocardial ischemia, activate the complement system and release cytokines, leading to an egress of neutrophils and fluid across endothelium. These triggers can also cause end-organ dysfunction, including organs such as the lungs.[7] Such changes in the lungs can lead to alterations in microcirculation and gas exchange that ultimately result in ventilation–perfusion mismatching, shunting, and atelectasis.[15]

Mechanical ventilation is required to achieve adequate oxygenation and ventilation. Adequate oxygenation is achieved by adjusting the level of oxygen delivered by the ventilator; the usual starting point is 40% to 50% oxygen. Effective oxygenation is monitored using pulse oximetry with intermittent ABG sampling. Positive end-expiratory pressure (PEEP) is a standard intervention used to help keep the alveoli open and improve oxygenation. PEEP usually starts at 5 cm H_2O but can be increased to 10 cm H_2O or more if hypoxemia is present. Care must be taken when increasing PEEP because it can decrease preload, thereby decreasing cardiac output and blood pressure. The initial mode for the ventilator is usually assist-control ventilation and is changed to continuous positive airway pressure (CPAP) when the patient is awake, stable, and ready to be weaned for extubation.

Adequate ventilation is maintained by selecting tidal volumes that are appropriate for body size as well as setting a sufficient rate for the ventilator tidal volumes. Monitoring of ventilation should include end-tidal carbon dioxide ($ETCO_2$) monitoring, which should be correlated with the partial pressure of carbon dioxide ($PaCO_2$) on an ABG analysis. $ETCO_2$ monitoring is also used to confirm proper endotracheal tube placement.

The recent implementation of newer techniques in cardiac anesthesia, which allows for faster recovery times, has led to shorter times on mechanical ventilation. Weaning from mechanical ventilation is a quick process in patients who have undergone CABG surgery. Once the patient has displayed the ability to follow commands and the strength to protect the airway, a short CPAP trial is instituted. The patient can be extubated if (1) cardiac performance is good (cardiac index more than 2.2 L/min/m^2), (2) adequate oxygenation and ventilation are achieved without acidosis, and (3) chest tube bleeding is minimal. Aggressive use of incentive spirometry and physical mobility ensures proper pulmonary function. Continual assessment by the critical care nurse is very important. Auscultation of breath sounds should be performed at frequent intervals and as the patient's condition dictates. Diminished breath sounds, especially in the left lower lobe, are common because left lower lobe atelectasis is an expected postoperative outcome. Observation of the work of breathing is also important, and signs, such as tachypnea, use of accessory muscles, and prolonged expiratory time can indicate compromised pulmonary function. Bronchodilator therapy may be indicated and should be continued if the patient was using bronchodilators at home.

Prolonged mechanical ventilation may be a complication of cardiac surgery. Protracted poor cardiac function requires continued mechanical ventilation. Phrenic nerve damage due to cold preservation techniques for myocardial protection or physical transection is another cause of ventilatory failure due to diaphragm dysfunction. Acute respiratory distress syndrome associated with SIRS, a hypoperfusion state, or both can also be a reason for prolonged ventilator days. A tracheostomy should be considered in patients with compromised pulmonary function because it can enhance the ventilator weaning process and promote patient comfort. Expert nursing and multidisciplinary care of the patient in ventilatory failure using weaning protocols may make a difference in patient outcome.[22,23]

Preventing Neurological Complications

Neurological recovery of the cardiac surgery patient depends on several factors, such as preoperative neurological state; age (more than 70 years); presence of conditions such as aortic atherosclerosis, hypertension, and diabetes mellitus; and use of the IABP.[1] The usual course of neurological recovery is much faster since anesthetic agents have changed. Narcotics and benzodiazepines, with neuromuscular blockade, are used now, and gases are used less.[15] There is little need for sedation when the patient is transported from the operating room unless hemodynamic instability is present. The patient is allowed to wake up and recover from the anesthesia as soon as possible. There may be some barriers to this process, including age and renal failure. The elderly patient is not able to metabolize narcotics and paralytics as quickly as a younger patient and may require a longer recovery time. If the patient is difficult to arouse and has pinpoint pupils, reversal of narcotics with naloxone (Narcan) may be indicated. Naloxone diluted 0.4 mg in 10 mL normal saline solution and given 1 to 2 mL IV every 5 minutes is a delicate method of regaining level of consciousness that does not reverse pain control. If the patient does not have good muscle strength, reversal of neuromuscular blockade is indicated. Glycopyrrolate, 0.6 mg IV, and neostigmine, 3 mg IV (or more), are used. The patient in renal failure is not able to clear these drugs and probably needs reversal of both narcotic and neuromuscular blockade agents to expedite extubation.

Once the patient is awake, continual evaluation using the standard neurological examination that assesses the level of consciousness and motor and sensory ability is mandatory. Postoperative neurological deficits are divided into two categories: (1) major focal deficits (stroke), stupor, or coma and (2) deterioration in intellectual function.[1] The best predictor of stroke is proximal aortic atherosclerosis, which is the source of emboli that are released with the manipulation of the aorta, especially during cannulation or cross-clamping. Hypoxia, hypoperfusion, hemorrhage, or metabolic abnormalities may also cause strokes.[1] Cognitive changes are more difficult to detect because there may be deficits in memory, language, and psychomotor function. The family of the patient may be helpful in detecting any subtle changes. These changes are most noticeable immediately after surgery but may still be present 12 to 36 months after the procedure.[24] Confirmation of a stroke can be performed with computed tomography or magnetic resonance imaging of the head, but these studies may need to be repeated; embolic events do not immediately appear on scans. Prevention of such a catastrophic event is difficult, but the risk can be reduced by patient selection and procedure selection.[24] If a patient has known carotid disease, maintaining higher blood pressure may help increase perfusion of the cerebral tissues. Thrombolytic therapy, which is used successfully in other patients with emboli, cannot be used after surgery in the patient who has just had CABG surgery because of bleeding concerns.

Monitoring Postoperative Bleeding

Postoperative bleeding is expected; the challenge is to know when and how to intervene. Anticoagulation interventions that have improved outcomes for the cardiac patient in general confound bleeding problems in the patient who has had CABG surgery. Timely correction of bleeding problems can decrease both the occurrence of complications and the cost of patient care. Preoperative anticoagulation such as therapy with thrombolytics and antiplatelet drugs (eg, aspirin and clopidogrel [Plavix]) hamper coagulation, and their effects are difficult to reverse; reversal may not even be an option, and postoperative bleeding may increase. It is recommended that if the patient is receiving clopidogrel, it should be stopped 5 days before surgery.[1]

Drainage and decompression of the pericardial and pleural spaces are required after cardiac surgery. Traditionally, chest tubes were large, rigid tubes, which were very uncomfortable for patients. Recently, smaller, more flexible chest tubes with bulb suction have been introduced to decrease discomfort, increase early ambulation, and decrease the accumulation of pleural effusions. Large postoperative pleural effusions may require increased hospital days or rehospitalization. These tubes are longer and more flexible to enhance pleural drainage; they have been found to decrease clinically significant pleural effusions at 6-week follow-up.[14]

Vigilant monitoring of chest tube drainage is imperative to anticipate impending problems. Chest tube drainage is monitored hourly. The usual chest tube output can range from 100 to 200 mL/h, with periods of increased drainage due to a change in position or temperature. Measurement of drainage may be required at more frequent intervals (every 15 or 30 minutes) if drainage is high.

If the chest tube output continues to be greater than 200 mL/h, then intervention is necessary. Protamine, the first level of intervention, is given at 1 mg for every 100 units of heparin to reverse the effects of heparin, which is used in the surgical process (both on and off bypass).[25] PTT is commonly used to monitor the intrinsic pathway of the coagulation cascade, which heparin affects. Additional protamine may be necessary, especially if the patient is hypothermic, because a "rebound" phenomenon may occur. Aggressive rewarming is very important in a patient who has increased bleeding because the coagulation cascade, with its enzymatic reactions, cannot function properly at hypothermic temperatures. However, as the patient's temperature rises, heparin is reactivated, causing increased bleeding. Platelet infusion (usually 6 units per infusion) is used next to help decrease bleeding. It is important to remember that a platelet infusion can cause a blood product reaction because each infusion may be from multiple donors. Before surgery, patients require therapy to make platelets dysfunctional, thus preventing thrombus formation in the coronary arteries. Causes of platelet dysfunction and postoperative bleeding include medications, such as aspirin; the bypass machine itself; the IABP, which mechanically destroys platelets; and heparin-induced thrombocytopenia, a recent phenomenon in which heparin exposure disables platelet function.

Follow-up coagulation studies act as a guide to the need for further infusions as well as monitoring blood loss, but they are not absolute parameters. If bleeding is increasing, a PT can be ordered to monitor the extrinsic pathway of the coagulation cascade and determine whether other factors need to be replaced. An elevated PT (more than 15 seconds) may indicate that bleeding is due to a lack of factors, such as fibrinogen, that can be replaced using fresh frozen plasma, usually 4 to 6 units/infusion. The overall goal is to determine whether bleeding is due to a coagulopathy or surgical bleeding. Chest tube bleeding that exceeds 500 mL/h is considered surgical bleeding and mandates surgical reexploration.

Other therapeutic interventions may also be used to decrease bleeding. Coagulation factors, such as cryoprecipitate (factors I and VIII) and factor VII, are indicated in severe bleeding. Various drugs, such as aminocaproic acid (Amicar), a potent inhibitor of fibrinolysis; aprotinin (Trasylol), a serine-protease inhibitor that blocks kallikrein at the beginning of the coagulation cascade; and desmopressin acetate (DDAVP), which influences factor VIII and enhances platelet adhesion, can be administered to promote coagulation.[25] Autotransfusion of mediastinal chest tube drainage using special drainage systems has been used to decrease blood transfusion requirements. However, the possibility that reinfusion can stimulate fibrinolysis and exacerbate bleeding is a concern; therefore, this treatment method is not recommended for routine use (especially in low-risk patients).[1]

Intraoperative measures to prevent bleeding include minimizing hemodilution, minimizing autologous losses, and optimizing coagulation status with full rewarming and antifibrinolytics.[26] Blood loss requires replacement, which should be considered carefully. Transfusion of red blood cells not only can increase exposure to infectious diseases, especially hepatitis and human immunodeficiency virus, but also is associated with increased immunosuppressive and microcirculatory complications.[27] The hemoglobin level indicated for transfusion is a controversial issue. Recent research indicates that a restrictive transfusion strategy (hemoglobin < 7 g/dL) has demonstrated a lower mortality rate in patients who are less critically ill. Autotransfusion of chest drainage also has

been used, but there are no clear data that support improved outcomes with this intervention.

Cardiac tamponade is a serious complication of increased postoperative bleeding that occurs when excessive fluid or blood accumulates in the pericardial space, resulting in increasing pressure on the right atrium and ventricle that can lead to collapse of those structures. Tamponade may develop rapidly or slowly, depending on how fast blood accumulates in the pericardial sac. When a patient is treated for excessive bleeding, it is important to monitor the chest tube drainage closely and maintain patency. Decreasing cardiac output and blood pressure and significant increases in pharmacological support (especially norepinephrine [Levophed]) are important warning signs. The mechanism of cardiac tamponade is the collapse of the lower pressure chambers of the right heart as a result of the increasing and equalizing of the CVP, pulmonary artery diastolic pressure, and the PAOP. This increase and equalization of the three values is classic evidence of cardiac tamponade. However, the clinical situation can be a late finding, and decreasing cardiac performance and blood pressure, despite volume resuscitation, is an earlier indicator. An arterial line waveform with significant respiratory variation (best illustration of an increased pulsus paradoxus) is another warning sign that cardiac tamponade is pending.[28] Definitive diagnosis is made with an echocardiogram (two-dimensional or transesophageal).

Interventions to prevent tamponade include stripping and milking chest tubes when the blood begins to clot, although stripping the tubes can generate increased negative pressure. Because the chambers (atria and ventricles) are being compressed, cardiac pressures, especially CVP, may be elevated. Another useful intervention involves the infusion of volume even with increased pressure to keep the structures from collapsing. Performing a pericardial window is the best surgical intervention.

Preventing Renal Complications

The postoperative course of renal function is influenced by preoperative function. Preoperative risk factors are age, history of moderate to severe congestive heart failure, prior CABG surgery, and preexisting conditions including type 1 diabetes mellitus and renal disease (serum creatinine 1.4 to 2.0 mg/dL).[1] The usual postoperative course also depends on whether the surgery was performed on or off bypass. After on-bypass CABG surgery, brisk initial urine output is expected because of the priming of the bypass circuit with mannitol and the possible use of diuretics. The output diminishes as these effects decrease with time. After OPCABG surgery, there is a smaller urine volume because patients are not exposed to these interventions. Some patients are able to autodiurese excess volume, but as the inflammatory response diminishes within 24 to 48 hours, the leaky capillary membranes seal and extra interstitial fluid shifts into the intravascular space, increasing the need for pharmacological diuresis with drugs, such as furosemide (Lasix). Electrolyte repletion with potassium and magnesium after diuresis is also important to maintaining a regular cardiac rhythm. A slight metabolic acidosis may be present in the patient with existing renal failure and may persist after surgery. If acidosis is present, the source (respiratory, metabolic, or combined) should be determined to intervene appropriately. The focus of interventions is to remove excess fluid while protecting metabolic and cardiac function.

OLIGURIA. Decreasing urine output (<0.5 mL/kg/h) is usually caused by decreased renal perfusion. Obvious causes, such as Foley catheter obstruction or malposition, may often be overlooked and should be considered initially, so that mechanical problems may be ruled out. Decreased cardiac function may also cause a decrease in urine output. Hypovolemia is a very common problem that can be addressed with fluid boluses, and monitoring pulmonary artery pressures and the cardiac output/cardiac index shortly after fluid infusions indicates whether the intervention was therapeutic. Caution must be exercised when adding volume because excess fluid can cause decreased function in compromised myocardial muscle. In that situation, inotropic agents or vasoactive drugs may be required. Determining the patient's baseline blood pressure is important so that control of vasoactive drugs can be titrated according to a perfusion pressure (MAP or systolic blood pressure) that the patient's kidneys require.

If none of these interventions is successful, diuresis may be necessary. Loop diuretics (eg, furosemide) are the usual first-line drugs. If urine output does not increase, larger doses may be indicated, or other diuretics that act on other areas of the renal tubular system, such as thiazides, may be added. Creatinine and BUN values are closely monitored.

RENAL FAILURE. If acute renal failure develops, dialysis is necessary. The method used depends on patient condition and practitioner preference. Continuous venovenous hemofiltration (CVVH) and hemodialysis are among the several methods that may be used. CVVH is preferred in the patient who is severely hemodynamically compromised because it is more gradual and minimizes preload compromise, which could decrease cardiac performance. Patients who undergo dialysis require fluid restriction, nutrition modification for prolonged renal dysfunction, and other standard interventions, such as dietary modifications to decrease protein and potassium intake. Chapter 31 provides a further discussion of renal failure and Chapter 30 discusses dialysis. Unfortunately, the mortality rate from acute renal failure in postoperative cardiac surgery patients is greater than 60%.[1]

Preventing Endocrine Complications

Diabetes mellitus is one of the major risk factors for the development of cardiovascular disease. This disease affects almost all systems in the body and requires that the vigilant clinician continually monitor blood glucose and maintain strict glucose control. In the initial postoperative period, a blood glucose level less than 200 mg/dL is particularly important in managing wound healing. Intervention using an insulin drip initially may reduce the incidence of deep sternal wound infections by 50%.[1] Once good glucose control has been achieved, insulin is given subcutaneously, and glucose levels are followed closely. Such insulin therapy also decreases the incidence of diabetic ketoacidosis or hyperosmolar coma. It is important to remember that whereas hyperglycemia is detrimental, severe hypoglycemia can be fatal. The need for vigilant blood glucose monitoring cannot be overemphasized.

Adrenal insufficiency may occur, especially in patients who were receiving steroids at regular intervals before surgery. The administration of steroids can suppress adrenal function. To prevent suppression, postoperative stress doses of hydrocortisone (100 mg every 8 hours) should be given, and the patient's regular dose should be restarted. If the

patient is taking vasoactive drugs and is not weaning off the drips, adrenal insufficiency is considered; this condition may be the result of hypoperfusion of the adrenal gland. Cortisol levels are low and confirm adrenal insufficiency.

Thyroid dysfunction, especially hypothyroidism, is common in elderly persons and women. Although perioperative effects do not result in dysfunction, preoperative function is an important consideration, and undiagnosed dysfunction may become apparent in the postoperative period because the thyroid hormones, especially triiodothyronine (T_3), can have cardiovascular effects.

Preventing Gastrointestinal Complications

Fortunately, the gastrointestinal aspects of the postoperative course are uneventful and similar to those of general surgery. After extubation, the patient takes nothing by mouth (NPO) for up to the first 8 hours, with a nasogastric tube in place to decompress the stomach. Then the patient is allowed to have small amounts of ice or water. This relatively simple aspect of nursing care is very important for the postoperative patient, who may experience significant thirst because of the anticholinergic drugs that were given before surgery. The use of ice pops and ginger ale can help with compliance and decrease the possibility of nausea, vomiting, and aspiration.

Complications such as cholecystitis, pancreatitis, and bowel infarction rarely occur. Their pathogenesis is not always clear but is attributed to splanchnic hypoperfusion and general gastrointestinal ischemia. Thorough assessment of the abdomen looking for pain, distention, or tympany may help discover subtle abnormalities. Lactate levels greater than 2.5 mmol/L may indicate splanchnic hypoperfusion. However, they may also be the result of nonpulsatile flow of the bypass machine, which may cause a release of angiotensin II, exacerbating splanchnic ischemia.[15] The need for further evaluation is dictated by the clinical presentation.

Monitoring for Infection

In the early postoperative period, hypothalamic resetting is the cause of temperature derangement. Febrile reactions are usually attributed to SIRS and to overshoot from rewarming. If the fever (temperature more than 100.4°F [38°C]) persists for more than 48 to 72 hours, infection should be considered. Infection prevention is the major goal of all programs. This goal is achieved through the prudent use of antibiotics, vancomycin, and a cephalosporin (eg, cefazolin, ceftazidime). Timing of antibiotic administration is pivotal. For optimal results, preoperative doses should be completely infused before the skin is cut. Antibiotic dosing depends on preoperative renal function. A short postoperative course should also be anticipated.

Mediastinitis is the major infection in patients who have undergone CABG surgery and may be a devastating complication that increases the length of hospital stay and mortality. Risk factors associated with mediastinitis are obesity; prior cardiac surgery; preexisting type 1 diabetes mellitus; and perioperative factors, such as excessive electrocautery and use of both internal mammary arteries resulting in compromised blood flow to the chest wall. Therapy is an extended antibiotic course and plastic surgery. The aggressive regulation of the blood glucose level using insulin drips initially instead of subcutaneous administration has been shown to decrease the occurrence of mediastinitis.[1] This intervention is also used in cases that do not involve CABG; a decrease in mortality has been demonstrated.[29] It is very important to instruct the patient not to use the arms excessively when moving and to use a "cough pillow" with coughing (a small pillow that is placed on the sternal incision and squeezed when coughing). Other interventions, such as having the patient sleep on his or her back, are also important. Following these instructions may help maintain the stability of the sternum.

Patient Teaching and Discharge Planning

With managed care, capitation, rising costs, and limited resources, the usual length of hospitalization after cardiac surgery is 4 to 7 days. Discharge planning that begins at admission is imperative because of the short length of hospital stays. The patient should be discharged with the following medications, unless contraindicated: aspirin, a beta blocker, an ACE inhibitor (if the ejection fraction is <40%), and a statin. If a medication is contraindicated, the rationale should be documented. Smoking cessation interventions, if applicable, should be included in discharge teaching. Box 22-8 summarizes patient teaching about cardiac postoperative care.

BOX 22-8 TEACHING GUIDE *Recovering From Cardiac Surgery*

General Instructions

- Avoid lifting heavy objects (10 to 15 pounds or more) for first 3 months.
- Avoid strenuous arm movement, such as golf or tennis. When getting in and out of chair or bed, use legs. Arms should not bear weight and should be used only for balance.
- Do not drive for 6 weeks after surgery. (May ride in automobile.)
- Follow physician's instructions for activity progression.
- Resume sexual activity when you can climb two flights of stairs without stopping (with physician's recommendations).
- Use alternative positions for 3 to 4 months to decrease stress on sternum; avoid side-lying and prone positions.
- Inspect and cleanse surgical incisions daily with soap and water.
- Understand medications, including reason for taking, dosage, frequency, and side effects.
- Follow dietary restrictions.
- Understand how much pain to expect and how to manage it.

Risk Factors

- Follow instructions on individual risk factors, understand their impact on health after cardiac surgery, and learn how to modify them.
- Seek referrals as appropriate (eg, for a weight loss program or a smoking cessation program).

Follow-Up With Physician

- Know how and when to schedule follow-up appointments.
- Be alert for signs and symptoms of infection, such as fever, increased redness, tenderness, drainage, or swelling of incisions.
- Report palpitations, tachycardia, or an irregular pulse (if normally regular) to the physician immediately.
- Seek follow-up care if you experience dizziness or increased fatigue, sudden weight gain or peripheral edema, shortness of breath, or chest pain.

▲ Carotid Endarterectomy

Stenosis or occlusion of the carotid arteries is usually due to atherosclerotic disease and may cause stroke, a leading cause of morbidity and mortality in the United States. Carotid endarterectomy is the most common noncardiac vascular procedure performed to restore flow to the carotid arteries and is designed to decrease the risk for stroke and stroke-related death.[30]

The right carotid artery is a branch of the innominate artery that arises from the right side of the aortic arch. The left common carotid artery arises directly from the aortic arch. At the level of the thyroid, the common carotids bifurcate into the external and internal carotids. Located near this bifurcation, in the carotid sinus, are the carotid chemoreceptors, which are sensitive to blood carbon dioxide and oxygen levels, and the baroreceptors, which help regulate blood pressure. The external carotid arteries supply blood to the structures in the head and neck, excluding the eyes and brain. The internal carotid arteries give rise to the ophthalmic arteries and the posterior communicating, anterior cerebral, and middle cerebral arteries, which help supply blood to the brain (Fig. 22-8).

Patients with carotid artery occlusive disease may have sudden dysphagia, unilateral motor weakness, expressive aphasia, dizziness, memory deficits, or monocular blindness.[31] They often exhibit signs of vascular disease in other parts of the body, such as the heart (CAD) or the legs (peripheral arterial disease). Risk factors for carotid artery occlusive disease are associated with stroke and should guide patient care. Hypertension is the most important risk factor for stroke, and blood pressure regulation is essential in the postoperative period. Cigarette smoking, hyperlipidemia, alcohol consumption, and postmenopausal use of estrogen may also affect patient care.[30]

Patients with risk factors for carotid artery occlusive disease must be examined carefully. A carotid bruit can usually be auscultated over the artery because of turbulent flow through the narrowed artery. Carotid Doppler ultrasonography is usually performed to estimate the presence and amount of stenosis, but angiography is the most reliable method to determine the exact amount of stenosis. Magnetic resonance angiography, which is less invasive, may also be used.

Indications for Carotid Endarterectomy

Carotid artery occlusive disease is part of the systemic atherosclerotic process, which is reviewed in Chapter 21. Carotid endarterectomy is indicated for recently symptomatic patients with 70% to 99% carotid artery stenosis.[31] This surgery should not be considered for symptomatic patients with less than 50% stenosis; these patients have better outcomes if treated medically.[31] To be considered for carotid endarterectomy, patients should be between 40 and 75 years of age, with a 5-year life expectancy.[31]

Surgical Procedure

A skin incision is made along the lower anterior border of the sternocleidomastoid muscle just below the angle of the jaw, and the common, internal, and external carotid arteries are isolated. The carotid arteries on the operative side must be clamped. Clamping puts the ipsilateral cerebral hemisphere and eye at risk for ischemia and infarct because the only perfusion to these areas occurs through the circle of Willis and collaterals, which may be inadequate. To prevent thromboembolus formation while the arteries are clamped, a heparin bolus may be given before clamping. Adequacy of circulation is

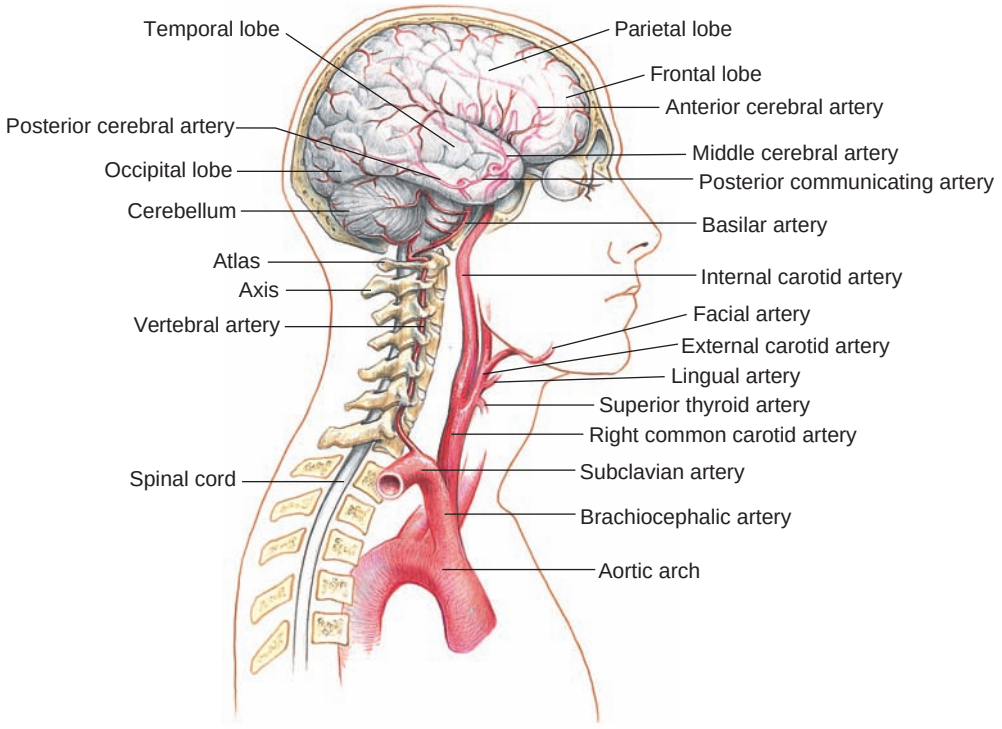


FIGURE 22-8 ▲ Branches of the right external carotid artery. (Courtesy of Anatomical Chart Company.)

determined by continuous electroencephalographic monitoring in the operating room. If circulation is determined to be inadequate, a temporary bypass or shunt may be placed from the common carotid artery to the distal portion of the internal carotid to provide continued intraoperative perfusion. Patients treated with shunts often include those with contralateral carotid stenosis, neurological deficits, history of cerebrovascular accidents, and stroke in evolution.

Endarterectomy or removal of the ulcerated or stenotic atheromatous plaque is then performed, and the artery is closed. If primary closure will cause a narrowing in the vessel, a patch may be used.

Postoperative Care

After extubation in the recovery room, patients are transferred to the ICU with ECG monitoring, an arterial line, CVP monitoring, and oxygen. Traditionally, patients stay in the ICU for 24 hours. However, cost concerns have led to monitoring patients in an intermediate care unit and reducing the hospital stay to 1 day.[32]

Controlling Blood Pressure

Blood pressure is commonly labile up to 24 hours after surgery because of surgically induced abnormalities of the carotid baroreceptor sensitivity. This is characterized as baroreflex failure syndrome and is usually associated with bilateral surgical procedures.[30] Preoperative hypertension is thought to be the most important determinant of postoperative hypertension, which means that the critical care nurse must be aware of the patient's preoperative blood pressure range. Increased blood pressure may also increase the risk for wound bleeding and possible hematoma formation. The goal of blood pressure regulation is a systolic blood pressure between 120 and 170 mm Hg. A systolic blood pressure greater than 170 mm Hg should be treated with nitroprusside or other IV agents, whereas one less than 120 mm Hg should be treated with IV fluid or a norepinephrine or phenylephrine drip if the patient is unresponsive to volume.

Wound Care

To minimize stress on the operative site, the patient's head and neck are kept in alignment. The dressing and the area behind the patient's neck and shoulders are assessed for the presence of blood. Persistent oozing from deep tissue, coughing, straining during extubation, and disruption of suture lines may all lead to bleeding into the operative site. The risk for bleeding can be further aggravated by anticoagulation with heparin, aspirin, or antiplatelet therapy. The nurse assesses the neck size, comparing the operative side with the nonoperative side. Swelling could indicate hematoma formation. Any patient complaints of difficulty talking, swallowing, or breathing should be reported to the physician immediately. If a hematoma is suspected because of tracheal compression by a hematoma, surgical evacuation may be indicated. Wound hematomas occur in about 5.5% of patients.[30]

Preventing Neurological Complications

Brain injury, local nerve injury, or both may occur. Perioperative stroke occurs in approximately 3% of patients and may be due to embolization of atheromatous debris, air from the operative site, or low flow during carotid artery clamping.[31] Neurological assessment includes monitoring level of consciousness, pupil reactivity, eye movement, orientation, appropriateness of response, and motor function (flexion, extension, and hand grips) for the first 24 hours. Abnormalities should be reported to the physician immediately.

Hyperperfusion syndrome occurs in patients with high-grade stenosis. Theoretically, the hemisphere distal to the stenotic area has suffered hypoperfusion that causes the small blood vessels to remain maximally dilated with a loss of autoregulation. Once the stenosis is repaired, autoregulation is still paralyzed, but a marked increase in blood flow occurs that cannot be controlled with vasoconstriction to protect the capillaries. Edema or hemorrhage to the area results.[31] Strict blood pressure control is imperative.

Several cranial nerves (CN) traverse the surgical area and can be exposed to trauma. The most commonly affected are CN VII (the facial nerve), CN X (the vagus nerve), CN XII (the hypoglossal nerve), and CN XI (the spinal accessory nerve). Specific functional assessment for each nerve should be performed after surgery, including those listed in Table 22-3. If a deficit is present, the nurse should notify the physician and explain to the patient how it occurred and that the deficit is usually temporary.

Table 22-3 Postoperative Functional Assessment of Cranial Nerves Following Carotid Endarterectomy

Nerve	Nerve Intervention	Functional Assessment	Functional Damage
Facial nerve (VII)	Motor function of facial muscles	Ability to smile and frown	Asymmetrical contraction of the mouth
Vagus nerve (X)	Motor and sensory function of larynx and throat	Quality and tone of voice and ability to swallow	Difficult swallowing, hoarseness, speech problems, loss of gag reflex
Hypoglossal nerve (XII)	Muscles to tongue	Movement of tongue	Difficult swallowing, speech problems, deviation of tongue, sometimes airway damage
Spinal accessory nerve (XI)	Trapezius and sternocleidomastoid muscles	Ability to shrug shoulders and raise arm to horizontal position	Shoulder may sag, difficulty raising shoulder against resistance, difficulty raising arm to horizontal position

BOX 22-9 TEACHING GUIDE Recovering From Carotid Endarterectomy

Risk Factor Reduction
- Stop smoking.
- Eat a low-fat diet.
- Control hypertension if present.

Activity
- There are usually no restrictions on activity. It is all right to move your neck in a normal manner.

Incision Care
- Bruising and discoloration are common.
- Wash the incision site with soap and water.

General
- Be familiar with signs and symptoms of incisional infection.
- Notify your physician of visual defects, changes in memory or sensation, or an inability to swallow or speak.
- Be knowledgeable about medication indications, including reason for taking, dosage, frequency, and side effects.
- Keep physician appointments.

Home Care Considerations

Patients are usually discharged on the first or second postoperative day. Aspirin (81 or 325 mg/d) should be prescribed postoperatively for at least 3 months to reduce the possibility of stroke, MI, or death.[31] The critical care nurse plays an essential role in the care of the patient who has had a carotid endarterectomy. Although considered a vascular surgical procedure, postoperative complications usually manifest as neurological symptoms, and the nurse must assess the patient for subtle neurological changes. Patient education is also a key component of care. Patients and their families should understand that the patient has an underlying cardiovascular disease and that risk factor modification is necessary. Education should include the items listed in Box 22-9.

▲ Clinical Applicability Challenges

CASE STUDY

Ms. W., a 54-year-old woman who has had CABG surgery with three grafts and good left ventricular function preoperatively (ejection fraction about 50%), has arrived in the intensive care unit (ICU) from the operating room after 5 hours in surgery. An internal mammary artery graft to the left anterior descending coronary artery and saphenous vein grafts to the right coronary artery and diagonal artery were performed using the standard bypass technique. Ms. W. has a history of arthritis and takes large doses of ibuprofen for pain relief. Her chest tube output upon arrival in the ICU was 200 mL and increased to 250 mL in the next 30 minutes × 2. Her blood pressure is 100/50 mm Hg, and her normal blood pressure averages 130/70 mm Hg.

1. What do you think caused the increased amount of bleeding in this patient situation?
2. What would be your immediate intervention for Ms. W.?
3. What interventions would be the most important to make the provider aware of?

References

1. Eagle K, Guyton R, Davidoff R, et al: ACC/AHA 2004 guideline update for coronary bypass graft surgery. J Am Coll Cardiol 110:e340–431, 2004
2. Felisky C, Paull D, Hill M, et al: Endoscopic greater saphenous vein harvesting reduces the morbidity of coronary artery bypass surgery. Am J Surg 183:576–579, 2002
3. Leavitt BJ, O'Connor GT, Olmstead EM, et al: Use of internal mammary artery graft and in-hospital mortality and other adverse outcomes associated with coronary artery bypass surgery. Circulation 93:507–512, 2001
4. Connolly MW, Torrillo LD, Stauder MJ, et al: Endoscopic radial harvesting: Results of the first 300 patients. Ann Thoracic Surg 74: 502–505, 2002
5. Shapira O, Xu A, Vita J, et al: Nitroglycerin is superior to diltiazem as a coronary bypass conduit vasodilator. J Thorac Cardiovasc Surg 117: 906–911, 1999
6. Emery RW, Arom KV, Holter AR, et al: Advances in coronary artery surgery. In Franco KL, Verrier E (eds): Advanced Therapy in Cardiac Surgery, 2nd ed. Hamilton, ON: BC Decker, 2003, pp 124–130
7. Vallely M, Bannon P, Kritharides L: The systemic inflammatory response syndrome and off-pump cardiac surgery. Heart Surg Forum 4(Suppl): S7–S13, 2001
8. Magee M, Jablouski K, Stamou S, et al: Elimination of cardiopulmonary bypass improves early survival for multivessel coronary artery bypass patients. Ann Thorac Surg 73:1196–1202, 2002

9. Peel GK, Stamou SC, Dullum MK, et al: Chronological distribution of stroke after minimally invasive versus conventional coronary artery bypass. J Am Coll Cardiol 43(5):752–756, 2004
10. Horvath KA: Transmyocardial laser revascularization. In Franco KL, Verrier E (eds): Advanced Therapy in Cardiac Surgery, 2nd ed. Hamilton, ON: BC Decker, 2003, pp 131–137
11. Lindsay MR: Transmyocardial laser revascularization revisited. Crit Care Nurs Q 26:69–75, 2003
12. Lytle BW: Coronary artery reoperations. In Cohn LH, Edmunds LH (eds): Cardiac Surgery in the Adult, 2nd ed. New York, NY: McGraw-Hill Medical Publishing Division, 2003, pp 659–679
13. Kaiser L, Kron I, Spray T: Mastery of Cardiothoracic Surgery. Philadelphia, PA: Lippincott-Raven, 2006
14. Lancey R, Gaca C, Vander Salm T: The use of smaller, more flexible chest drains following open heart surgery. Chest 119:19–24, 2001
15. Goldstein JP, Waulthy P. Cardiac surgery: Indications and Complications. In Fink MP, Abraham E, Vincent JL, et al (eds): Textbook of Critical Care, 5th ed. Philadelphia, PA: Elsevier Saunders, 2005, pp 889–896
16. Sladen R, Berend J, Fassero J, et al: Comparison of vecuronium and meperidine on the clinical and metabolic effects of shivering after hypothermic cardiopulmonary bypass. J Cardiothorac Vasc Anesth 9:147–153, 1995
17. Muckart D, Bhagwanjee S: American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference: Definitions of the systemic inflammatory response syndrome and allied disorders in relation to critically injured patients. Crit Care Med 25:1789–1795, 1997
18. Cheek DJ, Rodgers SC, Schulman SC: Systemic inflammatory response syndrome and multiorgan dysfunction syndrome. In Carlson K (ed): Advanced Critical Care Nursing. St. Louis, MO: Saunders Elsevier, 2009
19. Sirieix D, Hongnat M, Delayaance M, et al: Comparison of the acute hemodynamic effects of hypertonic or colloid infusions immediately after mitral valve repair. Crit Care Med 27:2159–2165, 1999
20. Le Conte P, Coutaut V, N'Guyeu J, et al: Prognostic factors in acute cardiogenic pulmonary edema. Am J Emerg Med 17:329–332, 1999
21. Brown T: Hibernating myocardium. Am J Crit Care 10:84–90, 2001
22. Ely E, Meade M, Haponik E, et al: Mechanical ventilator weaning protocols driven by non-physician health-care professionals: Evidence-based clinical practice guidelines. Chest 120(6 Suppl):454S–463S, 2001
23. Burns S, Dempsey E: Long-term ventilator management strategies: Experiences in two hospitals. AACN Clin Issues 11:424–441, 2000
24. Jarcia JP, Venkataramana V, Gold JP: Prevention of neurologic injury during cardiac and great vessel surgery. In Franco KL, Verrier E (eds): Advanced Therapy in Cardiac Surgery, 2nd ed. Hamilton, ON: BC Decker, 2003, pp 74–82
25. Brunton LL, Lazo JS, Parker KL (eds): Goodman & Gilman's the Pharmacological Basis of Therapeutics. New York, NY: McGraw-Hill, 2006
26. Rosengart TK: Blood conservation for open heart surgery. In Franco KL, Verrier E (eds): Advanced Therapy in Cardiac Surgery, 2nd ed. Hamilton, ON: BC Decker, 2003, pp 37–45
27. Hebert P, Wells G, Blajchman M, et al: and the Transfusion Requirements in Critical Care Investigators for the Canadian Critical Care Trials Group: A multicenter, randomized, controlled clinical trial of transfusion requirements in critical care. N Engl J Med 340: 409–417, 1999
28. Fink MP, Abraham E, Vincent JL, et al (eds): Textbook of Critical Care, 5th ed. Philadelphia, PA: Elsevier Saunders, 2005
29. Van den Berghe G, Vounters P, Weekers F, et al: Intensive insulin therapy in the critically ill patients. N Engl J Med 345:1359–1367, 2001
30. Bettmann M, Katzen B, Whisnant J, et al: A statement for health-care professionals from a special writing group of the Stroke Council, American Heart Association. Circulation 97:121–123, 1998
31. Chaturvedi S, Bruno A, Feasby T, et al: Carotid endarterectomy: An evidence-based review. Report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. Neurology 65:794–801, 2005
32. Dirks JL: Cardiac surgery. In Carlson K (ed): Advanced Critical Care Nursing. St. Louis, MO: Saunders Elsevier, 2009

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

PART 6

RESPIRATORY SYSTEM

23

Anatomy and Physiology of the Respiratory System

Megan Cecere Lynn and Karen L. Johnson

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Identify the major structures of the respiratory system that are located in the thorax.
2. Describe the movement of air through the airways from the nose to the alveoli.
3. Discuss the function of surfactant in maintaining alveolar inflation.
4. Differentiate the function of bronchial and pulmonary circulations.
5. Describe the mechanics of ventilation in terms of air movement into and out of the lungs, lung compliance, and airway resistance.
6. Explain four factors that affect the diffusion of gases across the alveolar–capillary membrane.
7. Identify physiological and pathophysiological conditions that produce a ventilation–perfusion mismatch.
8. Discuss conditions that affect the relationship described in the oxyhemoglobin dissociation curve and how these conditions affect oxygen exchange.
9. Describe the function of the chemoreceptors and lung receptors.

The structures of the respiratory system allow gases to move between the external environment and the internal environment. The cardinal function of the respiratory system is gas exchange, a process by which oxygen moves from the air into the blood and carbon dioxide moves out of the blood and is exhaled to the external environment. The respiratory system also has several other functions, including regulation of acid–base balance, metabolism of some compounds, and filtration of inhaled unwanted materials. Intact respiratory structures and proper functioning of the respiratory system are necessary for transport of gases in and out of the body. Knowledge of respiratory anatomy and physiology helps the nurse understand respiratory assessment techniques, principles of respiratory system management, and common disorders of the respiratory system.

▲ Anatomy of the Respiratory System

The Thorax

The thorax contains the major structures of the respiratory system. These structures include the bony thoracic cage, the muscles of ventilation, the lungs, the pleural space, and the mediastinum (Fig. 23-1). The thoracic cage is a rigid, yet flexible, structure. Its bony structure protects the major organs in the thoracic cavity. Flexibility allows for inhalation/inflation and exhalation/deflation of the lungs. The thoracic cage consists of 12 vertebrae, each with a pair of ribs. Posteriorly, each rib is attached to a vertebra (Fig. 23-2). Anteriorly, the first seven ribs are attached to the sternum (Fig. 23-3). The

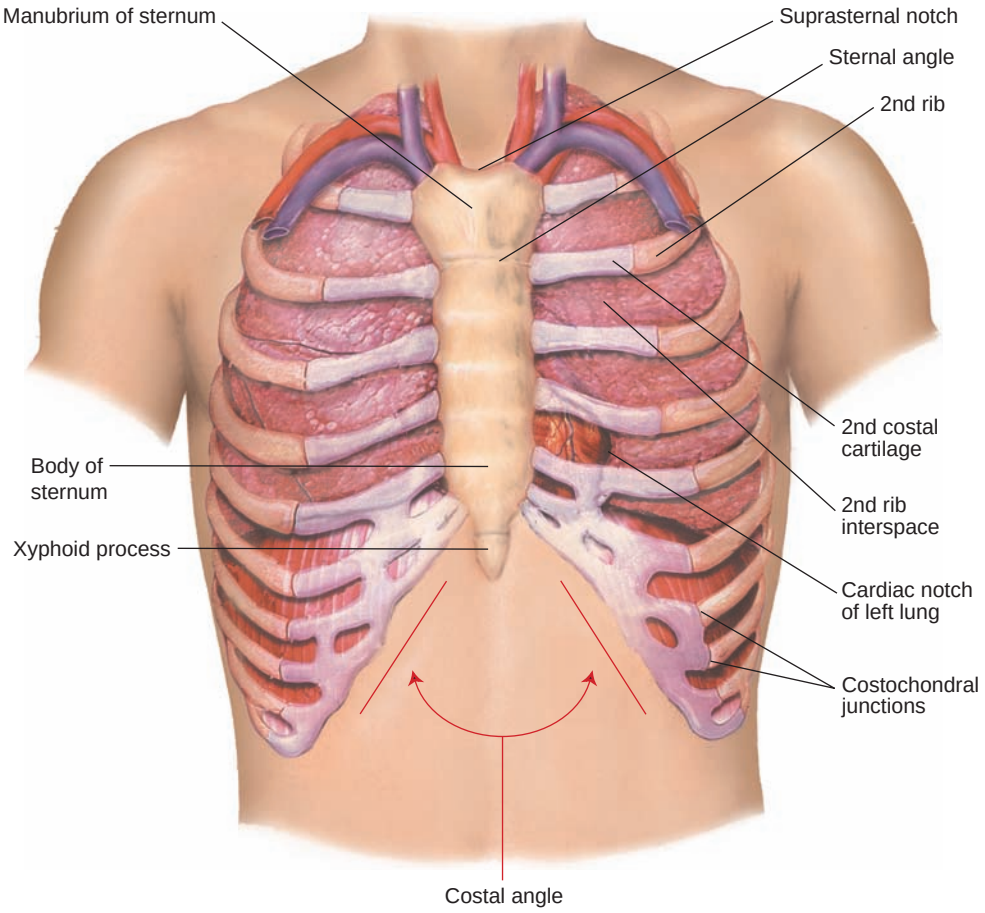


FIGURE 23-1 ▲ Anatomy of the chest wall. (From Bickley LS: Bates' Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 283.)

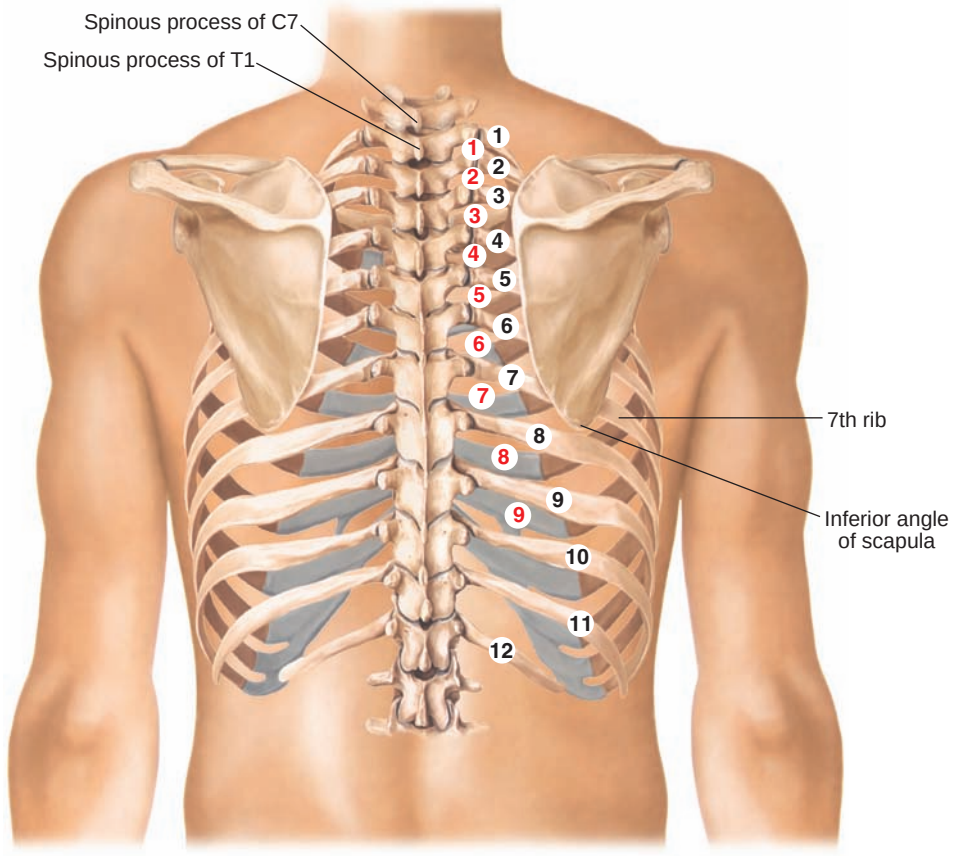


FIGURE 23-2 ▲ Posterior thoracic cage. (From Bickley LS: Bates' Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 285.)

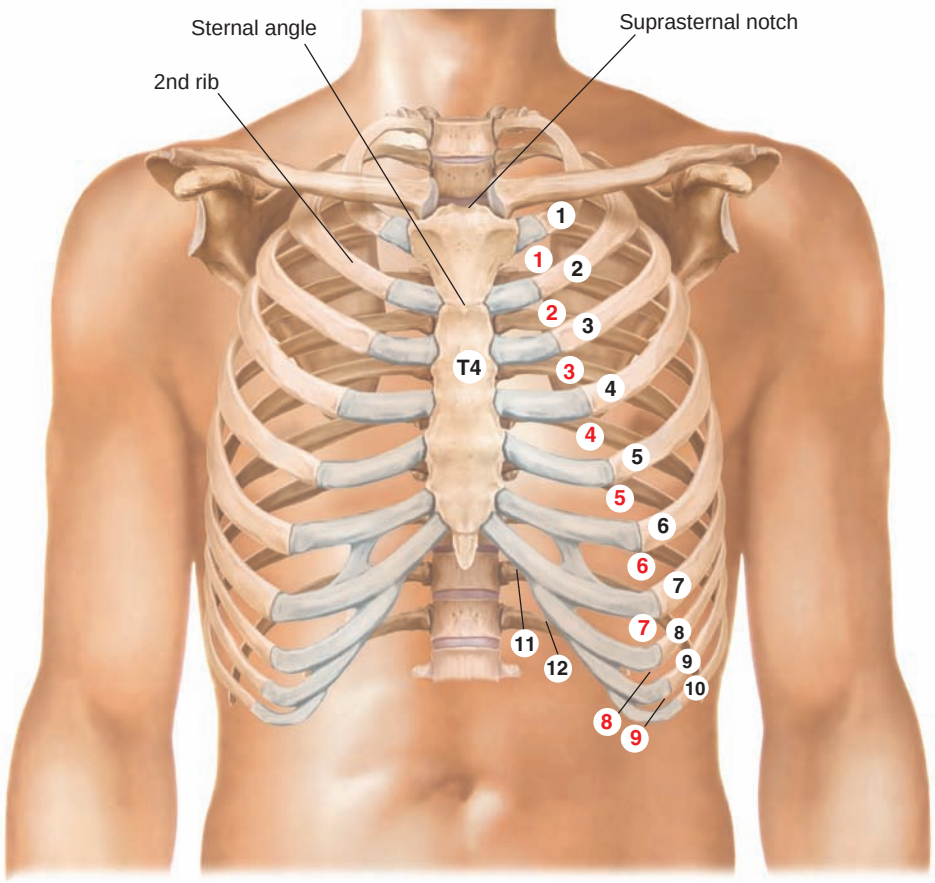


FIGURE 23-3 ▲ Anterior thoracic cage. (From Bickley LS: Bates' Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 284.)

8th, 9th, and 10th ribs are attached by cartilage to the ribs above them. The 11th and 12th ribs are called "floating ribs," because they are not attached anteriorly to another structure.

The Lungs, Mediastinum, and Pleural Space

Positioned within, and protected by, the thoracic cage, the lungs are located on either side of the chest. These air-filled, spongy structures are attached to the body only at the pulmonary ligament at the mediastinum. The right lung contains three lobes, and the left lung contains only two lobes because of the space limitation imposed by the heart. The base of each lung rests anteriorly at the level of the 6th rib at the mid-clavicular line and at the 8th rib at the mid-axillary line. The apices extend 2 to 4 cm above the inner aspects of the clavicles.

The space between the two lungs is the mediastinum. The mediastinum contains the heart, blood vessels, lymph nodes, thymus gland, nerve fibers, and esophagus.

Pleural membranes surround the lungs and line the thoracic wall. The parietal pleura is the membrane lining the chest wall, and the visceral pleura overlays the lung parenchyma (Fig. 23-4). A thin layer of serous fluid in the small space between these two pleurae allows the parietal and visceral pleurae to slide over each other during inspiration and expiration. The pressure within the pleural space is called the intrapleural pressure. The intrapleural pressure is normally less than the pressures within the lung. It is this negative pressure that keeps the lungs inflated. If the intrapleural space loses its negative pressure (by exposure to atmospheric pressure; eg, as a result of chest trauma), the lung collapses, a condition known as a pneumothorax. The pleural space is also a potential space for the accumulation of fluid. An abnormal collection of fluid in the pleural space is a pleural effusion.

The Respiratory Muscles

The muscles that elevate the thoracic cage are classified as *muscles of inspiration*.[1] The major muscle involved with inspiration is the diaphragm. The diaphragm is a thin, dome-shaped muscle that is innervated by the phrenic nerves. When it contracts, the abdominal contents are forced downward, and the chest expands vertically (Fig. 23-5). In normal breathing, the level of the diaphragm moves about 1 cm, but on forced inspiration, a total excursion of up to 10 cm may occur.[2] The external intercostal muscles also assist with inspiration (Fig. 23-6). These muscles attach to adjacent ribs and slope downward and forward. When the external muscles contract, the ribs are pulled forward and upward, and this increases the lateral and anterior–posterior diameters of the thoracic cage. The accessory muscles of inspiration include the scalene and sternocleidomastoid muscles. The scalene muscles elevate the first two ribs, and the sternocleidomastoid muscles raise the sternum.[2] During normal breathing, these muscles are not used, but during exercise, these muscles contract to aid in inspiration.

Muscles that depress the thoracic cage are classified as *muscles of expiration*.[1] Expiration is a largely passive process during normal breathing. During expiration, the diaphragm relaxes, and the elastic recoil of the lungs, chest wall, and abdominal structures compresses the lungs. Expiration can become an

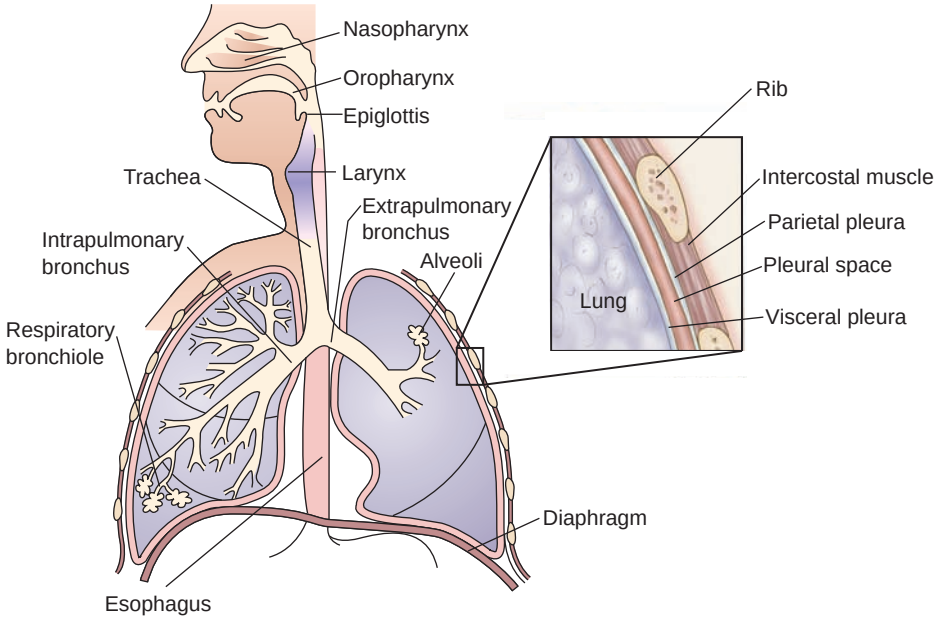


FIGURE 23-4 ▲ Structures of the respiratory system. (From Porth CM: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 641.)

active process during exercise. The abdominal and intercostal muscles can increase expiratory effort (see Fig. 23-5). When the abdominal muscles contract, the intra-abdominal pressure increases and pushes the diaphragm upward. These muscles are also used during defecation, vomiting, and coughing. When the internal intercostal muscles contract, the ribs are pulled downward and inward, decreasing the thoracic volume.

The Conducting Airways

The conducting airways include the nasopharynx, oropharynx, trachea, bronchi, bronchioles, and the terminal bronchioles (see Fig. 23-4). These airways—a series of tubes that become more numerous and narrow as they penetrate deeper into the lungs (Fig. 23-7)—warm, humidify, and filter air that is inhaled as it is channeled to the gas exchange region. Because the conducting airways contain no alveoli and do not participate in gas exchange, they constitute the *anatomic dead space*. The volume in the anatomic dead space is approximately 150 mL.[3]

The Nasopharynx and Oropharynx

The nasopharynx is the preferred route for entrance of air into the respiratory tract during normal breathing because it filters and warms inspired air.[4] The outer passages are lined with coarse hairs that filter large particles. The upper portion of the nasal cavity supplies warmth and moisture to the air inhaled. If nasal passages are plugged or when larger volumes of gases need to be exchanged (eg, during exercise), the oropharynx provides an alternate route. Obstruction of the oropharynx leads to immediate cessation of ventilation ("choking"). Foreign bodies and swelling of the pharyngeal airways from infection, injury, or allergic reaction can also cause airway obstruction.

The Epiglottis

The epiglottis is located posterior to the root of the tongue (see Fig. 23-4). It is a leaf-shaped piece of cartilage that moves up and down. During inhalation, the epiglottis moves upward to allow air to move through the trachea. During swallowing, it moves downward to cover the larynx and allow food and

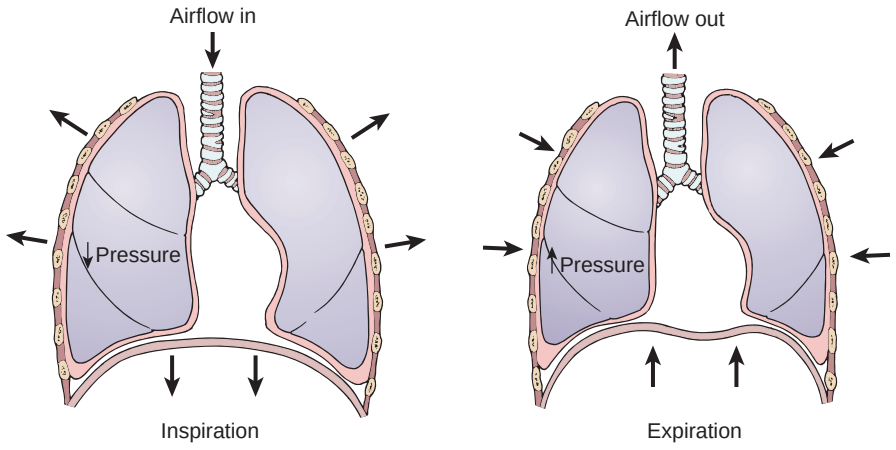


FIGURE 23-5 ▲ Frontal section of the chest showing the movement of the rib cage and diaphragm during inspiration and expiration. (From Porth CM: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 650.)

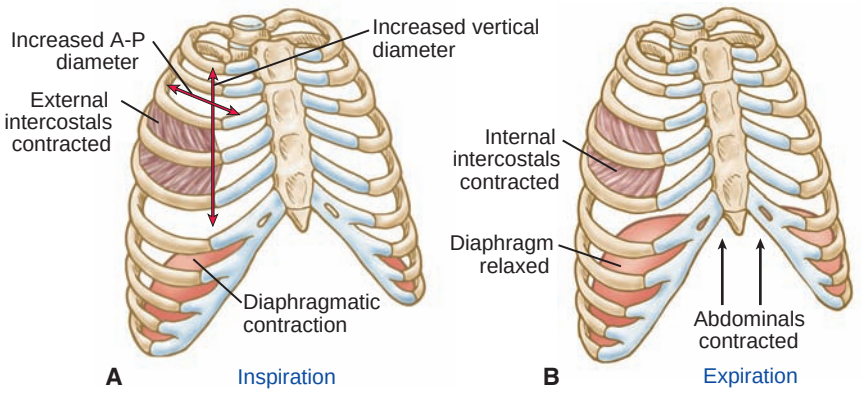


FIGURE 23-6 ▲ **A, B:** Contraction and expansion of the thoracic cage during expiration and inspiration, demonstrating diaphragmatic contraction, function of the intercostals, and elevation and depression of the rib cage. (From Porth CM: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 651.)

liquid to pass into the esophagus. During defecation, especially during defecation associated with straining and constipation, inhaled air is temporarily held in the lungs by closure of the glottis. Contraction of the intra-abdominal muscles causes an increase in the intra-abdominal and intrathoracic pressures. These collective processes are called the *Valsalva maneuver*. The Valsalva maneuver can be dangerous because the abrupt increase in intrathoracic pressure can significantly reduce venous return and therefore cardiac output.

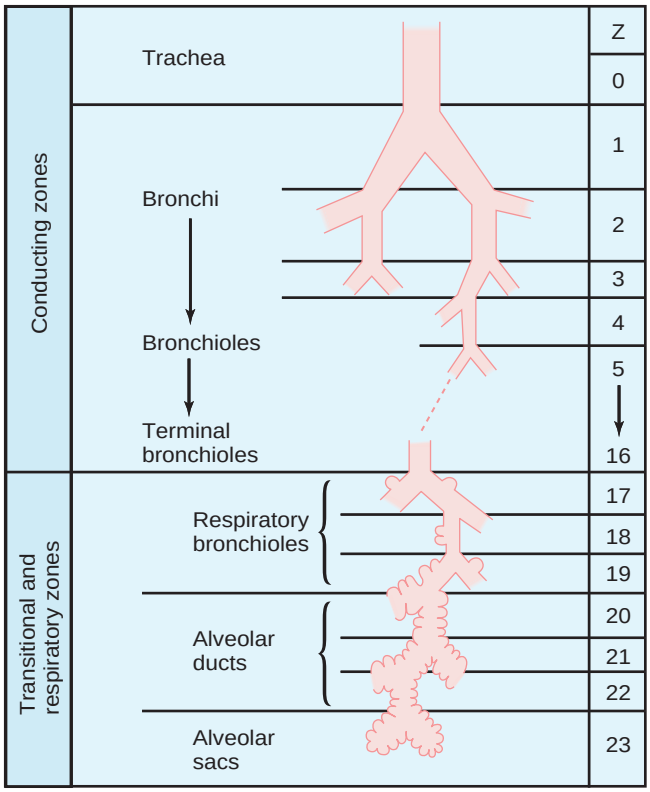


FIGURE 23-7 ▲ Idealization of the human airways. Note that the first 16 generations (Z) make up the conducting zone, and generations 17 to 23 make up the transitions and respiratory zones. Throughout childhood, the airways increase in diameter and length, and the number and size of the alveoli increase until adolescence, when respiratory development matures to that of an adult. (Adapted from West JB: Respiratory Physiology: The Essentials, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, p 6.)

The Tracheobronchial Tree

The tracheobronchial tree consists of the trachea, bronchi, and bronchioles. The trachea is a hollow tube, or "windpipe," that connects the larynx and the major bronchi of the lungs (see Fig. 23-4). The trachea is primarily smooth muscle and is supported by horseshoe-shaped rings of cartilage that prevent the trachea from collapsing during coughing or bronchoconstriction of the smooth muscle.

The end of the trachea divides, forming the two large mainstem bronchi. The point at which the trachea divides is called the *carina*. The carina is innervated with sensory neurons. When the carina is stimulated (eg, during tracheal suctioning), the cough reflex and bronchoconstriction are elicited. The right mainstem bronchus is wider and shorter than the left bronchus. Thus, the right mainstem bronchus is the most common site of aspiration of foreign bodies. The right and left mainstem bronchi divide into branches that become smaller and more numerous as they divide (Fig. 23-8). The right and left mainstem bronchi divide into lobar and segmental bronchi, which divide into bronchioles, which become terminal bronchioles. The terminal bronchioles are the smallest airways without alveoli. The mainstem bronchi are similar in structure to the trachea, in that they are airways supported by cartilage rings. However, as the bronchi extend into the lungs, the cartilage rings become irregular and smaller until they disappear at about the level of the respiratory bronchioles. Here, smooth muscle wraps around the bronchioles. Contraction of these muscles (bronchospasm) causes narrowing of the bronchioles and impairs gas flow.[4]

The Respiratory Airways

The terminal bronchioles branch into the respiratory airways. These airways include the respiratory bronchioles, the alveolar ducts, and the alveolar sacs (see Fig. 23-7). The respiratory zone makes up most of the lung, its volume being about 2.5 to 3 L.[5]

The Respiratory Bronchioles

Each respiratory bronchiole forms a lobule. The lobule is the smallest functional unit of the lung and is where gas exchange

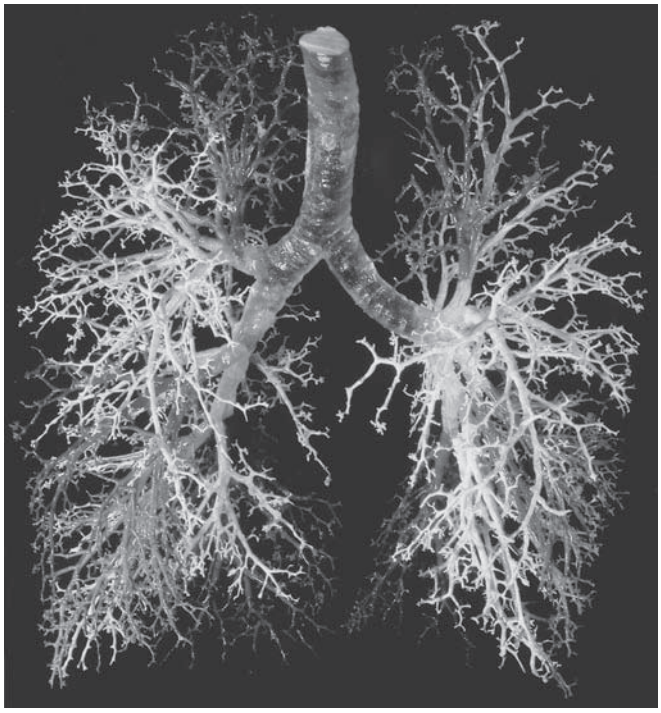


FIGURE 23-8 ▲ Cast of the airways of a human lung. The alveoli have been pruned away, allowing the conducting airways from the trachea to the terminal bronchioles to be seen. (From West JB: Respiratory Physiology: The Essentials, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, p 5.)

takes place. A lobule consists of an arteriole, the pulmonary capillaries, and a venule (Fig. 23-9). Blood enters through a pulmonary artery and exits through a pulmonary vein. This is the only place in the body where highly oxygenated blood flows through a vein.

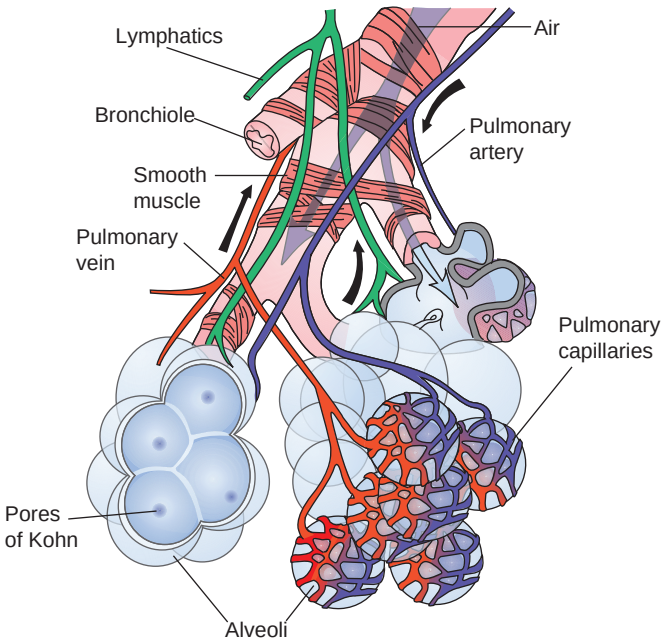


FIGURE 23-9 ▲ Lobule of the lung, showing the bronchial smooth muscle fibers, pulmonary blood vessels, and lymphatics. (From Porth CM: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 645.)

The Alveoli

Each respiratory bronchiole gives rise to several alveolar ducts that terminate in a cluster of alveoli, as shown in Figure 23-9. The alveolus is the end point of the respiratory tract, and it is here where gas exchange takes place. The alveoli are thin-walled, cup-shaped structures. There are approximately 300 million alveoli in the adult lung, with a total surface area of 85 m^2.[5] The alveoli also contain macrophages that perform a phagocytic role. These cells move from alveolus to alveolus, removing foreign substances and keeping the alveoli sterile.

Alveolar structures are composed of two types of cells: type I alveolar cells and type II alveolar cells. *Type I alveolar cells* are flat squamous epithelial cells and comprise approximately 90% of the total alveolar surface area. Gas exchange takes place along these cells. *Type II alveolar cells* secrete pulmonary surfactant. Surfactant is a lipoprotein that decreases surface tension in the alveoli. This prevents collapse of the smaller airways during expiration and makes it easier to inflate the alveoli during inspiration. Therefore, injury to type II alveolar cells leads to alveolar collapse and impaired pulmonary gas exchange.

The Lung Circulation

The lungs have a dual blood supply: the bronchial circulation and the pulmonary circulation. The bronchial circulation distributes blood to the airways, and the pulmonary circulation contributes to gas exchange.

The Bronchial Circulation

The bronchial arteries that perfuse the left side of the thorax arise from the aorta, and the arteries that perfuse the right side of the thorax branch from the internal mammary, subclavian, and intercostal arteries. The capillaries of the bronchial circulation drain into the bronchial veins and eventually empty into the vena cava or the pulmonary vein. The bronchial circulation does not participate in gas exchange. Blood that is emptied into the pulmonary vein is unoxygenated blood and mixes with oxygenated blood flowing to the left side of the heart. This contributes to the "anatomic shunt" and is why arterial oxygen saturation is always less than 100%. The flow through the bronchial circulation is minimal, and the lung can function fairly well without it, such as after lung transplantation.[5]

The Pulmonary Circulation

The pulmonary circulation arises from the pulmonary artery and provides for the gas exchange function of the lung (Fig. 23-10). As shown in Figure 23-10, deoxygenated blood leaves the right ventricle and enters the pulmonary artery. The blood passes from the pulmonary artery through a series of branching arteries to the capillaries, and then back through a series of venules to the pulmonary vein.

In the walls of the alveoli, the capillaries form a dense network (see Fig. 23-9). The diameter of a capillary segment is about 10 micrometers, just large enough for one red blood cell.[5] The extreme thinness of the blood–gas barrier is extremely efficient for gas exchange but also means these capillaries are easily damaged. Increasing pressure in the alveoli (such as occurs with high levels of positive

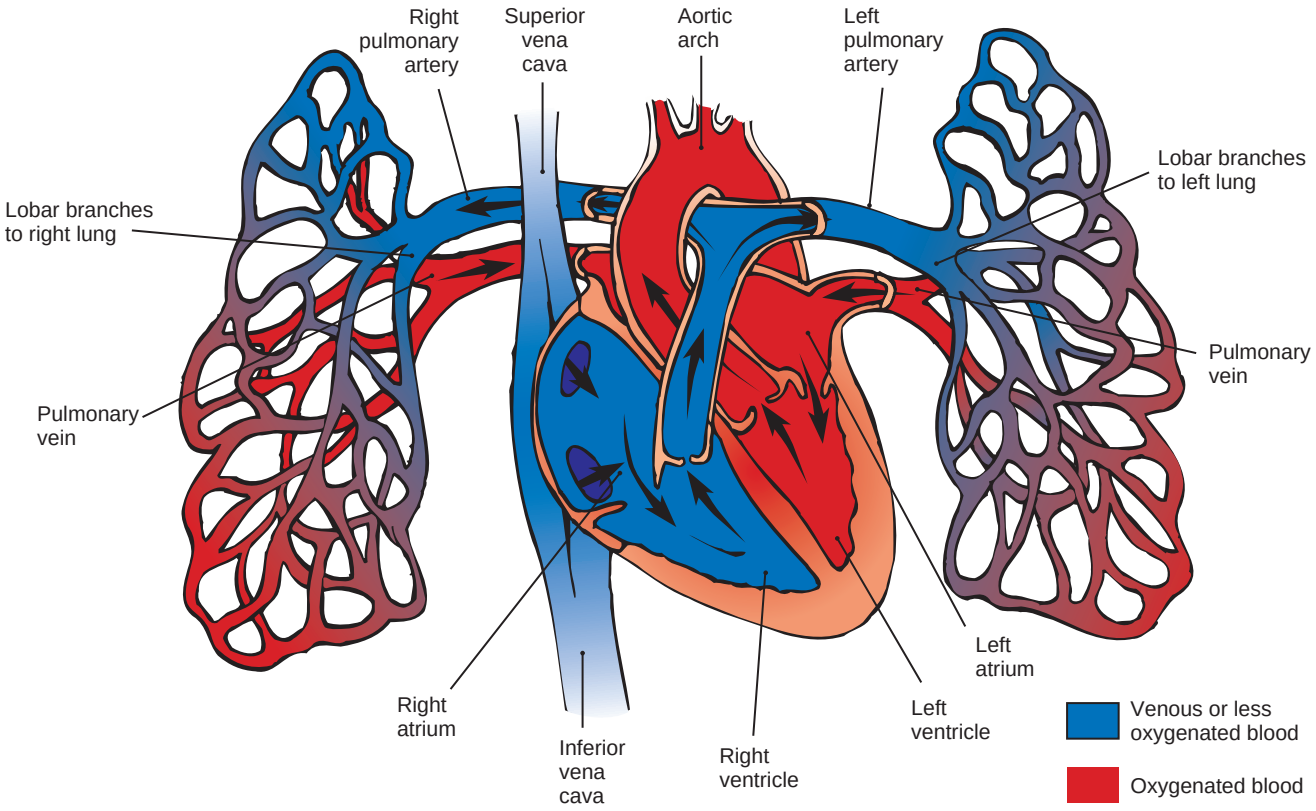


FIGURE 23-10 ▲ Circulation from the right heart to the lungs and left heart.

end-expiratory pressure) or increasing volume in the alveoli (such as occurs with mechanical ventilation with large tidal volumes) can damage capillaries, causing them to leak plasma into the alveolar spaces. Each red blood cell spends about 0.75 second in the capillary network and during this time probably transverses two or three alveoli.[5] Almost complete equilibrium of oxygen and carbon dioxide between alveolar gas and capillary blood can occur during this very brief time.

The pulmonary artery receives the whole output from the right ventricle. However, the resistance of the pulmonary circuit is extremely low, compared with the systemic vascular resistance, because the pulmonary vascular structures do not have vascular smooth muscle like the systemic vascular structures do. Thus, systolic and diastolic pressures in the pulmonary circulatory system are much lower. Normal pulmonary artery pressures are 20 to 30/8 to 15 mm Hg. Just as hypertension can develop in the systemic circulatory system, hypertension can occur within the pulmonary circulatory system and is called pulmonary hypertension.

The Pulmonary Lymphatics

The lungs represent the largest surface area of the body that is exposed to an increasingly hostile environment.[5] Fortunately, the lungs have multiple mechanisms to handle inhaled particles. The nose filters large particles. Particles that deposit in the conducting airways are removed by cilia that line the airways. The cilia brush the particles up toward the epiglottis, where they are then swallowed. By the process of phagocytosis, macrophages or leukocytes destroy foreign particles in the alveoli. Foreign materials that reach the alveoli are then removed by lymphatic tissue. The lungs have a vast supply of lymphatic tissue. The lymphatic vessels parallel the pulmonary vasculature (see Fig. 23-9). They surround the lobule and aid in the removal of particles and protein from the interstitial spaces. These vessels eventually drain into lymph nodes located at the hila of the lungs.

▲ Physiology of the Respiratory System

The goals of respiration are to provide oxygen to tissues and to remove carbon dioxide. The physiology of respiration involves the following three processes: (1) *ventilation*, or the movement of air between the atmosphere and the alveoli; (2) *diffusion* of oxygen and carbon dioxide between the pulmonary capillaries and the alveoli; and (3) *transport* of oxygen and carbon dioxide in the blood to and from the cells.[6]

Ventilation

During ventilation, the movement of air into the lungs is known as *inhalation*, and the movement of air out of the lungs is known as *exhalation*. Air flows from a region of higher pressure to a region of lower pressure. To initiate a breath, a drop in pressure in the alveoli must precipitate airflow into the lungs.

The Mechanics of Ventilation

Ventilation is a complex process with multiple variables, including the change in pressures and the integrity of the muscles responsible for moving air in and out of the lungs, the compliance of the lungs, and the resistance afforded by the airways. Collectively, these variables are referred to as the *mechanics of ventilation*.

MOVEMENT OF AIR INTO AND OUT OF THE LUNGS. The movement of air in and out of the lungs requires muscles to expand and contract the chest cavity and a change in gas pressures to facilitate movement of air from one compartment to another. The lungs can be expanded and contracted in two ways: (1) by downward and upward movement of the diaphragm to lengthen and shorten the chest cavity and (2) by elevation and depression of the ribs to increase and decrease the anterior–posterior diameter of the chest cavity.[1]

According to the laws of physics, the movement of gases is always from an area of higher pressure to lower pressure. Several pressures are involved in the process of respiration: airway pressure, intrapleural pressure, intra-alveolar pressure, and intrathoracic pressure (Fig. 23-11). The *airway pressure* is the pressure in the conducting airways. The *intrapleural pressure* is the pressure in the narrow space between the visceral and parietal pleurae. The *intra-alveolar pressure* is the pressure inside the alveoli. The pressure difference between the intra-alveolar pressure and the intrapleural pressure is called the *transpulmonary pressure*. The *intrathoracic pressure* is the pressure within the entire thoracic cavity.

Figure 23-12 illustrates the mechanics involved in ventilation. Figure 23-12A shows the pressures in the resting state. Pleural pressure, a slightly negative pressure, creates a suction that holds the lungs open to their resting level. Without this negative pressure to hold the lungs against the chest wall, the elastic recoil properties of the lungs would cause them to collapse. When the glottis is open and no air is flowing, the pressure in the conducting airways and alveoli equals atmospheric pressure. Figure 23-12B shows the pressures during inspiration. During inspiration, as the diaphragm and intercostal muscles contract, the volume of the chest cavity increases. Expansion of the chest wall pulls outward on the lungs, and the intrapleural pressure becomes more negative. As the alveolar pressure becomes more negative, air flows in from the atmosphere through the conducting airways to the alveoli. After inspiration, the muscles relax, and the chest cavity returns to its resting position. With this decrease in chest size and resultant compression of the lungs, the intra-alveolar pressure builds and forces air out of the lungs during expiration. One respiratory cycle consists of one inhalation and one exhalation. At rest, inhalation requires 1 second and exhalation lasts 2 seconds.

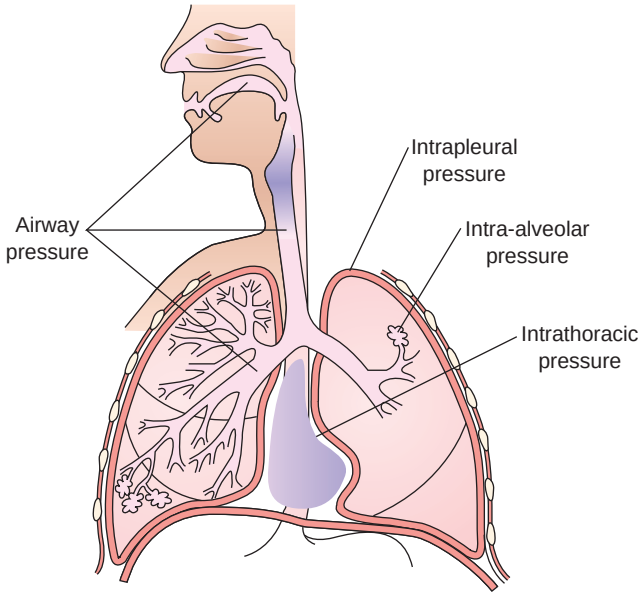


FIGURE 23-11 ▲ Partitioning of respiratory pressures. (From Porth CM: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 650.)

LUNG COMPLIANCE. The extent to which the lungs expand is called *compliance*. Compliance is a measurement of distensibility, or how easily a tissue is stretched. If compliance is reduced, it is more difficult to expand the lungs for inspiration. And conversely, if compliance is increased, it is easier to expand lung tissue. Compliance is expressed as the ratio of the change in lung volume to the change in lung pressure.

$$\text{Compliance} \quad \frac{\text{Change in lung volume (L)}}{\text{Change in lung pressure (cm } H_2O\text{)}}$$

Compliance can be appreciated by comparing the ease of blowing up a new balloon that is stiff and resistant with one that has been previously blown up and is more compliant. Lung compliance is determined by the elastin and collagen fibers of the lung and the surface tension in the alveoli.

Lung tissue is made up of elastin and collagen fibers. Collagen fibers resist stretching and make lung inflation difficult, whereas elastin fibers are easily stretched and increase the ease of lung inflation. When elastin fibers are replaced with scar tissue, such as that which occurs with pulmonary fibrosis or interstitial lung disease, the lungs become stiff and noncompliant.

The fluid lining the alveoli has a high surface tension. When the surface tension is high, the moist interior surfaces of an alveolus are difficult to separate from one another, and more energy is required to open and fill the alveolus with air during inspiration. When the surface tension is low, the alveoli walls separate more easily, requiring less effort for alveolar filling during inspiration. Recall that a lipoprotein substance called *surfactant*, which is secreted by type II alveolar cells, decreases the surface tension of these fluids in the alveoli.

Surfactant has four important effects on lung inflation: it lowers the surface tension, increases lung compliance and ease of inflation, provides for stability and more even inflation of the alveoli, and assists in preventing pulmonary edema by keeping the alveoli dry.[4] Without surfactant, lung inflation is extremely difficult. The type II alveolar cells that produce surfactant do not mature until the 26th to 28th weeks of gestation.[4] Premature infants do not have sufficient amounts of surfactant, which leads to alveolar collapse and severe respiratory distress, a condition known as *infant respiratory distress syndrome*. Lack of surfactant or inefficient surfactant production may also play a role in the development of *acute respiratory distress syndrome* (ARDS) in adults.

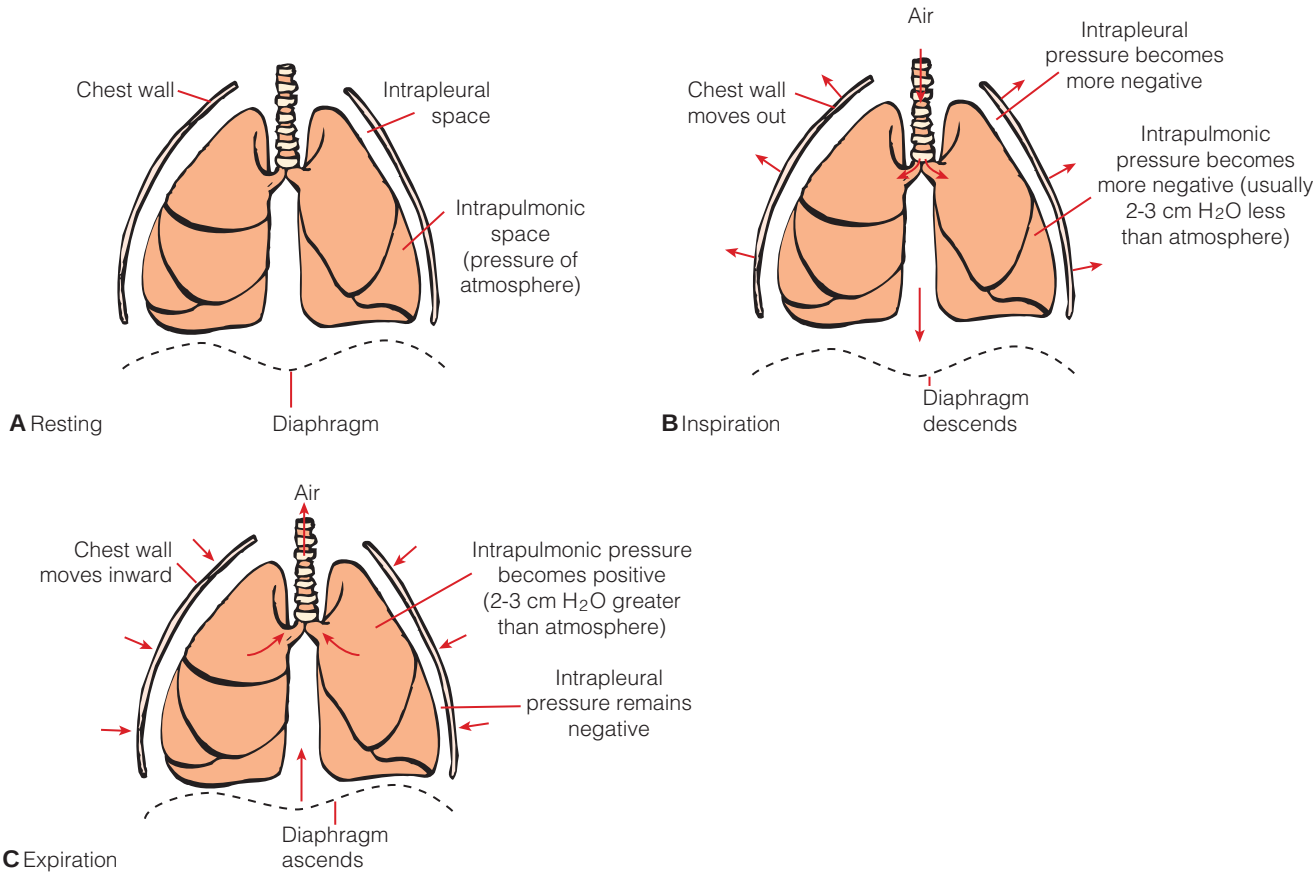


FIGURE 23-12 ▲ Phases of ventilation. **A:** No air movement (resting). **B:** Air moves from the environment to the intrapulmonic space (inspiration). **C:** Air moves from the intrapulmonic space to the environment (expiration).

AIRWAY RESISTANCE. Airflow in the conducting airways is affected not only by pressure differences between the atmosphere and alveoli but also by the resistance that air encounters as it moves through the airways. According to Poiseuille's law, the resistance to flow is inversely proportional to the fourth power of the radius ($R = 1/r^4$). If the radius of the tube that gas is flowing through is cut in half, the resistance is increased 16-fold ($2 \times 2 \times 2 \times 2 = 16$). In the respiratory airways, this means that small changes in airway diameter can have enormous effects on airflow resistance. Normally, airway resistance is so small that only small changes in pressure are needed to move large volumes of air into the lungs. But in conditions that decrease airway diameter, such as those caused by pulmonary secretions or bronchospasm, marked increases in airway resistance occur. To maintain the same rate of airflow as before the onset of increased airway resistance, people with these conditions must increase the driving pressure (or respiratory effort) to move air.

Assessment of Ventilation

Minute ventilation is the volume of air inhaled and exhaled per minute. It is calculated by multiplying tidal volume (VT) and respiratory rate. At rest, minute ventilation is approximately 7,500 mL/min.

Not all the air that enters the airways reaches the alveoli where gas exchange takes place. The part of VT that does not participate in alveolar gas exchange is called *dead space ventilation*. Dead space ventilation includes anatomical dead space volume and physiological dead space volume. Anatomical dead space is the amount of air in the conducting airways and is normally about 2 mL/kg, or about 150 mL.[7] Anatomical dead space depends on body posture and disease states. In certain disease states, such as chronic obstructive pulmonary disease (COPD), anatomical dead space is larger than normal. Physiological dead space occurs when ventilation is normal but perfusion to the alveoli is reduced or absent. This can occur with certain disease states, such as reduced cardiac output or pulmonary embolism. Dead space increases the partial pressure of arterial carbon dioxide ($PaCO_2$) because blood that is carrying carbon dioxide back from the tissues cannot reach the alveoli.

The *alveolar ventilation* is the volume of fresh gas entering the respiratory zone each minute. Alveolar ventilation is of key importance because it represents the amount of fresh inspired air available for gas exchange.[3] Alveolar ventilation is the minute ventilation minus dead space. It is inversely proportional to $PaCO_2$ levels. If one breathes excessively, alveolar ventilation is increased, and $PaCO_2$ decreases. If alveolar ventilation is decreased, $PaCO_2$ levels increase.

Pulmonary Volumes and Capacities

The flow of air in and out of the lungs provides tangible measures of lung volumes. Although referred to as "pulmonary function" measures, in reality, these volumes represent

BOX 23-1 CONSIDERATIONS FOR THE OLDER PATIENT

Anatomical and Physiological Changes in the Respiratory System That Occur With Aging

- The anterior–posterior diameter increases.
- Compliance is increased.
- Anatomical dead space is increased.
- The residual volume (RV) increases.
- Respiratory muscle strength decreases.
- The number of alveoli is reduced, resulting in decreased surface area for diffusion.
- Alveolar elasticity is decreased.
- Chest wall motility is decreased.
- The vital capacity (VC) decreases.
- Blood oxygen levels are decreased—subtract 1 mm Hg from a baseline arterial oxygen tension (PaO_2) of 80 mm Hg for every year over age 60.
- Anemia is common due to decreased hemoglobin and oxygen carrying capacity.

"pulmonary anatomy" measures. In the evaluation of ventilation, structure or anatomy often determines function.

Ventilatory or pulmonary function tests measure the ability of the chest and lungs to move air into and out of the alveoli. Pulmonary function tests include volume measurements, capacity measurements, and dynamic measurements. These measurements are influenced by exercise and disease. Age, sex, body size, and posture are other variables that are taken into consideration when the test results are interpreted. (For a summary of age-related changes affecting the anatomy and physiology of the respiratory system, see Box 23-1.) Figure 23-13 illustrates pulmonary function tests showing normal lung volumes and capacity. Volume measurements show the amount of air contained in the lungs during various parts of the respiratory cycle. Measures of lung volume include V_T, inspiratory reserve volume (IRV), expiratory reserve volume (ERV), and residual volume (RV), as shown in Table 23-1. Capacity measurements quantify a part of the pulmonary cycle. They are measured as a combination of the previous volumes and include inspiratory capacity (IC), functional residual capacity (FRC), vital capacity (VC), and total lung capacity (TLC; see Table 23-1).

The "Work" of Breathing

In normal quiet breathing, muscle contraction occurs during inspiration, and expiration is a passive process caused by elastic recoil of the lung. Thus, under normal resting conditions, muscle contraction (or work) is required only during inspiration. The work of inspiration can be divided into three categories: (1) work required to expand the lungs against lung and chest wall elastic forces, called *compliance work* or *elastic work*; (2) work required to overcome the viscosity of the lung and chest wall structures, called *tissue resistance work*; and (3) work required to overcome airway resistance during the movement of air into the lungs, called *airway resistance work*.[1] Normally during quiet respiration, only a small percentage of the total work is used to overcome tissue resistance, and a little more is used to overcome airway resistance; only 3% to 5% of the total energy expended by the body is required for ventilation. However, during heavy breathing when air must flow through the airways at a higher velocity, more work is used to overcome airway resistance.

All three types of work are frequently increased in pulmonary disease. Fibrosis of the lungs increases compliance work and tissue resistance work. Diseases that obstruct the airways increase airway resistance work. During heavy exercise, the amount of energy required can increase as much as 50-fold, especially if the person has any degree of increased airway resistance or decreased pulmonary compliance.[1]

Diffusion

After the alveoli are ventilated with fresh air, the next step in the respiratory process is diffusion of oxygen from the alveoli to the pulmonary capillaries and diffusion of carbon dioxide from the pulmonary capillaries to the alveoli. Diffusion, or movement of molecules, occurs from an area of high to low concentration. Fick's law describes the diffusion of gases through the alveolar–capillary membrane (Fig. 23-14). Fick's law states that the rate of transfer of gas through a semipermeable membrane is proportional to the tissue surface area and the difference in gas pressures between the two sides, and inversely proportional to the tissue thickness. Recall that the surface area of the alveoli is very large (50 to 100 m^2) and that the thickness of the alveolar membrane is 0.3 micrometers, so the dimensions of the blood–gas barrier are ideal for diffusion of gases.[8] Different gases also cross the barrier at different rates

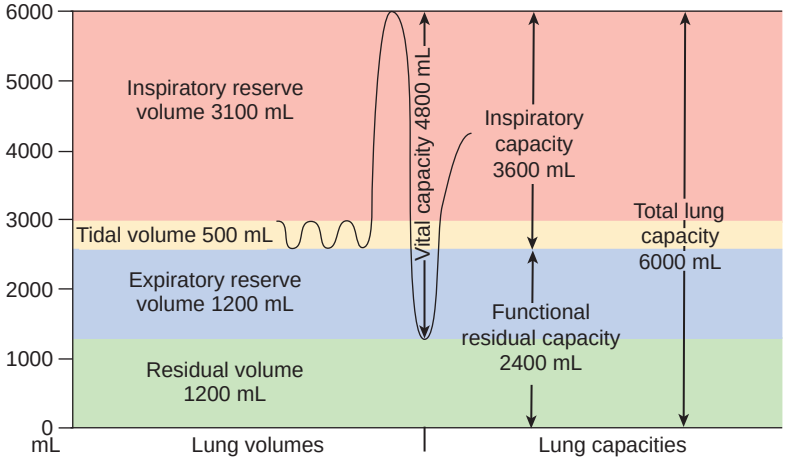


FIGURE 23-13 ▲ Tracings of the respiratory volumes (**left**) and lung capacities (**right**) as they would appear if made using a spirometer. The tidal volume (*yellow*) represents the amount of air inhaled and exhaled during normal breathing; the inspiratory reserve volume (*pink*), the maximal amount of air in excess of the tidal volume that be forcefully inhaled; the maximal expiratory reserved (*blue*), the maximal amount of air that can be exhaled in excess of the tidal volume; and the residual volume (*green*), the air that continues to remain in the lung after maximal expiratory effort. The inspiratory capacity represents the sum of the inspiratory reserve volume and the tidal volume; the functional residual capacity, the sum of the maximal expiratory reserve and residual volumes; and the total lung capacity, the sum of all the volumes. (From Porth CM: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 654.)

Table 23-1 Lung Volumes and Lung Capacities

Term Used	Symbol	Description	Remarks	Normal Values
Lung Volumes				
Tidal volume	V_T	Volume of air inhaled and exhaled with each breath	Tidal volume may not vary, even with severe disease.	500 mL
Inspiratory reserve volume	IRV	Maximum volume of air that can be inhaled after a normal inhalation		3,000 mL
Expiratory reserve volume	ERV	Maximum volume of air that can be exhaled forcibly after a normal exhalation	ERV is decreased with restrictive disorders, such as obesity, ascites, and pregnancy	1,100 mL
Residual volume	RV	Volume of air remaining in the lungs after a maximum exhalation	RV may be increased with obstructive diseases.	1,200 mL
Lung Capacities				
Vital capacity	VC	Maximum volume of air exhaled from the point of maximum inspiration	Decrease in VC may be found in neuromuscular disease, generalized fatigue, atelectasis, pulmonary edema, and COPD	4,600 mL
Inspiratory capacity	IC	Maximum volume of air inhaled after normal expiration	Decrease in IC may indicate restrictive disease.	3,500 mL
Functional residual capacity	FRC	Volume of air remaining in lungs after a normal expiration	FRC may be increased with COPD and decreased in ARDS.	2,300 mL
Total lung capacity	TLC	Volume of air in lungs after a maximum inspiration and equal to the sum of all four volumes (V_T, IRV, ERV, RV)	TLC may be decreased with restrictive disease (atelectasis, pneumonia) and increased in COPD.	5,800 mL

depending on their molecular characteristics. Carbon dioxide diffuses about 20 times more rapidly than oxygen. Thus, four factors affect alveolar–capillary gas exchange: (1) the surface area available for diffusion, (2) the thickness of the alveolar–capillary membrane, (3) the partial pressure of gas across the membrane, and (4) solubility and molecular characteristics of the gas (Table 23-2). Any condition or disease that affects one or more of these factors may impair diffusion of oxygen and carbon dioxide across the alveolar–capillary membrane.

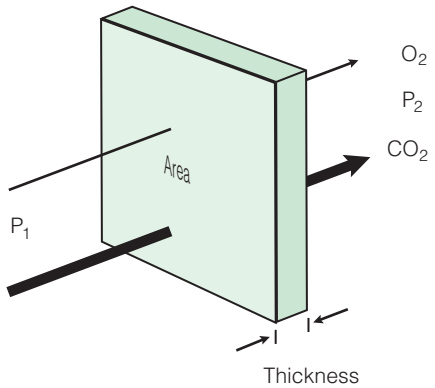


FIGURE 23-14 ▲ Fick's law describes diffusion through a tissue sheet. The amount of gas diffused is directly proportional to the surface area and the difference in partial pressures between the tissue sheets (P1 to P2). The amount of gas diffused is inversely proportional to the thickness of the tissue sheet. The molecular characteristics of carbon dioxide (CO_2) allow it to diffuse about 20 times more rapidly than oxygen (O_2). (From West JB: Respiratory Physiology: The Essentials, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, p 26.)

Perfusion

Once oxygen has diffused from the alveolus to the pulmonary capillary, it is carried away from the lung by the bloodstream. This gas exchange function of the lungs requires a constant flow of blood through the respiratory airways. The term *perfusion* is used to describe the flow of blood through the pulmonary capillary bed. As previously described in this chapter, pulmonary capillaries form a dense network around the alveolar wall, making an extremely efficient structure for gas exchange to take place (see Fig. 23-9, p. 474). The meshwork of the capillaries in the respiratory portion of the lungs is so dense that the flow in these vessels often is described as being similar to a "sheet" of blood.[4] When these blood vessels sense a low oxygen content in the alveoli, they vasoconstrict. The precise mechanism of this response, called *hypoxic vasoconstriction*, is not known.[9] Hypoxic vasoconstriction has the effect of directing blood flow away from hypoxic areas of the lung. By diverting blood flow from these areas, the deleterious effects on gas exchange are reduced.

Relationship of Ventilation to Perfusion

Distribution of Ventilation

Not all areas of the lung have the same ventilation. Body position affects distribution of ventilation. In a seated or standing position, lower regions of the lung ventilate better than upper zones. In a supine position, the apex and base of the lung ventilate about the same; however, ventilation in the lowermost (posterior) lung is greater than that of the uppermost (anterior) lung. In a lateral position, the dependent lung is best ventilated.[3]

Table 23-2 Factors Affecting Alveolar–Capillary Gas Exchange

Factors Affecting Gas Exchange	Examples
Surface area available for diffusion	Removal of a lung or diseases, such as emphysema and chronic bronchitis, which destroy lung tissue or cause mismatching of ventilation and perfusion
Thickness of the alveolar–capillary membrane	Conditions, such as pneumonia, interstitial lung disease, and pulmonary edema, which increase membrane thickness
Partial pressure of alveolar gas	Ascent to high altitudes where the partial pressure of oxygen is reduced. In the opposite direction, increasing the partial pressure of a gas in the inspired air (eg, oxygen therapy) increases the gradient for diffusion.
Solubility and molecular weight of the gas	Carbon dioxide, which is more soluble in the cell membranes, diffuses across the alveolar–capillary membrane more rapidly than oxygen

From Porth CM: Pathophysiology: Concepts of Altered Health States, 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2005, p 650.

Distribution of Perfusion

As with ventilation, the distribution of pulmonary blood flow is affected by body position and gravity. For a person in the upright position, blood flow is better at the base of the lungs than the apex of the lungs. For a person in a supine position, the blood flow from apex to base is almost uniform, but blood flow in the posterior (dependent) regions of the lung exceeds that of the anterior regions. For a person in the prone position, the same holds true: blood flow in the dependent region (now the anterior chest) exceeds that of the posterior chest.

Considerable inequality of blood flow exists within the human lung (Fig. 23-15). The uneven distribution of blood flow can be explained by the hydrostatic pressure differences in the blood vessels. In zone 1, alveolar pressures exceed pulmonary arterial and pulmonary venous pressures. The capillaries are basically squashed flat by the pressure in the alveoli, and there is no blood flow. In zone 2, pulmonary arterial pressures are greater than alveolar pressures, so some blood flow occurs. Blood flow here is determined by the differences in arterial and alveolar pressures. In zone 3, there is minimal alveolar pressure influence on the pulmonary vasculature, and blood flow is determined in the usual way by the arteriovenous pressure difference.

Matching of Ventilation to Perfusion

Effective pulmonary gas exchange depends on a balance or matching of ventilation to perfusion (Fig. 23-16A). Two factors may interfere with the matching of ventilation to perfusion: dead space and shunt. As previously described in this chapter, dead space refers to areas in the respiratory system that do not participate in gas exchange. The air in the conducting airways (about 150 mL) does not participate in gas exchange and is referred to as *anatomical dead space*. Anatomical dead space increases with intubation. In zone 1 of the lung, the region is ventilated but not perfused, and this is referred to as *alveolar dead space*. Other areas of the lung may also contain alveolar dead space, such as that which occurs with collapsed alveoli from atelectasis or pneumonia. Shunt refers to blood that bypasses, or shunts by, alveoli without picking up oxygen. There are two types of shunts: anatomical and physiological. With an anatomical shunt, blood moves from the right side to the left side of the heart without passing through the lungs. Anatomical shunts occur with congenital heart diseases. With a physiological shunt, blood is shunted past alveoli without picking up sufficient amounts of oxygen.

A ventilation–perfusion imbalance, known as a ventilation–perfusion mismatch, occurs when there is

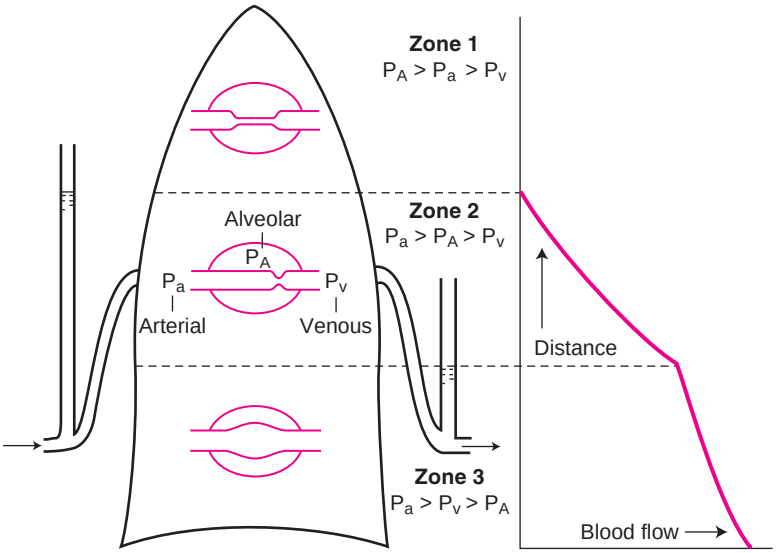


FIGURE 23-15 ▲ Explanation of the uneven distribution of blood flow in the lung, based on the pressures affecting the capillaries. P_A, alveolar pressure; Pa, arterial pressure; Pv, venous pressure. (From West JB: Respiratory Physiology: The Essentials, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, p 44.)

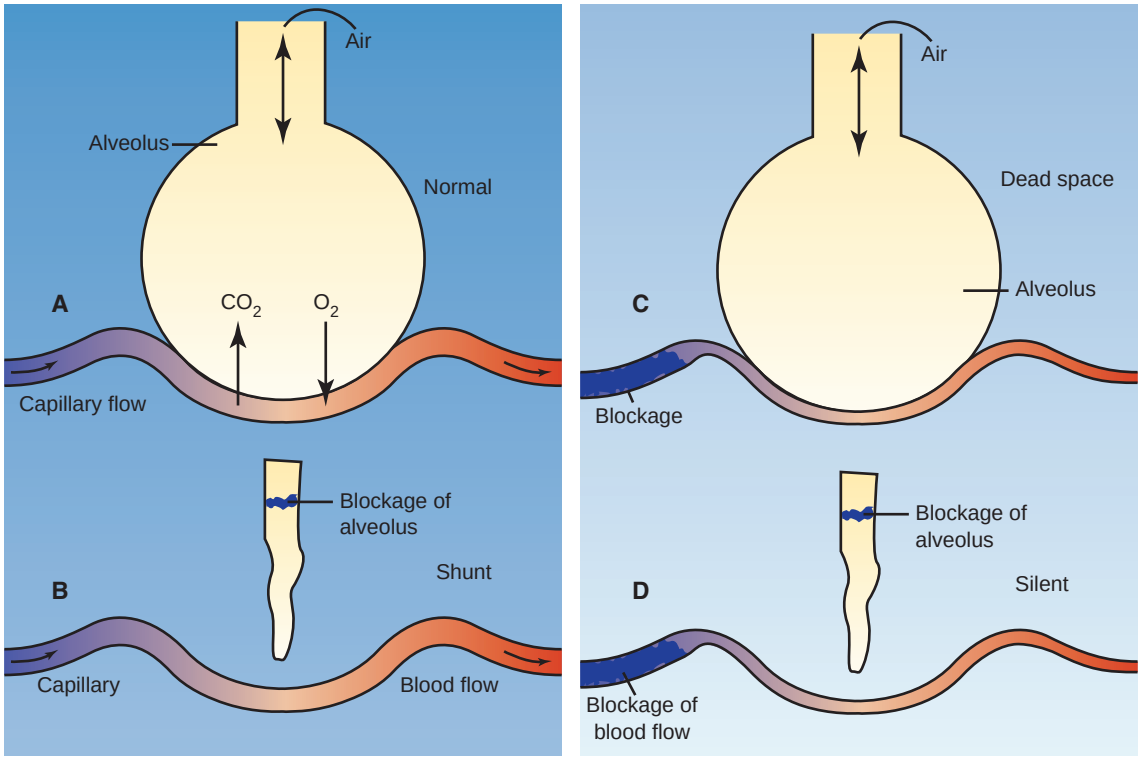


FIGURE 23-16 ▲ A schematic representation of various ventilation–perfusion situations. **A:** Normal unit with normal ventilation and normal perfusion. **B:** Low-ventilation/perfusion ratio—alveoli with no ventilation but normal perfusion. **C:** High-ventilation/perfusion ratio—alveoli with normal ventilation but no perfusion. **D:** Silent unit—alveoli with no ventilation and no perfusion. CO_2, carbon dioxide; O_2, oxygen. (From Smeltzer SC, Bare BG: Brunner and Suddarth's Textbook of Medical Surgical Nursing, 12th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 492.)

inadequate ventilation, inadequate perfusion, or both. Three types of ventilation–perfusion imbalances may occur:

- *Physiological shunt* (low ventilation/perfusion ratio). When perfusion exceeds ventilation, the ratio is low, and a shunt is present. A shunt means that blood passes by alveoli without gas exchange occurring. A low ventilation–perfusion ratio is seen with pneumonia, atelectasis, tumor, or a mucous plug (see Fig. 23-16B).
- *Alveolar dead space* (high ventilation/perfusion ratio). When ventilation exceeds perfusion, the ratio is high and an alveolar dead space develops. The alveolus has inadequate perfusion available, and gas exchange cannot occur. A high ventilation/perfusion ratio is seen with a pulmonary embolus, pulmonary infarction, cardiogenic shock, and mechanical ventilation associated with high tidal volumes (see Fig. 23-16C).
- *Silent unit*. When both ventilation and perfusion are decreased, a silent unit occurs. A silent unit is seen with pneumothorax and severe ARDS (see Fig. 23-16D).

Gas Transport

Oxygen

Oxygen is carried in the blood in two forms: dissolved and attached to hemoglobin. The partial pressure of oxygen in arterial blood (PaO_2) represents the level of dissolved oxygen in plasma. Less than 3% of all oxygen is carried in this form, whereas 97% of oxygen carried in the blood is bound to hemoglobin and is called *oxyhemoglobin*. Each gram of hemoglobin carries approximately 1.34 mL of oxygen when it is completely saturated. As oxygen diffuses across the alveolar–capillary membrane, it combines with hemoglobin in the red blood cell where it forms a reversible bond. Oxyhemoglobin is transported in arterial blood and made available to the tissues for use in cell metabolism. The saturation of oxygen in arterial blood (SaO_2) represents the percentage of hemoglobin molecules that are bound with oxygen.

The hemoglobin molecule is said to be fully saturated when oxygen is bound to all four of its oxygen-binding sites and only partially saturated when less than four molecules are bound to it. The term *affinity* is used to refer to the capacity of hemoglobin to combine with oxygen. When the affinity is high, hemoglobin binds readily with oxygen at the alveolar–capillary membrane. But, at the tissue level, hemoglobin does not readily release oxygen. When the affinity is low, hemoglobin does not bind readily with oxygen at the alveolar–capillary membrane. Instead, when affinity is low, hemoglobin releases oxygen more readily at the tissue level. The affinity of hemoglobin and oxygen is described by the oxyhemoglobin dissociation curve (Fig. 23-17).

The oxyhemoglobin dissociation curve is a graphic depiction of the relationship between oxyhemoglobin saturation (the percentage of hemoglobin combined with oxygen or the SaO_2) and the arterial oxygen tension (PaO_2) to which it is exposed. The initial part of the curve is very steep and then flattens at the top. The flat portion represents the binding of

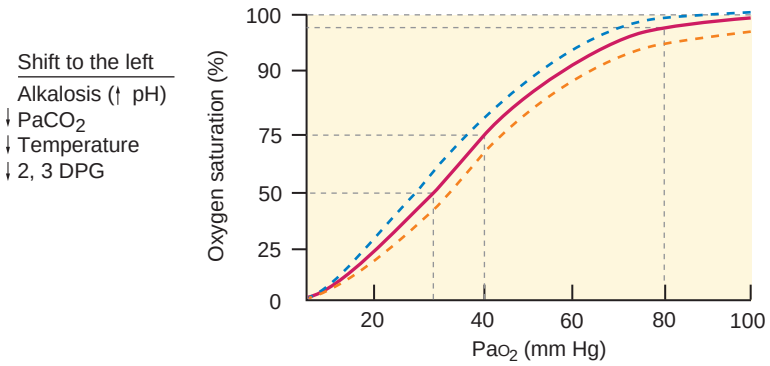


FIGURE 23-17 ▲ Oxyhemoglobin dissociation curve. The shift to the left indicates a higher oxygen saturation at any given arterial oxygen tension (PaO_2), an increased affinity of hemoglobin for oxygen, and a decreased release of oxygen to the tissues. A shift to the right indicates a lower oxygen saturation at any given PaO_2, a decreased affinity of hemoglobin for oxygen, and an increased release of oxygen to the tissues.

oxygen to hemoglobin in the lungs. The steep portion of the curve (between 40 and 60 mm Hg) represents the release of oxygen from the hemoglobin that occurs in the capillaries. At an arterial oxygen tension (PaO_2) of 40 mm Hg, hemoglobin molecules are still about 70% to 75% saturated with oxygen. This provides a reserve supply of oxygen that can be given to the tissues in cases of emergency or strenuous exercise.

Hemoglobin's affinity for oxygen is influenced by pH, carbon dioxide concentration, temperature, and 2,3-diphosphoglycerate (2,3-DPG). 2,3-DPG is a metabolically important phosphate compound found in the blood in different combinations under different metabolic conditions.[10] Hemoglobin binds more readily with oxygen under conditions of increased pH, decreased carbon dioxide, decreased body temperature, and decreased 2,3-DPG. This is represented on the oxyhemoglobin dissociation curve as a shift to the left (see Fig. 23-17). With a shift to the left, there is higher oxygen saturation for any given PaO_2, increased affinity of hemoglobin for oxygen, and decreased release of oxygen to tissues. Hemoglobin more readily releases oxygen under conditions of decreased pH, increased carbon dioxide, increased body temperature, and increased 2,3-DPG. This relationship is represented on the curve by a shift to the right (see Fig. 23-17). With a shift to the right, there is lower oxygen saturation for any given PaO_2, decreased affinity of hemoglobin for oxygen, and increased release of oxygen to the tissues.

Carbon Dioxide

Carbon dioxide is carried in the blood in three forms: as dissolved carbon dioxide (10%), attached to hemoglobin (30%), and as bicarbonate (60%).[4] Carbon dioxide is formed as a metabolic byproduct. It diffuses out of the cell and into the capillaries. Most of it diffuses into red blood cells, where it attaches to hemoglobin, and most of that is released from the red blood cell as bicarbonate. In the pulmonary capillaries, the concentration of carbon dioxide is greater in the capillaries than in the alveoli, so the carbon dioxide moves down this concentration gradient and diffuses into the alveoli and is exhaled. An increased rate of exhalation leads to greater elimination of carbon dioxide. The transport of carbon dioxide has a profound effect on the acid–base status of the blood and the body as a whole. The lung excretes more than 10,000 mEq of carbonic acid per day, compared with the kidney, which excretes less than 100 mEq of fixed acids per day.[11] Therefore, by altering alveolar ventilation (and subsequently, the elimination of carbon dioxide), the body is able to exert precise control over its acid–base balance.

Regulation of Respiration

Breathing is controlled by both the nervous system and chemical regulation. Nervous system regulation is achieved by the respiratory centers, which are located in the medulla and pons (ie, the brainstem). Chemical regulation of breathing occurs through chemoreceptors, which respond to blood pH and the levels of oxygen and carbon dioxide in the blood. Chemoreceptors are located near the respiratory center in the medulla, in the carotid arteries, and in the aortic arch.

Brainstem Centers and the Respiratory Cycle

Unlike the heart, the lungs have no spontaneous rhythm. Ventilation depends on rhythmic operation of brainstem centers and intact pathways to the respiratory muscles. There are two centers in the medulla: a center that stimulates inspiration by diaphragmatic contraction (by way of phrenic nerves) and another center that innervates both inspiratory and expiratory intercostal and accessory muscles (Fig. 23-18). The pons also contains two centers involved in controlling respiration: the pneumotaxic center and the apneustic center. The apneustic center produces sustained inspiration if stimulated. Voluntary control and involuntary control are further established by descending fibers from other brain centers. Neural control of ventilation is illustrated in Figure 23-18. In breathing at rest, the following sequence is thought to occur. The neurons innervating the inspiratory muscles fire bursts of impulses to these muscles, leading to inspiration. These neurons also stimulate the pneumotaxic center. This center, in turn, fires inhibitory impulses back to the inspiratory neurons, causing a halt in inspiration. Expiration follows passively. After expiration, the inspiratory neurons are again stimulated to fire automatically. During exercise or other occasions when more vigorous ventilation occurs, the expiratory neurons of the medulla are postulated to participate in this sequence, causing active exhalation.

Chemoreceptors

Chemoreceptors are like radar screens planted in the body to monitor blood levels of carbon dioxide and oxygen. Signals from these receptors are transmitted to the respiratory center, and ventilation is adjusted to maintain these gases in a normal range. There are two types of chemoreceptors: central chemoreceptors and peripheral chemoreceptors.

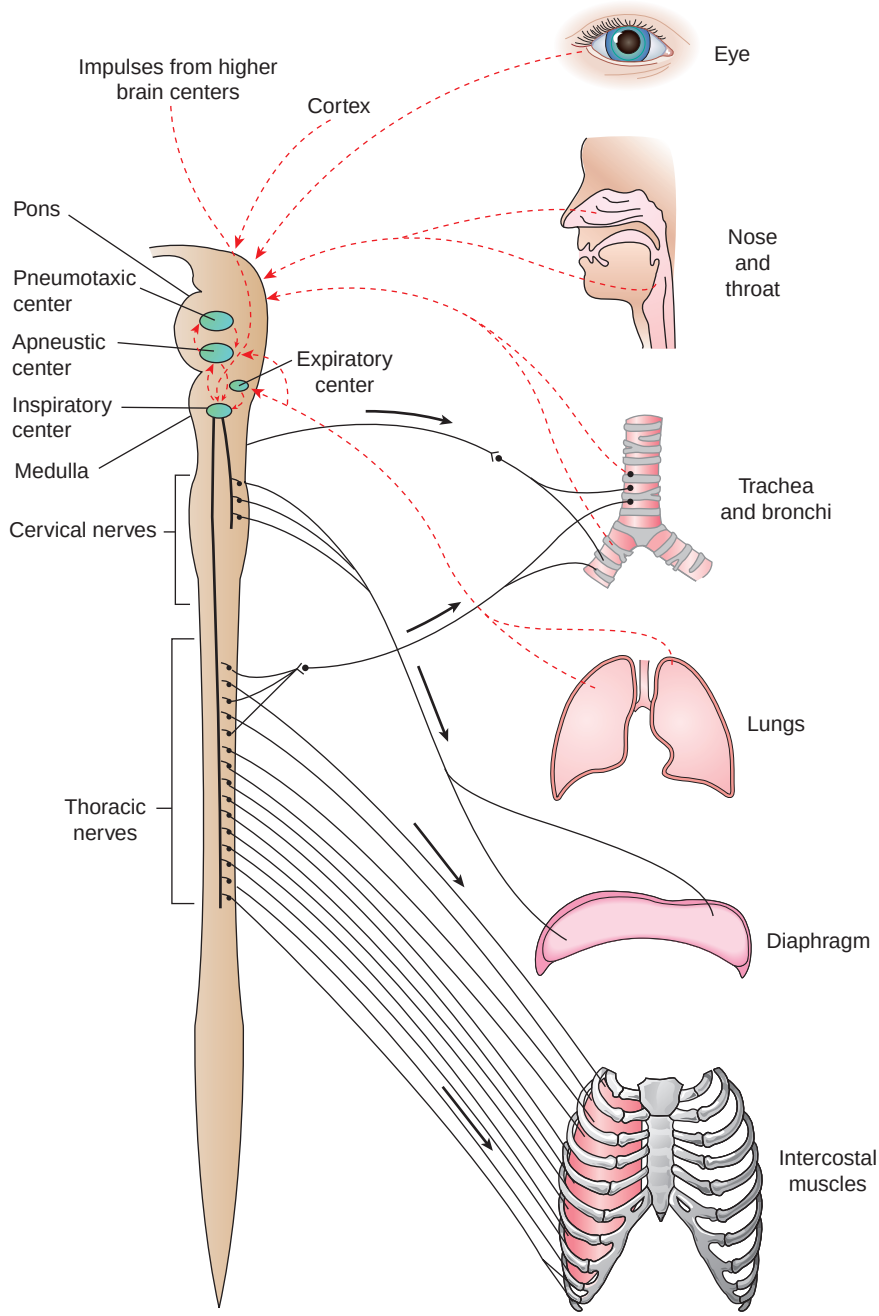


FIGURE 23-18 ▲ Schematic representation of activity in the respiratory center. Impulses traveling over afferent neurons activate central neurons, which activate efferent neurons that supply the muscles of respiration. Respiratory movements can be altered by a variety of stimuli.

Central chemoreceptors sense changes in carbon dioxide content. They are located near the respiratory center in the medulla and are in close contact with cerebrospinal fluid (CSF). Carbon dioxide freely diffuses across the blood–brain barrier into the CSF. As the level of carbon dioxide in the CSF increases and the pH decreases, the nearby respiratory center is stimulated to increase respirations to "blow off" more carbon dioxide.

Peripheral chemoreceptors are located in the arch of the aorta and in the carotid arteries. These chemoreceptors are sensitive to changes in oxygen content in arterial blood. These receptors exert little control over respirations until the PaO_2 is below 60 mm Hg.[4] When this occurs, the respiratory center is stimulated to increase the rate and depth of respirations to inhale more oxygen.

Lung Receptors

Recall the importance of airway resistance and lung expansion in the respiratory process. Lung and chest wall receptors provide information to the respiratory center on the status of these processes. There are three types of lung receptors: stretch, irritant, and juxtacapillary receptors. Stretch receptors, located in the smooth muscle layers of the conducting airways, respond to pressure changes in the airways. When the lungs are fully inflated, they inhibit further inspiration and trigger exhalation. These receptors are important because they establish respiratory patterns by adjusting respiratory rate and tidal volume in an attempt to respond to changes in airway resistance and lung compliance.

Irritant receptors, located in the airways, are stimulated by inhaled dust, smoke, chemicals, and cold air. Stimulation of these receptors triggers airway constriction and more rapid, shallow breathing. It is possible that these receptors play a key role in the bronchoconstriction that occurs with asthma.[4]

Juxtacapillary receptors are located in the alveolar wall close to the pulmonary capillaries. These receptors sense lung congestion. It may be stimulation of these receptors that produces the rapid, shallow breathing that is characteristic in patients with pneumonia and pulmonary edema.

▲ Clinical Applicability Challenges

SHORT ANSWER QUESTIONS

1. Discuss the process of inspiration and exhalation.
2. What are the four factors that affect the alveolar-capillary gas exchange?
3. Discuss the oxyhemoglobin saturation curve. What does a shift to the left indicate? What does a shift to the right indicate? Identify the four factors that are responsible for the shift in the oxyhemoglobin saturation curve.

References

1. Guyton AC, Hall JE: Pulmonary ventilation. In Guyton AC, Hall JE (eds): Textbook of Medical Physiology, 12th ed. Philadelphia, PA: Saunders Elsevier, 2011, pp 465–475
2. West JB: Mechanics of breathing—how the lung is supported and moved. In West JB (ed): Respiratory Physiology: The Essentials, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, pp 95–122
3. West JB: Ventilation—how gas gets to the alveoli. In West JB (ed): Respiratory Physiology: The Essentials, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, pp 13–23
4. Porth CB, Litwack K: Structure and function of the respiratory system. In Porth CB (ed): Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, pp 640–669
5. West JB: Structure and function—how the architecture of the lung subserves its function. In West JB (ed): Respiratory Physiology: The Essentials, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, pp 1–11
6. Smeltzer SC, Bare BG, Hinkle JL, et al: Gas exchange and respiratory function. In Smeltzner SC, Bare BG, Hinkle JL, et al (eds): Brunner & Suddarth's Textbooks of Medical-Surgical Nursing, 11th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, pp 552–587
7. Pierce NLP: Practical physiology of the pulmonary system. In Pierce LNB (ed): Management of the Mechanically Ventilated Patient. Philadelphia, PA: Lippincott Williams & Wilkins, 2007, pp 26–60
8. West JB: Diffusion—how gas gets across the blood–gas barrier. In West JB (ed): Respiratory Physiology: The Essentials, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, pp 25–34
9. West JB: Blood flow and metabolism—how the pulmonary circulation removes gas from the lungs and alters some metabolites. In West JB (ed): Respiratory Physiology: The Essentials, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, pp 35–53
10. Guyton AC, Hall JE: Transport of oxygen and carbon dioxide in blood and tissue fluids. In Guyton AC, Hall JE (eds): Textbook of Medical Physiology, 12th ed. Philadelphia, PA: Saunders Elsevier, 2011, pp 495–504
11. West JB: Gas transport by the blood—how gases are moved to the peripheral tissue. In West JB (ed): Respiratory Physiology: The Essentials, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, pp 75–93

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

24 Patient Assessment: Respiratory System

Patricia Gonce Morton and Kenneth J. Rempher

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Describe the components of the history for respiratory assessment.
2. Explain the use of inspection, palpation, percussion, and auscultation for respiratory assessment.
3. Discuss the purpose of pulse oximetry and end-tidal carbon dioxide monitoring.
4. Explain the components of an arterial blood gas and the normal values for each component.
5. Compare and contrast the causes, signs, and symptoms of respiratory acidosis, respiratory alkalosis, metabolic acidosis, and metabolic alkalosis.
6. Analyze examples of an arterial blood gas result.
7. Describe the purpose of mixed venous oxygen saturation monitoring.
8. Discuss the purpose of respiratory diagnostic studies and associated nursing implications.

Nurses contribute significantly to the care of patients with respiratory problems by taking a comprehensive history and performing a thorough physical examination. This information allows the nurse to establish a baseline level of assessment of the patient's status and provides a framework for detecting rapid changes in the patient's condition. Assessments are valuable if made before, during, and after interventions that are likely to alter or improve respiratory status. Because the nurse is with the patient more consistently than most other health care professionals, it is often the nurse who detects the patient's changing condition. High-quality assessments often uncover complications or changes that precede the information provided by other diagnostic tests.

▲ History

A thorough review of the patient's clinical history is an essential component of the overall physical assessment process. A properly conducted review of the patient's clinical history should serve as a guide for the remainder of the physical examination. In many cases, obtaining a clinical history is the first step in developing a relationship with the patient. Patients often suppress information or underreport personal experiences that may be essential in identifying the underlying cause of illness. As a result, constant subjective evaluation of the patient's report should also serve to guide the clinical history. The interviewer must conduct the examination in such a way that the patient feels as comfortable as possible.

The clinical history of the respiratory system is divided into six components: (1) chief complaint, (2) history of present illness, (3) past health history, (4) family history, (5) personal and social history, and (6) review of systems (Box 24-1). The patient's history starts with the chief complaint and information about the present illness. Often, if the patient is very ill, a relative or friend provides more information. Data about the present illness and any symptoms are thoroughly investigated using the mnemonic NOPQRST: normal (N), onset (O), precipitating and palliative factors (P), quality and quantity (Q), region and radiation (R), severity (S), and time (T), as described in Box 17-1. Principal symptoms that should be investigated in more detail commonly include dyspnea, chest pain, sputum production, and cough. An overview of the patient's past medical history and family's respiratory history, as well as personal and social history, may uncover elements that are contributing to the patient's current health problem. Because smoking has a significant impact on the patient's respiratory health, the patient's use of tobacco should be quantified by amount and how long the patient has smoked. Box 24-2 demonstrates the process for calculating this quantity, which is known as pack years.

Dyspnea

Dyspnea is commonly seen in patients with pulmonary or cardiac compromise. Information about the onset of symptoms

BOX 24–1 HEALTH HISTORY for Respiratory Assessment

Chief Complaint
- Patient's description of the problem

History of the Present Illness
- Complete analysis of the following signs and symptoms (using the NOPQRST format; see Box 17-1, p. 207).
- Dyspnea, dyspnea on exertion
- Shortness of breath
- Chest pain
- Cough
- Sputum production
- Hemoptysis
- Wheezing
- Orthopnea
- Clubbing
- Fatigue
- Cyanosis

Past Health History
- Relevant childhood illnesses and immunizations: whooping cough (pertussis), mumps, cystic fibrosis
- Past acute and chronic medical problems, including treatments and hospitalizations: streptococcal infection of the throat, upper respiratory infections, tonsillitis, bronchitis, sinus infection, emphysema, asthma, bronchiectasis, tuberculosis, cancer, pulmonary hypertension, heart failure, musculoskeletal and neurological diseases affecting the respiratory system
- Risk factors: age, obesity, smoking, environmental exposure such as asbestos, coal dust, chemicals, poison gas/vapors, dust, allergens
- Past surgeries: tonsillectomy, thoracic surgery, coronary artery bypass surgery, cardiac valve surgery, aortic aneurysm surgery, trauma surgery, tracheostomy
- Past diagnostic tests and interventions: tuberculin skin test, allergy tests, pulmonary function tests, chest radiograph, computed tomography scan, magnetic resonance imaging, bronchoscopy, cardiac stress test, ventilation–perfusion scanning, pulmonary angiography, thoracentesis, sputum culture
- Medications: use of oxygen, bronchodilators, antitussives, expectorants, mucolytics, antiinfectives, antihistamines, methylxanthine drugs, antiinflammatory drugs
- Allergies and reactions
- Transfusions

Family History
- Health status or cause of death of parents and siblings: tuberculosis, cystic fibrosis, emphysema, asthma, malignancy

Personal and Social History
- Tobacco, alcohol, and substance use
- Family composition
- Occupation and work environment: asbestos, chemical, and coal dust exposure
- Living environment: exposure to allergens and toxic substances, type of heating and ventilation system
- Diet
- Sleep patterns: use of pillows
- Exercise
- Cultural beliefs
- Spiritual, religious beliefs
- Coping patterns and social support systems
- Leisure activities
- Sexual activity
- Recent travel

Review of Systems
- HEENT: strep throat, sinus infections, ear infection, deviated nasal septum, tonsillitis
- Cardiac: heart failure, dysrhythmias, coronary artery disease, valvular disease, hypertension
- Gastrointestinal: weight loss, nausea, vomiting
- Neuromuscular: Guillain-Barré syndrome, myasthenia gravis, amyotrophic lateral sclerosis, weakness
- Musculoskeletal: scoliosis, kyphosis

gives clues as to the source and duration of the problem. The nurse asks questions such as the following:

- Does the dyspnea occur when the patient is lying flat (therefore requiring the patient to sit up, as is seen more commonly in heart failure)?
- Does the dyspnea awaken the patient at night (paroxysmal nocturnal dyspnea)?
- Does the dyspnea occur only with exertion?

Paroxysmal nocturnal dyspnea and orthopnea often signify heart failure but may occur in a variety of pulmonary disorders. Description of the entire course of dyspnea, including exacerbating factors, length of episodes, and any relief measures attempted, is warranted.

BOX 24-2 Steps for Calculating Pack Years

Pack years = (Number of packs smoked per day) × (Number of years smoking)
Example: The patient reports during the physical assessment that he has smoked two packs per day for 15 years.
(2 packs per day) × (15 years) = 30 pack years

Chest Pain

Dyspnea that occurs with primary lung disease is associated with an anterior chest discomfort that must be distinguished from angina. First, the nurse determines whether the patient experiences more than one type of pain. For each type of chest pain, the nurse asks the patient to describe the pain using the mnemonic NOPQRST. The detailed information obtained from using the mnemonic is key to determining the cause of the pain.

Sputum Production

A pulmonary illness often results in the production (or a change in the production) of sputum. The nurse questions the patient about the amount (eg, tablespoonful, one-half cup) and color of the sputum produced in 24 hours. The color of the sputum provides important information about infection. An increase in either the color or the amount of sputum often means infection. Yellow, green, or brown sputum typically signifies bacterial infection; clear or white sputum may signify absence of bacterial infection. The color comes from

white blood cells in the sputum. However, a yellow color may occur if there are many eosinophils in the sputum, thereby signifying allergy rather than infection. Rust-colored sputum (yellow sputum mixed with blood) may signify tuberculosis. Mucoid, viscid, or blood-streaked sputum is often a sign of a viral infection. Persistent slightly blood-streaked sputum is present in patients with carcinoma. Large amounts of clotted blood are present in the sputum of patients who have suffered a pulmonary infarction.

Occasionally, coughing does not yield sputum. Sometimes, the patient with an infection is unable to cough up sputum. For example, a decrease in sputum production associated with worsening hypoxemia may signify bronchiolitis. A cough without sputum production usually means that the problem is not bacterial in origin.

It is important to know whether the sputum comes from the nose, the chest, or sinus postnasal drainage. Chronic sputum production may indicate chronic obstructive pulmonary disease (COPD).

Sometimes, the patient is afraid to mention that there has been blood in the sputum; it is essential to ask the patient, family members, and care givers about the presence of blood. The amount of blood should be evaluated. Was it just streaks or specks, blood-colored mucus, or pure blood (bright red or dark)? Through careful questioning, the nurse determines whether the blood is associated with retching and vomiting or sputum production, as it often is in bronchitis and pneumonia, or whether it occurs alone, as is often true with a pulmonary embolus.

Cough

A cough is a frequent respiratory symptom with varying significance. External agents, inflammation of the respiratory mucosa, or pressure on an airway caused by a tumor may stimulate a cough. Specifically, smoking, allergies, heartburn, asthma, and certain medications, including angiotensin-converting enzyme inhibitors and β-blockers, may cause a cough.

▲ Physical Examination

Physical assessment of the respiratory system is a reliable means of gathering essential data and is guided by the information obtained through the history. A thorough physical assessment includes inspection, palpation, percussion, and auscultation.

Inspection

Inspection of the patient involves checking for the presence or absence of several factors (Box 24-3).

Cyanosis refers to a bluish discoloration of the skin or mucous membranes. Cyanosis is notoriously difficult to detect in a patient with anemia. The patient with polycythemia may have cyanosis in the extremities even if oxygen tension is normal. Peripheral cyanosis occurs in the extremities or on the tip of the nose or ears. Even with normal oxygen tensions, peripheral cyanosis may appear if there is diminished blood flow to these areas, particularly if they are cold or in a dependent position. Central cyanosis is apparent on the tongue or lips and usually means the patient has low oxygen tension. Unfortunately, the presence of cyanosis is a late and often ominous sign.

Labored breathing is an important marker of respiratory distress. As part of the inspection, the nurse determines whether the patient is using the accessory muscles of respiration (the scalene and sternocleidomastoid muscles). *Intercostal retractions* (ie, sucking in of the muscles and skin between the ribs during inspiration) usually mean that the patient is making a larger effort at inspiration than normal. The nurse also observes the patient for use of the abdominal muscles during the usually passive expiratory phase.

BOX 24-3 Components of the Inspection Process in the Physical Assessment of the Respiratory System

General
- Mentation
- Anxiety level
- Speech
  - Staccato
  - Coherence
  - Aphasia
  - Articulation
  - Hoarseness
- Skin turgor
- Skin integrity
  - Scars
  - Rash
  - Wounds
- Skin color
  - Pallor
  - Cyanosis
- Weight
  - Obese
  - Malnourished
- Body position
  - Leaning forward
  - Arms elevated

Thorax
- Symmetry of thorax
- Position of sternum
- Anteroposterior diameter less than transverse by at least half
- Rate, pattern, rhythm, and duration of breathing
- Use of accessory muscles
- Synchrony of chest and abdomen movement
- Alignment of spine
- Supernumerary nipples
- Superficial venous patterns

Head and Neck
- Nasal flaring
- Pursed-lip breathing
- Mouth breathing versus nose
- Use of neck and shoulders
- Tracheal position

Extremities
- Clubbing
- Edema
- Peripheral cyanosis

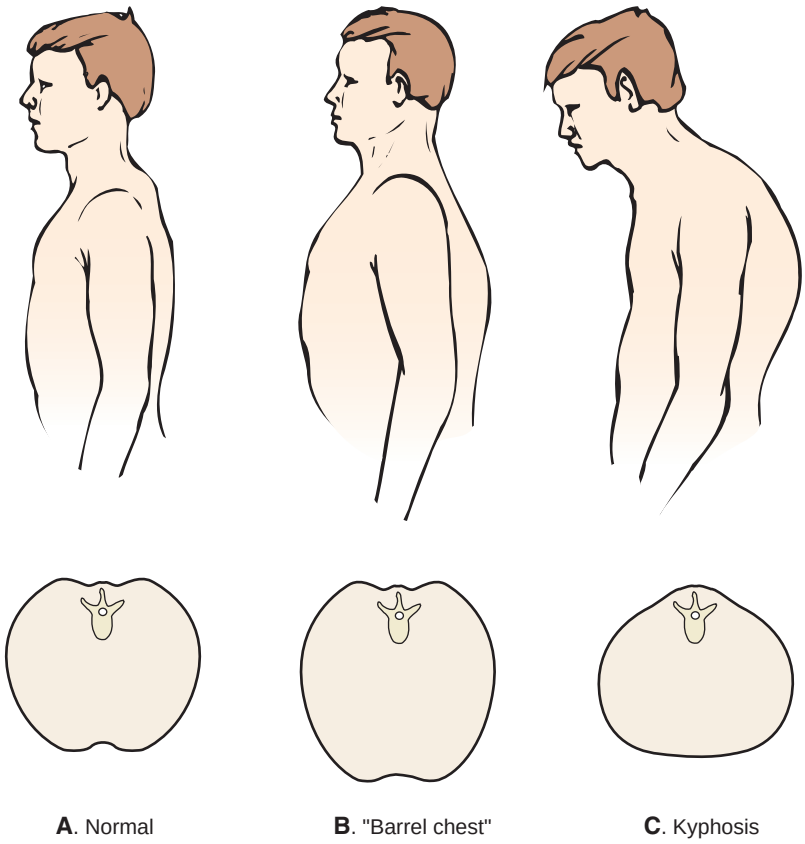


FIGURE 24-1 ▲ Deformities and configurations of the human thorax. **A:** Normal chest. **B:** "Barrel chest," a chest deformity that typically results from emphysema. **C:** Kyphosis, a chest deformity that is most common in older adults.

Staccato speech, in which the patient's speech pattern is frequently interrupted as he or she gulps for air, may accompany labored breathing. Sometimes, the number of words a patient can say before having to gasp for another breath is a good measure of the degree of labored breathing.

The *anterior–posterior diameter of the chest* (ie, the size of the chest from front to back) is also checked (Fig. 24-1). Often, the cause of an increased anterior–posterior diameter is overexpansion of the lungs from obstructive pulmonary disease. Patients with kyphosis (curvature of the spine) may also have an increase in anterior–posterior diameter.

Chest deformities and scars are important in helping to determine the reason for respiratory distress. A chest deformity, such as kyphoscoliosis or flail chest from trauma, may indicate why the patient has respiratory distress. A scar may signify recent or old injuries to the chest and provides clues to possible sources of distress. For example, evidence of recent trauma to the chest, such as a stabbing or compression injuries from an automobile collision, could be responsible for the present distress.

Observation of the *patient's posture* is necessary. Patients with obstructive pulmonary disease often sit and prop themselves up on outstretched arms or lean forward with their elbows on a table in an effort to elevate their clavicles. This posture gives the patient a slightly greater ability to expand the chest.

It is important to observe the *position of the trachea*. The nurse determines whether the trachea is in the midline as it should be or if it is deviated off to one side. Pleural effusion, hemothorax, pneumothorax, or a tension pneumothorax can deviate the trachea away from the affected side (toward the opposite side). However, atelectasis, fibrosis, and phrenic nerve paralysis often pull the trachea toward the affected side.

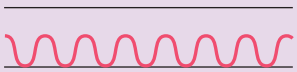
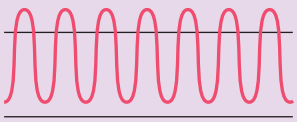
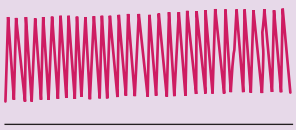
The *respiratory rate* is an important parameter to follow. It should be counted over at least a 15-second period for stable patients and over a full minute for critically ill patients. The patient's rate must be compared with his or her usual rate. Breathing 24 to 26 times a minute may be normal in one patient but abnormal in another. The patient's family or friends may provide additional important information about the patient's usual rate of breathing.

The *respiratory effort* is often as meaningful as the respiratory rate. For instance, if a patient is breathing 40 times per minute, the nurse might think a severe respiratory problem is the source of patient distress. However, the rate may be the result of Kussmaul respirations caused by diabetic acidosis. If a patient's respiration is shallow at a rate of 40 breaths/min (tachypnea), the indication may be severe respiratory distress from a primary pulmonary problem. Deep, rapid respirations, known as hyperventilation, may indicate compensation for acidosis. The pattern of respirations also should be noted because it may correlate with various disease processes. Table 24-1 provides a description of respiratory patterns and their clinical implications.

The *duration of inspiration versus the duration of expiration* helps determine the presence of obstructive lung disease. In patients with any of the obstructive lung diseases, expiration is more than one and a half times as long as inspiration.

Observation of *thoracic expansion* is an integral part of examining a patient. Normally, chest expansion of about 3 inches occurs from maximal expiration to maximal inspiration. Motion of the abdomen in breathing efforts (more likely to be normal in men than women) may be

Table 24-1 Respiration Patterns

Type	Description	Pattern	Clinical Indication
Normal	12–20 breaths/min and regular		Normal breathing pattern
Tachypnea	More than 24 breaths/min and shallow		May be a normal response to fever, anxiety, or exercise Can occur with respiratory insufficiency, alkalosis, pneumonia, or pleurisy
Bradypnea	Less than 10 breaths/min and regular		May be normal in well-conditioned athletes Can occur with medication-induced depression of the respiratory center, diabetic coma, neurologic damage
Hyperventilation	Increased rate and increased depth		Usually occurs with extreme exercise, fear, or anxiety. Causes of hyperventilation include disorders of the central nervous system, an overdose of the drug salicylate, or severe anxiety.
Kussmaul	Rapid, deep, labored		A type of hyperventilation associated with diabetic ketoacidosis
Hypoventilation	Decreased rate, decreased depth, irregular pattern		Usually associated with overdose of narcotics or anesthetics
Cheyne-Stokes respiration	Regular pattern characterized by alternating periods of deep, rapid breathing followed by periods of apnea		May result from severe congestive heart failure, drug overdose, increased intracranial pressure, or renal failure May be noted in elderly persons during sleep, not related to any disease process
Biot's respiration	Irregular pattern characterized by varying depth and rate of respirations followed by periods of apnea		May be seen with meningitis or severe brain damage
Ataxic	Significant disorganization with irregular and varying depths of respiration		A more extreme expression of Biot's respirations indicating respiratory compromise
Air trapping	Increasing difficulty in getting breath out		In chronic obstructive pulmonary disease, air is trapped in the lungs during forced expiration

observed. Ankylosing spondylitis, a chronic condition resulting in painful, progressive inflammatory arthritis that typically affects the spine and sacroiliac joints, may be present. General chest expansion is limited in this condition. During the inspection, the nurse compares the expansion of the upper chest with that of the lower chest. The nurse also observes the movement of the diaphragm to determine whether the patient with obstructive pulmonary disease is concentrating on expanding the lower chest and using the diaphragm properly. Expansion of one side of the chest versus the other side is important to note because atelectasis, especially that caused by a plug of mucus, may cause unilaterally diminished chest expansion because the air cannot move equally through the pulmonary bed. Abnormal chest expansion may also occur with flail chest, in which the chest collapses instead of expanding during inspiration. Flail chest may result from broken or fractured ribs that cannot maintain the integrity of the chest wall during respiration. The nurse also notes whether the abdomen and chest rise and fall together as they should, or if the effort is not coordinated, is there symmetry of respiratory effort? Asynchronous respiratory effort decreases the quality of respiration at the cost of increased work of breathing and often precedes the need for ventilatory support.

A pulmonary embolus, pneumonia, pleural effusion, pneumothorax, or any problem associated with chest pain, such as fractured ribs, may lead to diminished chest expansion. An endotracheal or nasotracheal tube positioned beyond the trachea into one of the mainstem bronchi (usually the right) is a serious cause of diminished expansion of one side of the chest. If the tube slips into the right mainstem bronchus, the left lung is not expanded, and the patient may experience atelectasis on the left side and hypoxemia.

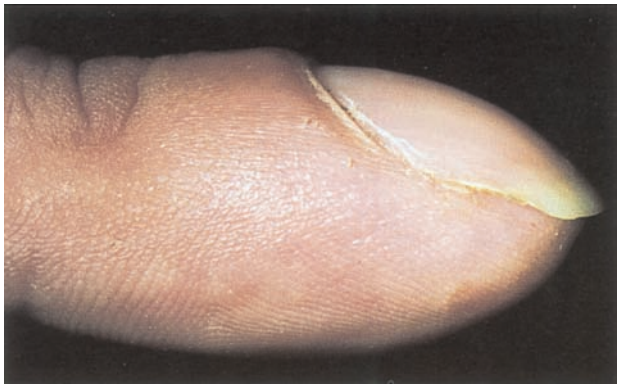


FIGURE 24-2 ▲ In clubbing, the angle between the nail plate and the proximal nail fold increases to 180 degrees or more. Clubbing of the fingers is seen in patients with respiratory and cardiovascular disease. (Photo from Bickley LS: Bates' Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 193. Line art from Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 203.)

Examination of the *patient's extremities* may provide additional information about the patient's respiratory status. Clubbing of the fingers is an enlargement of the distal portion of the finger and is seen in many patients with respiratory and cardiovascular diseases (Fig. 24-2). Although the exact cause is not known, chronic hypoxia is a contributing factor. It is also important to assess the extremities for edema and peripheral cyanosis.

Palpation

Chest palpation may indicate lung or chest abnormalities. To palpate the chest, the nurse places his or her hand flat against the patient's chest. When the patient speaks, sounds are generated by the larynx, and these sounds travel along the bronchial tree, resulting in a resonant motion of the chest wall. *Tactile fremitus* is the ability to feel the sound on the chest wall. Tactile fremitus is more easily palpated over the large bronchi and is more difficult to palpate over the distant lung fields.

To assess tactile fremitus, the nurse asks the patient to say "ninety-nine" while moving his or her hands over the posterior surfaces of the chest wall (Fig. 24-3). Tactile fremitus should be symmetrical. Tactile fremitus may be diminished or absent if there is an increase in air per unit volume of lung because air impedes the transmission of sound. For example, patients with emphysema have little or no tactile fremitus on physical examination. Tactile fremitus is slightly increased by the presence of solid substances, such as the consolidation of a lung from pneumonia. Other respiratory conditions causing an alteration in tactile fremitus are listed in Table 24-2.

Palpation is also used to assess for subcutaneous emphysema, a condition in which air "leaks" out of the alveolus and moves through the subcutaneous tissue. By moving the fingers in a gentle rolling motion across the chest and neck, it is possible to feel the pockets of air underneath the skin. Feeling subcutaneous emphysema is often likened to the "crunch" of Rice Krispies under the skin. Subcutaneous

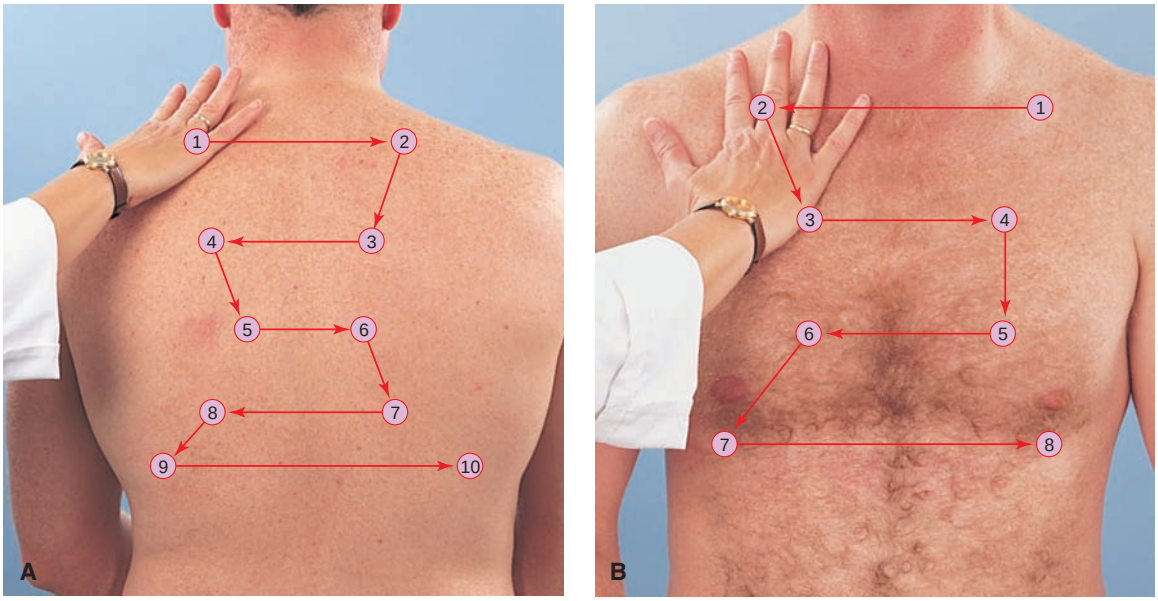


FIGURE 24-3 ▲ Palpating the thorax is performed in a sequential fashion, starting near the neck and moving systematically downward. **A:** Posterior thorax. **B:** Anterior thorax. (Adapted from Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2007, p 321.)

Table 24-2 Physical Findings in Selected Chest Disorders

Condition	Percussion Note	Trachea	Breath Sounds	Adventitious Sounds	Tactile Fremitus and Transmitted Voice Sounds
Normal The tracheobronchial tree and alveoli are clear; pleurae are thin and close together; mobility of the chest wall is unimpaired.	**Resonant**	Midline	Vesicular, except perhaps bronchovesicular and bronchial sounds over the large bronchi and trachea, respectively	None, except perhaps a few transient inspiratory crackles at the bases of the lungs	Normal
Chronic Bronchitis The bronchi are chronically inflamed and a productive cough is present. Airway obstruction may develop.	**Resonant**	Midline	Vesicular (normal)	None; or scattered coarse *crackles* in early inspiration and perhaps expiration; or *wheezes* or *rhonchi*	Normal
Left-Sided Heart Failure *(Early)* Increased pressure in the pulmonary veins causes congestion and interstitial edema (around the alveoli); bronchial mucosa may become edematous.	**Resonant**	Midline	Vesicular	*Late inspiratory crackles* in the dependent portions of the lungs; possibly *wheezes*	Normal
Consolidation Alveoli fill with fluid or blood cells, as in pneumonia, pulmonary edema, or pulmonary hemorrhage.	**Dull** over the airless area	Midline	*Bronchial* over the involved area	*Late inspiratory crackles* over the involved area	*Increased* over the involved area, with *bronchophony, egophony,* and *whispered pectoriloquy*
Atelectasis *(Lobar Obstruction)* When a plug in a mainstem bronchus (as from mucus or a foreign object) obstructs air flow, affected lung tissue collapses into an airless state.	**Dull** over the airless area	May be *shifted toward involved side*	*Usually absent* when bronchial plug persists. Exceptions include right upper lobe atelectasis, where adjacent tracheal sounds may be transmitted.	None	*Usually absent* when the bronchial plug persists. In exceptions (eg, right upper lobe atelectasis) may be increased
Pleural Effusion Fluid accumulates in the pleural space, separates air-filled lung from the chest wall, blocking the transmission of sound.	**Dull** to flat over the fluid	*Shifted toward opposite side* in a large effusion	*Decreased to absent,* but bronchial breath sounds may be heard near top of large effusion.	None, except a *possible pleural* rub	*Decreased to absent, but may be increased* toward the top of a large effusion

(continued on page 492)

Table 24-2 Physical Findings in Selected Chest Disorders (continued)

Condition	Percussion Note	Trachea	Breath Sounds	Adventitious Sounds	Tactile Fremitus and Transmitted Voice Sounds
Pneumothorax When air leaks into the pleural space, usually unilaterally, the lung recoils from the chest wall. Pleural air blocks transmission of sound.	**Hyperresonant** or tympanitic over the pleural air	*Shifted toward opposite side* if much air	*Decreased to absent* over the pleural air	None, except a *possible pleural rub*	*Decreased to absent* over the pleural air
Chronic Obstructive Pulmonary Disease Slowly progressive disorder in which the distal air spaces enlarge and lungs become hyperinflated. Chronic bronchitis is often associated.	Diffusely **hyperresonant**	Midline	Decreased to absent	None, or the crackles, wheezes, and rhonchi of associated chronic bronchitis	Decreased
Asthma Widespread narrowing of the tracheobronchial tree diminishes air flow to a fluctuating degree. During attacks, air flow decreases further, and lungs hyperinflate.	**Resonant** to diffusely **hyperresonant**	Midline	Often obscured by wheezes	Wheezes, possibly crackles	Decreased

The black boxes in this table suggest a framework for clinical assessment. Start with the three boxes under Percussion Note: resonant, dull, and hyperresonant. Then move from each of these to other boxes that emphasize some of the key differences among various conditions. The changes described vary with the extent and severity of the disorder. Abnormalities deep in the chest usually produce fewer signs than superficial ones, and may cause no signs at all. Use the table for the direction of typical changes, not for absolute distinctions.

From Bickley LS: Bates' Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, pp 320–321.

emphysema may result from a pneumothorax, small pockets of alveoli that have burst with increased pulmonary pressure, or the use of positive end-expiratory pressure. In severe cases, the subcutaneous emphysema may spread into the lower thorax, arms, and face.

Evaluation of thoracic expansion during respiration also requires palpation (Fig. 24-4). To perform this procedure, the nurse stands behind the patient, identifies the level of the 10th rib, and places his or her thumbs along the spine using the bony processes as a guide, letting the palms come in light contact with the posterolateral surface. The nurse asks the patient to breathe normally and then deeply, both times watching as the thumbs diverge. Expansion of the chest wall should be symmetrical. Asymmetrical expansion may be indicative of a collapsed lung or unilateral disease. Retractions may be a sign of obstruction to inspiration and require immediate attention.

Palpation of the patient's trachea is an important element in the physical assessment of the respiratory system. To palpate the trachea to evaluate the midline position, the nurse positions his or her index finger in the suprasternal notch, feeling each side of the notch and palpating the tracheal rings (Fig. 24-5). The trachea should be in the midline position directly above the suprasternal notch.

Percussion

Percussion of the chest results in slight motion of the chest wall and underlying structures, causing audible and tactile vibrations. To percuss a patient's chest, the nurse presses one finger from the nondominant hand flat against the chest and uses a fingertip from the dominant hand to strike the knuckle pressed against the chest (Fig. 24-6). Normally, the chest has a resonant or hollow percussion note. In diseases in which there is increased air in the chest or lungs, such as pneumothorax and emphysema, there can be hyperresonant percussion notes. However, these loud, low-pitched sounds are sometimes difficult to detect.

More important is a flat percussion note (eg, the sound that is heard when percussing over a part of the body that contains no air). A flat percussion note is a soft, high-pitched sound that is more easily distinguished by noting the change in sound when one moves from percussing an area with air to an area with no

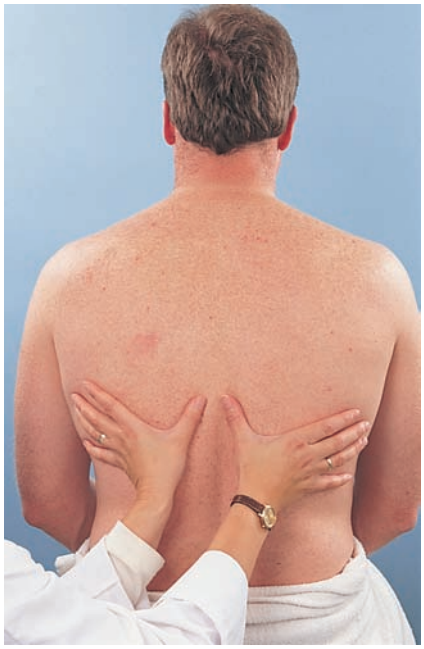


FIGURE 24-4 ▲ Palpating chest expansion. The thumbs are positioned at the level of the 10th rib. (From Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 315.)

air. It is more likely to be heard if a large pleural effusion is present in the lung beneath the examining hand. A dull percussion note is medium in intensity and pitch. It is heard if atelectasis or consolidation resulting from pneumonia, pulmonary edema, or pulmonary hemorrhage is present. A tympanic drum-like sound is a high-pitched noise heard if asthma or a large pneumothorax

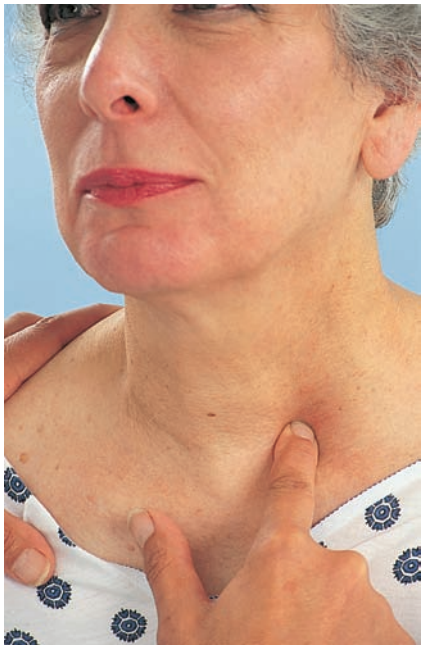


FIGURE 24-5 ▲ Palpating the trachea. The trachea should be midline, above the suprasternal notch. (Photograph B. Proud. From Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 220.)

is present. See Table 24-2 for a description of percussion sounds associated with various respiratory pathologies.

Auscultation

In chest auscultation, the diaphragm of the stethoscope is pressed firmly against the chest wall. The sequence for auscultating the posterior and anterior thorax is given in Figure 24-7. The nurse listens to the intensity or loudness of breath sounds. Normally, there is a fourfold increase in loudness of breath sounds when a patient takes a maximal deep breath as opposed to quiet breathing. Sounds are louder in the upper and central chest when listening to the larger bronchus and become quieter as the smaller airways are auscultated. The intensity of the breath sounds may be diminished because of decreased flow through the airways or the presence of substances between the lungs and the stethoscope. In pleural thickening, pleural effusion, pneumothorax, and obesity, an abnormal substance (fibrous tissue, fluid, air, or fat) lies between the stethoscope and the underlying lung; this substance insulates the breath sounds from the stethoscope, making the breath sounds seem less loud. In airway obstruction, such as COPD or atelectasis, the intensity of breath sounds is diminished. With shallow breathing, there is diminished air movement through the airways, and the breath sounds are not as loud. With restricted movement of the thorax or diaphragm, there are diminished breath sounds in the restricted areas.

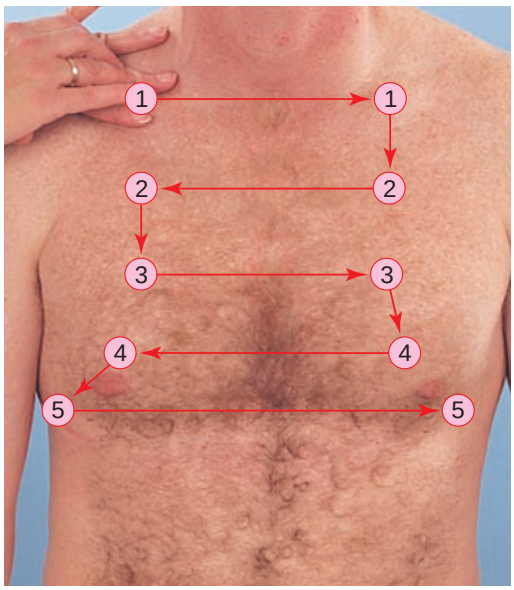
In general, four types of sounds are heard in the normal chest (Table 24-3). *Vesicular breath sounds* are quiet, low-pitched sounds, and the inspiratory phase is longer than the expiratory phase. *Bronchovesicular breath sounds* are medium in pitch, and the inspiratory and expiratory phases are of equal length. *Bronchial breath sounds* are higher pitched and louder compared with vesicular sounds, and the expiratory phase is longer than the inspiratory phase. *Tracheal breath sounds* are loud, high-pitched sounds, and the inspiratory and expiratory phases are about equal in length.[1]

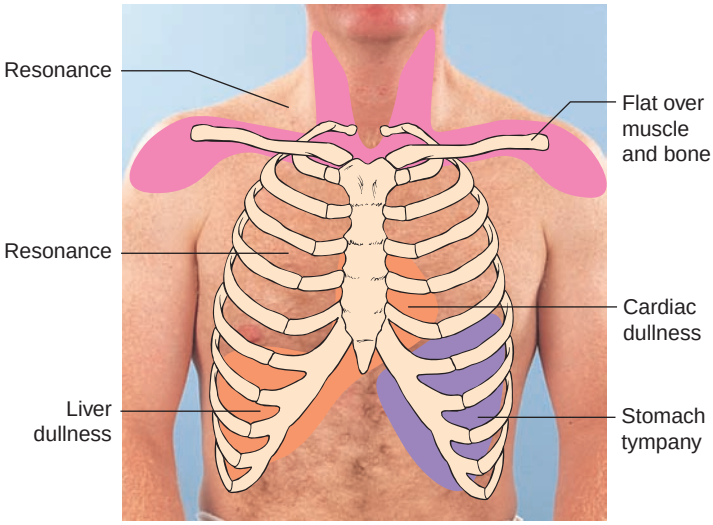
Bronchial breath sounds are heard over the manubrium not only in the normal state but when consolidation is present, as in pneumonia. Bronchial breath sounds are also heard above a pleural effusion in which the normal lung is compressed and sounds are transmitted through the tissue, which is not participating in airflow. Wherever there is bronchial breathing, there also may be two associated changes: E to A changes and whispered pectoriloquy.

An *E to A change* occurs when the patient says "E" and the nurse listening with a stethoscope actually hears an "A" sound rather than an "E" sound. This occurs if consolidation is present. *Egophony* is the term used to describe voice sounds that are distorted.

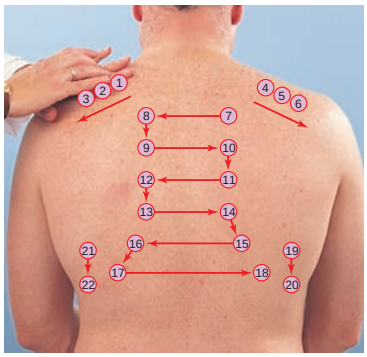
Whispered pectoriloquy is the presence of loud, clear sounds heard through the stethoscope when the patient whispers. Normally, the whispered voice is heard faintly and indistinctly through the stethoscope. The increased transmission of voice sounds indicates that air in the lungs has been replaced by fluid as a result of pneumonia, pulmonary edema, or hemorrhage.

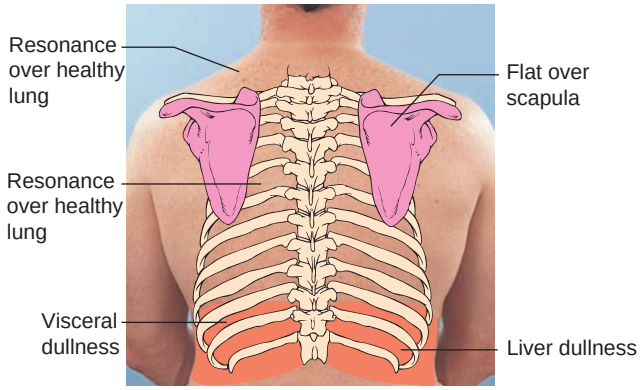
Adventitious sounds are additional breath sounds heard with auscultation and include discontinuous sounds, continuous





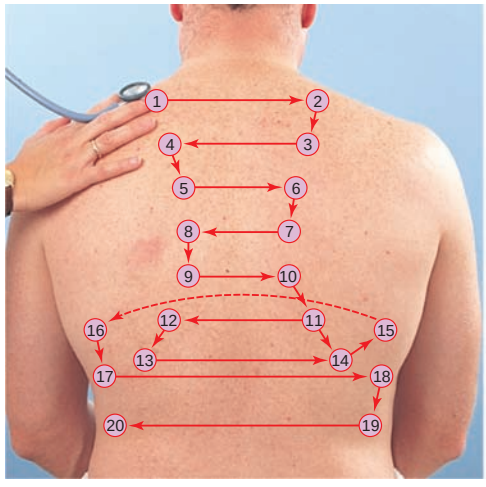
A

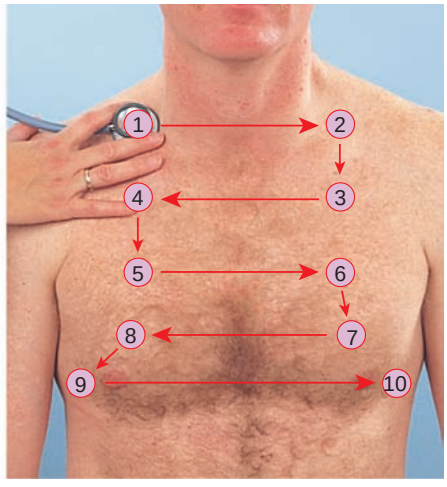




B

FIGURE 24-6 ▲ Percussing the thorax is performed in a sequential fashion, starting near the neck and moving systematically downward. **A:** Anterior thorax. **B:** Posterior thorax. (From Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, pp 315, 316, 322, 323.)





A B

FIGURE 24-7 ▲ Auscultating the chest is performed in a sequential fashion, starting near the neck and moving systematically downward. **A:** Posterior thorax. **B:** Anterior thorax. (From Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, pp 317, 323.)

Table 24-3 Characteristics of Breath Sounds

	Duration of Sounds	Intensity of Expiratory Sound	Pitch of Expiratory Sound	Location Where Heard Normally
Vesicular* 	Inspiratory sounds last longer than expiratory ones.	Soft	Relatively low	Over most of both lungs
Bronchovesicular 	Inspiratory and expiratory sounds are about equal.	Intermediate	Intermediate	Often in the first and second interspaces anteriorly and between the scapulae
Bronchial 	Expiratory sounds last longer than inspiratory ones.	Loud	Relatively high	Over manubrium, if heard at all
Tracheal 	Inspiratory and expiratory sounds are about equal.	Very loud	Relatively high	Over the trachea in the neck

*The thickness of the bars indicates intensity; the steeper their incline, the higher the pitch.From Bickley LS: Bates' Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 303.

sounds, and rubs. Discontinuous sounds are brief, nonmusical, intermittent sounds and include fine and coarse *crackles*. (Crackles were formerly known as *rales*.) Fine crackles are soft, high-pitched, very brief popping sounds that occur most commonly during inspiration. Crackles result from fluid in the airways or alveoli, or from the opening of collapsed alveoli. Restrictive pulmonary disease results in crackles during late inspiration, whereas obstructive pulmonary disease results in crackles during early inspiration. Crackles become coarser as the air moves through larger fluid accumulations, as in bronchitis or pneumonia. Crackles that clear with coughing are not associated with significant pulmonary disease. When assessing crackles, the nurse also notes their loudness, pitch, duration, amount, location, and timing in the respiratory cycle.[2]

Continuous adventitious breath sounds are longer in duration than crackles and include wheezes and rhonchi. *Wheezes* are continuous musical sounds that are longer than crackles in duration and persist throughout the respiratory cycle. Wheezes (also known as sibilant wheezes) are continuous, high-pitched adventitious sounds that have a shrill quality. They are caused by the movement of air through a narrowed or partially obstructed airway, such as in asthma, COPD, or bronchitis.

Rhonchi, another type of continuous adventitious breath sound, are deep, low-pitched rumbling noises that are sometimes referred to as sonorous wheezes or gurgles. The presence of rhonchi indicates the presence of secretions in the large airways.[1] Conditions such as bronchitis cause sonorous wheezing. These sounds may clear somewhat with coughing.

A *friction rub* is a crackling, grating sound heard more often with inspiration than expiration. The sound of friction results from the visceral and parietal pleurae rubbing against each other. A friction rub can be heard with pleural effusion, pneumothorax, or pleurisy. It is important to distinguish a pleural friction rub from a pericardial friction rub. To determine the origin of the rub, the nurse asks the patient to hold his or her breath while the lungs are auscultated. If the sounds continue while the patient is holding his or her breath, it is most likely a pericardial friction rub; a pleural friction rub stops when breathing stops.

In elderly people, unique anatomical and physiological characteristics manifest in different assessment findings. Box 24-4 shows specific respiratory assessment findings in older patients.

BOX 24-4 CONSIDERATIONS FOR THE OLDER PATIENT

Respiratory Assessment

- Decreased ability to hold breath during examination
- Increased hyperresonance (caused by increased distensibility of the lungs)
- Decreased chest wall expansion
- Decreased use of respiratory muscles
- Increased use of accessory muscles secondary to calcification of rib articulations
- Less subcutaneous tissue
- Possible pronounced dorsal curvature
  - Kyphosis (abnormal convexity of the spine—see Fig. 24-1C, p. 488)
  - Gibbus (severe kyphosis)
- Presence of basilar crackles in the absence of disease (should clear after a few coughs)

▲ Respiratory Monitoring

Pulse Oximetry

Approximately 3% of oxygen is dissolved in the plasma (Box 24-5). The partial pressure of oxygen dissolved in the arterial blood is measured by the PaO_2. The normal PaO_2 is 80 to 100 mm Hg at sea level. The remaining 97% of oxygen is attached to hemoglobin molecules in red blood cells. Each gram of hemoglobin can carry a maximum of 1.34 mL of oxygen. The percentage of saturation of hemoglobin is defined as

BOX 24-5 How Oxygen Is Carried in the Blood

Oxygen dissolved in the plasma measured as PaO_2	0.3 mL/100 mL of blood
Oxygen combined with hemoglobin measured as SaO_2	19.4 mL/100 mL of blood
Total oxygen in blood	19.7 mL/100 mL of blood

the amount of oxygen that hemoglobin is carrying compared with the amount of oxygen that hemoglobin (Hgb) can carry, expressed as a percentage:

$$\text{Percent } O_2 \text{ saturation of Hgb} = \frac{\text{Amount of } O_2 \text{ hemoglobin is carrying}}{\text{Amount of } O_2 \text{ hemoglobin can carry}} \times 100$$

Because the amount of oxygen that hemoglobin can carry is a constant 1.34 mL/g,

$$1.34 \text{ mL/g} \times \text{g Hgb} \times \% \text{ saturation Hgb} = \text{mL of } O_2 \text{ that Hgb is carrying}$$

The arterial oxygen saturation of hemoglobin is known as the SaO_2. The normal SaO_2 ranges from 93% to 99%.

The relationship between PaO_2 and SaO_2 is depicted by the oxyhemoglobin dissociation curve (Fig. 24-8). The initial part of the curve is very steep and flattens at the top. The flattened part means that large changes in the PaO_2 result in only small changes in SaO_2. A critical point of the curve occurs when the PaO_2 drops below 60 mm Hg. At this point, the curve drops sharply, signifying that a small decrease in PaO_2 is associated with a large decrease in SaO_2.

When the curve shifts to the right, there is a reduced capacity for hemoglobin to combine with oxygen, resulting in more oxygen released to the tissues. When the curve shifts to the left, there is an increased capacity for hemoglobin to combine with oxygen, resulting in less oxygen released to the tissues. See Chapter 23 for a more detailed discussion of the oxyhemoglobin dissociation curve.

A pulse oximeter, illustrated in Figure 24-9, is a device used to measure a value known as SpO_2 (oxygen saturation as measured by pulse oximetry). The SpO_2 reflects the arterial oxygen saturation of hemoglobin. Through oximetry, light-emitting and light-receiving sensors quantify the amount of light absorbed by oxygenated/deoxygenated hemoglobin in arterial blood. The value displayed on the pulse oximeter is an average of numerous readings taken in a 3- to 10-second period. This reduces the effects of pressure waveform variation caused by patient activity. Usually, the sensors are in a clip placed on a finger or ear lobe and allow for evaluation of the quality of the pulsatile waveform. The oximeter sensor in some devices is placed on the forehead. For assessment of pulse oximetry in infants, flexible probes can measure saturation when placed on the palm, arm, penis, or foot.

Oximetry should not be used in place of arterial blood gas (ABG) monitoring. Instead, pulse oximetry may be used to assess trends in oxygen saturation when the correlation between arterial blood and pulse oximetry readings has been established. Values obtained by pulse oximetry are unreliable when vasoconstricting medications or intravenous dyes are used and when shock, cardiac arrest, or severe anemia is present. Pulse oximetry has limited usefulness in patients with known dyshemoglobins, such as carboxyhemoglobin, which is elevated in smokers, and methemoglobin, which is seen in patients undergoing nitrate and lidocaine therapy. These limitations should be considered when interpreting pulse oximetry readings in certain patients.

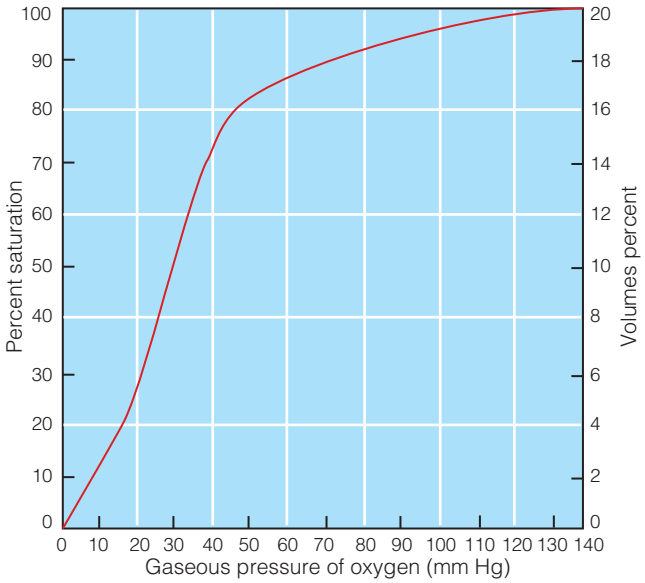


FIGURE 24-8 ▲ Oxyhemoglobin dissociation curve.

End-Tidal Carbon Dioxide Monitoring

End-tidal carbon dioxide ($ETCO_2$) monitoring measures the level of carbon dioxide at the end of exhalation, when the percentage of carbon dioxide dissolved in the arterial blood

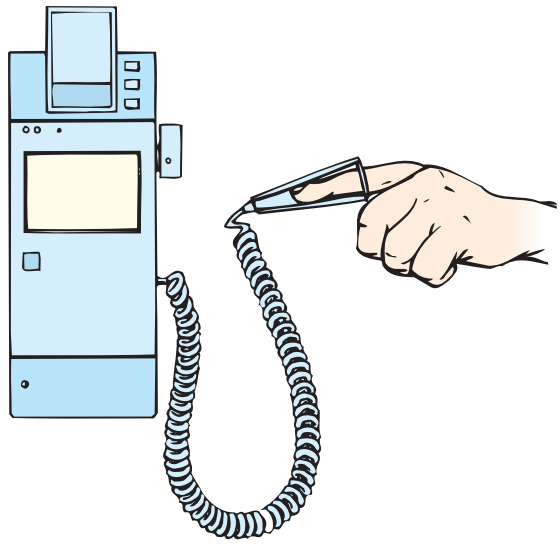


FIGURE 24-9 ▲ Pulse oximetry monitor.

($PaCO_2$) approximates the percentage of alveolar carbon dioxide ($PACO_2$). Therefore, samples of exhaled carbon dioxide measured at the end of exhalation ($ETCO_2$) can be used to approximate levels of $PACO_2$. Levels of alveolar carbon dioxide and arterial carbon dioxide are similar; therefore, $ETCO_2$ can be used to estimate $PaCO_2$. Although $PaCO_2$ and $ETCO_2$ values are similar, $ETCO_2$ is usually lower than $PaCO_2$ by 2 to 5 mm Hg. The difference between $PaCO_2$ and $ETCO_2$ ($PaCO_2$–$ETCO_2$ gradient) may be attributed to several factors; pulmonary blood flow is the primary determinant.

$ETCO_2$ values are obtained by monitoring samples of expired gas from an endotracheal tube, an oral airway, or a nasopharyngeal airway. Because $ETCO_2$ provides continuous estimates of alveolar ventilation, its measurement is useful for monitoring the patient during weaning from a ventilator, in cardiopulmonary resuscitation, and in endotracheal intubation.

The accuracy of the $ETCO_2$ readings may be affected by high concentrations of oxygen and water vapor. The nurse using $ETCO_2$ technology must be aware of these conditions and their effect on the monitor being used. Impaired infrared absorption from the interaction of carbon dioxide and oxygen in high concentrations may cause falsely low $ETCO_2$ measurements, and the interference of water vapor with the absorption of infrared light may cause falsely elevated measurements. The nurse must combine $ETCO_2$ readings with a variety of other clinical data.

The exhaled carbon dioxide waveform is displayed on the monitor as a plot of $ETCO_2$ versus time called a capnogram, which provides the nurse with a continuous graphic reading of the patient's $ETCO_2$ level with each exhaled breath. Changes in the waveform indicate clinical abnormalities, mechanical abnormalities, or both and require immediate assessment by the nurse or other trained professional.

On a capnogram, the waveform is composed of four phases, each one representing a specific part of the respiratory cycle (Fig. 24-10):

1. The first phase is the baseline phase, which represents both the inspiratory phase and the very beginning of the expiratory phase, when carbon dioxide–free air in the anatomical dead space is exhaled. This value should be zero in a healthy adult.
2. The second phase is the expiratory upstroke, which represents the exhalation of carbon dioxide from the lungs. Any process that delays the delivery of carbon dioxide from the patient's lungs to the detector prolongs the expiratory upstroke. Conditions such as COPD and bronchospasm are known physiological causes of prolonged expiratory upstroke. Mechanical obstructions, such as kinked ventilator tubing, may also cause prolonged expiratory upstroke.
3. The third phase begins as carbon dioxide elimination rapidly continues; a plateau on the capnogram indicates the exhalation of alveolar gases. The $ETCO_2$ is the value generated at the very end of exhalation, indicating the amount of carbon dioxide exhaled from the least ventilated alveoli.
4. The fourth phase is known as the inspiratory downstroke. The downward deflection of the waveform is caused by the washout of carbon dioxide that occurs in the presence of the oxygen influx during inspiration.

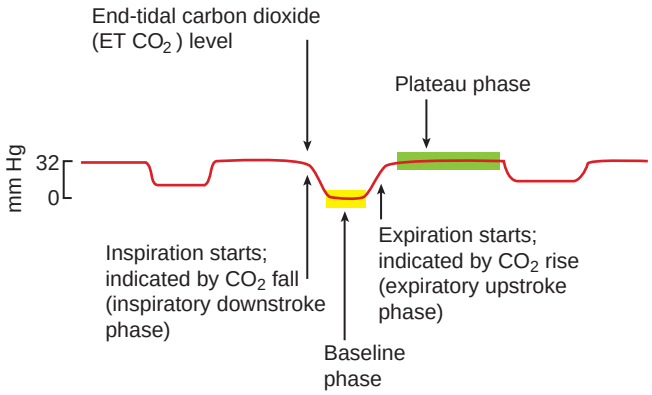


FIGURE 24-10 ▲ Capnogram tracing, with four phases labeled. CO_2, carbon dioxide.

Arterial Blood Gases

In an ABG test, a sample of arterial blood is drawn and analyzed to help determine the quality and extent of pulmonary gas exchange and acid–base status. The ABG test measures PaO_2, SaO_2, $PaCO_2$, pH, and the bicarbonate (HCO_3) level. The procedure involves obtaining arterial blood from a direct arterial puncture or from an arterial line often placed in the radial artery. More recent technology allows the continuous monitoring of ABGs using a fiberoptic sensor placed in the artery. Normal ABG values are given in Box 24-6.

Measuring Oxygen in the Blood

Oxygenation may be measured using an ABG by evaluating the PaO_2 and the SaO_2. As mentioned previously, only 3% of oxygen is dissolved in the arterial blood, and the remaining 97% is attached to hemoglobin in the red blood cells.

The normal PaO_2 is 80 to 100 mm Hg at sea level (barometric pressure of 760 mm Hg). For people living at higher altitudes, the normal PaO_2 is lower because of the lower barometric pressure. PaO_2 tends to decrease with age. For patients who are 60 to 80 years of age, a PaO_2 of 60 to 80 mm Hg is normal. An abnormally low PaO_2 is referred to as hypoxemia. Hypoxemia may result from many conditions, which are most commonly grouped according to their origin: intrapulmonary (disturbances in the lung), intracardiac (disturbance of flow to or from the heart, which impedes pulmonary flow or function), or perfusion deficits (inadequate perfusion of the lung tissues, which causes decreased oxygen uptake from the alveoli).

BOX 24-6 Normal Values for an Arterial Blood Gas

PaO_2: 80 to 100 mm Hg
SaO_2: 93% to 99%
pH: 7.35 to 7.45
$PaCO_2$: 35 to 45 mm Hg
HCO_3: 22 to 26 mEq/L

The normal SaO_2 ranges between 93% and 97%. SaO_2 is an important oxygenation value to assess because most oxygen supplied to tissues is carried by hemoglobin.

Measuring pH in the Blood

The pH is a measure of the hydrogen ion concentration in the blood and provides information about the acidity or alkalinity of the blood. A normal pH is 7.35 to 7.45. As hydrogen ions accumulate, the pH drops, resulting in acidemia. Acidemia refers to a condition in which the blood is too acidic. Acidosis refers to the process that caused the acidemia.

A decrease in hydrogen ions results in an elevation of the pH and alkalemia. Alkalemia refers to a condition in which the blood is too alkaline. Alkalosis refers to the process that causes the alkalemia. Box 24-7 reviews the terms used in acid–base balance.

ACIDS. An acid is a substance that can donate a hydrogen ion (H^+) to a solution. There are two different types of acids: volatile acids and nonvolatile acids.

Volatile acids are those that can move between the liquid and gaseous states. Once in the gaseous state, these acids can be removed by the lungs. The major acid in the blood serum is carbonic acid (H_2CO_3). This acid is broken down into carbon dioxide and water by an enzyme produced in the kidneys.

Nonvolatile ("fixed") acids are those that cannot change into a gaseous form and therefore cannot be excreted by the lungs. They can only be excreted by the kidneys (a metabolic process). Examples of nonvolatile acids are lactic acid and ketoacids.

An acid–base disorder may be either respiratory or metabolic in origin. Table 24-4 lists the possible causes and signs and symptoms of acid–base disorders. An excess of either kind of acid results in acidemia. Also see Table 29-6 in Chapter 29. If carbon dioxide from volatile acids accumulates, then respiratory acidosis exists. If nonvolatile acids accumulate, then metabolic acidosis exists.

Alkalemia may be the result of losing too many acids from the serum. If too much carbon dioxide is lost, the result is respiratory alkalosis. If there are less than normal amounts of nonvolatile acids, the result is metabolic alkalosis.

BASES. A base is a substance that can accept a hydrogen ion (H^+), thereby removing it from the circulating serum. The main base found in the serum is bicarbonate (HCO_3). The amount of bicarbonate that is available in the serum is regulated by the kidney (a metabolic process). If there is too little bicarbonate in the serum, the result is metabolic acidosis. If there is too much bicarbonate in the serum, the result is metabolic alkalosis.

Conditions leading to acidemia or alkalemia are influenced by a multitude of physiological processes (see Table 24-4). Some of these processes include respiratory and renal function or dysfunction, tissue oxygenation, circulation, lactic acid production, substance ingestion, and electrolyte loss from the gastrointestinal tract. The identification of a pH abnormality should lead to the investigation of possible contributing factors.

BOX 24-7 Clinical Terminology

Acid: A substance that can donate hydrogen ions (H^+). Example: H_2CO_3 (an acid) $\rightarrow H^+ + HCO_3$
Base: A substance that can accept hydrogen ions, H^+; all bases are alkaline substances. Example: HCO_3 (base) + $H^+ \rightarrow H_2CO_3$
Acidemia: Acid condition of the blood in which the pH is <7.35
Alkalemia: Alkaline condition of the blood in which the pH is >7.45
Acidosis: The process causing acidemia
Alkalosis: The process causing alkalemia

Measuring Carbon Dioxide in the Blood

The $PaCO_2$ refers to the pressure or tension exerted by dissolved carbon dioxide gas in arterial blood. Carbon dioxide is the natural byproduct of cellular metabolism. Carbon dioxide levels are regulated primarily by the ventilatory function of the lung. The normal $PaCO_2$ is 35 to 45 mm Hg. In interpretation of ABGs, $PaCO_2$ is thought of as an "acid." Elimination of carbon dioxide from the body is one of the main functions of the lungs, and an important relationship exists between the amount of ventilation and the amount of carbon dioxide in blood.

If a patient hypoventilates, carbon dioxide accumulates, and the $PaCO_2$ value increases above the upper limit of 45 mm Hg. The retention of carbon dioxide results in respiratory acidosis. Respiratory acidosis may occur even with normal lungs if the respiratory center is depressed and the respiratory rate or quality is insufficient to maintain normal carbon dioxide concentrations.

If a patient hyperventilates, carbon dioxide is eliminated from the body, and the $PaCO_2$ value decreases below the lower limit of 35 mm Hg. The loss of carbon dioxide results in respiratory alkalosis.

Measuring Bicarbonate in the Blood

Bicarbonate (HCO_3), the main base found in the serum, helps the body regulate pH because of its ability to accept a hydrogen ion (H^+). The concentration of bicarbonate is regulated by the kidneys and is referred to as a metabolic process of regulation. The normal bicarbonate level is 22 to 26 mEq/L. Bicarbonate may be thought of as a "base" (alkaline). When the bicarbonate level increases above 26 mEq/L, a metabolic alkalosis exists. Metabolic alkalosis results from a gain of base (alkaline) substances or a loss of metabolic acids. When the bicarbonate level decreases below 22 mEq/L, a metabolic acidosis exists. Metabolic acidosis results from a loss of base (alkaline) substances or a gain of metabolic acids.

Alterations in Acid–Base Balance

Disturbances in acid–base balance result from an abnormality of the metabolic or respiratory system. If the respiratory system is responsible, it is detected by the carbon dioxide in the serum. If the metabolic system is responsible, it is detected by the bicarbonate in the serum.

RESPIRATORY ACIDOSIS. Respiratory acidosis is defined as a $PaCO_2$ greater than 45 mm Hg and a pH of less

Table 24-4 Possible Causes and Signs and Symptoms of Acid–Base Disorders

Condition	Possible Causes	Signs and Symptoms
Respiratory Acidosis		
$PaCO_2$ > 45 mm Hg pH < 7.35	Central nervous system depression Head trauma Oversedation Anesthesia High cord injury Pneumothorax Hypoventilation Bronchial obstruction and atelectasis Severe pulmonary infections Heart failure and pulmonary edema Massive pulmonary embolus Myasthenia gravis Multiple sclerosis	Dyspnea Restlessness Headache Tachycardia Confusion Lethargy Dysrhythmias Respiratory distress Drowsiness Decreased responsiveness
Respiratory Alkalosis		
$PaCO_2$ < 35 mm Hg pH > 7.45	Anxiety and nervousness Fear Pain Hyperventilation Fever Thyrotoxicosis Central nervous system lesions Salicylates Gram-negative septicemia Pregnancy	Light-headedness Confusion Decreased concentration Paresthesias Tetanic spasms in the arms and legs Cardiac dysrhythmias Palpitations Sweating Dry mouth Blurred vision
Metabolic Acidosis		
HCO_3 < 22 mEq/L pH < 7.35	*Increased acids* Renal failure Ketoacidosis Anaerobic metabolism Starvation Salicylate intoxication *Loss of base* Diarrhea Intestinal fistulas	Headache Confusion Restlessness Lethargy Weakness Stupor/coma Kussmaul respiration Nausea and vomiting Dysrhythmias Warm, flushed skin
Metabolic Alkalosis		
HCO_3 > 26 mEq/L pH > 7.45	*Gain of base* Excess use of bicarbonate Lactate administration in dialysis Excess ingestion of antacids *Loss of acids* Vomiting Nasogastric suctioning Hypokalemia Hypochloremia Administration of diuretics Increased levels of aldosterone	Muscle twitching and cramps Tetany Dizziness Lethargy Weakness Disorientation Convulsions Coma Nausea and vomiting Depressed respiration

than 7.35. Respiratory acidosis is characterized by inadequate elimination of carbon dioxide by the lungs and may be the result of inefficient pulmonary function or excessive production of carbon dioxide.

RESPIRATORY ALKALOSIS. Respiratory alkalosis is defined as a $PaCO_2$ less than 35 mm Hg and a pH of greater than 7.45. Respiratory alkalosis is characterized by excessive elimination of carbon dioxide from the serum.

METABOLIC ACIDOSIS. Metabolic acidosis is a bicarbonate level of less than 22 mEq/L and a pH of less than 7.35. Metabolic acidosis is characterized by an excessive production of nonvolatile acids or an inadequate concentration of bicarbonate for the concentration of acid within the serum.

METABOLIC ALKALOSIS. Metabolic alkalosis is a bicarbonate level of greater than 26 mEq/L and a pH of greater than 7.45. Metabolic alkalosis is characterized by excessive loss of nonvolatile acids or excessive production of bicarbonate.

Interpreting Arterial Blood Gas Results

When interpreting ABG results, three factors must be considered: (1) oxygenation status, (2) acid–base status, and (3) degree of compensation. A suggested approach for interpreting ABG results is presented in Box 24-8, along with sample values for interpretation.

EVALUATING OXYGENATION. It is necessary to examine the patient's oxygenation status by evaluating the PaO_2 and the SaO_2. If the PaO_2 value is less than the patient's norm, hypoxemia exists. If the SaO_2 is less than 93%, inadequate amounts of oxygen are bound to hemoglobin.

EVALUATING ACID–BASE STATUS. The first step in evaluating acid–base status is the examination of the arterial pH. If the pH is less than 7.35, acidemia exists. If the pH is greater than 7.45, alkalemia exists.

The second step in evaluating acid–base status is examination of the $PaCO_2$. A $PaCO_2$ of less than 35 mm Hg indicates a respiratory alkalosis, whereas a $PaCO_2$ of greater than 45 mm Hg signifies a respiratory acidosis.

The third step in evaluating acid–base status is examination of the bicarbonate level. If the bicarbonate value is less than 22 mEq/L, metabolic acidosis is present. If the bicarbonate value is greater than 26 mEq/L, metabolic alkalosis exists.

Occasionally, patients present with both respiratory and metabolic disorders that together cause an acidemia or alkalemia. For example, alkalosis could result from an increase in bicarbonate and a decrease in carbon dioxide, or an acidosis could result from a decrease in bicarbonate and an increase in carbon dioxide. A patient with metabolic acidosis from acute renal failure could also have a very slow respiratory rate that causes the patient to retain carbon dioxide, creating a respiratory acidosis. Therefore, the ABG reflects a mixed respiratory and metabolic acidosis. Box 24-9 lists examples of mixed gases.

DETERMINING COMPENSATION. If the patient presents with an alkalemia or acidemia, it is important to determine whether the body has tried to compensate for the abnormality. If the buffer systems in the body are unable to maintain normal pH, then the renal or respiratory systems attempt to compensate. If the problem is respiratory in origin, the kidneys work to correct it. If the problem is renal in origin, the lungs try to correct it. It may take as little as 5 to 15 minutes for the lungs to recognize a metabolic presentation and start to correct it. It may take up to 1 day for the kidneys to correct the respiratory-induced problem. One system will not overcompensate; that is, a compensatory mechanism will never make an acidotic patient alkalotic or an alkalotic patient acidotic.

The respiratory system responds to metabolic-based pH imbalances in the following manner:

- Metabolic acidosis: increase in respiratory rate and depth
- Metabolic alkalosis: decrease in respiratory rate and depth

The renal system responds to respiratory-based pH imbalances in the following manner:

- Respiratory acidosis: increase in hydrogen secretion and bicarbonate reabsorption
- Respiratory alkalosis: decrease in hydrogen secretion and bicarbonate reabsorption

ABGs are defined by their degree of compensation: uncompensated, partially compensated, or completely compensated. To determine the level of compensation, the pH, carbon dioxide, and bicarbonate are examined. First, it is determined whether the pH is acidotic or alkalotic. In some cases, the pH is not within the normal range, indicating an acidosis or alkalosis. If it is within the normal range, it

BOX 24-8 Interpretation of Arterial Blood Gas Results

Approach

1. Evaluate oxygenation by examining the PaO_2 and the SaO_2
2. Evaluate the pH. Is it acidotic, alkalotic, or normal?
3. Evaluate the $PaCO_2$. Is it high, low, or normal?
4. Evaluate the HCO_3. Is it high, low, or normal?
5. Determine whether compensation is occurring. Is it complete, partial, or uncompensated?

Examples

Sample Blood Gas: Case 1

PaO_2	80 mm Hg	Normal
SaO_2	95%	Normal
pH	7.30	Acidemia
$PaCO_2$	55 mm Hg	Increased (respiratory cause)
HCO_3	25 mEq/L	Normal

Conclusion: Respiratory acidosis (uncompensated)

Sample Blood Gas: Case 2

PaO_2	85 mm Hg	Normal
SaO_2	90%	Low saturation
pH	7.49	Alkalemia
$PaCO_2$	40	Normal
HCO_3	29 mEq/L	Increased (metabolic cause)

Conclusion: Metabolic alkalosis with a low saturation (uncompensated)

BOX 24-9 Arterial Blood Gases in Mixed Respiratory and Metabolic Disorders

Mixed Acidosis
pH: 7.25
$PaCO_2$: 56 mm Hg
PaO_2: 80 mm Hg
HCO_3: 15 mEq/L

Mixed Alkalosis
pH: 7.55
$PaCO_2$: 26 mm Hg
PaO_2: 80 mm Hg
HCO_3: 28 mEq/L

is important to determine on which side of 7.40 (midpoint of the normal pH range) the pH lies. For example, a pH of 7.38 is tending toward acidosis, whereas a pH of 7.41 is tending toward alkalosis. Next, an evaluation is made to see whether carbon dioxide or bicarbonate has changed to account for the acidosis or alkalosis. Finally, it is determined whether the opposite system (metabolic or respiratory) has worked to try to shift back toward a normal pH. The primary abnormality (metabolic or respiratory) is correlated with the abnormal pH (acidotic or alkalotic). The secondary abnormality is an attempt to correct the primary disorder. By using the rules for defining compensation in Box 24-10, it is possible to determine the compensatory status of the patient's ABGs.

BOX 24-10 Compensatory Status of Arterial Blood Gases

Uncompensated: pH is *abnormal* and *either* the CO_2 or HCO_3 is also abnormal. There is no indication that the opposite system has tried to correct for the other.

In the example below, the patient's pH is alkalotic as a result of the low (below the normal range of 35 to 45 mm Hg) CO_2 concentration. The renal system value (HCO_3) has not moved out its normal range (22 to 26 mEq/L) to compensate for the primary respiratory disorder.

PaO_2:	94 mm Hg	Normal
pH:	7.52	Alkalotic
$PaCO_2$:	25 mm Hg	Decreased
HCO_3:	24 mEq/L	Normal

Partially compensated: pH is *abnormal*, and *both* the CO_2 and HCO_3 are also abnormal; this indicates that one system has attempted to correct for the other but has not been completely successful.

In the example below, the patient's pH remains alkalotic as a result of the low-CO_2 concentration. The renal system value (HCO_3) has moved out its normal range (22 to 26 mEq/L) to compensate for the primary respiratory disorder but has not been able to bring the pH back within the normal range.

PaO_2:	94 mm Hg	Normal
pH:	7.48	Alkalotic
$PaCO_2$:	25 mm Hg	Decreased
HCO_3:	20 mEq/L	Decreased

Completely compensated: pH is *normal* and *both* the CO_2 and HCO_3 are abnormal; the normal pH indicates that one system has been able to compensate for the other.

In the example below, the patient's pH is normal but is tending toward alkalosis (>7.40). The primary abnormality is respiratory because the $PaCO_2$ is low (decreased acid concentration). The bicarbonate value of 18 mEq/L reflects decreased concentration of base and is associated with acidosis, not alkalosis. In this case, the decreased bicarbonate has completely compensated for the respiratory alkalosis.

PaO_2:	94 mm Hg	Normal
pH:	7.44	Normal, tending toward alkalosis
$PaCO_2$:	25 mm Hg	Decreased, primary problem
HcO_3:	18 mEq/L	Decreased, compensatory response

Mixed Venous Oxygen Saturation

Mixed venous oxygen saturation (SvO_2) is a parameter that can be measured to evaluate the balance between oxygen supply and oxygen demand. Blood obtained from a vein in an extremity gives information mostly about that extremity; it can be quite misleading if the metabolism in the extremity differs from the metabolism of the body as a whole. This difference is accentuated if the extremity is cold or underperfused (eg, in shock), if the patient has performed local exercises with the extremity (eg, opening and closing the fist), or if there is local infection in the extremity.

Sometimes blood is sampled through a central venous pressure (CVP) catheter in the hope of obtaining mixed venous blood, but even in the superior vena cava or right atrium where a CVP catheter ends, there is usually incomplete mixing of venous return from various parts of the body. For complete mixing of the blood, it is necessary to obtain a blood sample from a pulmonary artery catheter. Use of the pulmonary artery catheter provides a sample of blood that has returned from the extremities and has been mixed in the right ventricle.

Oxygen measurements of mixed venous blood indicate whether the tissues are being oxygenated, but SvO_2 does not distinguish the independent contributions of the heart and the lungs. SvO_2 indicates the adequacy of the supply of oxygen relative to the demand for oxygen at the tissue levels. Normal SvO_2 is 60% to 80%; this means that supply of oxygen to the tissues is adequate to meet the tissue's demand. However, a normal value does not indicate whether compensatory mechanisms were needed to maintain the perfusion. For example, in some patients, an increase in cardiac output is needed to compensate for a low supply of oxygen.

A low SvO_2 may be caused by a decrease in oxygen supply to the tissues or an increase in oxygen use caused by a high demand. A decrease in oxygen supply results from low hemoglobin, hemorrhage, or low cardiac output. An increase in oxygen demand results from hyperthermia, pain, stress, shivering, or seizures. An SvO_2 of 40% to 60% may occur in heart failure, and values less than 40% may indicate profound shock. A decrease in SvO_2 often occurs before other hemodynamic changes and, therefore, is an excellent clinical tool in assessing and managing critically ill patients. The goals of interventions for a low SvO_2 include increasing the oxygen supply by blood transfusions or by increasing cardiac output. Treatment may also be aimed at eliminating the cause of the high demand.

A high SvO_2 value indicates that oxygen supply exceeds demand or a decrease in the demand. Elevated SvO_2 values are associated with increased delivery of oxygen (high fraction of inspired oxygen [FiO_2]) or with decreased demand from hypothermia, hypothyroidism, or anesthesia. An elevated SvO_2 also is seen in the early stages of septic shock when the tissues are unable to use the oxygen. Table 24-5 summarizes possible causes of abnormalities in SvO_2.

A pulmonary artery catheter with an oximeter built into its tip that allows continuous monitoring of SvO_2 provides ongoing assessment of oxygen supply and demand imbalances. If a catheter with a built-in oximeter is not available, the nurse can draw blood from the pulmonary artery through

Table 24-5 Possible Causes of Abnormalities in Mixed Venous Oxygen Saturation (SvO_2)

Abnormality	Possible Cause
Low SvO_2 < 60%	***Decreased oxygen supply***
	Low hematocrit from anemia or hemorrhage
	Low arterial saturation and hypoxemia from lung disease, ventilation–perfusion mismatches
	Low cardiac output from hypovolemia, heart failure, cardiogenic shock, myocardial infarction
	Increased oxygen demand
	Increased metabolic demand, such as hyperthermia, seizures, shivering, pain, anxiety, stress, strenuous exercise
High SvO_2 > 80%	***Increased oxygen supply***
	Supplemental oxygen
	Decreased oxygen demand
	Anesthesia, hypothermia, early stages of sepsis
	Technical problems
	False high reading because of wedged pulmonary artery catheter
	Fibrin clot at end of catheter

a regular pulmonary artery catheter, send the sample to the laboratory for blood gas and SvO_2 analysis, and use the information in the same manner.

▲ Respiratory Diagnostic Studies

Chest Radiography

Chest radiography is a valuable diagnostic tool that clinicians frequently use to assess anatomical and physiological features of the chest and to detect pathological processes. X-rays pass through the chest wall and make it is possible to visualize various structures. Dense tissues, such as bones, absorb the x-ray beam and appear as opaque or white on the radiograph. Blood vessels and blood-filled organs, such as the heart, are moderately dense structures and appear as gray areas on the radiograph. During inspiration, normal lungs fill with air and appear black on the radiograph. When parts of the lungs fill with fluid, which is a more dense material, the lungs appear white.

The nurse uses the radiograph as an assessment parameter to validate clinical findings and suspected abnormalities. Using a systematic approach, the nurse examines the radiograph by comparing the film with previous films. The approach can be to examine the film starting in the periphery and then to move toward the center of the chest or to start centrally and move outward toward the soft tissue. Whatever the method, the objects of scrutiny are the soft tissue areas, the bony structures, the inner layers just under the bone, and the internal structures. See Figure 17-15 in Chapter 17. The nurse examines the soft tissues on the radiograph by looking for homogeneity, beginning with lateral areas and moving medially. Air visualized in the lateral soft tissue may indicate a pneumothorax.

Bony structures inspected on the chest film include the ribs, clavicles, sternum, manubrium, spine, and vertebrae. Approximately eight to nine ribs should overlie lung tissue on the normal chest film. The nurse examines the ribs for fractures by following the curve of each rib, beginning anteriorly and moving around posteriorly. Like the ribs, the other bony structures are examined for correct position and intactness.

The contour of the diaphragm is also visible on the radiograph. Normally, the diaphragm is rounded with sharp, pointed costophrenic angles. Pleural effusions may cause the angles to become blunted. The top of the diaphragm is apparent at about the sixth rib. A lowered diaphragm may indicate hyperinflation caused by emphysema.

The nurse assesses lung parenchyma by comparing right and left sides, moving top to bottom. Normal air-filled lungs should appear black or very dark compared with the bones and heart. It is important in the evaluation to look for symmetry. Abnormally high density on one side of the chest may indicate edema, a mass, pleural effusion, or pneumonia.

Interlobar fissures separate the lobes of the lungs. The minor fissure in the right lung is usually visible in the frontal film. Displacement of the normal fissures seen on the film may indicate atelectasis or lobar collapse.

The trachea should appear midline over the thoracic vertebrae. The trachea can shift toward areas of atelectasis and away from areas of pneumothorax or pleural effusion.

Ventilation–Perfusion Scanning

Ventilation–perfusion scanning is a nuclear imaging test used to evaluate a suspected alteration in the ventilation–perfusion relationship. (See Chapter 23 for a discussion of ventilation–perfusion relationships.) A ventilation–perfusion scan is helpful in detecting the percentage of each lung that is functioning normally, diagnosing and locating pulmonary emboli, and assessing the pulmonary vascular supply.

The ventilation–perfusion scan consists of two parts: a ventilation scan and a perfusion scan. In the ventilation scan, the patient inhales radioactive gas, which follows the same pathway as air in normal breathing. In pathological conditions, the diminished areas of ventilation are visible on the scan. In the perfusion scan, a radioisotope is injected intravenously, enabling visualization of the blood supply to the lungs. When a pulmonary embolus is present, the blood supply beyond the embolus is restricted, revealing poor or no visualization of the affected area.

Ventilation–perfusion scans are often not useful in patients who depend on mechanical ventilation because the ventilation component of the scan is difficult to perform. Ventilation–perfusion mismatches may make interpretation of ventilation–perfusion scans difficult in patients with lung diseases, such as pneumonia. Because of these limitations, pulmonary angiography may be appropriate in the critically ill patient, especially if a pulmonary embolus is suspected.

Pulmonary Angiography

Pulmonary angiography involves the rapid injection of a radiopaque substance for radiographic studies of the pulmonary vasculature. Suspected pulmonary embolus is the most common indication for pulmonary angiography. A radiopaque substance is injected into one or both arms, the femoral vein, or a catheter that has been placed in the pulmonary artery. A positive test result is indicated by the impaired flow of the radiopaque substance through a narrowed vessel or by the abrupt cessation of flow of the substance in a vessel.

Bronchoscopy

Bronchoscopy involves the direct visualization of the larynx, trachea, and bronchi through a flexible fiberoptic bronchoscope. Bronchoscopy is used diagnostically to examine tissues, collect secretions, determine the extent and location of a pathologic process, and obtain a biopsy. In addition, bronchoscopy is used therapeutically as a means to remove foreign bodies or secretions from the tracheobronchial tree, treat postoperative atelectasis, and excise lesions.

In preparation for a bronchoscopy, a history and physical examination should be performed. A chest radiograph, clotting studies, and ABGs are also obtained. The patient often receives intravenous sedation or analgesia before the procedure. If the purpose of the bronchoscopy is therapeutic, medications that suppress a cough or diminish secretions are avoided (eg, intratracheal topical anesthetics, atropine, and codeine).

Careful monitoring of the patient is indicated after a bronchoscopy. The nurse assesses for any evidence of complications, which may include laryngospasm, fever, hemodynamic changes, cardiac dysrhythmias, pneumothorax, hemorrhage, or cardiopulmonary arrest.

Thoracentesis

In thoracentesis, a needle is inserted into the pleural space to remove air, fluid, or both; obtain specimens for diagnostic evaluation; or instill medications. A chest radiograph, coagulation studies, and patient education are essential before a thoracentesis. Some patients may require medication to reduce anxiety. Unlike bronchoscopy, thoracentesis requires the cooperation of the patient; therefore, a local anesthetic, rather than moderate sedation, is used to minimize the pain and discomfort that accompanies the procedure. During the procedure, the patient is placed either in a chair or on the edge of the bed in an upright position with arms and shoulders raised so that the ribs lift and separate, allowing easier needle insertion. If a patient is unable to lift his or her arms, sitting on the bed with the arms placed above the head on a table is an alternative position.

During thoracentesis, the nurse's primary function is to provide comfort for the patient, perform ongoing assessment of the patient's respiratory system, dress the wound with sterile dressings on completion of the procedure, and send labeled specimens to the laboratory as ordered. Postthoracentesis nursing care includes assessment for complications, including pneumothorax, pain, hypotension, and pulmonary edema.

Sputum Culture

Sputum specimens are often part of the respiratory assessment. Because healthy patients do not produce sputum, obtaining a specimen requires the patient to cough to bring up sputum from the lungs. It is essential that the nurse distinguishes sputum from saliva before sending the specimen to the laboratory.

In most cases, sputum specimens are obtained for culture and sensitivity study. The specimen is examined for specific microorganisms and their corresponding drug sensitivities. In addition, sputum specimens are also required for studies of cytology and acid-fast bacilli. Culture of acid-fast bacilli requires serial collection (usually over 3 days) and is used to identify tuberculosis and mycobacteria.

Pulmonary Function Tests

The flow of air in and out of the lungs provides tangible measures of lung volumes. Although these volumes are referred to as measures of "pulmonary function," in reality, they are measures of pulmonary anatomy. In the evaluation of ventilation, structure or anatomy often determines function. Ventilatory or pulmonary function tests measure the ability of the chest and lungs to move air into and out of the alveoli.

Pulmonary function tests include volume measurements, capacity measurements, and dynamic measurements. These measurements are influenced by exercise and disease. Age, sex, body size, and posture are other variables that are taken into consideration when the test results are interpreted. Figure 23-13 in Chapter 23 illustrates normal lung volumes and capacity.

Volume Measurements

Volume measurements show the amount of air contained in the lungs during various parts of the respiratory cycle. Measures of lung volume include tidal volume (V_T), inspiratory reserve volume (IRV), expiratory reserve volume (ERV), and residual volume (RV; see Chapter 23, Table 23-1).

Capacity Measurements

Capacity measurements quantify part of the pulmonary cycle. They are a combination of the previous volumes and include inspiratory capacity (IC), functional residual capacity (FRC), vital capacity (VC), and total lung capacity (TLC; see Chapter 23, Table 23-1).

Dynamic Measurements

The following measurements, called dynamic measurements, provide data about airway resistance and the energy expended in breathing (work of breathing).

- Respiratory rate or frequency is the number of breaths per minute. At rest, the respiratory rate is about 15 breaths/min.
- Minute volume, sometimes called minute ventilation, is the volume of air inhaled and exhaled per minute. It is calculated by multiplying tidal volume by respiratory

rate. At rest, the minute volume is approximately 7,500 mL/min.

- Dead space is the part of the tidal volume that does not participate in alveolar gas exchange. The dead space (measured in milliliters) is the air contained in the airways (anatomical dead space) plus the volume of alveolar air that is not involved in gas exchange (physiological dead space; eg, air in an unperfused alveolus from pulmonary embolism or, more commonly, air in underperfused alveoli). Adult anatomical dead space is usually equal to the body weight in pounds (eg, 140 mL in a 140-pound person). In a healthy person, dead space is composed only of anatomical dead space. Physiological dead space occurs in certain disease states. Dead space is calculated by subtracting the partial pressure of arterial carbon dioxide ($PaCO_2$) from the partial pressure of alveolar carbon dioxide (P_ACO_2). The normal value of dead space in healthy adults is typically less than 40% of the tidal volume. The dead space/tidal volume ratio is used to follow the effectiveness of mechanical ventilation.
- Alveolar ventilation, the complement of dead space, is expressed as the volume of tidal air that is involved in alveolar gas exchange. This volume is represented as volume per minute by the symbol $\dot{V}_A$. $\dot{V}_A$ is a measure of ventilatory effectiveness. It is more relevant to the blood gas values than either the dead space or tidal volume because these last two measures include physiological dead space. $\dot{V}_A$ is calculated by subtracting the dead space (V_D) from the tidal volume (V_T) and multiplying the result by the respiratory rate (f):

$$\dot{V}_A = (V_T - V_D) \times f$$

About 2,300 mL of air (FRC) remains in the lung at the end of expiration. Each new breath introduces about 350 mL of air into the alveoli. The ratio of new alveoli air to total volume of air remaining in the lungs is

$$\frac{350\,mL}{2,300\,mL}$$

Therefore, new air is only about one seventh of the total volume contained in the lungs. The normal is 5,250 mL/min (350 mL/breath × 15 breaths/min = 5,250 mL/min). A normal breath (V_T) can replace 7,500 mL of air/min (500 mL/breath × 15 breaths/min = 7,500 mL/min), requiring 0.008 s/mL:

$$\frac{1\,minute}{7,500\,mL} \times \frac{60\,seconds}{1\,minute} = 0.008\ seconds/mL$$

Therefore, the FRC of the lungs can be completely replaced in 18.4 seconds (2,300 mL × 0.008 s/mL = 18.4 seconds) if air diffusion is uniform. This slow turnover rate prevents rapid fluctuations of gas concentrations in the alveoli with each breath.

▲ Clinical Applicability Challenges

CASE STUDY

Mrs. S. is a 56-year-old white female admitted to the intermediate care unit from the postanesthesia care unit (PACU), status: post laproscopic cholecystectomy. Her procedure was uneventful and she has been placed on patient-controlled analgesia (PCA) to help control her pain. She has a medical history significant for hypertension, morbid obesity, and hypercholesterolemia. Her PCA pump is set to deliver a PCA dose of hydromorphone at 0.2 mg with a 10-minute lockout, basal rate at zero, and a maximum of 1.2 mg/h.

At change of shift assessment, Mrs. S. has stable vital signs with heart rate at 68 beats/min, respiratory rate at 18 breaths/min, blood pressure at 149/175 mm Hg, and temperature at 36.7°C. She is in sinus rhythm with occasional premature atrial contractions (PACs). Her breath sounds are clear to auscultation and her pulse oximetry reading shows that she is saturating at 96% on 2 liters of oxygen. Because Mrs. S. is receiving PCA pain control, her end-tidal CO_2 is being monitored and is at 36 mm Hg. Mrs. S. is rating her pain at 9/10 and asks for additional pain medication. After consultation with the physician, an order is received to increase the hydromorphone by adding a basal rate of 0.3 mg/h with a maximum of 1.5 mg/h maximum. One hour after the change in pain medication, you assess Mrs. S. and notice that there is a change in her level of sedation and her respiratory rate has dropped to 8 breaths/min. In addition, her SpO_2 has decreased to 86% and the $ETCO_2$ has increased to 48 mm Hg.

1. What is the primary factor contributing to the change in the patient's respiratory status?
2. What is the explanation for the increase in the $ETCO_2$?
3. In addition to the increased infusion of narcotic medication, what other factor may have contributed to respiratory depression in Mrs. S.?

References

1. Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010

2. Bickley LS, Szilagyi PG: Bates' Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

25 Patient Management: Respiratory System

John C. Hagen, Tracey Wilson, Charles Fisher, Donna Charlebois, Sidenia S. Tribble, and Paul K. Merrel

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Summarize the desired outcomes of the various bronchial hygiene therapies.
2. Compare and contrast situations in which chest physiotherapy (including postural drainage) is useful versus contraindicated situations.
3. Describe the nursing assessment and management of patients on oxygen therapy.
4. Compare and contrast indications for, and complications of, orotracheal intubation versus nasotracheal intubation.
5. Compare and contrast the principles governing chest tube drainage systems.
6. Discuss nursing management of the patient with a chest tube drainage system.
7. Discuss the pharmacology for the treatment of bronchospasm in asthma and chronic obstructive pulmonary disease.
8. Differentiate between the principles of negative-pressure ventilation and positive-pressure ventilation.
9. Differentiate between pressure-cycled and volume-cycled ventilators in positive-pressure ventilation.
10. Compare and contrast the following ventilator modes: assist-control, synchronized intermittent mandatory, pressure-support, and pressure-controlled ventilation.
11. Summarize strategies to maximize oxygen delivery with the goal of achieving a nontoxic FiO_2 setting.
12. Summarize adverse effects of positive end-expiratory pressure, how they are identified, and the appropriate treatment.
13. Compare and contrast the advantages and disadvantages of tracheostomy versus endotracheal intubation.
14. Describe the nursing management of the ventilated patient, and explain how to prevent complications.
15. Discuss the differences between short-term and long-term ventilation weaning.

Respiration is necessary to sustaining life, and the nurse plays an important role in helping the critically ill patient breathe. The nurse must be knowledgeable and skilled in assessing patient needs, providing quick and efficient care, evaluating results of intervention, and supporting and teaching the patient and family. Techniques, equipment, and procedures vary according to the patient's respiratory status.

▲ Bronchial Hygiene Therapy

Bronchial hygiene therapy (BHT), also known as pulmonary toilet, is helpful in preventing and treating pulmonary complications. The primary phases of lung function that BHT aims to improve are ventilation and diffusion (Fig. 25-1).

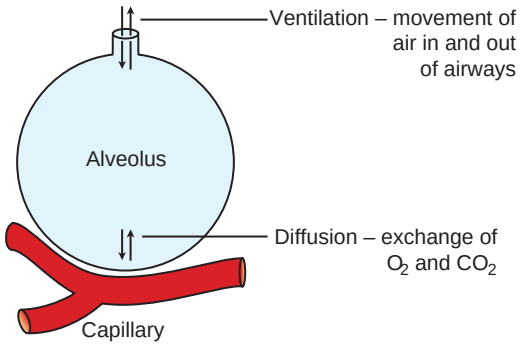


FIGURE 25-1 ▲ Primary lung functions: ventilation and diffusion.

These are accomplished through the therapeutic goals of (1) secretion mobilization and removal and (2) improved gas exchange.

Specific BHT depends on existing pulmonary dysfunction. The normal airway has a functioning mucociliary "escalator" with a cough reflex and normal mucous production. In contrast, the hospitalized patient may have pneumonia, atelectasis, or inability to perform deep breathing, cough, or clear mucus effectively because of weakness, sedation, or pain. The patient may also have chronic conditions such as chronic obstructive pulmonary disease (COPD), cystic fibrosis, pulmonary fibrosis, or quadriplegia.

The need for and the effectiveness of various methods of BHT are based on physical assessment, chest radiography, measurement of arterial blood gases (ABGs), and additional sources of information as indicated. Any one or a combination of the following measures is used: coughing and deep-breathing maneuvers, airway clearance adjunct devices, chest physiotherapy (CPT), and bronchodilator aerosol therapy (pharmacology is discussed later in the chapter).

Coughing and Deep Breathing

Effective coughing is necessary for the patient to clear secretions. The objectives of deep breathing and coughing are to promote lung expansion, mobilize secretions, and prevent the side effects of retained secretions (eg, atelectasis and pneumonia). These techniques are effective only if the patient is able to cooperate and has the strength to cough productively.

The patient is positioned seated and upright on the edge of the bed or chair with the feet supported. The nurse instructs the patient to take a slow, deep breath; hold it for 2 to 3 seconds; and exhale slowly for auscultation. If adventitious sounds are auscultated, indicating the presence of secretions, the patient must be made to maximally inhale and cough. Even if secretions are not auscultated, the patient should be encouraged to cough and deep breathe as a prophylactic measure every hour. The patient must be taught the effective use of the incentive spirometer (IS) to have immediate visual feedback on the breath depth, and coached to increase the volume. Ideally, the patient uses the IS hourly while awake, completing 10 breaths each session followed by coughing, and then the patient progressively increases breath volumes. The nurse coaches the patient to maximize the deep breaths, followed by coughing, and documents the IS volume results. IS, along with coughing and deep-breathing exercises, improves inhaled volumes and prevents atelectasis.

Airway Clearance Adjunct Therapies

Various adjunct therapies may be useful, in general, for patients who require mucous removal and, in particular, when coughing efforts are limited by a disease process, injury, or surgery. The Acapella and Flutter valves, two such methods of airway clearance, provide intermittent positive expiratory pressure (PEP) therapy, which improves mucous removal by causing airway vibration to loosen secretions that can be cleared with a cough.[1] The Acapella valve is just as effective as the Flutter valve and may be easier to use, especially in elderly patients. Both produce PEP and oscillatory vibrations in the airways to loosen mucus, but the Acapella valve is adjustable for the patient, with two types for patients who can sustain flow of greater than or equal to 15 L/min and less than or equal to 15 L/min; this provides flexibility, especially in those with very low expiratory flows. The nurse assists the patient's cough with positive pressure on the abdominal costal margin during exhalation to increase the cough force, thus producing a manually assisted cough. Various specialized BHTs are used for patients with cystic fibrosis and other chronic pulmonary diseases, including autogenic drainage (AD), which may be used with the huff cough.[1] The nurse teaches AD to patients who have reactive airway disease with likelihood of wheezing with normal cough. AD is a series of controlled breaths and uses low-pressure cough with minicoughs instead of one to two big coughs.

The Vest system (Hill-Rom, Batesville, IN), another method of airway clearance, is a chest wall oscillation device that creates chest wall motion through a machine that rapidly alternates air into sections of a vest that is placed circumferentially around the chest. The method is called high-frequency chest wall oscillation, which results in improved secretion removal. This is an alternative to traditional CPT. The Vest system has been used in trials involving patients with bronchiectasis, cystic fibrosis, COPD, lung transplantation, and even spinal cord injury or quadriplegia, as well as in postoperative intensive care units (ICUs). The Vest system has been shown to improve the removal of mucus and improve pulmonary function. It is well tolerated by surgical patients and can be self-administered at home.

Other therapies include EzPAP, bilevel positive airway pressure (BiPAP), and IS, which may be given before any of the BHTs to improve mucous removal. EzPAP and BiPAP are positive airway pressure devices that enable airway recruitment and prevent atelectasis using between 5 and 20 cm H_2O with variable flow of oxygen during therapy. Both work to reduce atelectasis using positive-pressure therapy, often in combination with aerosol pharmacology agents. EzPAP has only one setting of continuous positive airway pressure (CPAP), and BiPAP has both inspiratory high-pressure and expiratory lower pressure levels. Both are used in patients when IS or other therapies are not sufficient to reduce or prevent atelectasis.

Chest Physiotherapy

Postural drainage, positioning, and chest percussion and vibration are methods of CPT used to augment the patient's efforts and to improve pulmonary function. These may be used in sequence in different lung drainage positions and should be preceded by bronchodilator therapy and followed by deep breathing and coughing or other BHT. Changing the patient's position from supine to upright affects gas exchange, and positioning the patient in the lateral position may improve gas exchange, especially in unilateral lung disease. Positioning the patient with the "good" lung down improves oxygenation.[2] This improvement occurs because shunting is decreased when the "good" lung is in the dependent position.

Postural Drainage

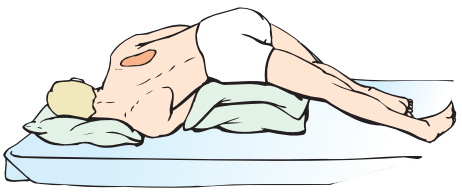
Postural drainage positions facilitate gravitational drainage of pulmonary secretions into the main bronchi and trachea based on anatomy of the lung segments (Fig. 25-2). The focus of postural drainage should be on the lobes affected by atelectasis and on increasing mucous removal with suctioning or by cough effort. Postural drainage is not indicated in all positions for all critically ill patients. Contraindications are listed in Box 25-1. The nurse must closely monitor the patient who is in a head-down position for aspiration, respiratory distress, and dysrhythmias. Alternate techniques may include gentle percussion and using a mechanical percussor to stimulate mucous movement while avoiding surgical areas.

Chest Percussion and Vibration

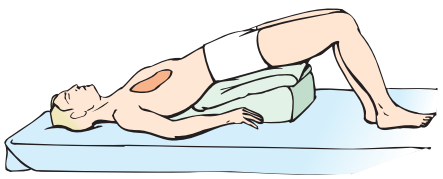
Chest percussion (tapotement) and vibration, performed by a trained health care professional, are used to dislodge secretions. Percussion involves striking the chest wall with the hands formed into a cupped shape by flexing the fingers and placing the thumb tightly against the index finger. The patient's position depends on the segment of lung to be percussed. A towel or pillowcase is draped over the area to be percussed, and percussion is performed for 3 to 5 minutes per position. Percussion is never performed over the spine, over the sternum, or below the thoracic cage. Percussion and vibration are performed only on the rib cage. If performed correctly, percussion does not hurt the patient or redden the skin. A clapping sound (as opposed to slapping) indicates correct hand position. Mechanical percussors are also available.

Vibration takes place during a prolonged pursed-lip exhalation. It increases the velocity and turbulence of exhaled air to

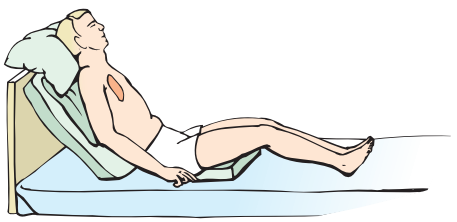
A. Face-lying—hips elevated 16–18 inches on pillows, making a 30°–45° angle.
Purpose: to drain the posterior lower lobes.



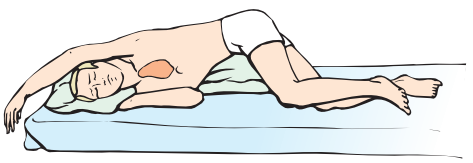
B. Lying on the left side—hips elevated 16–18 inches on pillows.
Purpose: to drain the right lateral lower lung segments.



C. Back lying—hips elevated 16–18 inches on pillows.
Purpose: to drain the anterior lower lung segments.



D. Sitting upright or semireclining.
Purpose: to drain the upper lung field and allow more forceful coughing.



E. Lying on the right side—hips elevated on pillows forming a 30°–45° angle.
Purpose: to drain the left lower lobes.

FIGURE 25-2 ▲ Positions used in lung drainage.

BOX 25-1 *PATIENT SAFETY*

Contraindications to Chest Physiotherapy

Contraindications to Postural Drainage

- Increased intracranial pressure (ICP)
- After meals/during tube feeding
- Inability to cough
- Hypoxia/respiratory instability
- Hemodynamic instability
- Decreased mental status
- Recent eye surgery
- Hiatal hernia
- Obesity

Contraindications to Percussion/Vibration

- Fractured ribs/osteoporosis
- Chest/abdominal trauma or surgery
- Bronchopleural fistula
- Pulmonary hemorrhage or embolus
- Coagulopathy
- Chest malignancy/mastectomy
- Pneumothorax/subcutaneous emphysema
- Cervical cord trauma
- Tuberculosis
- Pleural effusions/empyema
- Pulmonary edema
- Asthma

loosen secretions. This technique is accomplished by placing the hands side by side with fingers extended and applying the flat of the palm over the affected chest area. The patient inhales deeply and then slowly exhales. While the patient exhales, the nurse vibrates the patient's chest by quickly contracting and relaxing the arm and shoulder muscles. Vibration is used instead of percussion if the chest wall is extremely painful.

Modern ICU beds have options to percuss, vibrate, or provide continuous lateral rotation therapy (CLRT), using either an added module or a system integrated into the bed. These bed features can be used to provide BHT to critically ill patients who may not tolerate manual therapy. The nurse assesses patients for tolerance to both position changes and the level of therapy because most bed systems allow variable settings of high to low frequency of percussion or vibration. Continuous lateral rotation is effective for certain patients, especially ventilated patients.[3]

Contraindications and Adaptations

No single method of CPT has been shown to be superior, and there are many contraindications to using these techniques (see Box 25-1). Studies have questioned the efficacy of CPT, except in segmental atelectasis caused by mucous obstruction and diseases that result in increased sputum production (at least 30 mL/d), such as cystic fibrosis and bronchiectasis.[4] Bronchoscopy is an alternative treatment to remove mucous plugs that result in atelectasis. CPT may produce bronchospasm in asthmatics and spread infected material to uninfected lung tissue in patients with unilateral pneumonia.

The inclusion of CPT in the plan of care should be individualized and evaluated in terms of derived benefit versus potential risks. In addition, CPT should be discontinued when it fails to promote treatment goals. In patients who cannot tolerate CPT, turning the patient laterally every 2 hours aids in mobilizing secretions for removal with cough or suctioning. Progressive mobility from sitting up in a chair to weight bearing and to ambulation is used in all ventilated patients as part of pulmonary hygiene as well as increasing patient strength and endurance. Patients with an artificial airway or an ineffective cough may require suctioning after CPT.

To be effective, CPT must be accompanied by the postural drainage position specific to the affected area of the lung. Patients with unilateral disease are positioned with the healthy lung down for better ventilation and perfusion. Positioning the patient with the diseased lung down is likely to cause hypoxemia with ventilation–perfusion mismatching and shunting. Positioning is altered if the patient has a lung abscess. In such cases, the preferred position is with the diseased lung down because the abscessed lung in a gravity-dependent position can drain its purulent contents into the opposite lung. The abscessed lung would then contaminate the healthy lung.

Patient Positioning

Studies demonstrate improved oxygenation in patients with acute respiratory failure who were placed in the prone position, although this maneuver may not ultimately improve survival.[5] Prone positioning is an advanced technique used with critically ill ventilated patients who have acute lung injury (ALI) or acute respiratory distress syndrome (ARDS). ALI is defined by a PaO_2/FiO_2 ratio less than 300. ARDS is defined by a PaO_2/FiO_2 ratio less than 200. The enhanced oxygenation is attributed to recruitment of collapsed lung areas related to body position change, allowing dependent lung regions to have improved perfusion and ventilation. Prone positioning involves multiple personnel and specialized beds or equipment, and it should be performed only by specially trained staff to prevent the many complications related to prone positioning.

Patients who are ventilated benefit from having the head of the bed (HOB) elevated 30 degrees at all times. The rationale is to promote lung expansion, prevent the aspiration that can occur in the recumbent position in intubated patients, and prevent ventilator-associated pneumonia (VAP). Keeping the HOB elevated 30 degrees, with the associated reduction in VAP, is included in the ventilator bundle to prevent VAP and is part of the Institute for Healthcare Improvement's 5 Million Lives Campaign.[6] Mobilization of the patient contributes to improved oxygenation, secretion removal, and airway patency. Using lateral rotational therapy beds is more effective than the inconsistent nursing care of turning every 2 hours at minimum. (See the *American Association of Critical-Care Nurses* [AACN] *Protocols for Practice, Care of Mechanically Ventilated Patients*, 2nd edition, for a complete explanation of prone therapy.)

Mobilization of the patient using CLRT improves oxygenation and blood flow to the lung tissue in affected regions. CLRT is defined as continuous lateral positioning of less than 40 degrees for 18 of 24 hours daily. The lateral positioning improves blood flow and ventilation in the superior lung regions. CLRT may help reduce incidences of pneumonia, although it may not reduce days on the ventilator or the length of hospital stay. The results do not show improved survival for ventilated patients using either prone positioning or rotational therapy. However, with CLRT, the improved

oxygenation and recruitment lead to a reduction in VAP and in the cost associated with VAP, and with prone positioning, there is improved oxygenation. The rotation should be at the maximum to each side, and rotation should be continuous for 18 of 24 hours to obtain best outcomes. Rotation therapy with kinetic therapy refers to beds that rotate greater than or equal to 40 degrees and to CLRT beds that rotate to less than 40 degrees.[7] Both types of beds may include percussion and vibration modules that allow frequent use of those functions to further improve secretion mobilization.

Positioning of critically ill patients remains a nursing intervention for ventilated patients not only to improve oxygenation but also to prevent pressure ulcers. The additional benefit of using CLRT over conventional patient position changes by nursing staff is prevention of skin ulceration. Turning by nurses has become more significant, with the Joint Commission-mandated safety goal of requiring the risk assessment and reassessment for pressure ulcers followed by implementing actions to prevent them.[8] Turning every 2 hours allows the nurse to assess pressure points on the torso and extremities, including the back of the head, and this is even more important in patients with low perfusion. Guidelines should be in place for the use of a scale such as the Braden scale (see Chapter 51) to assess for pressure ulcer risk.[9] The Braden scale is a tool used to reassess increased risk factors daily; its use should be followed by consulting the wound care team and providing additional actions to treat pressure ulcers. Repositioning manually, using CLRT or prone positioning, requires care to avoid causing tissue injury when positioning for extended periods. Prolonged positioning in any one position leaves a patient at risk for developing a pressure ulcer, and turning can result in the dislodging of various tubes or lines. The critical care staff should be well trained in prone positioning, monitoring tubes and lines during rotation therapy, and preventing prolonged pressure in lateral positions. Low-pressure airflow mattresses may help reduce skin ulcer occurrence but should not be relied on as a primary prevention method. Mobilization of ventilated patients using rotation therapy specialty beds is one method for nurses to improve patient outcomes of improved oxygenation; this technique also helps prevent VAP and skin ulcers. Ultimately, the critically ill patient should progress to weight-bearing positions, sitting up in a chair, and, with physical therapy, to ambulation that improves overall physical reconditioning toward a return to independent functioning.

▲ Oxygen Therapy

The administration of oxygen therapy to a patient is designed to correct hypoxemia (low oxygen blood levels). When tissue oxygen availability is decreased, it is referred to as hypoxemia. Hypoxia is reduced oxygen supply, which can be generalized or region limited, for example, tissue hypoxia. If external or internal respiration is impaired, supplemental oxygen is vital to maintain the patient's cellular function. Oxygen therapy corrects hypoxemia, decreases the work of breathing, and decreases myocardial work. Any disease process that alters the gas exchange can cause hypoxemia.

Asthma, bronchitis, pneumonia, ALI and ARDS, COPD, and emphysema are disease processes that alter oxygen supply. Traumatic events that lead to pneumothorax or hemothorax, as well as surgical events, such as pneumonectomy and lobectomy, or events causing large pleural effusions can significantly alter gas exchange. Oxygen delivery by nasal cannula may provide sufficient additional oxygen to reduce air hunger and shortness of breath. A patient with COPD may also need continuous oxygen because of permanent alterations in the lungs, which result in lowered oxygen delivery, especially with stress, illness, infection, and exercise. Patients with COPD and emphysema require close monitoring for carbon dioxide retention and narcosis/stupor associated with the delivery of too high a concentration of oxygen. These patients normally tolerate higher levels of carbon dioxide because their chemoreceptors no longer respond to the normally accepted partial pressure of carbon dioxide (PCO_2) levels and serum pH. These patients' primary drive to breathe comes from their oxygen, rather than carbon dioxide, levels. The desired goals for all patients on oxygen therapy are stable arterial oxygen saturation (SaO_2) level, nonlabored respirations, and a decrease in anxiety and shortness of breath. These goals should be accomplished through delivery of the least amount of supplemental oxygen needed, so the nurse continuously monitors the patient on oxygen for the desired result and complications (Box 25-2). Appropriate physician or advanced practice nurse orders are necessary to initiate this therapy.

Patient Assessment

Assessment of the patient's oxygen need is based on the disease process and the severity of the hypoxemia. The nursing assessment considers the patient's level of consciousness, vital signs (including the rate and depth of breathing), nail bed color, airway patency or presence of an artificial airway, SaO_2, and ABGs. The use of accessory muscles or abdominal breathing may indicate severe distress, and the inability to speak (or the tendency to respond using only one-syllable words) is ominous. A patient with asthma who comes in with bilateral wheezes but who can communicate in complete sentences is less likely to have increased dyspnea and require intubation than the patient with asthma who sits bolt upright, only nods the head to questions, and is using the shoulder and neck accessory muscles to breathe. Accessory muscle use in any patient is usually a sign of respiratory fatigue. Laboratory data, including hemoglobin, hematocrit, electrolyte panel, ABGs, and chest radiographs, may be obtained to assist in correcting electrolyte and pH

BOX 25-2 PATIENT SAFETY

Complications of Oxygen Therapy

- Respiratory depression/arrest
- Discomfort with skin breakdown from straps and masks
- Dry mucous membranes, epistaxis, or infection in the nares
- Oxygen toxicity (prolonged high levels seen in acute lung injury or acute respiratory distress syndrome [ARDS])
- Absorptive atelectasis
- Carbon dioxide narcosis (manifested by altered mental status, confusion, headache, somnolence)

imbalances. A low phosphorus level, or hypophosphatemia, is associated with muscle weakness including the diaphragm impacting ventilation. A low hemoglobin level affects oxygen transport and delivery to tissues. A full assessment, which may include collection of blood for blood gas analysis, takes time, whereas assessment of the person's vital signs, SaO_2, respiratory effort, and symptoms is possible to do quickly and repeatedly. To establish baseline activity tolerance and respiratory function, it may be necessary to involve the family if the patient cannot communicate in complete sentences. Clinicians need to compare the usual symptoms exhibited by a patient with asthma or COPD with the presenting symptoms to establish the severity of the patient's illness. They should initiate oxygen therapy for distress and hypoxemia. After a thorough assessment including laboratory data, it is necessary to adjust the oxygen delivery method to meet the therapeutic goal.

The patient's acuity and underlying disease process dictate the level of oxygen delivery required. The choice of oxygen delivery method is based on the assessment and presentation of the patient, the SaO_2 on room air, and the desired outcome. The desired oxygen level for a patient with COPD may be much lower than that for a patient with pneumonia but without COPD. The patient with pneumonia tolerates higher levels of oxygenation for longer periods than the patient with COPD, who is susceptible to carbon dioxide narcosis.

After giving oxygen, it is necessary to reassess the patient. Signs of improvement include reduced respiratory rate, a more comfortable breathing pattern, increased SaO_2, and the patient's own subjective statement of improved breathing with decreased anxiety or distress. Altered mental status may indicate hypoxemia but may also be due to pH, electrolyte, or carbon dioxide abnormalities. The nurse assesses the patient's respiratory status as often as needed until the desired results are achieved. ABG values guide therapy, especially in patients known to have carbon dioxide retention or continued lethargy or sedation and in those unable to clear secretions. Ultimately, the ABG values indicate success or failure of efforts to correct the underlying hypoxemia.

Oxygen Delivery Systems

Several methods of oxygen delivery are available. The choice of a delivery method depends on the patient's condition. Oxygen delivery systems are traditionally divided into high-flow and low-flow systems (Box 25-3).

BOX 25-3 Oxygen Delivery Methods With Delivered Fraction of Inspired Oxygen (FiO_2)

Nasal Cannula—Low-Flow Device

Flow (L/min)	FiO_2
1	21%–25%
2	25%–28%
3	28%–32%
4	32%–36%
5	36%–40%
6	40%–44%

High-Flow Nasal Cannula

Flow (L/min)	FiO_2
1–40	21%–100%

The high-flow nasal cannula (eg, AquinOx system or Vapotherm) is adjusted for the desired clinical effect depending on the arterial blood gas (ABG), SaO_2, and breaths per minute. These high-flow systems allow for humidification at 100%, with high levels of oxygen delivery maintaining nasal mucosa moisture not possible with a low-flow nasal cannula. The nurse should monitor the SaO_2 closely for at least 30 to 60 minutes when switching from another oxygen delivery device, evaluate ABG as needed, and assess patient tolerance. Be aware of clinical contraindications with increased oxygen delivery.

Face Mask—Low-Flow Device

Flow (L/min)	FiO_2
5–6	40%
6–7	50%
7–10	60%

Face Tent—Low-Flow Device

Variable oxygen delivery of 21% to 50% depends on patient breathing (21% delivered with compressed air and up to 50% delivered with 10 L/min oxygen flow attached). Air is mixed with the oxygen flow in the mask, resulting in variable delivery with humidification. This is often used for humidification as well as oxygen delivery in patients who do not like the claustrophobic feeling associated with more traditional masks.

Venturi Mask—Low-Flow Device

Oxygen Flow (Minimal Rate) (L/min)	FiO_2 Setting*
4	25%
4	28%
6	31%
8	35%
8	40%
10	50%
15	60%

*FiO_2 setting is based on Venturi setting/adapter used and oxygen flow.

Nonrebreather Mask–Low-Flow Device

The nonrebreather mask is used in severe hypoxemia to deliver the highest oxygen concentration. The one-way valve on one side allows for the exhalation of carbon dioxide. The mask delivers 80% to 95% FiO_2 at a flow rate of 10 L/min depending on the patient's rate and depth of breathing, with some room air entrained through the open port on the mask. However, the mask should fit snugly to prevent additional entrainment of room air.

Tracheostomy Collar and T-Piece—Low-Flow Device

The T-piece is a T-shaped adapter used to provide oxygen to either an endotracheal or tracheostomy tube. The flow rate should be at least 10 L/min with humidification. Flow can also be provided by a ventilator. The tracheostomy collar may also be used and is generally the preferred method because it is more comfortable than the T-piece. The strap on the tracheostomy collar is adjusted to keep the collar on top of the tracheostomy. With both the T-piece and tracheostomy collar, the goal is to provide a high-enough flow rate to ensure that there is a minimal amount of entrained room air.

Low-flow oxygen devices work by supplying oxygen at flow rates less than the patient's inspiratory volume, usually 1 to 10 L/min. The rest of the volume is pulled from room air (entrained). Because of this oxygen and room air mixing (entrainment), the actual fraction of inspired oxygen (FiO_2) delivered to the patient is difficult to specify. Low-flow oxygen devices are suitable for patients with normal respiratory patterns, rates, and ventilation volumes. High-flow oxygen devices supply flow rates high enough to accommodate two to three times the patient's inspiratory volume, at 1 to 40 L/min. These devices are suitable for patients with high oxygen requirements because high-flow devices deliver 100% O_2 and maintain 100% humidification essential to prevent drying of the nasal mucosa.

Oxygen delivery devices all deliver different levels of oxygen. Device selection is based on the desired FiO_2. For example, a patient admitted with pneumonia who has an SaO_2 of 88% might improve to the desired level with a nasal cannula at 2 L/min. In contrast, a patient who has an SaO_2 of 88% but who also has a PaO_2 of 52 mm Hg and is using accessory muscles may require a higher liter flow or a nonrebreather oxygen mask. Both patients are monitored for improved SaO_2, respiratory rate and pattern, and ABG improvement. If increased distress, desaturation, or both are noted, more extreme interventions (such as intubation) may be necessary.

If lower concentrations of oxygen are needed, the system selected is usually nasal cannula. The cannula can be used even with mouth breathers because oxygen fills the nasopharynx and, with inspiration, oxygen is entrained. A patient who is primarily a mouth breather with sinusitis needs to be monitored closely for nasal cannula effectiveness or placed on mask oxygen delivery. The exact concentration of oxygen depends on the patient's inspired tidal volume (VT). If the patient hypoventilates, the oxygen concentration increases in the upper airway. In contrast, if hyperventilation occurs, the concentration of oxygen decreases because of large amounts of room air diluting the oxygen delivered. A simple calculation for nasal cannula delivery is to add another 4% for each liter of FiO_2 delivered to the room air value of 21% (see Box 25-3). Each of the other oxygen delivery devices delivers a variable FiO_2, based on breathing pattern and what device is used, as well as the oxygen flow in liters per minute.

If the oxygen concentration must be constant, as is the case for patients with COPD, Venturi systems (eg, the Venturi mask) are used. The Venturi mask delivers an exact percentage of oxygen regardless of the patient's tidal volume. Patients with COPD may require oxygen delivery by the Venturi system. These patients are "sensitive" to oxygen, and a small increase in the percentage of FiO_2 delivered may result in an elevated $PaCO_2$ and respiratory depression. The patient with COPD may have a respiratory drive based on his or her PaO_2, and with this disease process, ventilation decreases with an increased FiO_2, resulting in hypercapnia. The carbon dioxide level can be detected through serial ABG monitoring, which may reveal large increases in $PaCO_2$ with small increases in oxygen flow.

As higher concentrations of oxygen are required, the nasal cannula is replaced by a mask system. A simple mask delivers the lowest concentrations of oxygen, and a nonrebreather mask delivers the highest concentration. The alternative to a nonrebreather for high FiO_2 delivery is the high-flow nasal cannula system. The high-flow nasal cannula is used on a variety of patients with higher oxygen requirements. For example, patients may be those just off the ventilator with low oxygen saturation for prevention of dry secretions with increased humidity (100%), those in pulmonary rehabilitation with decreased exercise tolerance, and those with COPD and asthma, for whom high-flow nasal cannula systems improve breathing rate and dyspnea. It must be stated that patients who have high oxygen requirements on mechanical ventilation should not be extubated until their clinical condition improves. The AquinOx system is an example of a high-flow oxygen device. It allows high-liter oxygen flow through a nasal cannula with humidification at flows of 35 L/min. The high-flow nasal cannula can be more comfortable and allow improved tolerance by providing a constant temperature and high humidity without condensation and moisture buildup in the tubing, which would enter the nose in a low-flow nasal cannula. The other advantage of the high-flow nasal cannula is the ability to deliver a range of FiO_2, up to 100%, to meet patient oxygen demand. If a patient's PaO_2 and SaO_2 cannot be maintained using the nonrebreather mask or high-flow nasal cannula, respiratory failure, with the need for intubation and mechanical ventilation, is imminent.

Complications of Oxygen Delivery

The delivery of oxygen can cause discomfort, skin breakdown, and other complications. Long-term oxygen by nasal cannula, even with humidification, can cause dry mucous membranes, epistaxis, or infection in the sinuses. Nasal cannula tubing, face masks (including the straps), and tracheostomy collars can cause skin breakdown along the face, bridge of the nose, back of the neck, or behind the ears. Oxygen delivery can fail if the tubing is disconnected from the wall, leading to hypoxemia with dysrhythmias or increased dyspnea. The edematous or malnourished patient is at higher risk for alteration in skin integrity, and contaminated oxygen delivery devices should be replaced. Contamination can occur with copious secretions coughed onto a tracheostomy collar or with mucus on any other device used. To prevent a fire-related injury, a "no smoking" rule must be enforced for all patients receiving oxygen therapy.

The nurse routinely inspects the skin and mucous membranes of the mouth and nares for signs of breakdown. Should skin injury occur, further breakdown can be prevented by providing skin barriers or cushions and possibly changing to another type of device. For example, if the bridge of the nose is irritated by a face mask, then switching to a nasal cannula may relieve the discomfort to the nose, as long as the patient receives the same level of oxygen. The mask may cause some patients anxiety, with feelings of suffocation, and, as with all devices, the nurse should ensure the patients' comfort. The nasal cannula can cause breakdown behind the ears or on the upper lip, and even in the nares. Because oxygen delivered by nasal cannula is not highly humidified, except from high-flow devices, it may dry the mucous membranes in the nose. Finally, it is necessary to change disposable humidification systems according to manufacturer specifications to prevent system-related infections. Oxygen humidification sets for any device need routine changing at least every 72 hours. The key to preventing any complication, including

hypoxemia, is accurate and timely assessment of oxygenation parameters and monitoring for complications of the therapies by the nurse.

Oxygen toxicity starts to occur in patients breathing a concentration of more than 50% for longer than 24 hours. Patients who are ventilated with high oxygen concentration for prolonged periods are at risk for oxygen toxicity. To prevent the pathological cellular changes of oxygen toxicity, the patient's FiO_2 should be decreased as tolerated to the lowest possible setting as long as the PaO_2 remains greater than 60 mm Hg. The pathophysiological changes that occur with oxygen toxicity occur at the alveolar level and may progress from capillary leaking to pulmonary edema and possibly to ALI if the high FiO_2 continues for several days. Once the oxygen concentration is decreased to safer levels, the pathophysiological cellular changes may reverse, but if high FiO_2 levels continue, there may be permanent cellular changes and impairment of pulmonary function.

Carbon dioxide narcosis is a risk in patients with COPD who are "sensitive" to oxygen, and an increased FiO_2 may result in an elevated $PaCO_2$, hypoventilation, and respiratory depression or respiratory arrest. Oxygen must therefore be administered with caution to patients with COPD and often at low levels to prevent respiratory depression. Patients on high FiO_2 may develop absorptive atelectasis resulting from less nitrogen in the delivered gas mixture. Because nitrogen is not absorbed, it normally exerts a pressure within the alveoli, keeping the alveoli open. When nitrogen is "washed out," the oxygen replacing it is absorbed, resulting in alveolar collapse (atelectasis).

Respiratory arrest is a complication that can occur even in patients on oxygen therapy. Nurses prevent this complication by monitoring the patient's overall respiratory status, neurological status, vital signs with SaO_2, and ABG values to evaluate for signs of impending respiratory failure. Respiratory arrest can also occur for reasons such as mucous plugging within the tracheobronchial airways or plugging of a tracheostomy or endotracheal tube (ETT). Respiratory failure from fatigue caused by the increased work of breathing can occur quickly in a patient with pulmonary compromise, such as a patient with COPD and new-onset pneumonia. Risk of aspiration of food or gastric contents can occur in hospitalized patients with dysphagia (eg, from a stroke, sedation, or secondary to prolonged intubation with vocal cord paralysis). The nurse must monitor the vulnerable patient more frequently when there is a clear comorbidity that may result in respiratory failure or arrest.

▲ Artificial Airways

Rigorous BHT and carefully monitored oxygen therapy may eliminate the need for an artificial airway or ventilatory support. An artificial airway and ventilatory support become mandatory if these measures fail to provide adequate oxygenation and removal of carbon dioxide. Artificial airways have a fourfold purpose:

- Establishment of an airway
- Protection of the airway, with the cuff inflated
- Provision of continuous ventilatory assistance with ETT and tracheostomy
- Facilitation of airway clearance

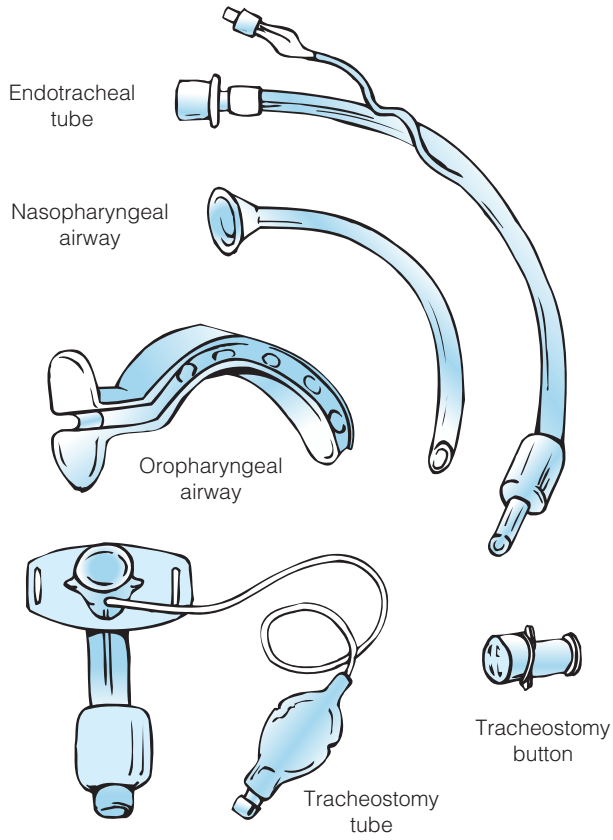


FIGURE 25-3 ▲ Five frequently used artificial airways.

Knowledgeable, aggressive nursing care is required to maintain airway patency, maximize therapeutic effects, and minimize damage to the patient's natural airway.

The selection of the appropriate artificial airway is important. Because all artificial airways increase airway resistance, it is essential that the largest tube possible be used for intubation. The cuff on the endotracheal or tracheostomy tube must be very compliant (soft) so that trauma to the trachea, vocal cords, and subglottic area is minimized. The competency of the cuff must be established before intubation. Approximately 10 mL of air is injected into the cuff before use, and the clinician checks for leaks.

If a patient is sedated and lying supine or becomes unconscious, tongue and airway muscle tone is decreased, causing the tongue to occlude the airway. Although an oropharyngeal or nasopharyngeal airway will maintain the air passage, it will not eliminate the potential for aspiration. Figure 25-3 illustrates some frequently used artificial airways. The nasopharyngeal airway (nasal trumpet) is a flexible tube that is inserted nasally past the base of the tongue to maintain airway patency. This airway may be better tolerated than the oropharyngeal airway in patients with an intact gag reflex.

Oropharyngeal Airway

An oropharyngeal airway is never placed in a conscious patient because it stimulates the gag reflex and can cause vomiting and aspiration. Before placing an artificial airway,

the nurse makes sure any possible obstruction is cleared. Insertion of an oropharyngeal airway follows three steps:

1. Gently open the patient's mouth using a crossed-finger technique or a modified jaw thrust.
2. Hold down the tongue with a depressor and guide the airway over the back of the tongue. (An optional method is to position the tip of the airway toward the roof of the mouth, with the curved end toward the roof, and gently advance the airway by rotating it 180 degrees.)
3. Monitor the patient frequently for airway patency by listening to breath sounds. Provide oropharyngeal suction as needed for emesis or oral secretions.

Oral suctioning is important to maintain oral hygiene when the patient is intubated because the patient's ability to swallow can be limited. The nurse performs oral suctioning as needed for copious oral secretions and after suctioning the endotracheal or tracheostomy tubes to maintain oral hygiene and comfort.

To perform oral suctioning, use a Yankauer device (tonsil-tip suction apparatus). The larger openings on the Yankauer tip allow for suctioning of thick or copious secretions better than other suction catheters designed for suctioning through endotracheal or nasotracheal tubes, which are smaller in diameter. In addition, the smaller suction catheters are flexible, which may cause them to kink. The Yankauer device is angled to allow it to follow the contour of the oral cavity along the palate. This facilitates suctioning in the posterior oropharynx and the buccal pouches, where secretions may collect. After suctioning, the nurse rinses the tubing with tap water to clear it of thick secretions and ensure that the suction will continue to function in the future. To remove an oropharyngeal airway, the oropharynx is suctioned, and the airway is gently removed.

Nasopharyngeal Airway

The insertion of a nasopharyngeal airway involves the following steps:

1. Determine and select the correct tube length by measuring from the tip of the nose to the earlobe. Use a tube with the largest outer diameter that fits the patient's nostril.
2. Lubricate the tube with water, water-soluble jelly, or lidocaine jelly, which alleviates discomfort.
3. Reassure the patient and familiarize him or her with the procedure.
4. Insert the airway into the nostril up to the end of the nasal trumpet.
5. Have the patient exhale with the mouth closed. (If the tube is in the correct position, air can be felt exiting from the tube opening.)
6. Open the patient's mouth, depress the tongue, and look for the tube's tip just behind the uvula.

In patients who require frequent nasotracheal suctioning, nasopharyngeal airways are frequently used to prevent patient discomfort and airway trauma from repeated suction catheter introduction through the nares. Nasotracheal suctioning is best done using a red rubber suction catheter, which is more flexible and better tolerated than standard plastic catheters. Nasotracheal suctioning is done as a sterile procedure. The catheter is lubricated with water-soluble jelly and passed to the back of the nasopharynx initially. Supplemental oxygen is given before suctioning and in between each suction attempt. The oxygen can be given using a bag–valve–mask (BVM) apparatus or manual resuscitation bag (MRB) and gently bagging with each inspiration to provide a high FiO_2. Other high-flow devices, such as a Venturi mask, may also be used to provide more accurate concentrations of oxygen. The nurse or respiratory therapist then asks the patient to cough, which opens the epiglottis and allows the catheter to be advanced. A change in the sound of the cough and the return of sputum with suctioning indicates passage into the tracheal tree. The technique of nasotracheal suctioning is difficult and should only be attempted by experienced practitioners. The novice ICU nurse should seek the help of experienced clinicians to learn this skill.

The nasopharyngeal airway may have to be gently rotated to withdraw it from the nares. It is important to be prepared for the potential of epistaxis with nasopharyngeal airway removal. Prior history of epistaxis or known coagulopathy should be carefully reviewed before placing a nasopharyngeal airway or performing nasotracheal suctioning that may lead to bleeding.

Endotracheal Tubes

An ETT is inserted if the patient needs ventilation or protection of the airway from aspiration. Equipment listed in Box 25-4 is assembled before intubation. The ETT can be inserted nasally or orally.

To reduce the incidence of complications, personnel with specialized training must perform tracheal intubation. The nurse explains rationale for the procedure to the patient and family. The patient is positioned on his or her back with a small blanket under the shoulder blades to hyperextend the neck and open the airway. Air is injected (10 mL) into the endotracheal cuff before insertion to ensure an intact cuff, and then the cuff is deflated.

The nurse's role in intubation includes patient assessment; monitoring vital signs, pulse oximetry, and intubation and suction equipment; and collaborating with additional support staff as needed.

Before the procedure, the nurse confirms that the suction is working properly. Using an MRB and mask, the nurse preoxygenates the patient. The physician may use topical anesthetics, sedatives, or a short-acting neuromuscular blocking (NMB) agent to facilitate rapid and nontraumatic

BOX 25-4 Equipment for Endotracheal Intubation

- Laryngoscope with curved and straight blades and intact bulb
- Suction setup with Yankauer suction
- Correct size endotracheal tube (ETT) with stylet*
- 10-mL syringe for cuff inflation
- Adhesive tape, twill tape, or commercial ETT holder
- Magill forceps (may be used with nasal intubation)
- Pulse oximetry
- Oxygen source
- Manual resuscitation bag (MRB) with mask
- End-tidal CO_2 ($ETCO_2$) monitor or disposable detector
- Sedation and paralytic medication

*In adults, tube size is usually 8.0 initially unless the procedure is difficult, the patient is small, or a difficult intubation is anticipated, in which case smaller sizes are used. An ETT larger than 7.0 mm facilitates bronchoscopy.

intubation. Newer short-acting intravenous (IV) anesthetics facilitate rapid intubation. Sedation with neuromuscular blockade must always be provided.

The nurse assists during intubation by providing suction as necessary and monitoring the patient's SaO_2 by pulse oximetry as well as the patient's heart rate and blood pressure. Intubation attempts should be withheld and the patient oxygenated with the MRB if the SaO_2 falls below 90%. Hypoxemia during intubation may cause bradycardia, hypotension, dysrhythmias, cardiac arrest, and other complications.

After placement of the ETT, the cuff is inflated. It is necessary to auscultate the chest bilaterally for equal breath sounds, which may indicate a right mainstem intubation, and the abdomen for evidence of esophageal intubation. Waterproof tape is used to secure the ETT, and the centimeter mark is noted at the lips, teeth, or nostril for the nasotracheal tube. The level of the ETT must be noted to prevent changing of position, which could result in either right mainstem bronchus placement with left lung collapse or self-extubation. A portable chest radiograph is obtained immediately after the insertion to confirm proper tube placement, which is about 2 to 3 cm above the carina.

Complications of ETT placement are noted in Box 25-5. Initially during intubation, complications of hypoxemia, gastric intubation, mainstem intubation, and oral or tracheal tissue damage can occur. Vomiting during the procedure can lead to aspiration, which may result in lung injury. If the patient has prolonged hypoxemia and hypercapnia (as might result with a difficult intubation), dysrhythmias such as bradycardia or tachycardia can occur, possibly leading to hemodynamic instability.

Once the patient is intubated, potential complications include disconnection, failure of the ventilator, tube obstruction, sinusitis, and tracheoesophageal fistula. Vocal cord paralysis or laryngeal or tracheal stenosis may present after extubation. Accidental extubation in a critically ill patient is a preventable complication. The most challenging cases involve confused patients who attempt to self-extubate. Orienting the patient to the need for the ETT and reassuring the patient that you will help make him or her more comfortable are the first interventions. Occasionally, physical or pharmacological restraints are necessary.

Many complications can be avoided by ensuring adequate fixation of the ETT, securing ventilator tubing properly, suctioning only as needed, and following other care maintenance protocols. These include providing oral care to remove secretions and maintaining the HOB elevated at 30 degrees to help prevent aspiration. Following aseptic policies decreases nosocomial infections, and maintaining proper cuff pressure helps prevent tracheal erosion. Ultimately, the long-term ventilated patient, intubated longer than 72 hours, may require a tracheostomy to continue ventilatory support and weaning.

BOX 25-5 PATIENT SAFETY

Complications of Intubation

- Laryngospasm/bronchospasm
- Hypoxemia/hypercapnia during intubation
- Laryngeal edema resulting in stridor with extubation
- Trauma/bleeding to nasal, oral, esophageal, tracheal, or laryngeal sites
- Fractured teeth
- Nosocomial infection (pneumonia, sinusitis, abscess)
- Displacement of tube (right mainstem intubation, gastric intubation)
- Aspiration of oral or gastric contents
- Tracheal stenosis/tracheomalacia
- Laryngeal damage, paralysis, and necrosis
- Dysrhythmias, hypertension, hypotension

Suctioning

The presence of an artificial tube prevents glottic closure. As a result, the patient is unable to use the normal clearing mechanism (ie, effective coughing). Additionally, the foreign object increases production of secretions. Suctioning, therefore, becomes paramount to removing secretions and maintaining airway patency. Suctioning is not without risks and should be done only when needed. Possible complications of suctioning are listed in Box 25-6. Indications for suctioning include observing secretions in the airway, identifying secretions or mucous plugs by chest auscultation, coughing, increasing peak airway pressure, decreasing tidal volume during pressure ventilation, or deteriorating oxygenation as noted by decreased SaO_2.

The procedure for suctioning is presented in Box 25-7. The nurse performs suctioning as a sterile procedure, using practices recommended by the Centers for Disease Control and Prevention (CDC). In-line suction catheters are available for use in patients on high levels of positive end-expiratory pressure (PEEP) who do not tolerate disconnection of the ventilator tubing for suctioning. Additionally, in-line suction catheters are used for patients with copious secretions requiring frequent suctioning and for those with grossly bloody secretions. Patients who are identified as having the potential for long-term mechanical ventilation and patients who have been reintubated following a failed extubation are candidates for the use of the continuous aspiration of subglottic secretions (CASS) ETT. This device is used to prevent the subglottic accumulation of secretions above the ETT cuff that may lead to aspiration and is discussed as part of the VAP section.

Hyperoxygenation and Saline Instillation

The patient must be hyperoxygenated using the ventilator set to 100% if an in-line system is used. Patients not on ventilators also need to be hyperoxygenated before suctioning. The patient should be instructed to take deep breaths while

BOX 25-6 PATIENT SAFETY

Complications of Suctioning

- Hypoxemia
- Dysrhythmias
- Vagal stimulation (bradycardia, hypotension)
- Bronchospasm
- Elevated ICP
- Atelectasis
- Tracheal mucosal trauma
- Bleeding
- Nosocomial infection

BOX 25-7 Procedure for Suctioning

Equipment

Sterile suction catheter*
Sterile gloves
Sterile normal saline for irrigation, only when indicated
Sterile disposable container

Technique

1. Perform routine procedures before suctioning: Administer medication, assemble equipment, explain the procedure to the patient, adjust bed to comfortable working position, prepare suction pressure, wash hands, prepare and open equipment and supplies, and don gloves.
2. Hyperoxygenate the patient with 100% oxygen using an MRB or the ventilator. If the ventilator method is used, preoxygenation must last at least 2 minutes. Return to the previous oxygen setting after suctioning is completed. In patients who do not tolerate suctioning with hyperoxygenation, a positive end-expiratory pressure (PEEP) attachment should be on the MRB at the appropriate setting, or in-line suctioning should be used to avoid loss of PEEP and desaturation.
3. Quickly but gently, insert the catheter as far as possible into the artificial airway without application of suction. For tracheostomy patients, limit the distance to just beyond the end of the tracheostomy device.
4. Withdraw the catheter 1 to 2 cm, and apply intermittent suction while rotating and removing the catheter. Limit suction pressure to 80 to 120 mm Hg. Aspiration should not exceed 10 to 15 seconds. (Prolonged aspiration can lead to severe hypoxemia, hemodynamic instability, and ultimately cardiac arrest.) Tracheostomy patients are usually suctioned for a briefer period of 3 to 5 seconds because of the very short device.
5. Do not instill sterile normal saline solution unless the patient has thick secretions and trial use has shown that it improves secretion removal. Routine instillation has been shown to decrease oxygenation and have other negative effects.[28–31]
6. Hyperoxygenate the patient before and after each subsequent pass of the catheter for at least 30 seconds, and before reconnection to the ventilator.
7. Monitor heart rate and rhythm and pulse oximetry during and after suctioning.
8. Discontinue the procedure if the patient does not tolerate it, as evidenced by dysrhythmias, bradycardia, or a drop in SaO_2.
9. Remove equipment.
10. Perform oral hygiene. Cleanse suction tubing with a water rinse to remove secretions into the suction container.
11. Wash your hands.
12. Document procedure.

*Suction catheter sized for either ETT or tracheostomy; with tracheostomy, the red rubber or more flexible suction catheter brand is used to prevent tracheal bleeding.

connected to a 100% oxygen source. Patients incapable of taking a deep breath should be assisted using an MRB with mask, timing a squeeze on the MRB with the patient's own breath. The presence of epiglottitis or croup is an absolute contraindication to any kind of suctioning of patients without an artificial airway as this may worsen the patient's condition.

The routine instillation of normal saline solution has become increasingly questionable. In a test tube, saline and sputum act as oil and water, and they do not form a mixture. Therefore, it is unlikely that saline instillation liquefies or increases the amount of sputum obtained during suction. In addition, instilling saline solution causes oxygenation to decrease and may predispose patients to nosocomial infection by transporting bacteria to lower airways.[10]

ETT care and cuff pressure monitoring are discussed in more detail later in this chapter, under Ventilatory Support, Assessment and Management beginning on page 537. After the patient is intubated, the patient loses the ability to communicate easily. This inability to communicate can become a major stressor during the ventilated period.

▲ Chest Tubes

The chest tube is a drain. Its purposes are to remove air, fluid, or blood from the pleural space; restore negative pressure to the pleural space; reexpand a collapsed or partially collapsed lung (pneumothorax); and prevent reflux of drainage back into the chest. Chapter 23 provides a review of the anatomical and physiological principles of the lungs and chest that helps explain how chest tube systems work.

Equipment

Equipment needed for chest tube insertion is listed in Box 25-8.

Most chest tubes are multifenestrated transparent tubes with distance and radiopaque markers. This enables the physician or other qualified health care professional to visualize the tube on chest radiograph and position it correctly in the pleural space. All openings in the tube must be placed within the rib cage to ensure that air leaks do not develop either in subcutaneous tissue or outside the chest wall. Chest tubes may be pleural or mediastinal, depending on distal tip location. Patients can have more than one tube in different locations, depending on the purpose of each tube.

Larger tubes (20 to 36 French) are used to drain blood or thick pleural drainage. Smaller tubes (16 to 20 French) are used to remove air.

Drainage Systems

To reestablish intrapleural negative pressure, a seal for the chest tube that prevents outside air from entering the system

BOX 25-8 Equipment for Chest Tube Insertion

- Chest tube tray or thoracotomy tray (with scalpel)
- Chest tube
- 1% lidocaine
- Syringe for lidocaine infiltration
- Topical antiseptic
- Sterile gloves
- Large curved hemostats
- Suture material (0-0 or 2-0 silk) on a cutting needle
- Bacteriostatic ointment or petrolatum gauze
- Sterile gauze with a slit
- Tape—both wide and narrow, or an occlusive dressing
- Chest tube drainage system and suction
- Sterile water for water seal systems
- Medication for pain and sedation

is required. The simplest way to accomplish this is to use an underwater system of drainage. A review of multichamber systems can provide a basis for understanding all the commonly used disposable drainage units. Knowledge of these systems enables the nurse to safely manage the most complex chest tube drainage setup. Modern chest drainage systems are composed of disposable materials and may be configured in either two- or three-chamber systems. The two-chamber system has a water seal and a collection chamber, whereas the three-chamber system adds a suction control chamber.

Two-Chamber System

In a two-chamber system, the first chamber is the collection receptacle and the second chamber is the water seal. In a disposable system that requires water, sterile water is added to the second chamber to the 2-cm level to achieve the seal. This level represents the negative pressure that is exerted on the pleural space as the water closes the chest drain to outside air, acting as a one-way valve. The water seal allows air to escape while preventing outside air from entering the pleural space. A fluid level higher than 2 cm H_2O exerts a greater negative pressure on the pleural space and may prevent resolution of the air leak. In addition, a higher column of water in the water seal chamber can make breathing more difficult because the patient has a longer column of fluid to move during respiration. Figure 25-4 depicts disposable chest drainage systems.

The patient's chest tube is connected to a 6-ft length of latex tubing that is attached to an outlet on the top of the drainage collection chamber. The second chamber (the water seal) has a vent that remains open, allowing air from the pleural space to escape as it bubbles through the water seal to the atmosphere. Except for the vented cap, the drainage system from the chest tube insertion site to the bottle is airtight.

The fluid level in the water seal fluctuates ("tidals") during respiration. During inspiration, pleural pressures become more negative, causing the fluid level in the water seal chamber to rise. During expiration, pleural pressures become more positive, causing the fluid level to descend. If the patient is receiving mechanical ventilation, this process is reversed. Bubbling should be seen only in the underwater seal chamber during expiration (or during inspiration with positive-pressure ventilation) as air and fluid drain from the pleural cavity. Constant bubbling indicates an air leak in either the system or a bronchopleural fistula; this is discussed further under Assessment and Management.

Three-Chamber System

In the three-chamber system, a suction control chamber is added to the two-chamber system. This is the safest way to regulate the amount of suction. In a disposable system that requires water, suction is achieved by adding water to the prescribed level in the suction chamber, usually −20 cm H_2O, and newer waterless suction systems adjust with a dial to the desired suction in centimeters of water.

In this system, it is the height of the water column in the third chamber, not the amount of wall suction, that determines the suction amount applied to the chest tube, most commonly −20 cm H_2O. Once the wall suction exceeds the force necessary to "lift" this column of fluid, any additional suction simply pulls air from a vented cap atop the chamber up through the water. The amount of wall suction applied to the third chamber should be sufficient to create a "gently rolling" bubble in the suction control chamber. Vigorous bubbling results in water loss through evaporation, changing suction pressure and increasing the noise level in the patient's room. It is important to assess for water loss and to add sterile water as necessary to maintain the prescribed level of suction. The bubbling should be assessed for gentle action, and the water level (−20 cm H_2O) is assessed every 8 hours and when the patient's clinical status changes.

Suction

Dry suction (waterless) systems use a spring mechanism to control the suction level and can provide higher levels of suction with easier setup. The dry suction systems can be easily adjusted for any setting between −10 and −40 cm H_2O and allow for safer use if the device is accidentally tipped over. If this happens, the drainage can be returned to the correct collection chamber without replacing the unit, resulting in cost savings. This system affords the patient a quieter environment. Dry suction systems that can deliver higher levels of suction may be necessary in patients with large bronchopleural fistulas, hemorrhage, or obesity.

The Emerson pleural suction pump may be used instead of wall suction. It can be set up using a two- or three-bottle system as well as a disposable chest drainage system. In contrast to the wall unit, the pressure-control knob on the front of the pump controls the suction generated. The amount of pressure is registered on the suction dial.

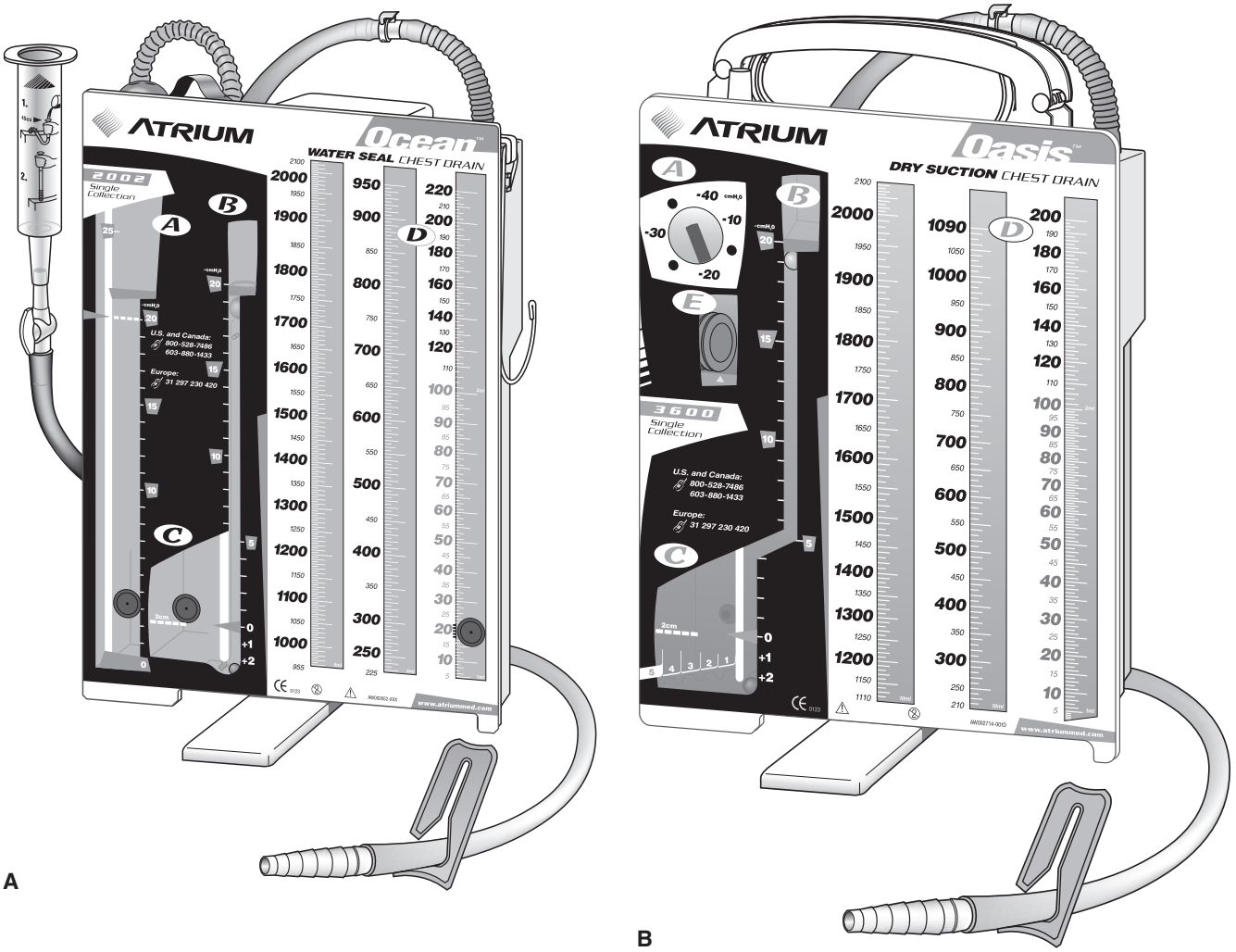
Heimlich valves are reserved for treating pneumothoraces on an outpatient basis or by emergency medical providers to treat blunt or penetrating chest trauma. The valve is composed of a small-bore chest tube attached to a one-way valve, which is enclosed in a plastic case. The one-way valve allows air to escape but not reenter the pleural space. The Heimlich valve is not appropriate for fluid removal.

Chest Tube Placement

If injury, surgery, or any disruption in the integrity of the lungs and chest cavity occur, placement of a chest tube is warranted. In addition, iatrogenic pneumothorax can occur in the ICU during thoracic central line placement, thoracentesis, high mechanical ventilation pressures, or cardiopulmonary resuscitation (CPR), or after transbronchial lung biopsy. Indications for chest tube placement are listed in Table 25-1.

Chest tube insertion can be accomplished in the operating room, in the emergency department, or at the bedside. Placement is based on the principle that, because of their different densities and weights, air rises and liquid sinks. The insertion site for air removal is near the second intercostal space along the midclavicular line. The insertion site for liquid drainage is near the fifth or sixth intercostal space on the midaxillary line. Fluid may occasionally become loculated (walled off), requiring ultrasound or computed tomography guidance for drainage tube placement. After heart surgery, placement can be in the mediastinum to drain blood from around the heart.

The nurse prepares the patient and family for the procedure, answering any questions they may have. The nurse also prepares the patient physically. Because parietal pleurae are



A

B

FIGURE 25-4 ▲ Chest tube drainage systems. **A:** The Atrium Ocean is an example of a water seal chest drain system composed of a drainage chamber and water seal chamber. The suction control is determined by the height of the water column in that chamber (usually 20 cm). (*A*, suction control chamber; *B*, water seal chamber; *C*, air leak zone; *D*, collection chamber). **B:** The Atrium Oasis is an example of a dry suction water seal system that uses a mechanical regulator for vacuum control, a water seal chamber, and a drainage chamber. (*A*, dry suction regulator; *B*, water seal chamber; *C*, air leak monitor; *D*, collection chamber; *E*, suction monitor bellows.) (Courtesy of Atrium Medical Corporation, Hudson, New Hampshire.)

Chest drainage units (CDUs) work with the same three chambers shown in the examples above. The collection chamber collects fluid with air passing through, the water seal prevents air going back into the patient, and a suction control chamber allows suction level settings depending on the medical condition. A collection chamber allows fluid volume up to 2,000 mL. This allows for assessment of type of drainage, amount of drainage, and rate changes, with some models having a sampling port. The water seal chamber is the one-way valve action that prevents air from returning to the chest while allowing air removal as in pneumothorax. The water seal chamber is filled to the –2 cm H_2O level that maintains a slight negative pleural pressure and prevents air entering the pleural space when the CDU is off suction and on water seal. The suction control chamber is either the dry suction or a water-filled chamber. Water-filled suction is set by adjusting the water level. With continuous high suction, evaporation occurs, changing the volume of water that will change the suction level. Nursing care includes assessing the water level daily with suction off to refill the chamber to the desired level. Dry suction uses a mechanical system that allows the set suction level to be maintained regardless of the external suction level applied. The bellow window on dry suction systems allows a visual check that correct suction is applied; the colored indicator rises when sufficient suction is applied to maintain the set suction level from –10 to –40 cm H_2O. The wall suction regulator should be adjusted to –80 mm Hg, with a continuous gentle bubbling in the water seal chamber. All CDUs follow these same functions of collection, water seal, and suction regulation chambers, and the nurse adheres to manufacturer instructions concerning proper setup and monitoring of the system.

Table 25-1 Indications for Chest Tube Placement

Indication	Cause
Hemothorax	Chest trauma Neoplasms Pleural tears Excessive anticoagulation Postthoracic surgery/open lung biopsy
Pneumothorax	
Spontaneous: >20%	Bleb rupture Symptomatic patient Presence of lung disease
Tension	Mechanical ventilation Penetrating puncture wound Prolonged clamping of chest tubes Lack of seal in chest tube drainage system
Bronchopleural fistula	Tissue damage Tumor (esophageal cancer) Aspiration of toxic chemicals Boerhaave's syndrome (spontaneous rupture)
Pleural effusion	Neoplasms Cardiopulmonary disease, congestive heart failure Inflammatory conditions Recurrent infections/pneumonia
Chylothorax	Trauma or thoracic surgery Malignancy Congenital abnormalities

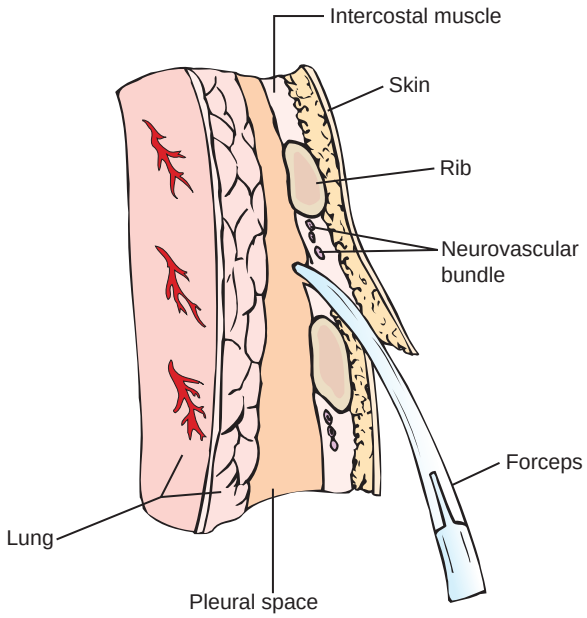


FIGURE 25-5 ▲ Forceps penetrate the pleural space and are spread to create a track within the pleural space for the chest tube to be positioned. A gloved finger may also be used to create this track.

innervated from the intercostal and phrenic nerves, this is a painful procedure, and administration of analgesics is indicated. The patient is placed in Fowler's or semi-Fowler's position. After the skin has been cleaned and anesthetized, the physician or other qualified health care professional makes a small skin incision. A hemostat is used to penetrate the pleural space (Fig. 25-5). The tract made by the hemostat is then dilated with a sterile, gloved finger. The proximal end of the tube is clamped with the hemostat and then inserted into the pleural space. If the placement is difficult, a metal trocar can be used to penetrate the chest wall, leaving the tube in place and removing the trocar.

After insertion, the external end of the tube is connected to a chest drainage unit (CDU). It is important to remember that the ends of both the chest tube and the drainage system tubing must remain sterile as they are connected. To prevent the tube from dislodging, the tube is sutured to the skin around the insertion site. The ends of the suture are wrapped around the tube and tied off. Bacteriostatic ointment or petrolatum gauze can be applied to the incision site. Petrolatum gauze has been preferred because it is thought to prevent air leaks; however, it also has the potential to macerate the skin and predispose the site to infection. A 4″ × 4″ drain sponge is positioned over the tube and taped occlusively to the chest. All connections from the insertion site to the drainage collection system are securely taped to prevent air leaks as well as inadvertent disconnection. The proximal tube is taped to the chest to prevent traction on the tube and sutures if the patient moves.

A postinsertion chest radiograph is always ordered to confirm proper positioning. The lungs are auscultated, and the condition of the tissue around the insertion site is evaluated for the presence of subcutaneous air. This assessment provides a baseline for determining improvement or worsening of the patient's condition. Daily chest radiographs may be necessary to assess the clinical picture. Pain management continues to be an issue throughout the duration of chest tube use. Narcotics, nonsteroidal anti-inflammatory drugs, or a lidocaine transcutaneous patch may help reduce pain. The application of a lidocaine-infused patch proximal to the incision or at the chest tube incision site, which slowly releases medication over 12 to 24 hours, is an additional pain relief tool. Dressings are changed per institutional protocol, or as needed if soiled or loose. It is necessary to assess chest tube output every 2 hours, looking for sudden cessation or increase to greater than 200 mL/h or a sudden change in the character of the drainage.

Chest tubes are removed after drainage is minimal and 12 to 24 hours after the chest tube is on water seal as ordered by the health care practitioner. Placement to water seal only (without suction) identifies persistent air leaks or reaccumulation of fluid with a repeat chest radiograph. Other indications for removal of the chest tube are listed in Box 25-9. When the tube is connected to water seal, disconnect the suction tube to facilitate atmospheric venting. Premature clamping or removal of the tube may cause reaccumulation of the pneumothorax.

Before removing the chest tube, the patient is placed in a Fowler's or semi-Fowler's position (head of bed elevated 45 to 90 degrees). Premedication is recommended to alleviate pain and discomfort. The dressing over the insertion site is removed, and the area is cleaned. The suture is clipped. The tube is removed in one quick movement at peak inspiration or during expiration to prevent entraining air back into the

BOX 25-9 Indications for Chest Tube Removal

- One day after cessation of air leak
- Drainage of less than 50 to 100 mL of fluid/d
- One to three days after cardiac surgery
- Two to six days after thoracic surgery
- Obliteration of empyema cavity
- Serosanguineous drainage from around the chest tube insertion site
- Chest tube partially migrated out with holes visible (may require a new chest tube insertion)

pleural cavity through the chest tube eyelets. Immediately after tube removal, the lung fields are auscultated for any change in breath sounds, and an occlusive sterile dressing is applied over the site. A chest radiograph is usually obtained several hours later to look for the presence of residual air or fluid.

Assessment and Management

Nursing care is directed at maintaining patency and proper functioning of the chest tube drainage system. Vigilant and expert nursing care can prevent serious complications in the patient with a chest tube and drainage system. The latex tubing is frequently drained into the collection container. Coiling the latex tubing loosely on the bed prevents kinks and pooling of blood or drainage in a dependent loop hanging on the floor. Ensure that the patient does not inadvertently lie on the tubing. The chest tube drainage system is never raised above the chest or the drainage will back up into the chest. At frequent intervals, check the chest tube drainage system for drainage, suction level, and water seal integrity. The system should be secured to the foot of the patient's bed or taped to the floor to avoid accidental overturning and possible reaccumulation of the pneumothorax. Inspect all tubing connections for leaks, and secure them with tape to prevent accidental disconnection.

To check for chest tube patency and respiratory cycle fluctuations, it is necessary to momentarily disconnect the suction (system placed only to water seal—not clamped). A step-by-step system can be used to evaluate and troubleshoot the system:

1. Assess cardiopulmonary status and vital signs every 2 hours and as needed.
2. Check and maintain tube patency every 2 hours and as needed.
3. Monitor type and amount of drainage.
4. Mark amount of drainage on collection chamber in hourly or shift increments and document in output record.
5. Prevent dependent loops from forming in tubing; ensure that the patient does not inadvertently lie on the tubing.
6. Refill water systems with sterile water to the water seal level and prescribed suction level (secondary to evaporation).
7. Assess for "tidaling" in the water seal chamber with respiration or mechanical ventilation breaths.
8. Assess for the location of air leaks (constant bubbling in the water seal chamber). Turn off the suction. Begin at the insertion site; occlude the chest tube or drainage tube (briefly) below each connection point until the drainage unit is reached.
9. Check that all tubing connections are securely sealed and taped. Another technique for sealing the connections is to use a banding gun that secures a plastic loop around the connections to prevent air leakage.[11]
10. Assess the patient for pain and medicate as needed.
11. Assess the actual chest tube insertion site for signs of infection and subcutaneous emphysema with dressing changes.
12. Change the dressing twice daily or per unit guideline, when soiled, and when ordered.

Drainage Monitoring

The nurse assesses and documents the color, consistency, and amount of drainage while remaining alert to significant changes. A sudden increase indicates hemorrhage or sudden patency of a previously obstructed tube. A sudden decrease indicates chest tube obstruction or failure of the chest tube or drainage system. The following nursing actions are recommended to reestablish chest tube patency:

- Attempt to alleviate the obstruction by repositioning the patient.
- If the clot is visible, straighten the tubing between the chest and drainage unit and raise the tube to enhance the effect of gravity.

Studies suggest that milking and stripping techniques may not be beneficial for maintaining chest tube patency.[11]

These techniques may excessively increase intrapleural and intrapulmonary pressures, affecting ventricular function or causing trauma from aspiration of lung tissue into chest tube eyelets. However, under health care practitioner direction, this procedure may be necessary in cases of active bleeding to prevent blood clotting in the tubing that could lead to cardiac or pleural tamponade.

Water Seal Monitoring

Monitoring the water seal of the chest tube drainage system is as important as observing the drainage. Visual checks are made to ensure water seal chambers are filled to the 2-cm water line. If suction is applied, the nurse ensures that the water line in a water-controlled suction chamber is at the ordered level (usually −20 cm H_2O) because water evaporates over time, decreasing the amount of suction being applied. It is important to add only sterile water to the system. If an Emerson pleural suction pump is used, the nurse checks the suction gauge for the desired suction level. It is essential that the air vent opening is never occluded. The suction tubing is disconnected briefly to accurately assess the water level in the chamber (water suction control) only after clamping the tubing. Then, the tubing is reattached and the clamp opened. The tubing should never be left clamped because this can result in pneumothorax or buildup of fluid in the chest, leading to respiratory distress.

Respiratory fluctuations are observed in the water seal chamber. The absence of fluctuations can indicate that the lung is reexpanded or that there is an obstruction in the system. Continuous vigorous bubbling in the water seal chamber, without suction, indicates continued pneumothorax, or it can indicate the tube has been displaced or disconnected, or the drainage system is damaged. It is necessary to check

the entire system for disconnections and to inspect the chest tube to see if it is displaced outside the chest. In the setting of mechanical ventilation at high volumes and pressures, bubbling in a chest tube system that persists may indicate a bronchopleural fistula when there is no pneumothorax or other known cause.

Positioning

The ideal position for a patient with a chest tube is semi-Fowler's. Turning the patient every 2 hours enhances air and fluid evacuation. The nurse teaches patients how to support or "splint" the chest wall near the tube insertion site using a pillow, bath blanket, or their arms placed firmly against the chest. The nurse also encourages coughing, deep breathing, and ambulation. Administration of pain medication before these exercises decreases pain and enhances lung expansion.

Complications

The most serious complication resulting from chest tube placement is tension pneumothorax, which can develop if there is any obstruction in the chest tube drainage system. Clamping chest tubes as a routine practice predisposes patients to this complication. Clamping of chest tubes is recommended in only two situations:

- To locate the source of an air leak if bubbling occurs in the water seal chamber (*clamping is only momentary*)
- To replace the chest tube drainage unit (*clamping is only momentary*)

If the tube must be clamped, padded hemostats are used to avoid lacerating the vinyl chest tube.

Occasionally, the chest tube may become dislodged or be accidentally removed. In such a circumstance, the insertion site is quickly sealed off using petrolatum gauze covered with dry gauze and occlusive tape dressing to prevent air from entering the pleural cavity.

Transporting the Patient With Chest Tubes

As in any transport situation for critically ill patients, constant assessment is necessary to prevent inadvertent chest tube removal, resulting in recurrent pneumothorax. Maintain chest drainage system integrity by positioning the drainage system below the level of the chest. Secure the system to the foot of the bed, and ensure that the tubing does not become crushed or kinked. If the system requires suction to evacuate the pleural space, then portable suction must be implemented. The nurse performs frequent assessment of the patient and the drainage system per unit guidelines as needed to check for air leaks, dressing integrity, water seal integrity, water level, and drainage.

▲ Pharmacological Agents

Bronchodilator Therapy

Asthma is characterized by recurrent airway inflammation and increased hypersensitivity to a wide range of stimuli (noxious fumes and gases, air pollutants, animal dander, extreme cold, and exercise). The hypersensitivity leads to hyperreactivity of the airways with obstruction, with widely variable symptoms even in the same person. Asthma is an episodic disease with recurrent exacerbations and periods without symptoms. Management goals include symptom control to maintain normal activities, prevention of exacerbations, and minimization of pharmacological side effects and toxicity. Pharmacological therapy is designed around multiple classes of drugs and is aimed at reducing inflammation, treating acute symptoms, and maintaining a plan for the short- and long-term therapy. Bronchospasm may also be present in COPD and can be treated with the same pharmacological agents.

Delivery of agents typically has been by propellant inhalers, with dosage dependent on the number of puffs per treatment using a metered-dose inhaler (MDI). The breath, depth, inspiratory flow rate, and use of a spacer (vs. administering just into the mouth) create variable medication delivery. Cleaning and proper fitting together of the actuator to the valve stem, and onto a spacer (if used), are necessary. The preferred MDI method involves using a spacer—a tube attached to the inhaler to hold the medication until it is breathed in by the patient. In many patients, this makes MDIs easier to use and deposits medications into the lungs better. Dry powder inhalers (DPIs) allow some asthma medications to be taken in a dry powder form. The difference between DPIs and MDIs is that DPIs do not use a propellant, only a medication. Patient inhalation ensures delivery into the lungs. Patients must practice the proper use of these devices, maintain consistent inspiratory flow, and consistently use a spacer or aim the device directly into the mouth to achieve consistent medication dosage. Patients may range in age from 5 years to elderly, although they must be able to inhale forcefully enough to breathe in the medication. As with MDIs, patients need training to follow the directions and process for each form of DPI inhaler.

The Diskus inhaler is a version of the DPI. Usually, it contains a set number of metered doses, such as a long-acting β_2-agonist (eg, Serevent) or the newer combination of Flovent and Serevent called Advair. The Diskus type of medication is used for asthma control and not for an acute asthma attack because the medication is long acting for daily use. A spacer is not used with the Diskus inhaler. The patient has to breathe in deeply and steadily, hold the breath for up to 10 seconds, and then slowly exhale. With the Diskus inhaler, it is important to point out that the mouthpiece should never be washed in water and that the inhaler should not be placed in water. In addition, the patient does not breathe into the Diskus before inhaling.

Bronchodilators

Bronchodilators act principally to dilate the airways by relaxing bronchial smooth muscles. The goals of bronchodilator therapy are to relax the airways, mobilize secretions, and reduce mucosal edema. Bronchodilator therapy can be delivered through MDIs, preferably with a spacer attachment, or nebulization. Regardless of the mode of delivery, assessment before, during, and after the therapy is essential.

Assessment before and after treatment includes breath sounds, pulse, and respiratory rate. The last two commonly increase during bronchodilator therapy and can remain

elevated for as long as 1 to 1.5 hours after treatment. (In people with asthma, measurement of peak expiratory flow rate with a peak flow meter before and after a treatment measures the improvement in severity of airway obstruction.) Objective evaluation is crucial, but subjective information is also valuable. Evaluation of patient response should be ascertained by asking about improvement in breathing, presence of wheezing, and side effects, such as tremor or palpitations.

Bronchodilators may be divided into three categories based on their mechanism and site of action. These are β_2-adrenergic agonists, anticholinergic agents, and methylxanthines.

β_2-Adrenergic Agonists

The bronchodilator effects of β-adrenergic agonists result from the stimulation of β_2-adrenergic receptors in the lung bronchial smooth muscle. In addition, these agents may decrease the release of mediators from mast cells and basophils. β_1-Adrenergic receptors in the heart may also be stimulated and lead to undesired cardiac effects. Newer β-agonists are more specific for the β_2-receptor, although they retain some β_1 activity.

β-Agonists may be administered orally or inhaled. Aerosolized or inhaled therapy is preferred and has been shown to produce comparable bronchodilation and fewer systemic adverse effects.

β-Agonists are the bronchodilators of choice for treating acute exacerbation of asthma because of their rapid onset of action. They produce less bronchodilation in patients with COPD than in those with asthma. Albuterol (2.5 to 5 mg diluted in 3 mL normal saline solution) is the bronchodilator of choice in the acute setting and may be administered by continuous or intermittent frequent nebulization (every 15 to 20 minutes) then scheduled on an "as-needed" basis depending on patient response. Until recently, all available inhaled β-agonists, such as albuterol, had short durations of action (4 to 6 hours). Salmeterol is the first long-acting β-agonist, with duration of action of 12 hours. Salmeterol cannot be used for acute exacerbations of asthma because of its slow onset of action. The medication DuoNeb combines albuterol and ipratropium, allowing for a synergistic effect of both a bronchodilator and an anticholinergic agent, as discussed later. In addition, Advair Diskus combines a long-acting β_2-adrenergic agonist, salmeterol, with fluticasone propionate, an inhaled corticosteroid, to provide twice-daily dosing for patients who do not achieve adequate control using other asthma medications.

Anticholinergic Agents

Anticholinergic agents produce bronchodilation by reducing intrinsic vagal tone to the airways. They also block reflex bronchoconstriction caused by inhaled irritants.

Atropine is the prototype anticholinergic agent but is used infrequently. It is readily absorbed from the respiratory tract but produces unwanted systemic effects (eg, blurred vision, drying of respiratory secretions, tachycardia, and anxiety). Ipratropium, a quaternary amine that is not well absorbed from the respiratory tract, produces fewer systemic adverse effects and has taken the place of atropine. It is most effective in patients with COPD when used on a regular basis. It decreases submucosal gland secretion and relaxes bronchial smooth muscle. Ipratropium should not be used alone in acute exacerbations because of its slower onset of effect compared with β-agonists. It has been shown to be effective during status asthmaticus when administered through a nebulizer in combination with β-agonists as in DuoNeb.

Methylxanthines

Using methylxanthines for treating bronchospastic airway disease is controversial. The agents' mechanism of action is poorly understood. They inhibit phosphodiesterase, an enzyme that catalyzes the breakdown of cyclic adenosine monophosphate. They may also possess some degree of anti-inflammatory activity and may augment respiratory muscle contractility.

Theophylline, the prototype methylxanthine, may be used chronically in treating bronchospastic disease but is usually considered third- or fourth-line therapy. Some patients with severe disease that is not controlled with β-agonists, anticholinergics, or anti-inflammatory agents may benefit from theophylline. Aminophylline, the IV form of theophylline, is rarely used in acute exacerbations because of the lack of evidence that it is beneficial in this situation.

Theophylline has a narrow therapeutic index. Depending on the clinical situation, serum drug concentration should be monitored to ensure efficacy and prevent toxicity. The accepted therapeutic range is 10 to 20 mcg/mL, although some references use 5 to 15 mcg/mL.[12] Theophylline interacts with a variety of other medications that may alter its serum concentration. These include erythromycin, ciprofloxacin, and cimetidine. Patients with liver disease or congestive heart failure eliminate theophylline more slowly and may be at an increased risk for toxicity. The level should be monitored 12 to 24 hours after the loading dose is administered and frequently as clinical condition and liver and renal function dictate.

Anti-Inflammatory Agents

Anti-inflammatory agents interrupt the development of bronchial inflammation and have a prophylactic or preventive action. They may also reduce or terminate ongoing inflammation in the airway. Anti-inflammatory agents include corticosteroids, mast cell stabilizers, and leukotriene receptor antagonists.

Corticosteroids

Corticosteroids are the most effective anti-inflammatory agents for treating reversible airflow obstruction. Corticosteroid therapy should be initiated simultaneously with bronchodilator therapy because the onset of action may be 6 to 12 hours. They may be administered parenterally, orally, or as aerosols. In acute exacerbations, high-dose parenteral steroids (eg, IV methylprednisolone) are used and then tapered as the patient tolerates. Short courses of oral therapy may be used to prevent the progression of acute attacks. Long-term oral therapy is associated with systemic adverse effects and should be avoided if possible. If necessary, the chronic use of inhaled corticosteroids, such as fluticasone (Flovent) or budesonide (Pulmicort), is preferred because of the decreased risk for systemic adverse effects.

Mast Cell Stabilizers

The two available mast cell stabilizers are cromolyn and nedocromil. They are thought to stabilize the membrane and prevent the release of mediators from mast cells. These agents are not indicated for acute exacerbations of asthma because they are used *prophylactically* to prevent acute airway narrowing after exposure to allergens (eg, exercise, cold air). A 4- to 6-week trial may be required to determine the efficacy in individual patients. The desired endpoint is to reduce the frequency and severity of asthma attacks and enhance the effects of concomitantly administered bronchodilator and steroid therapy. As a result, it may be possible to decrease the dose of bronchodilators or corticosteroids in patients who respond to mast cell stabilizers.

Leukotriene Receptor Antagonists

Leukotriene receptor antagonists, such as montelukast, may be used in managing exercise-induced bronchospasm, asthma, allergic rhinitis, and urticaria. These agents block the activity of endogenous inflammatory mediators, particularly leukotrienes. These mediators cause increased vascular permeability, mucous secretion, airway edema, bronchoconstriction, and other inflammatory cell process activities. Leukotriene receptor antagonists are administered once daily and are usually well tolerated. They are not to be administered for acute conditions but as a part of an ongoing program of therapy.

Cystic Fibrosis Agent (DNase)

DNase is used in cystic fibrosis patients to break down molecules in tenacious secretions to facilitate expectoration as well as decrease the amount of medium for bacterial growth. This also improves gas flow through airways. It is administered in an inhaled form either daily or twice daily.

Antibiotics

Pneumonia is often treated empirically until the results of cultures and sensitivities are available. Then, the antibiotic regimen is tailored to eradicate the specific pathogenic organism (Table 25-2). Commonly, broad-spectrum antibiotics or combination therapy is used. The critically ill patient is at increased risk for developing pneumonia due to mechanical ventilation, decreased immune responses, use of corticosteroids, debilitated general health, and cross-infection by health care workers. Antibiotic therapy should be driven by institutional protocols to prevent antibiotic overuse and following guidelines for antimicrobial selection to limit resistance.

Empiric therapy for community-acquired pneumonia includes therapy directed toward the most common organisms associated with this type of pneumonia. These organisms include *Streptococcus pneumoniae* and *Haemophilus influenzae*. Methicillin-resistant *Staphylococcus aureus* should be suspected in patients admitted to the hospital from a nursing home. *Legionella* species should be suspected in patients with severe multilobar pneumonia. Patients infected with the human immunodeficiency virus require empiric treatment if there is suspected *Pneumocystis jiroveci* pneumonia, previously referred to as *Pneumocystis carinii* pneumonia.

Hospital-acquired pneumonia (HAP) or health care–associated pneumonia is often associated with Gram-negative bacilli, such as *Pseudomonas aeruginosa*, or it may be polymicrobial. HAP is the term for pneumonia that develops at least 48 hours after hospitalization; formerly, it was known as nosocomial pneumonia, and it includes VAP. Aspiration is a concern in mechanically ventilated patients or patients unable to protect their airway. Aspiration pneumonia is associated with anaerobic organisms (eg, *Actinomyces* species). Atypical organisms (*Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, and *Legionella* species) should also be considered, as should viral infection. Patients should have a quantitative sputum culture to identify species on admission, and they may require bronchoscopy for specimen collection.

Consensus guidelines for pneumonia management are constantly under revision. The causes of pneumonia may stem from multiple factors and these must be taken under consideration when treating the critically ill patient. Because of the prevalence of multi-drug resistance and evolving antibiotic regimens, priority must be placed on obtaining a sputum culture and defining the susceptibility of the organism to several antibiotics. This will further ensure appropriate antibiotic use.

Table 25-2 Antibiotic Therapy in Pulmonary Disease

Pulmonary Infection	Empiric Therapy
Community-Acquired Pneumonia (CAP)	
Outpatient	Macrolide or doxycycline. If cardiac disease gives beta-lactamase inhibitor and either a macrolide or doxycycline
Hospitalized with CAP	Beta-lactamase inhibitor and either a macrolide or doxycycline
Methicillin-resistant *Staphylococcus aureus* pneumonia	Vancomycin, linezolid, or anti-staphylococcal penicillin. Adjust dose for weight and renal function
Legionella pneumonia	Fluoroquinolone, azithromycin or doxycycline
Pneumocystis jiroveci pneumonia	Sulfa drug; adjust dose for renal function Prednisone if acutely ill
Hospital-acquired (nosocomial) pneumonia	Antipseudomonal quinolone in addition to a beta-lactamase inhibitor or carbapenem
Anaerobe (aspiration)	Beta-lactam or carbapenem
Mycoplasma pneumoniae	Macrolide, a tetracycline or fluoroquinolone

Sedative Agents

Critically ill patients frequently require pharmacological intervention for analgesia, sedation, control of anxiety, and facilitation of mechanical ventilation. The selection of appropriate pharmacological agents is based on the cause of the agitation (Box 25-10), underlying illness, possible adverse effects, history of previous drug use, and cost. Agents most commonly used in the ICU include opiates, benzodiazepines,

BOX 25-10 Etiologies of Agitation in Critically Ill Patients

Pain
Mechanical ventilation
Dyspnea
Hypoxemia
Metabolic disarray
Withdrawal from alcohol or drugs
Anxiety
Sleep deprivation
Immobility
Sepsis
Age
Steroid administration
Alzheimer's disease
Hearing or vision deficit (severe)

haloperidol (Haldol), and propofol (Diprivan). Specifically, haloperidol is recommended for patients with delirium, and opiates are used synergistically to treat pain.

Several agents can be given as bolus doses, by continuous infusion, or by using a combination of the two approaches, although some drugs, such as haloperidol, are limited to bolus dosing. When administering these agents by continuous infusion, it is important to monitor the patient's response closely and adjust the dose to meet his or her individual needs. This is best accomplished by using an objective sedation rating scale for consistent assessment and documentation of the medication's efficacy. Such a protocol can help prevent the prolonged use of these agents and can lower the cumulative amount required for the control of pain or agitation. This can contribute to a decreased length of hospital stay and decreased length of mechanical ventilation.

When using a continuous infusion, if an increase in dosage is necessary, an additional small bolus dose should be given to facilitate rapid increase to the new desired blood level. To prevent withdrawal symptoms, dosages given to patients who have received large amounts of opiates or benzodiazepines for 2 or more weeks must be tapered gradually. For example, the dose may be decreased by 25% a day. Some protocols promote the conversion of benzodiazepine infusions to the enteral route before stopping the infusion. Enteral administration is performed to maintain an appropriate level of sedation and to wean patients who have required prolonged sedation, usually over 7 days. Protocols that include daily tapering or weaning of infusions along with daily sedation interruption are recommended. Another method of nurse-controlled monitoring, bispectral index monitoring, has been studied and found to be useful for controlling sedation in critically ill patients who are receiving sedation.[13]

Neuromuscular Blocking Agents

If metabolic demands and work of breathing continue to compromise ventilatory or hemodynamic stability after maximization of sedation, NMB agents may be required. The goal of therapy with NMB agents is to maximize oxygenation and prevent complications such as barotrauma (alveolar rupture that can result in death), which can be caused by high ventilatory pressures.

The use of NMB agents is usually required if the pressure-controlled inverse ratio mode of ventilation is used. NMB drugs do *not* possess analgesic or sedative properties. When NMB agents are used, sedation and analgesia are required, along with patient and family education. Do not leave a chemically paralyzed patient unattended.

Recent reports of prolonged paralysis following use of NMB agents have prompted many institutions to initiate protocols for instituting, monitoring, and withdrawing these drugs. These range from the use of peripheral nerve stimulators to assess the level of neuromuscular blockade to the routine daily discontinuation of NMB agents to assess neurological status and the need for continued administration.

Commonly used NMB agents are vecuronium (Norcuron), atracurium (Tracrium), and cisatracurium (Nimbex). Each has advantages and disadvantages related to concomitant drug effects, underlying illness, and cost. Both atracurium and cisatracurium have fewer side effects than other NMB agents. Both atracurium and cisatracurium are useful for patients with renal failure because metabolic breakdown of the drugs is in the plasma independent of renal or hepatic function. These two NMB agents are eliminated by ester hydrolysis and Hoffman elimination in the plasma, which is a spontaneous nonenzymatic degradation that is optimal with physiological pH and temperature. Atracurium and cisatracurium may have prolonged duration in the setting of acute acidosis or hypothermia. Atracurium and cisatracurium are good for patients with multisystem organ failure because the other NMB drugs may have more prolonged effects with both renal and hepatic failure than these two. Cisatracurium is less prone than atracurium to trigger histamine release.

▲ Ventilatory Support

When a patient is unable to maintain a patent airway, adequate gas exchange, or both, despite aggressive management with the interventions discussed previously, more invasive support with intubation and mechanical ventilation must be considered. This step carries its own risks and imposes significant physical and psychological burdens on the patient and family. Every effort should be made to avoid mechanical ventilation, but it is usually necessary at the point when respiratory distress becomes respiratory failure.

Respiratory failure is defined as the inability to maintain adequate respiration as measured by arterial blood pH, $PaCO_2$, and PaO_2. Respiratory failure may be categorized as hypoxemic or hypoxemic hypercapnic. Hypoxemic respiratory failure is when PaO_2 is less than 60 mm Hg. Hypoxemic hypercapnic respiratory failure is when PaO_2 is less than 60 mm Hg and $PaCO_2$ exceeds 55 mm Hg. If the ABG values deteriorate beyond these parameters, mechanical ventilatory support is often indicated. Patients are predisposed to developing acute respiratory failure if any of the systems involved in respiration are compromised or overwhelmed (Table 25-3). The degree of risk for developing respiratory failure depends on the patient's ability to move air, secretions, and oxygenated blood. The nurse plays a key role in recognizing the onset of acute respiratory failure.

Table 25-3 Possible Events Leading to Respiratory Failure

Body System	Event
Nervous system	Head trauma
Brainstem	Poliomyelitis
Spinal cord and nerves	Cervical (C1–C6) fractures
	Overdose
Muscular system	
Primary—diaphragm	Myasthenia gravis
Secondary—respiratory	Guillain-Barré
Skeletal system	Flail chest
Thorax	Kyphoscoliosis
Respiratory system	Obstruction
Airways	Laryngeal edema
	Bronchitis
	Asthma
Alveoli	Emphysema
	Pneumonia
	Fibrosis
Pulmonary circulation	Pulmonary embolus
Cardiovascular system	Congestive heart failure
	Fluid overload
	Cardiac surgery
	Myocardial infarction
Gastrointestinal system	Aspiration
Hematological system	Disseminated intravascular coagulation
Genitourinary system	Renal failure

Identification of high-risk patients, serial monitoring and evaluation of respiratory status, and institution of appropriate measures may forestall or negate the need for ventilatory assistance. Before intubation and ventilation, the patient may have required increased FiO_2 to meet his or her oxygen demand.

When ventilatory assistance is required, the objective of mechanical ventilation is to support the patient through an episode of illness. Clinical goals of mechanical ventilation may include reversal of hypoxemia; reversal of acute respiratory acidosis; relief of respiratory distress; prevention or reversal of atelectasis; resting of ventilatory muscles; reduction in systemic oxygen consumption, myocardial oxygen consumption, or both; reduction in intracranial pressure (ICP); and stabilization of the chest wall. Mechanical ventilation is not curative and can actually cause complications (as discussed later in this chapter).

Physiological Principles

To understand the effects of modern mechanical ventilation, the reader is encouraged to review the physiology of normal respirations and lung compliance, as discussed in Chapter 23. The relationship between intrapulmonary pressures during inspiration and expiration is reversed during mechanical ventilation. The ventilator delivers air by pumping it into the patient; therefore, pressures during inspiration are positive. The positive pressure pumped into the lungs results in increased intrathoracic pressures and decreased venous return during inspiration. With the institution of PEEP, even greater pressures are generated during inspiration. During expiration, the pressure in the lungs decreases to the "baseline" PEEP level and continues to be positive throughout expiration. Most patients compensate for this hindrance to venous return by increasing peripheral venous tone. If conditions of decreased sympathetic response (eg, hypovolemia, sepsis, heart disease, drugs, or older age) are present, hypotension may develop. In addition, a large tidal volume (>10 to 12 mL/kg) that generates pressures greater than or equal to 35 cm H_2O not only reduces cardiac output but also increases the risk for pneumothorax.

Positive pressure can result in barotrauma. Barotrauma occurs when air leaks from the alveoli into the pleural space; this is called a pneumothorax. Another form of lung injury is called volutrauma and is caused by delivery of large tidal volumes in patients with stiff, noncompliant lungs. With volutrauma, the alveoli develop fractures that allow fluid and protein to seep into the lungs. This phenomenon is a form of noncardiogenic pulmonary edema. Lung damage from either barotrauma or volutrauma can increase mortality, especially in susceptible patients (such as those with asthma or ARDS). To prevent lung injury, it is important to determine lung compliance so that the ventilator can be appropriately adjusted to minimize airway pressures.

Ventilator-associated lung injury (VALI) and *ventilator-induced lung injury* (VILI) are terms used to describe the damage to lungs from prolonged ventilation. Other causes of VALI and VILI are volutrauma resulting from the overexpansion of alveoli with high ventilation pressures and atelectrauma, which is shear-induced injury from the repeated opening and closing of alveoli. In addition, prolonged high levels of FiO_2, high volumes and pressures leading to the loss of surfactant as well as inflammation of the lung tissue and alveoli, and the primary injury of pneumonia or aspiration result in lung injury while the patient's ventilator is on settings that lead to damage of the lung tissue. Vulnerable patients with ALI or ARDS may be more prone to VALI and VILI. The medical community in the United States has developed a research system to study the diagnosis and treatment of ARDS known as ARDSNet.[14] The ARDSNet protocols for protecting the lungs (from VALI and VILI) when the patient is on mechanical ventilation have recommended the following:

- Keep the plateau pressures at 30 cm H_2O or less.
- Reduce the FiO_2 to 50%.
- Maintain V_T at 5 to 6 mL/kg ideal body weight or less.
- Maintain PEEP to avoid collapse of alveoli at the end of expiration.[15]

Compliance

Compliance refers to the ability of the lung to distend. In terms of its compliance, the lung is frequently compared to a balloon. Initially, it is difficult to inflate (noncompliant) until it is stretched. After repeated inflations, this elastic resistance is lost (overly compliant), and the balloon becomes very easy to blow up. In conditions that reduce the lung's elasticity, such as inflammation, fibrotic changes, or edema, the lung requires more force to inflate. A patient who has

BOX 25-11 Factors Decreasing Compliance

Airway Factors
Peak flow
Size of airways
Airway obstructions
External obstructions (kinked ventilator tubing or water in the tubing)

Lung Factors
Elasticity (stiffness) of the lung
Presence of auto-PEEP
Shunt (ARDS)

Chest Wall Factors
Chest wall deformities
Position of patient
External compression of chest wall or diaphragm (distended abdomen, obesity)

normal lungs and who is on a ventilator should have compliance near 100 mL/cm H_2O (normal). In contrast, a patient who has pulmonary disease and who is on a ventilator that causes "stiff" lungs (eg, ARDS, sarcoidosis) has a compliance as low as 20 to 30 mL/cm H_2O, indicating a severely compromised lung.

As the volume of gas is delivered to a patient on a mechanical ventilator, the ventilator's pressure gauge slowly rises from zero to peak inspiratory pressure (PIP). The rise in pressure is caused by airway resistance (to flow) as well as by lung and chest wall compliance (Box 25-11). A graph of pressure over time, depicting inspiration, would look like the example shown in Figure 25-6. Dynamic pressures and PIP can give an indication of both airway resistance and lung compliance.

Static Pressure

One of the measurements used to obtain compliance is static pressure (SP) or plateau pressure. Plateau pressure is obtained by pushing the end-inspiratory hold button on a ventilator at the end of a maximal inspiration while on a volume mode of ventilation. This holds the volume of delivered air in the patient's chest by preventing exhalation. The PIP drops to a plateau pressure with this maneuver, which reflects the pressure necessary to hold the lungs open. A graph depicting SP and PIP can be seen in Figure 25-7. Static compliance is determined by dividing the tidal volume by the plateau pressure minus the total PEEP:

$$\text{Exhaled } V_T / [\text{plateau pressure} - \text{PEEP}] = \text{static compliance}$$

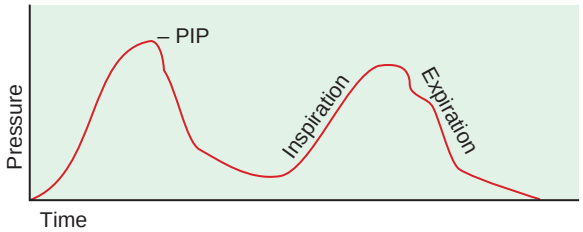


FIGURE 25-6 ▲ Graph displaying peak inspiratory pressure (PIP).

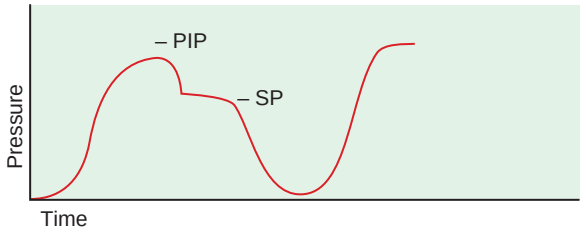


FIGURE 25-7 ▲ Graph depicting static pressure (SP) and peak inspiratory pressure (PIP).

A higher compliance means that the lung is more easily distended, whereas a lower compliance means that the lung is stiffer and difficult to distend. In other words, a higher compliance is better. Low compliance may be due to stiff lungs as with ARDS, a restrictive chest wall (ie, kyphoscoliosis), or ventilation of only a small portion of lung, such as occurs with partial lung collapse from consolidation. Serial measurements of compliance performed by the respiratory care professional can alert the nurse to sudden decreases, which may be due to pneumothorax, mucous plugging, or pulmonary edema.

Equipment

Many different ventilatory support systems are available. Manual resuscitators are typically used in emergencies, such as acute respiratory failure. Several types of mechanical ventilators are in use, and they offer a variety of modes.

Manual Resuscitators

The nurse's first line of defense for acute respiratory failure is the MRB, sometimes referred to as an Ambu bag or BVM apparatus. During CPR or hyperinflation with bagging and suctioning of any mechanically ventilated patient, MRBs with reservoirs must be used and connected to an oxygen source to deliver 74% to 100% concentrations of oxygen. MRBs without reservoirs may deliver a lower FiO_2 but must also be connected to an oxygen source.

Knowledge of the bag and skill in using it are vital. The function of this simple ventilator can be compared with that of the more sophisticated models. The following guidelines pertain to the manual resuscitator:

- The force of squeezing the bag determines the tidal volume delivered to the patient.
- The number of hand squeezes per minute determines the assisted respiratory rate.
- The force and rate that the bag is squeezed determine the peak flow.

While the bag is being used, the nurse must carefully observe the patient's chest rise to determine whether the bag is ventilating properly and whether any gastric (abdominal) distention is developing. In addition, the amount of resistance encountered can roughly indicate lung compliance. If a patient becomes progressively more difficult to ventilate, an increase in secretions, pneumothorax, worsening bronchospasms, or other condition that might decrease the patient's compliance must be considered. Breaths delivered to a conscious patient must be timed to coincide with spontane-

ous inspiratory effort, or the discomfort of dyssynchronous breathing will create anxiety. Then the patient will not tolerate the additional ventilation.

When delivering breaths with an MRB, the nurse allows time for complete exhalation between breaths to prevent air trapping (referred to as auto-PEEP), which can cause hypotension and barotrauma, especially in patients with obstructive airway disease.

Mechanical Ventilators

The goal of mechanical ventilation is to maintain alveolar ventilation appropriate for the patient's metabolic needs and to correct hypoxemia and maximize oxygen transport. Ventilators are classified into two categories: negative-pressure ventilators and positive-pressure ventilators. Regardless of which type or model is used, the nurse must be familiar with the ventilator's function and limitations. The following discussion of ventilators is in order of evolution of ventilator technology and subsequent use in clinical practice.

Negative-Pressure Ventilators

Early negative-pressure ventilators were known as "iron lungs." The patient's body was encased in an iron cylinder, and negative pressure was generated by a large piston to enlarge the thoracic cage. This caused alveolar pressures to fall, and a pressure gradient was formed so that air flowed into the lungs. The iron lung was used most frequently during the poliomyelitis epidemics of the 1930s and 1940s, but iron lungs are rarely used today. Intermittent short-term negative-pressure ventilation is sometimes used in patients with chronic diseases. Rarely, this method of support is chosen for patients who are not candidates for aggressive mechanical ventilation as provided through an artificial airway. These patients suffer from a wide variety of conditions such as COPD, diseases of the chest wall (kyphoscoliosis), and neuromuscular diseases (Duchenne's muscular dystrophy, amyotrophic lateral sclerosis [ALS]).

The iron lung is cumbersome to use and very large. Most negative-pressure ventilators in use today are more portable. To improve mobility and comfort, there is a device that fits like a tortoise shell, forming a seal over the chest. A hose connects the shell to a negative-pressure generator. The thoracic cage is literally pulled outward to initiate inspiration. However, the use of negative-pressure ventilators is restricted in clinical practice; they limit positioning and movement and lack adaptability to large or small body torsos.

Positive-Pressure Ventilators

VOLUME VENTILATORS. The volume ventilator is commonly used in critical care settings. The basic principle of this ventilator is that a designated volume of air is delivered with each breath. The amount of pressure required to deliver the set volume depends on the patient's lung compliance and patient–ventilator resistance factors. Therefore, PIP must be monitored in volume modes because it varies from breath to breath. With this mode of ventilation, a respiratory rate, inspiratory time, and tidal volume are selected for the mechanical breaths.

PRESSURE VENTILATOR. The use of pressure ventilators is increasing in critical care units. A typical pressure mode delivers a selected gas pressure to the patient early in inspiration and sustains the pressure throughout the inspiratory phase. By meeting the patient's inspiratory flow demand throughout inspiration, patient effort is reduced and comfort increased. Although pressure is consistent with these modes, volume is not. Volume will vary based on changes in resistance or compliance. Therefore, exhaled tidal volume is the variable to monitor closely. With pressure modes, the pressure level to be delivered is selected, and with some mode options (described later), rate and inspiratory time are preset as well. Figure 25-8 shows a typical ventilator with computer-controlled system and multiple screens showing monitoring data and mode of ventilation.

HIGH-FREQUENCY VENTILATOR. The high-frequency ventilator accomplishes oxygenation by the diffusion of oxygen and carbon dioxide from high to low gradients of concentration. This diffusion movement is increased if the kinetic energy of the gas molecules is increased. High-frequency ventilators use small tidal volumes (1 to 3 mL/kg) at frequencies greater than 100 breaths/min. The breathing pattern of a person on a high-frequency ventilator is somewhat analogous to the breathing pattern of a panting dog; panting entails moving small volumes of air at a very fast rate.

Theoretically, a high-frequency ventilator would be used to achieve lower peak ventilatory pressures, thereby reducing the risk for barotrauma and improving ventilation–perfusion matching because of its different flow delivery characteristics. Clinical data showing that use of high-frequency oscillatory ventilation improves outcomes in adults are limited so far; however, studies have revealed improvement in oxygenation with the ability to transfer back to conventional ventilation.[16] Potential adverse effects associated with high-frequency ventilators include gas trapping and necrotizing tracheobronchitis, when used in the absence of adequate humidification.

Ventilator Modes

Several different modes of ventilatory control are available on ventilators. Figure 25-9 and Table 25-4 compare these modes. Volume modes include assist-control (A/C) mode and synchronized intermittent mandatory ventilation (SIMV) mode. Pressure modes include pressure-support ventilation (PSV) mode, pressure-controlled ventilation (PCV) mode, airway pressure release ventilation (APRV) mode, volume-guaranteed pressure options (VGPO) mode, CPAP/PEEP mode, and noninvasive BiPAP mode. There is no one best mode for managing patients in respiratory failure, although each mode has its advantages and disadvantages.

Volume Modes

ASSIST-CONTROL MODE. In assist control, or volume-control mode as it is often termed, a mandatory (or "control") rate is selected. If the patient wishes to breathe faster, he or she can trigger the ventilator and receive a full-volume breath. This mode of ventilation is often used to fully support a patient, such as when the patient is first intubated or when the patient is too weak to perform the work of breathing (eg, when emerging from anesthesia).

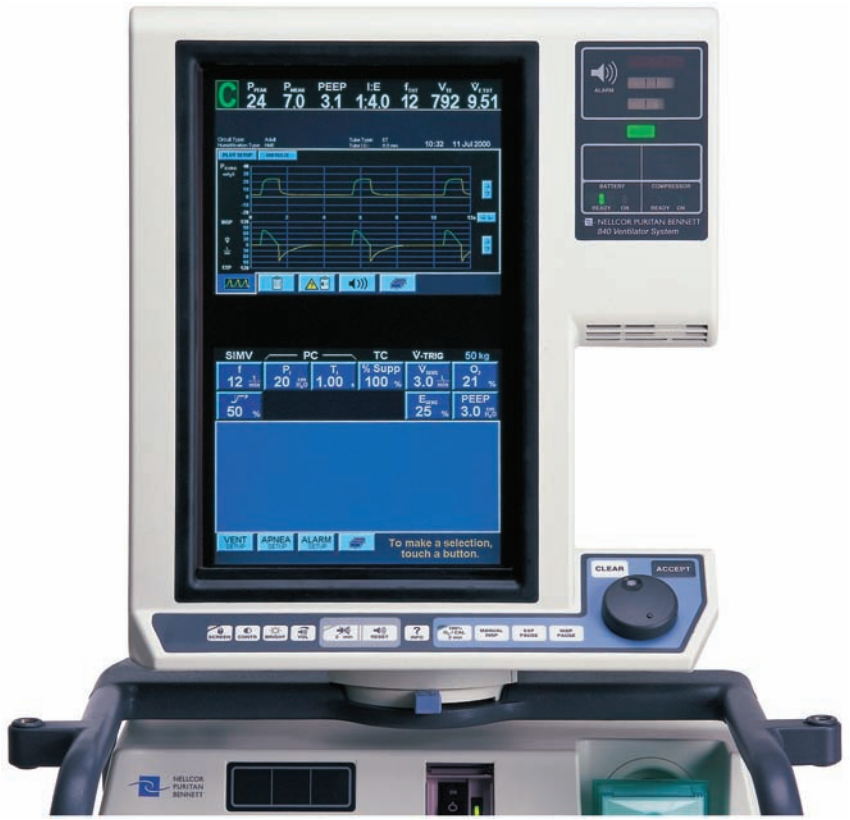


FIGURE 25-8 ▲ The Puritan-Bennett 840 (PB 840) Ventilator System. The close-up photos of the screens and controls are an example of the type of controls used with computer-controlled ventilators.

The controls on the PB 840 are simple to use. This ventilator has a battery system allowing for transport with bottled oxygen. Dual screens display monitoring and alarm data in the upper screen and ventilator settings in the lower screen. On the top of the upper screen are the patient's ventilator parameters, indicating respiratory rate, tidal volume, minute volume, PEEP, IPL, and other values. Primary ventilator controls are accessed by screen touch boxes, and then a value or mode is selected by turning the select knob on the right followed by touching the "Accept" button for changes made. There is also a cancel button to remove changes before entering them. The nurse should be familiar with each ventilator system; each uses similar controls, but configurations of screens and touch buttons differ. The PB 840 also has additional quick action buttons along the bottom of the controls for 100% FiO_2 setting that lasts 2 minutes. This function allows preoxygenation before suctioning, or if the SaO_2 drops, it can improve oxygenation until troubleshooting of the patient and ventilator identifies the problem. An alarm pause button along the bottom controls only stops alarms for 2 minutes during therapy such as suctioning. Current alarms are viewed on the upper display screen, with the two most urgent alarms visible for review. There is a three-tiered system of warning lights on the right upper corner, with red (three lights blink rapidly) for high-urgency situations that require immediate attention, yellow (two lights blink slowly) for medium-urgency situations that require prompt attention, and yellow (one light is steadily lit) for low-urgency situations that indicate a change in the ventilator system. Alarm sounds also increase to the highest level continuous with reduced intensity or duration sounds for the other alarm levels. There is an alarm reset button that resets the alarms, but if the condition persists, the alarm reactivates until the problem is corrected with the patient or the ventilator. Only registered respiratory therapists should enter the ventilator setting controls for adjustments. The nurse should be familiar with the alarm pause and 100% FiO_2 setting. He or she should know the alarm levels to respond immediately for high-urgency situations and be able to know the meaning of the alarm problem displayed in the upper screen for this ventilator. Familiarity with this and other ventilator manufacturers' alarm systems and controls is essential for every nurse who cares for ventilated patients.

SYNCHRONIZED INTERMITTENT MANDATORY VENTILATION MODE. In SIMV mode, the rate and tidal volume are preset. If the patient wants to breathe above this rate, he or she may. However, unlike the A/C mode, any breaths taken above the set rate are spontaneous breaths taken through the ventilator circuit. The tidal volume of these breaths can vary drastically from the tidal volume set on the ventilator because the tidal volume is determined solely by the patient's spontaneous effort. Adding pressure support (discussed in the next section) during spontaneous breaths can minimize the risk for increased work of breathing. In the past, SIMV was used as a popular weaning mode. To wean the patient, the mandatory breaths were gradually decreased, thereby allowing the patient to assume more and more of the work of breathing.

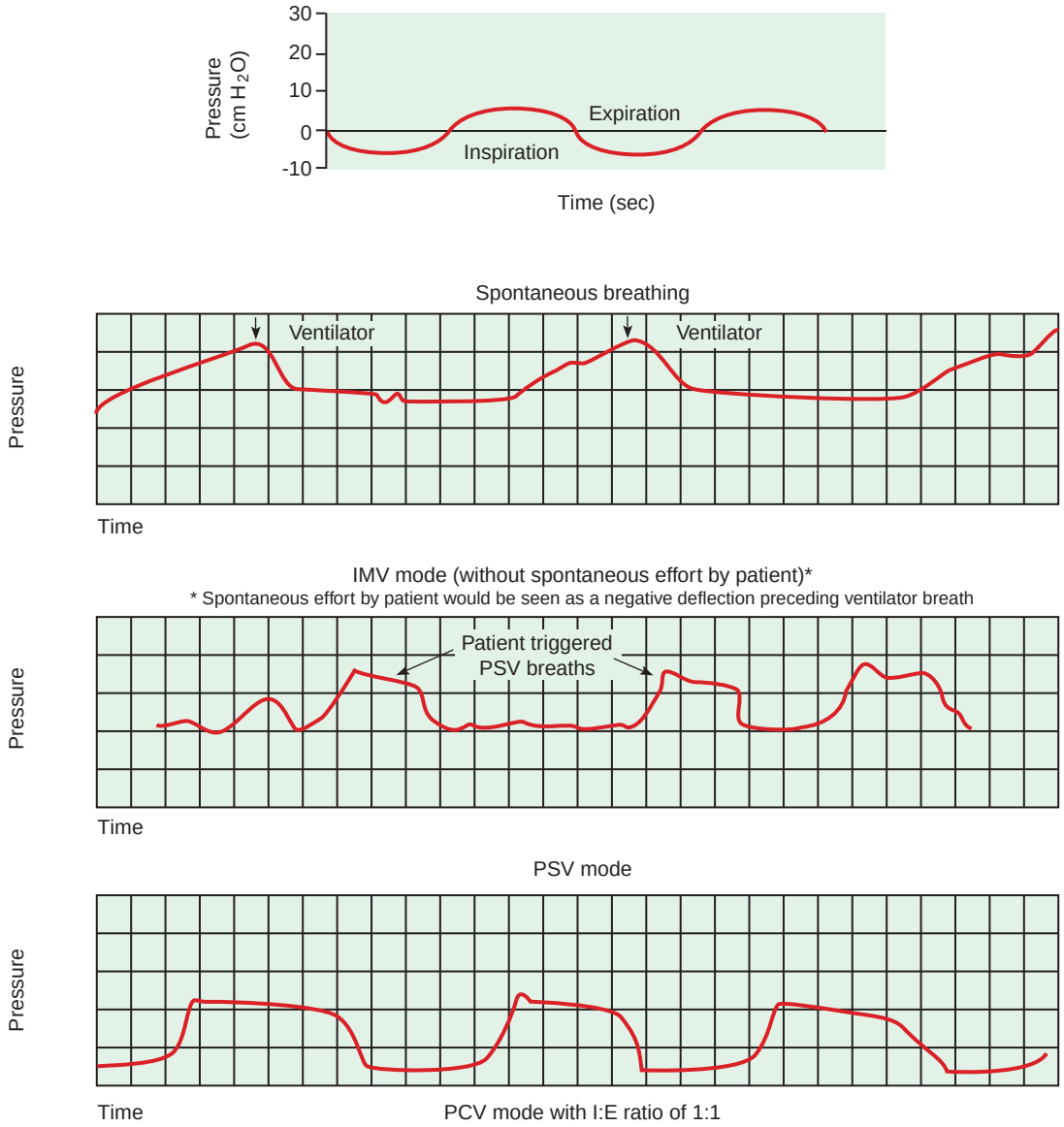


FIGURE 25-9 ▲ Comparison of ventilatory modes using continuous airway pressure monitoring.

Pressure Modes

PRESSURE-SUPPORT VENTILATION (PSV) MODE. PSV mode augments or assists spontaneous breathing efforts by delivering a high flow of gas to a selected pressure level early in inspiration and maintaining that level throughout the inspiratory phase. The patient's effort determines the rate, inspiratory flow, and tidal volume. When PSV mode is used as a stand-alone mode of ventilation, the pressure-support level is adjusted to achieve the approximate targeted tidal volume and respiratory rate. At high-pressure levels, PSV mode provides nearly total ventilatory support.

Specific uses of PSV are to promote patient comfort and synchrony with the ventilator, to decrease the work of breathing necessary to overcome the resistance of the ETT, and for weaning. As a weaning tool, PSV is thought to increase the endurance of the respiratory muscles by decreasing the physical work and oxygen demands during spontaneous breathing. Because the level of pressure support can be gradually decreased, endurance conditioning is enhanced.

In PSV mode, the inspired tidal volume and respiratory rate must be monitored closely in order to detect changes in lung compliance. Generally, if compliance decreases or resistance increases, tidal volume decreases and respiratory rate increases. PSV mode should be used with caution in patients with bronchospasm or other reactive airway conditions.

PRESSURE-CONTROLLED VENTILATION (PCV) MODE. The PCV mode is used to control plateau pressures in conditions, such as ARDS, in which compliance is decreased and the risk for barotrauma is high. It is used when the patient has persistent oxygenation problems despite a high FiO_2 and high levels of PEEP. The inspiratory pressure level, respiratory rate, and inspiratory/expiratory (I:E) ratio must be selected. Tidal volume varies with compliance, and airway resistance must be closely monitored. Sedation and

Table 25-4 Comparison of Modes of Ventilation

Ventilatory Mode	Indications	Advantages/Disadvantages	Special Monitoring
Assist-control (A/C)	Often used as initial mode of ventilation	Advantages: Ensures vent support during every breath Each breath same tidal volume Disadvantages: Hyperventilation, air trapping; may require sedation and paralysis	Work of breathing may be increased if sensitivity or flow rate is too low.
Synchronized intermittent mandatory ventilation (SIMV)	Often used as initial mode of ventilation and for weaning	Advantages: Allows spontaneous breaths (tidal volume determined by patient) between vent breaths; weaning is accomplished by gradually lowering the set rate and allowing the patient to assume more work Disadvantages: Patient–ventilator asynchrony possible	
Pressure-support ventilation (PSV)	Intact respiratory drive in patient necessary Used as a weaning mode, and in some cases of dyssynchrony	Advantages: Decreases work of breathing; increases patient comfort; can be combined with SIMV to allow a more comfortable mode Disadvantages: Should not be used in patients with acute bronchospasm or with altered mental status with reduced spontaneous breathing	Adjust PSV level to maintain desired respiratory rate and tidal volume. Monitor for changes in compliance, which can cause tidal volume to change. Monitor respiratory rate and tidal volume at least hourly.
Pressure-controlled ventilation (PCV)	Used to limit plateau pressures that can cause barotrauma Severe ARDS	Disadvantages: Patient–ventilator asynchrony possible, necessitating sedation/paralysis	Monitor tidal volume at least hourly. Monitor for barotrauma, hemodynamic instability.
Inverse ratio ventilation (IRV)	Usually used in conjunction with PCV	Advantages: Increases I:E ratio to allow for recruitment of alveoli and improve oxygenation Disadvantages: Almost always requires paralysis	
Volume-guaranteed pressure options (VGPO)	Combines advantages of pressure ventilation with guaranteed tidal volume	Advantages: Ensures a delivered tidal volume Disadvantages: Requires sophisticated knowledge of the mode and waveform analysis	Monitor for auto-positive end-expiratory pressure, barotrauma, and hemodynamic instability.
Continuous positive airway pressure (CPAP)	Constant positive airway pressure for patients who breathe spontaneously	Advantages: Used in intubated or nonintubated patients Disadvantages: On some systems, no alarm if respiratory rate falls	Monitor for increased work of breathing.
Noninvasive bilateral positive-pressure ventilation (BiPAP)	Nocturnal hypoventilation in patients with neuromuscular disease, chest wall deformity, obstructive sleep apnea, and chronic obstructive pulmonary disease; to prevent intubation; to prevent reintubation initially after extubation	Advantages: Decreased cost when patients can be cared for at home; no need for artificial airway Disadvantages: Patient discomfort or claustrophobia	Monitor for gastric distention, air leaks from mouth, aspiration risk.

the use of NMB agents are frequently indicated because any patient–ventilator asynchrony usually results in profound drops in the SaO_2. This is especially true when inverse ratios are used. The "unnatural" feeling of this mode often requires muscle relaxants to ensure patient–ventilator synchrony.

Most ventilators operate with a short inspiratory time and a long expiratory time (1:2 or 1:3 ratio). This promotes venous return and allows time for air to passively exit the lungs. Inverse ratio ventilation mode reverses this ratio so that inspiratory time is equal to, or longer than, expiratory time (1:1 to 4:1). Inverse I:E ratios are used in conjunction with pressure control to improve oxygenation in patients with ARDS by expanding stiff alveoli using longer distending times, thereby providing more opportunity for gas exchange and preventing alveolar collapse.

As expiratory time is decreased, the nurse must monitor for the development of hyperinflation or auto-PEEP. Regional alveolar overdistention and barotrauma may result from excessive total PEEP. When the PCV mode is used, the mean airway and intrathoracic pressures rise, potentially resulting in a decrease in cardiac output and oxygen delivery. Therefore, it is necessary to monitor the patient's hemodynamic status closely.

Airway Pressure Release Ventilation (APRV) Mode

APRV has been used in trauma and ARDS patients to reduce airway pressure and lower minute volume while allowing spontaneous breathing throughout the ventilator cycle, all with decreased sedation and NMB agent use. APRV mode allows lung protective strategies to be followed with limitation of plateau and peak pressures. The mode functions by having a time-triggered, pressure-limited, time-cycled mode of ventilation. It consists of a high-pressure setting and a low-pressure setting, with recruitment and oxygenation occurring during the high-pressure setting at a long set time interval (eg, 5 seconds) followed by a brief controlled release (eg, 0.6 second) to the low-pressure setting. What this means is that the patient spontaneously breathes both at a set high pressure with preset brief times and at low pressure, which is synchronized during exhalation. Because the patient is breathing spontaneously throughout both high- and low-pressure phases, sedation may be limited. Weaning from APRV is done by decreasing the high-pressure limit while increasing the time at high pressure. At the same time, the low-pressure limit may be dropped, allowing for reduced mean airway pressure. Usually, the low-pressure limit is reduced to 5 cm H_2O, and as the high pressure is lowered, this allows release to a PEEP level that prevents derecruitment. When the patient tolerates an FiO_2 of 50% or less, the patient can be switched to PSV and further weaning. This mode may improve oxygenation and prevent VALI and VILI in patients with ARDS or ALI.[17]

Volume-Guaranteed Pressure Options (VGPO) Mode

VGPO mode ensures delivery of a prescribed tidal volume while using a decelerating flow pattern by means of a "pressure" breath. The options include both spontaneous and control rate parameters, and the volume guarantee is provided differently, depending on the ventilator. VGPO can be used in acutely ill patients as well as more stable, weaning patients. Some examples include the volume support and pressure-regulated volume-control options (Siemens Medical) as well as pressure augmentation (Bear Medical Systems).

In the acutely ill, unstable patient, this option may provide pressure ventilation while guaranteeing tidal volume and minute ventilation (MV) at a set rate. In the spontaneously breathing patient, the option is used as a "safety" when pressure ventilation is desired. The use of a volume guarantee in the spontaneously breathing patient may be especially important at night (when respiratory rates and volumes normally decrease) and in patients for whom secretions are a problem (because secretions increase resistance and result in decreased spontaneous volumes).

Continuous Positive Airway Pressure (CPAP)/ Positive End-Expiratory Pressure (PEEP) Mode

CPAP is the term used when PEEP is supplied during spontaneous breathing. PEEP is the term used to describe positive end-expiratory pressure with positive-pressure breaths. CPAP assists spontaneously breathing patients to improve their oxygenation by elevating the end-expiratory pressure in the lungs throughout the respiratory cycle. CPAP can be used for intubated and nonintubated patients. It may be used as a weaning mode and for nocturnal ventilation (nasal or mask CPAP) to splint open the upper airway, preventing upper airway obstruction in patients with obstructive sleep apnea.

PEEP is positive pressure exerted at the end of exhalation. It is common practice to use low levels of PEEP (2 to 5 cm H_2O) in the intubated patient. PEEP is increased in 2- to 5-cm H_2O increments when FiO_2 levels are greater than 50% to attain an acceptable SaO_2 (>90%) or PaO_2 (>60 to 70 mm Hg). PEEP is most often necessary in patients with refractory hypoxemia (such as those with ARDS) in whom the PaO_2 deteriorates rapidly, despite greater concentrations of oxygen administration.

PEEP is used to keep alveoli stent open, and it may recruit alveolar units that are totally or partially collapsed during any mode of ventilation. This end-expiratory pressure increases the functional residual capacity (FRC) by reinflating collapsed alveoli, maintains the alveoli in an open position, and improves lung compliance. This decreases shunt and improves oxygenation. In addition, there is some evidence that keeping the alveoli open enhances surfactant regeneration. High levels of PEEP should rarely be interrupted because it may take several hours to recruit alveoli again and restore the FRC; until this occurs, oxygenation may suffer. In the patient who does not have adequate circulating blood volume, institution of PEEP decreases venous return to the heart, decreases cardiac output, and decreases oxygen delivery to the tissues. If hypotension or decreased cardiac output results from PEEP application, restoring circulating intravascular volume with administration of IV fluids may correct the hypotension. Another serious complication of PEEP is barotrauma. It can occur in any mechanically ventilated patient but is most common when high levels of PEEP are used (10 to 20 cm H_2O or more) in lungs with high ventilating pressures and low compliance and in patients with obstructive airway disease. The development of barotrauma is an emergency and usually requires placement of a chest tube in the event of pneumothorax.

Noninvasive Bilateral Positive-Pressure Ventilation Mode

BiPAP is a noninvasive form of mechanical ventilation provided by means of a nasal mask, nasal prongs, or a full face mask. It is used in the treatment of patients with chronic respiratory insufficiency to manage acute or chronic

respiratory failure without intubation and conventional mechanical ventilation. It is also used as a bridge to weaning patients from mechanical ventilation and as an alternative to conventional mechanical ventilation in patients who are ventilated in their homes. The system allows the clinician to select two levels of positive-pressure support: an inspiratory pressure-support level (referred to as IPAP) and an expiratory pressure called EPAP (PEEP/CPAP level). Because BiPAP allows for the provision of assisted inspiration with ventilator rate set, application of this mode to those patients who hypoventilate as well as obstruct during sleep is possible.

BiPAP is beneficial in patients with worsening hypoventilation, obstructive apneic episodes, or both. It is also useful to prevent intubation in patients with respiratory failure and hypercarbia as well as to prevent reintubation following extubation in borderline cases. Use of a full face mask may increase the risk for aspiration and risk for rebreathing carbon dioxide; therefore, ventilation with a full face mask should be used cautiously. Thick or copious secretions and poor cough may be relative contraindications for BiPAP.

Use of Mechanical Ventilators

Setting Ventilator Controls

The nurse must know how to monitor the various ventilators, modes, and controls before giving mechanical ventilatory support to a patient. The following section discusses these various controls and settings and their implications for nursing care. In some institutions, the respiratory therapists share or have complete responsibility for managing the ventilator, but the nurse still needs to be fully aware of the implications for the patient of the mode and level of mechanical support.

Ventilator settings must be frequently evaluated against patient response. Iatrogenically induced complications include overventilation (which causes respiratory alkalosis) and underventilation (which causes respiratory acidosis or hypoxemia). ABG studies determine the effectiveness of mechanical ventilation. Patients with chronic pulmonary disease, however, should be ventilated to stay relatively close to their normal ABG values. This usually means accepting relatively high carbon dioxide levels, lower-than-average oxygenation, or both.

Fraction of Inspired Oxygen

Ventilators allow for adjustment of oxygen percentage (FiO_2) with in-circuit or external oxygen analyzers, thus allowing the nurse to ascertain the FiO_2 being delivered. Changes in FiO_2 are based on ABG values and the SaO_2. Usually, the FiO_2 is adjusted to maintain an SaO_2 of greater than 90% (roughly equivalent to a PaO_2 > 60 mm Hg). Oxygen toxicity is a concern when an FiO_2 of greater than 60% is required for more than 24 hours; therefore, most clinicians attempt to use strategies to allow for maintaining an FiO_2 of 60% or less.

Respiratory Rate

The number of breaths per minute delivered to the patient can be directly set on most ventilator models. Ventilator monitoring should be performed frequently as to appropriate settings, patient response, and airway patency. In the pressure ventilator, the inspiratory time determines the duration of inspiration by regulating the gas flow rate. The higher the flow rate, the faster peak airway pressure is reached and the shorter the inspiration; conversely, the lower the flow rate, the longer the inspiration. A very high flow rate may produce turbulence, shallow inspirations, and uneven distribution of volume.

Respiratory rate times tidal volume equals minute ventilation (RR × VT = MV). In turn, minute volume determines alveolar ventilation. These two parameters are adjusted according to the $PaCO_2$. Increasing the minute volume decreases the $PaCO_2$; conversely, decreasing the minute volume increases the $PaCO_2$. In special cases, hypoventilation or hyperventilation is desired. For example, in a patient with a head injury, respiratory alkalosis may be required to promote cerebral vasoconstriction, with a resultant decrease in ICP. In this case, the tidal volume and respiratory rate are increased to achieve the desired alkalotic pH by manipulating the $PaCO_2$. In contrast, a patient with COPD whose baseline ABG values reflect an elevated $PaCO_2$ should not be hyperventilated. Instead, the goal should be restoration of the baseline $PaCO_2$. These patients usually have a large carbonic acid load, and lowering their carbon dioxide levels rapidly may result in seizures. Rate adjustments may also be necessary to enhance patient comfort or when rapid rates cause air trapping that results in auto-PEEP.

Tidal Volume

In the volume ventilator, the number of milliliters of air to be delivered with each breath is set by the clinician. Traditionally, tidal volumes of 10 to 15 mL/kg of body weight were used. Research has identified a phenomenon of iatrogenic lung injury (VILI or VALI), in which forces produced in the lungs by the large tidal volumes may aggravate the damage inflicted on the lungs by the pathological process that necessitated mechanical ventilation.[18] For this reason, lower tidal volume targets (5 to 8 mL/kg) are now recommended.

Peak Flow

Peak flow is the velocity of gas flow per unit of time and is expressed as liters per minute. On many volume ventilators, this is a separate setting. If auto-PEEP (due to inadequate expiratory time) is present, peak flow is increased to shorten inspiratory time so that the patient may exhale completely. However, increasing peak flow increases turbulence, which is reflected in increasing airway pressures.

Inspiratory Pressure Limit

On volume-cycled ventilators, the inspiratory pressure limit (IPL) control limits the highest pressure allowed in the ventilator circuit. Once the high-pressure limit is reached, inspiration is terminated. Therefore, if the IPL is being constantly reached, the designated tidal volume is not being delivered to the patient. The cause of this can be coughing, accumulation of secretions, kinked ventilator tubing, pneumothorax, decreasing compliance, or a pressure limit alarm set too low. IPL is used with PSV to adjust the pressure during spontaneous breathing, providing reduced work of breathing. Weaning can be accomplished in the PSV mode, reducing IPL to low levels as the patient increases the work of breathing.

Positive End-Expiratory Pressure

The PEEP control adjusts the pressure that is maintained in the lungs at the end of expiration. PEEP and CPAP can be visualized on the respiratory pressure gauge or display. Instead of returning to zero (atmospheric pressure) at the end of expiration, the pressure value drops to the PEEP/CPAP level. PEEP reduction is considered if the patient has a PaO_2 of 80 to 100 mm Hg or an FiO_2 of 50% or less, is hemodynamically stable, and has stabilization or improvement of the underlying illness. To evaluate whether the effects of PEEP are beneficial, monitoring ABG values, SaO_2, compliance, and hemodynamic pressures (including cardiac output and blood pressure) is necessary. Baseline values are obtained before changes in PEEP are made. PEEP is usually increased in increments of 2 to 5 cm H_2O. The patient is monitored for adverse effects, such as hypotension and dysrhythmias. If these occur, the PEEP is reduced. If higher PEEP is tolerated, the patient is stabilized on the new PEEP settings for approximately 15 minutes. The monitored parameters are then repeated.

Hemodynamic measurements (cardiac output, pulmonary artery pressure [PAP], central venous pressure, and pulmonary artery occlusion pressure) are taken at end expiration with the patient on PEEP. Accuracy in selecting the point of end expiration on the waveform tracing is facilitated by using continuous airway monitoring (Fig. 25-10). PEEP does not need to be discontinued before obtaining hemodynamic measurements. Hemodynamic measurements can be inaccurate (as an indicator of volume status) if a patient is on high PEEP or the position of the transducer is not leveled at the phlebostatic axis. The position of the catheter within the pulmonary circulation should also be verified on a chest radiograph.

Attempts are made to minimize removing the patient from the ventilator when using high levels of PEEP. Oxygenation can deteriorate and be slow to rebound because it takes a significant amount of time for the effects of PEEP to be reestablished. Therefore, if the patient is being oxygenated using an MRB, it must be equipped with a valve that allows levels of PEEP to be dialed in. An in-line suction apparatus may be helpful to prevent breaking the PEEP circuit to suction the patient.

Sensitivity

The sensitivity function controls the amount of patient effort needed to initiate an inspiration, as measured by negative inspiratory effort. Increasing the sensitivity (requiring less negative force) decreases the amount of work the patient must do to initiate a ventilator breath. Likewise, decreasing the sensitivity increases the amount of negative pressure that the patient needs to initiate inspiration and increases the work of breathing.

Responding to Alarms

Mechanical ventilators are used to support life. Alarm systems are necessary to warn the nurse of developing problems. Alarm systems can be categorized according to volume and pressure, high and low. Low-pressure alarms warn of disconnection of the patient from the ventilator or circuit leaks. High-pressure alarms warn of rising pressures. Electrical failure alarms are necessary for all ventilators. A nurse or respiratory therapist must respond to every ventilator alarm. Alarms must never be ignored or disarmed. Some clinical troubleshooting guidelines are presented in Table 25-5.

Ventilator malfunction is a potentially serious problem. Nursing or respiratory therapists perform ventilator checks every 2 to 4 hours, and recurrent alarms may alert the clinician to the possibility of an equipment-related issue. When device malfunction is suspected, a second person manually ventilates the patient while the nurse or therapist looks for the cause. If a problem cannot be promptly corrected by ventilator adjustment, a different machine is procured so that the ventilator in question can be taken out of service for analysis and repaired by technical staff.

Ensuring Humidification and Thermoregulation

Mechanical ventilation bypasses the upper airway, thereby negating the body's protective mechanism for humidifying and warming inspired air. These two processes must be added to the ventilator circuit in the form of a humidifier with a temperature control. All air delivered by the ventilator passes through the water in the humidifier, where it is warmed and saturated, and this decreases insensible water loss. In most instances, the temperature of the air is about the same as body temperature. In some rare instances (severe hypothermia), the air temperatures can be increased. Caution is advised because prolonged inhalation of gas at high temperatures can cause tracheal burns. An empty humidifier contributes to drying the airway, often with resultant mucous plugging and less ability to suction out secretions. A heat and moisture

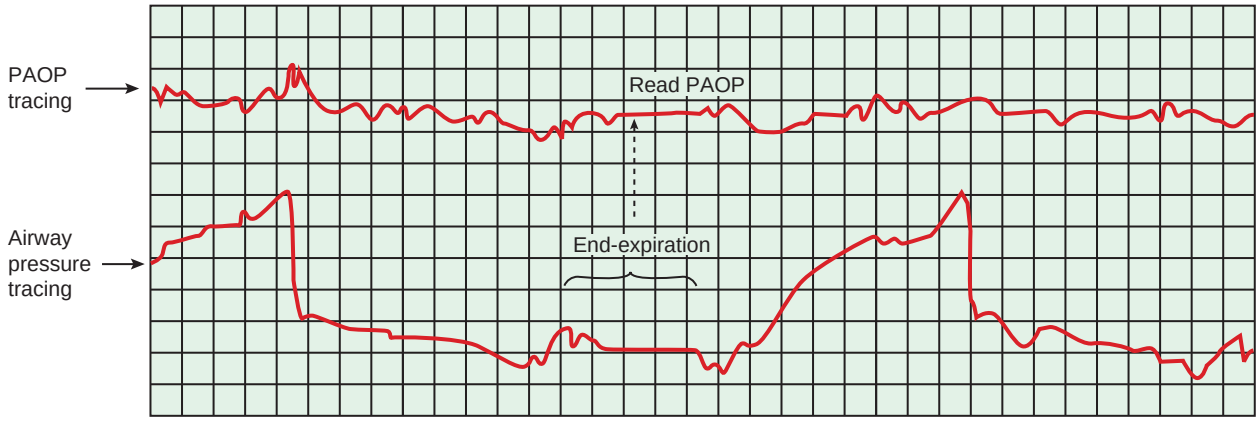


FIGURE 25-10 ▲ Use of continuous airway pressure monitoring to assist in identifying point of end expiration.

Table 25-5 Troubleshooting the Ventilator

Problem	Possible Causes	Action
Volume or pressure alarm	*Patient related*	
	Patient disconnected from ventilator	Reconnect STAT. Auscultate neck for possible leak around endotracheal tube (ETT) cuff.
	Loss of delivered V_T	Review chest radiograph for ETT placement—may be too high. Check for loss of V_T through chest tube.
	Decrease in patient-initiated breaths	Evaluate patient for cause: check respiratory rate, arterial blood gases (ABGs), last sedation.
	Increased compliance	May be due to clearing of secretions or relief of bronchospasms.
	Ventilator related	
	Leaks	Check all tubing for loss of connection, starting at patient and moving toward humidifier. Check for change in ventilator settings. (*Note*: If problem is not corrected STAT, use mandatory resuscitation bag until ventilator problem is corrected.)
High-pressure or peak-pressure alarm	*Patient related*	
	Decreased compliance Increased dynamic pressures	Suction patient. Administer inhaled β-agonists. If sudden, evaluate for pneumothorax. Evaluate chest radiograph for ETT placement in right mainstem bronchus. Sedate if patient is bucking the ventilator or biting the ETT.
	Increased static pressure	Evaluate ABG values for hypoxia, fluids for overload, chest radiograph for atelectasis. Auscultate breath sounds.
	Ventilator related	
	Tubing kinked	Check tubing.
	Tubing filled with water	Empty water into a receptacle: Do not drain back into the humidifier.
	Patient–ventilator asynchrony	Recheck sensitivity and peak flow settings. Provide sedation/paralysis if indicated.
Abnormal (ABGs)	*Patient related*	
Hypoxemia	Secretions	Suction. Increase FiO_2.
	Increase in disease pathology	Evaluate patient and chest radiograph.
	Positive fluid balance	Evaluate intake and output.
Hypocapnia	Hypoxia	Evaluate ABG values and patient.
	Increased lung compliance	Evaluate potential for weaning.
Hypercapnia	Sedation Fatigue	Increase respiratory rate or V_T settings.
	Ventilator related	
Hypoxemia	FiO_2 drift	Check ventilator with oxygen analyzer.
Hypocapnia	Settings not correct	Decrease respiratory rate, V_T, or minute ventilation (MV).
Hypercapnia	Settings not correct	Increase respiratory rate, V_T, or MV.
Heater alarm	Adding cold water to humidifier	Wait.
	Altered setting	Reset.
	Cold air blowing on humidifier	Redirect air flow.

exchanger unit may be attached to the airway to act as an artificial nose in lieu of a humidifier.

As air passes through the ventilator to the patient, water condenses in the corrugated tubing. This moisture is considered contaminated and must be drained into a receptacle and not back into the sterile humidifier. If the water is allowed to build up, resistance is developed in the circuit, and PEEP is generated. In addition, if moisture accumulates near the ETT, the patient can aspirate the water. The nurse and respiratory therapist jointly are responsible for preventing this condensation buildup. The humidifier is an ideal medium for bacterial growth. Institutional policies should describe the frequency of ventilator circuit changes.

Complications of Mechanical Ventilation

Complications that can occur with mechanical ventilation are listed in Box 25-12. Although all these adverse consequences occur over time in some ventilated patients, the incidence of these complications can be minimized by good preventive care practices.

Aspiration

Aspiration can occur before, during, or after intubation. The potential for developing nosocomial pneumonia or ARDS increases if aspiration occurs. The risk for aspiration after

BOX 25-12 Complications of Mechanical Ventilation

Airway
- Aspiration
- Decreased clearance of secretions
- Ventilator-acquired pneumonia

Endotracheal Tube
- Tube kinked or plugged
- Rupture of piriform sinus
- Tracheal stenosis or tracheomalacia
- Mainstem intubation with contralateral lung atelectasis
- Cuff failure
- Sinusitis
- Otitis media
- Laryngeal edema

Mechanical
- Hypoventilation with atelectasis
- Hyperventilation with hypocapnia and respiratory alkalosis
- Barotrauma (pneumothorax or tension pneumothorax, pneumomediastinum, subcutaneous emphysema)
- Alarm "turned off"
- Failure of alarms or ventilator
- Inadequate nebulization or humidification
- Overheated inspired air resulting in hyperthermia

Physiological
- Fluid overload with humidified air and sodium chloride retention
- Depressed cardiac function and hypotension
- Stress ulcers
- Paralytic ileus
- Gastric distention
- Starvation
- Dyssynchronous breathing pattern

intubation can be minimized by maintaining appropriate cuff inflation, evacuating gastric contents and relieving distention with suction, suctioning the oropharynx (especially before cuff deflations), and elevating the head of the patient's bed 30 degrees or more at all times. Elevation of the HOB may be limited when the patient has femoral central venous catheters; however, the bed can be raised up to 15 to 20 degrees and then placed in slight reverse Trendelenburg to approximately 30 degrees of elevation.

Barotrauma and Pneumothorax

Mechanical ventilation involves "pumping" air into the airway filling the chest, creating positive pressures during inspiration that may lead to barotrauma. If PEEP is added, the pressures are increased and continued throughout expiration. These positive pressures, especially with PEEP greater than 10 to 15 cm H_2O, can spontaneously rupture an alveolus or emphysematous bleb in the patient with COPD. Air then escapes into, and is trapped in, the pleural space, accumulating until it begins to collapse the lung. Eventually, the collapsing lung impinges on the mediastinal structures, compressing the trachea and eventually the heart; this is called tension pneumothorax. Signs and symptoms of tension pneumothorax are listed in Box 25-13. Signs of pneumothorax include extreme dyspnea, hypoxemia, and an abrupt increase

BOX 25-13 PATIENT SAFETY

Signs and Symptoms of Tension Pneumothorax
- Tachycardia
- Tachypnea
- Agitation
- Diaphoresis
- Tracheal shift from midline
- Muffled heart tones
- Absent breath sounds over affected lung
- Hyperresonance to percussion over affected lung
- Elevation in peak airway pressures in ventilated patients
- Decrease in saturation of oxygen in arterial blood (SaO_2) or arterial oxygen tension (PaO_2)
- Hypotension
- Cardiac arrest

in PIP. Breath sounds may be decreased or absent on the affected side; however, this sign may not be reliable in the patient on positive-pressure ventilation. Observation of the patient may reveal a tracheal deviation (to the opposite side) or the sudden development of subcutaneous emphysema. The PIP may become elevated and a ventilator alarm will be activated due to increased intrathoracic pressure. The most ominous signs of tension pneumothorax are hypotension and bradycardia that can deteriorate into a cardiac arrest without timely medical intervention. The physician or other qualified health care professional may decompress the chest by inserting a needle to evacuate the trapped air until a chest tube can be inserted.

Ventilator-Associated Pneumonia

VAP is the second most common hospital-acquired infection.[19] The incidence of nosocomial pneumonia is increased 10-fold in intubated patients, and the risk for developing VAP is especially great in critically ill patients who are mechanically ventilated. Factors that lead to nosocomial pneumonia are oropharyngeal colonization, gastric colonization, aspiration, and compromised lung defenses. Mechanical ventilation, reintubation, self-extubation, presence of a nasogastric tube, and supine position are a few of the associated risk factors for VAP. Maintenance of the natural gastric acid barrier in the stomach plays a major role in decreasing incidence and mortality from nosocomial pneumonia. The widespread use of antacids or histamine (H_2) blockers can predispose the patient to nosocomial infections because they decrease gastric acidity (increase alkalinity). These medications are used to guard against stress bleeding and may increase colonization of the upper gastrointestinal tract by bacteria that thrive in a more alkaline environment.

VAP is defined as nosocomial pneumonia in a patient who has been mechanically ventilated (by ETT or tracheostomy) for at least 48 hours at the time of diagnosis. A patient should be suspected of having a diagnosis of VAP if the chest radiograph shows new or progressive and persistent infiltrates. Other signs and symptoms can include a temperature higher than 100.4°F (38°C), leukocytosis, new-onset purulent sputum or cough, and worsening gas exchange.

There are numerous strategies for preventing VAP. The first step is to prevent colonization by pathogens of the oropharynx and gastrointestinal tract. Basic nursing care principles, such

as meticulous handwashing and wearing gloves when suctioning patients orally or through the ETT, are essential. Gloves should also be worn when suctioning through closed-suction devices. In addition, critically ill patients have an increased risk for colonization by the microorganisms associated with poor oral hygiene. Oral care for a mechanically ventilated patient involves brushing the patient's teeth (at least every 8 hours), using antimicrobial solutions and alcohol-free mouthwash to cleanse the mouth, applying a water-based mouth moisturizer to maintain the integrity of the oral mucosa, and thoroughly suctioning oral and subglottic secretions. Chlorhexidine oral rinse is one agent that provides antimicrobial action and is used in many institutions. An oral care protocol should be in place for every adult critical care unit using the current evidence-based research and practice. See Evidence-Based Practice Highlight 25-1.

In patients receiving enteral feedings, the HOB should be elevated 30 to 45 degrees (unless contraindicated) to decrease the risk for aspiration.[20] Long-term nasally placed endotracheal and gastric tubes (ie, longer than 3 days) should be placed orally unless contraindicated or not tolerated by the patient. This intervention reduces the risk for the patient developing sinusitis, which is associated with the development of VAP. Sinusitis is relatively common in nasally intubated patients and can cause bacteremia and sepsis. Signs of sinusitis (fever, purulent nasal drainage) must be reported immediately. Lastly, the use of an ETT that provides a port for the CASS appears to prevent the development of VAP in the first week of intubation, and it may decrease the overall incidence of VAP but does not affect mortality or length of stay.[21] The use of the CASS ETT is typically reserved for those patients who can be identified as potentially requiring long-term ventilation.

The advent of the ventilator bundle of standard orders, which incorporates gastrointestinal and deep venous thrombosis prophylaxis, along with getting the patient out of bed, oral care, and keeping the HOB elevated 30 to 45 degrees, has reduced the incidence of VAP in many institutions.[22] These procedures should be included as an integral part of the care of ventilated patients.

Decreased Cardiac Output

Decreased cardiac output, as reflected by hypotension, may be observed at the initiation of mechanical ventilation. Although this is often attributed to the drugs used for intubation (narcotics, sedatives, and NMB agents all reduce blood pressure), the most important contribution to this phenomenon is lack of sympathetic tone and decreased venous return due to the effects of positive pressure within the chest. In addition to hypotension, other signs and symptoms can include unexplained restlessness, decreased levels of consciousness,

EVIDENCE-BASED PRACTICE HIGHLIGHT 25-1
Ventilator-Associated Pneumonia

△ Expected Practice

- All patients receiving mechanical ventilation, as well as those at high risk for aspiration (eg, decreased level of consciousness, with enteral tube in place), should have the HOB elevated at an angle of 30 to 45 degrees unless medically contraindicated.[1–7] (Level VI)
- Use an ETT with a dorsal lumen above the endotracheal cuff to allow drainage by continuous suctioning of tracheal secretions that accumulate in the subglottic area.[1,2,8–13] (Level VI)
- Do not routinely change, on the basis of duration of use, the patient's ventilator circuit.[1,14–17] (Level VI)

△ Supporting Evidence

- Critically ill patients who are intubated for more than 24 hours are at 6 to 21 times the risk of developing ventilator-associated pneumonia (VAP),[1,2,18–20] and those intubated for less than 24 hours are at three times the risk of VAP.[20] Other risk factors for VAP include decreased level of consciousness, supine positioning with HOB flat, use of H_2 antagonists and antacids, gastric distention, presence of gastric or small intestine tubes, enteral feedings, and a trauma or chronic obstructive pulmonary disease diagnosis.[1,18–22] VAP is reported to occur at rates of 10 to 35 cases per 1,000 ventilator days, depending on the clinical situation.[1,19]
- Morbidity and mortality associated with the development of VAP is high, with mortality rates ranging from 20% to 41%.[20,23–25] Development of VAP increases ventilator days, critical care, and hospital lengths of stay by 4, 4, and 9 days, respectively,[18,23,26] and results in more than $11,000 of additional costs per VAP case.[18,25,27]
- Micro- or macroaspiration of oropharyngeal and/or gastric fluids is presumed to be an essential step in the development of VAP.[1,2,12,28] Pulmonary aspiration is increased by supine positioning and poking of secretions above the ETT cuff.[1,3,19]
- Compared to supine positioning, studies have shown that simple positioning with HOB elevation to 30 degrees or higher significantly reduces gastric reflux and VAP,[3–7] yet national surveys and reports in the literature describe poor compliance rates with HOB elevation in critical care units.[20,29–34]
- Studies on the use of special ETTs, which remove secretions pooled above the cuff with continuous suction, found that it decreases VAP by 45% to 50%.[8–11]
- Studies on the frequency of ventilator circuit changes have found no increase in VAP with prolonged use.[14–17]
- National regulatory and expert consensus groups include the AACN VAP Practice Alert interventions as critical to decreasing VAP rates.[1,2,35–37]

AACN Grading Level of Evidence

Level A Manufacturer's recommendations only
Level B Theory based—no research data to support recommendations; recommendations from expert consensus group may exist
Level C Laboratory or bench data only—no clinical data to support recommendations
Level D Limited clinical studies to support recommendations
Level E Clinical studies in more than one or two different populations or situations to support recommendations
Level F Clinical studies in a variety of patient populations and situations to support recommendations

Excerpted from American Association of Critical-Care Nurses Practice Alert. Available online at http://aacn.org. All references cited in this alert are available with the associated resources related to this chapter. Visit: http://thepoint.lww.com

decreased urine output, weak peripheral pulses, slow capillary refill, pallor, fatigue, and chest pain. Increasing fluids to correct the relative hypovolemia usually treats hypotension. However, in this setting, vasopressors may be needed.

Water Imbalance

The decreased venous return to the heart is sensed by the vagal stretch receptors located in the right atrium. This sensed hypovolemia stimulates the release of antidiuretic hormone from the posterior pituitary. The decreased cardiac output, leading to decreased urine output, compounds the problem by stimulating the renin–angiotensin–aldosterone response. A mechanically ventilated patient may be hemodynamically unstable and may require large amounts of fluid resuscitation. This patient can experience extensive edema, including limb, scleral, and facial edema.

Complications Associated With Immobility

Many complications that contribute to the morbidity and mortality of mechanically ventilated patients are the result of immobility. These complications include muscle wasting and weakness, contractures, loss of skin integrity, pneumonia, and deep venous thrombosis that can result in pulmonary embolus, constipation, and ileus.

Gastrointestinal Problems

Gastrointestinal complications associated with mechanical ventilation include distention (from air swallowing), hypomotility and ileus (from immobility and the use of narcotic analgesics), vomiting, and breakdown of the intestinal mucosa from the lack of normal nutritional intake. This breakdown allows translocation of bacteria from the gut into the bloodstream, leading to increased risk for bacteremia in patients who cannot be fed enterally. Maintenance of an adequate bowel elimination pattern is necessary to prevent abdominal distention with resulting impingement on diaphragmatic excursion.

Many mechanically ventilated patients are already malnourished because of underlying chronic disease. Research verifies that the many side effects of clinical starvation can lead to pulmonary complications and death, as listed in Box 25-14. Early enteral nutrition is advocated for trauma and critically ill patients with either small-bore or Salem-sump tubes that end in the stomach or small intestine.[23] The advantage of small-bore feeding tubes is comfort, and with postpyloric placement, enteral feeding goal rates can be achieved sooner.

Muscle Weakness

The muscles used in respiration, like other muscles, become deconditioned and may even atrophy with prolonged disuse. The ventilated patient's respiratory muscles may not be used (other than passive movement) while on the ventilator, especially if muscle relaxants, heavy sedation, or both have been part of the care plan. A retraining period to exercise and strengthen the respiratory muscles may be necessary before ventilatory support can be discontinued. Especially at risk for "critical illness myopathies" are patients who have been on corticosteroids in combination with NMB agents as well as those with multiorgan failure, sepsis, and ARDS.[24]

BOX 25-14 Side Effects of Clinical Starvation

- Atrophy of respiratory muscles
- Decreased protein
- Decreased albumin
- Decreased cell-mediated immunity
- Decreased surfactant production
- Decreased replication of respiratory epithelium
- Intracellular depletion of adenosine triphosphate
- Impaired cellular oxygenation
- Central respiratory depression

Muscle weakness also occurs as a result of muscle fatigue. Those patients requiring mechanical ventilation typically have one or more reasons for an increase in the work of breathing. These include an increase in carbon dioxide production, physiological dead space (non-gas-exchanging air passages), or both; decreased lung compliance; and increased airway resistance, as with bronchospasm or thick secretions. When the work of breathing exceeds the capacity of weakened muscles, the patient begins to display abnormal respiratory mechanics with inefficient use of these muscles. This often occurs during a weaning trial after prolonged ventilation. The accepted intervention for fatigue in this setting is returning to muscle rest on the ventilator. However, this carries the risk for contributing further to muscle atrophy. The diaphragm muscle also requires sufficient electrolytes of calcium, magnesium, and phosphorus to optimize function during weaning. The nurse should review electrolyte values daily to ensure these diaphragm-essential electrolytes are kept normalized.

Assessment and Management

The patient who needs ventilatory support also needs primary nursing care. One of the greatest contributions the nurse can make to decreasing costs, length of stay, and mortality in patients with respiratory problems is to implement interventions that will prevent or minimize complications. Because mechanical ventilation is supportive rather than curative, the focus of care for the mechanically ventilated patient is holistic. The nurse must interact effectively with each member of the health care team to achieve desired patient outcomes. Box 25-15 gives examples of nursing diagnoses for the patient who needs ventilatory support. Box 25-16 summarizes care of the patient on a ventilator. The mechanical ventilator, the artificial airway, and the care necessary to maintain mechanical ventilation require specialized nursing knowledge and skills, which are discussed in the following sections.

Endotracheal Tube Care

To prevent tube movement, tube migration, or inadvertent extubation, ETTs must be anchored securely. Anchoring can be accomplished with adhesive tape or with commercially manufactured tube immobilization appliances. Usual practice is to retape the ETT every 1 to 2 days or when it is soiled or insecure. In orally intubated patients, the position of the ETT should be changed from side to side to facilitate oral

BOX 25–15 EXAMPLES OF NURSING DIAGNOSES

- Ineffective Coping
- Excess Fluid Volume
- Impaired Gas Exchange
- Ineffective Breathing Pattern
- Ineffective Airway Clearance
- Ineffective Peripheral Tissue Perfusion
- Risk for Increased Cardiac Tissue Perfusion
- Disturbed Body Image related to intubation or tracheostomy
- Risk for Infection
- Risk for Activity Intolerance
- Ineffective Airway Clearance
- Anxiety
- Risk for Aspiration
- Ineffective Breathing Pattern
- Impaired Bed Mobility
- Sleep Deprivation
- Dysfunctional Ventilatory Weaning Response

care and to prevent areas of pressure necrosis on the lips, mouth, and tongue. The disadvantage of frequent retaping is that patients with fragile skin or prolonged intubation may incur skin breakdown. Twill tape can be substituted for adhesive tape in these situations and for patients with heavy beards. Retaping by two people is desirable to prevent accidental tube displacement. The final step in retaping is to check tube placement in comparison to placement before retaping. ETT placement is verified by radiography following initial intubation. The position in centimeters at the lips/teeth or nostril is recorded; this placement is verified every shift to detect inadvertent position changes. Tube placement is checked, following retaping, by comparing the centimeter markings at the lips/teeth or nostril with the last radiological documentation of position. Placement of an oral bite block can prevent biting on the tube, which can cause airway narrowing or tube displacement. The use of a swivel connector (connecting the tube to the ventilator circuit), along with anchoring a large loop of tubing to the bed, facilitates patient movement without ETT movement. Oral inspection and hygiene are of paramount importance when a bite block is used.

Persistent coughing may suggest that the ETT has migrated to touch the carina, requiring the tube to be withdrawn to an appropriate level. The pilot cuff balloon is protected from inadvertent disruption; cuff rupture or ETT occlusion with a mucous plug usually requires reintubation. If a patient is prematurely extubated for any reason, the airway must be kept patent. Oxygenation and ventilation may be provided with an MRB and mask until reintubation can be accomplished.

BOX 25–16 COLLABORATIVE CARE GUIDE for the Patient on Mechanical Ventilation

Outcomes	Interventions
Oxygenation/Ventilation	
A patent airway is maintained. Lungs are clear to auscultation. Patient is without evidence of atelectasis. Peak, mean, and plateau pressures are within normal limits. ABG values are within normal limits.	• Auscultate breath sounds every 2–4 h and as needed. • Suction as needed for rhonchi, coughing, or oxygen desaturation. • Hyperoxygenate and hyperventilate before and after each suction pass. • Monitor airway pressures every 1–2 h • Monitor airway pressures after suctioning. • Administer bronchodilators and mucolytics as ordered. • Perform chest physiotherapy (CPT) if indicated by clinical examination or chest radiograph. • Turn patient side to side every 2 h • Consider kinetic therapy or prone positioning as indicated by clinical scenario. • Get patient out of bed to chair or standing position when stable. • Monitor pulse oximetry and $ETCO_2$. • Monitor ABG values as indicated by changes in noninvasive parameters, patient status, or weaning protocol.
Circulation/Perfusion	
Blood pressure, heart rate, cardiac output, central venous pressure, and pulmonary artery pressure remain stable on mechanical ventilation.	• Assess hemodynamic effects of initiating positive-pressure ventilation (eg, potential for decreased venous return and cardiac output). • Monitor electrocardiogram for dysrhythmias related to hypoxemia. • Assess effects of ventilator setting changes (inspiratory pressures, V_T, PEEP, and fraction of inspired oxygen [FiO_2]) on hemodynamic and oxygenation parameters. • Administer intravascular volume as ordered to maintain preload.
Fluids/Electrolytes	
Intake and output (I & O) measurements are balanced. Electrolyte values are within normal limits.	• Monitor hydration status in relation to clinical examination, auscultation, amount, and viscosity of lung secretions. • Assess patient weight, I & O totals, urine specific gravity, or serum osmolality to evaluate fluid balance. • Administer electrolyte replacements (intravenous or enteral) per physician's order.

(*continued on page 539*)

BOX 25–16 *COLLABORATIVE CARE GUIDE for the Patient on Mechanical Ventilation (continued)*

Outcomes	Interventions
Mobility	
Patients will maintain/regain baseline functional status related to mobility and self-care. Joint range of motion is maintained.	• Collaborate with physical/occupational therapy staff to encourage patient effort/participation to increase mobility. • Progress activity to sitting up in chair, standing at bedside, ambulating with assistance as soon as possible. • Assist patient with active or passive range-of-motion exercises of all extremities at least every shift. • Keep extremities in physiologically neutral position using pillows or appropriate splint/support devices as indicated.
Safety	
ETT will remain in proper position. Proper inflation of ETT cuff is maintained. Ventilator alarm system remains activated.	• Securely stabilize ETT in position; use respiratory therapy expertise for best method. • Note and record the "cm" line on ETT position at lip or teeth. • Use patient self-protective devices or sedation per hospital protocol. • Evaluate ETT position on chest radiograph daily (by viewing film or by report). • Keep emergency airway equipment and MRB readily available, and check each shift. • Inflate cuff using minimal leak technique or pressure <25 mm Hg by manometer. • Monitor cuff inflation/leak every shift and as needed. • Protect pilot balloon from damage. • Perform ventilator setting and alarm checks every 4 h (minimum) or per hospital protocol.
Skin Integrity	
Patient is without evidence of skin breakdown.	• Assess and document skin integrity at least every shift. • Turn patient side to side every 2 h; reassess bony prominences for evidence of pressure injury. • When patient is out of bed to chair, provide pressure relief to sitting surfaces at least hourly. • Remove self-protective devices from wrists, and monitor skin per hospital policy.
Nutrition	
Nutritional intake meets calculated metabolic need (eg, basal energy expenditure equation). Patient will establish regular bowel elimination pattern.	• Consult dietitian for metabolic needs assessment and recommendations. • Provide early nutritional support by enteral or parenteral feeding. • Monitor actual delivery of nutrition daily with I & O calculations. • Weigh patient daily. • Administer bowel regimen medications as ordered, along with adequate hydration.
Comfort/Pain Control	
Patients will indicate/exhibit adequate relief of discomfort/pain while on mechanical ventilation.	• Document pain assessment, using numerical pain rating or similar scale when possible. • Provide analgesia as appropriate, document efficacy after each dose. • Prevent pulling and jarring of the ventilator tubing and endotracheal or tracheostomy tube. • Provide meticulous oral care every 1–2 h with oropharynx suctioning and mouth moisturizer as needed; teeth brushing scheduled at least three times daily, antimicrobial irrigation twice daily, oral assessment at least daily. • Administer sedation as indicated.
Psychosocial	
Patient participates in self-care and decision making related to own activities of daily living (ADLs) (eg, turning, bathing). Patient communicates with health care providers and visitors.	• Encourage patient to move in bed and attempt to meet own basic comfort/hygiene needs independently. • Establish a daily schedule for bathing, time out of bed, treatments, and so forth, with patient input. • Provide a means for patient to write notes and use visual tools to facilitate communication. • Encourage visitor conversations with patient in normal tone of voice and subject matter. • Teach visitors to assist with range of motion and other simple care delivery tasks to facilitate normal patterns of interaction.
Teaching/Discharge Planning	
Patient cooperates with and indicates understanding of need for mechanical ventilation. Potential discharge needs are assessed.	• Provide explanations to patient/significant others regarding: Rationale for use of mechanical ventilation Procedures such as suctioning, airway care, CPT Plan for and progress toward weaning and extubation • Initiate early social worker involvement to screen for needs, resources, and support systems.

Tracheostomy Care

In patients requiring long-term mechanical ventilation, the airway is converted to a tracheostomy at some point to prevent the complications of endotracheal intubation, such as tracheal stenosis and vocal cord paralysis. The preferred method of airway management is the tracheostomy tube for long-term ventilation. Past practice involved tracheostomy after 11 and up to 21 days on the ventilator. Current practice promotes earlier tracheostomy at 72 hours after intubation. Earlier tracheostomy (eg, after 3 to 7 days on the ventilator) is performed to facilitate earlier weaning, particularly if the patient has multiple comorbidities and demonstrates difficulty weaning or has trauma or neurological diagnoses associated with prolonged need for an artificial airway. Tracheostomy is also performed for patient comfort and safety when mobilizing the patient and may lead to decreased ventilator weaning time. In addition to long-term ventilation, indications for tracheostomy include upper airway obstruction, airway edema from anaphylaxis, failed intubation, multiple intubations (high risk for complications), complications of ETT intubation, absence of protective reflexes, home care, conditions in which ETT intubation is not possible (eg, facial trauma, cervical fractures), and the desire for improved patient comfort.

The advantages of tracheostomy over endotracheal intubation include faster weaning (at least in part because of decreased dead space), enhanced patient comfort, enhanced communication, and the possibility of oral feeding. The tracheostomy is inserted into the trachea, thereby avoiding the mouth, upper airway, and glottis, and this decreases problems of airway resistance and occlusion.

Tracheostomy is not without disadvantages. These include hemorrhage, infection, pneumothorax, and the need for an operative procedure that is itself a risk. Box 25-17 presents complications of tracheostomy. The most serious complication is erosion into the innominate artery, which can result in exsanguination. If bleeding occurs, the cuff can be hyperinflated in an attempt to control bleeding until emergency surgery can be performed. The practice of bedside percutaneous tracheostomy using a progressive dilation technique has been touted to decrease the morbidity and cost incurred with an operative procedure because it is often earlier than surgical tracheostomy. Although there is no major difference in mortality risk, it has been found that early tracheostomy resulted in decreased ventilator days. Less infection and bleeding have also been given as advantages over the standard procedure performed in the operating room.[25]

BOX 25-17 PATIENT SAFETY

Complications of Tracheostomy

- Acute hemorrhage at the site
- Air embolism
- Aspiration
- Tracheal stenosis
- Erosion into the innominate artery with exsanguination
- Failure of the tracheostomy cuff
- Laryngeal nerve damage
- Obstruction of tracheostomy tube
- Pneumothorax
- Subcutaneous and mediastinal emphysema
- Swallowing dysfunction
- Tracheoesophageal fistula
- Infection
- Accidental decannulation with loss of airway
- False placement of cannula (not in trachea)
- Weak voice/hoarseness

The nurse can prevent complications by assessing for them with each patient interaction and during tracheostomy care. Proper fixation of the tracheostomy tube reduces the movement of the tube in the airway and limits friction injury to the tracheal wall or larynx. Maintaining the cuff pressure at the minimum required to prevent air leak on the ventilator reduces the risk for tissue breakdown due to excessive pressure on the trachea wall. The tracheostomy tube must be firmly secured. The ventilator tubing should have enough length to allow movement without pulling on the tracheostomy and to allow for procedures. A tracheostomy swivel connector, with or without flex tubing, reduces the tension on the tracheostomy while the patient is on the ventilator. A confused or very mobile patient can easily self-decannulate; patient restraints may be needed to prevent accidental decannulation. Orienting the patient to the need for an artificial airway and providing pain control and sedation are measures that are taken before resorting to restraint application. If restraints are needed, it is necessary to obtain a physician's order, with regular review of continued need. The nurse must monitor the patient closely for potential injury and must perform circulatory checks with removal of restraints frequently.

Tracheostomy care includes frequent changing of tracheal ties and dressing, although initial ties are not changed until at least 24 to 48 hours after placement to allow for hemostasis of the site. The sutures from either a percutaneous or surgical tracheostomy are left in place for 48 to 72 hours or even up to a week (per hospital protocol) to prevent decannulation. As with retaping of the ETT, changing of tracheostomy ties should be a two-person procedure. The ties should be tied so that one to two fingers can be inserted between the ties and the skin, allowing minimal movement of the tracheostomy tube but maintaining comfort. It is mandatory to maintain a midline position for the tracheostomy to prevent pressure on surrounding tissue. The stoma is cleansed with half-strength hydrogen peroxide, followed by rinse with sterile saline solution, and observed for wound healing, bleeding, and signs of infection The routine practice of inner cannula cleaning or changes may not be necessary with a disposable inner cannula that can be changed daily. The routine care for tracheostomy is cleaning the tracheostomy site at least every 8 to 12 hours and as needed, changing the inner cannula daily (or according to facility policy), and changing soiled tracheostomy ties as needed, progressing to daily and as-needed care. This longer care interval usually occurs after 7 to 10 days or when secretion and tracheostomy drainage are minimal. The routine care of tracheostomies is always performed as a sterile procedure while in the hospital.

If decannulation occurs within the first 7 days of tracheostomy insertion, the patient may be reintubated with an ETT if emergent tracheostomy tube replacement cannot be done safely. An obturator and a new, appropriately sized

tracheostomy tube are kept at the bedside. If inadvertent decannulation occurs after a tract has developed, the tube is carefully replaced using the obturator.

Tube Cuff Pressure Monitoring

Tube cuff pressures are monitored every shift to prevent overdistention and excess pressure on the tracheal wall mucosa, which can cause complications such as tracheal stenosis. If a patient is on the ventilator, the best pressure is the lowest possible pressure without having a loss of inspiratory volume. Physiologically, pressures of about 20 to 30 mm Hg obliterate capillary circulation to the tracheal mucosa. If a cuff leak is suspected, auscultation at the neck for the sound of air escaping above the cuff can determine whether the seal is adequate.

One method used to inflate a cuff is called the minimal occluding volume. Air is injected slowly into the pilot balloon during ventilator inspiration while auscultation is performed over the trachea. When the harsh "squeak" of air escaping is no longer audible, the minimal occluding volume has been reached, and the tube cuff is occluding the airway without excessive pressure on the trachea. Extra air should not be added. In the ICU, the best practice is actual measurement of cuff pressure using a manometer. This device is attached to the ETT pilot balloon to obtain a reading, which should ideally be 20 to 25 mm Hg. If a leak is still present above this level of inflation, slight repositioning of the ETT within the patient's airway may correct the problem. Changing to a larger or longer ETT may be necessary with increasing pressures to seal the airway. The cuff pressure is assessed with the manometer every 6 to 8 hours and, when a leak is noted, to help prevent aspiration of subglottic secretions. Whenever a cuff leak is found, the medical team and respiratory care practitioner should be notified. Recurrent cuff leaks may indicate the need for an extra long tube or a larger size to provide for ventilation.

Discharge Planning and Patient Teaching

Discharge planning is necessary for patients who will be discharged to home with tracheostomies. Rationales for tracheostomy care include promotion of ostomy healing, prevention of infection, maintenance of a patent airway, and increased patient comfort.

Teaching the patient and family care giver tracheostomy care allows for independence and self-care. This is an essential component of discharge teaching. Communication about the procedure and reassurance during the training process reduce anxiety and improve cooperation.

Nutritional Support

Respiratory muscles, like all other body muscles, need energy to work. If energy needs are not met, muscle fatigue occurs, leading to discoordination of respiratory muscles and a decrease in tidal volume. Hypomagnesemia and hypophosphatemia have been implicated in muscle fatigue caused by depleted levels of adenosine triphosphate. Electrolyte imbalances must be corrected and monitored daily for optimal muscle functioning during ventilator weaning. In prolonged starvation, the body cannibalizes the intercostal and diaphragmatic muscles for energy.

Metabolic needs in critically ill patients are much higher than in normal subjects. Basic caloric requirements are usually increased by 25% for hospital activity and stress associated with treatment. Adequate nutrition is a prerequisite for weaning from mechanical ventilation; nutritional support should be instituted early. If the gastrointestinal tract is intact, enteral nutrition is preferred and can be provided through a small-bore feeding tube.

Initial tube feeding is started slowly, with close monitoring of blood glucose and electrolyte levels. The nurse observes the patient for signs of intolerance, such as diarrhea and hyperosmolar dehydration. If the patient tolerates feedings, the rate is gradually increased until the goal rate is achieved. If tube feedings cannot be tolerated, parenteral hyperalimentation should be considered (see Chapter 40).

Patients who require long-term mechanical ventilation typically need additional protein and calories per day. When available, metabolic cart testing (also called indirect calorimetry) or a 24-hour urine nitrogen test can assess individual nutritional requirements. The monitoring of the prealbumin level may give an indication of recent nutritional state. Nutritionists are invaluable in determining the caloric needs of critically ill patients.

Eye Care

Eye care of the ventilator patient is important. Many patients in the ICU are comatose, sedated, or chemically paralyzed and therefore have lost the blink reflex or ability to close their eyelids completely. This can lead to corneal dryness and ulceration.

Few studies have established the efficacy of one eye care measure over another. Current practices include instillation of lubricating drops or ointment, taping the eyes, applying eye shields, or applying a moisture chamber. Eye care should be scheduled and not on an as-needed basis to ensure 24-hour application. Scleral edema is common in the ventilated patient. Raising the HOB may help reduce scleral edema.

Oral Care

Frequent oral care must be performed on all mechanically ventilated patients. See Evidence-Based Practice Highlight 25-2. Oral care not only increases comfort but also preserves the integrity of the oropharyngeal mucosa. An intact mucosa helps prevent infection and colonization of organisms that leads to VAP. As noted in the VAP discussion, evidence-based studies note the use of oral care protocols for ventilated adult patients in preventing VAP.[26] Nursing skill manuals do present guidelines for oral care in patients with and without teeth as well as in those who are incapacitated. However, these general oral care guidelines are not suitable for patients with an ETT and do not promote VAP prevention. The current literature includes oral care guidelines using every 2-, 4-, and 8-hour interventions with specific interventions of tooth brushing, oral and subglottic suctioning, moisturizer, and oral rinses. The CDC recommends that every ICU implement a complete oral care program with use of an antimicrobial oral rinse to prevent oral colonization.[27] What is still needed is a definitive research study regarding the optimal frequency, products, type of

EVIDENCE-BASED PRACTICE HIGHLIGHT 25-2

Oral Care for Patients at Risk for Ventilator-Associated Pneumonia

△ Expected Practice

- Develop and implement a comprehensive oral hygiene program for patients in critical care and acute care settings who are at high risk for VAP.
  - Brush teeth, gums, and tongue at least twice a day using a soft pediatric or adult toothbrush. (Level E)
  - Provide oral moisturizing to oral mucosa and lips every 2 to 4 hours.
  - Use an oral chlorhexidine gluconate (0.12%) rinse twice a day during the perioperative period for adult patients who undergo cardiac surgery. (Level D)
  - Routine use of oral chlorhexidine gluconate (0.12%) in other populations is not recommended at this time.

△ Supporting Evidence

- Colonization of the oropharynx is a critical factor in the development of nosocomial pneumonia.[1–3] Growth of potentially pathogenic bacteria in dental plaque provides a nidus of infection for microorganisms that have been shown to be responsible for the development of VAP.[2–4] Dental plaque provides a microhabitat for organisms and provides opportunity for adherence either to the tooth surface or to other microorganisms. These microorganisms in the mouth translocate and colonize the lung, which can result in VAP.[3,5] Dental plaque can be removed by brushing.
  - There are no data associated with critically ill patients; however, the American Dental Association recommends that healthy people brush teeth twice daily to remove plaque from all tooth surfaces.[6]
  - The use of an oral care protocol (brushing with a pediatric toothbrush, mouthwash, and moisturizing gel) reduces oral inflammation and improves oral health.[7]
- Chlorhexidine oral rinse reduced respiratory infections in cardiac surgery patients who received chlorhexidine before intubation as well as postoperatively[8] and reduced nosocomial pneumonia in patients who were intubated for more than 24 hours.[9] However, when chlorhexidine was tested in a more varied ICU population, no difference was observed in VAP mortality or length of stay. Although oropharyngeal colonization by VAP pathogens was reduced with chlorhexidine, its efficacy was insufficient to reduce the incidence of respiratory infections.[10,11] A 2005 meta-analysis of chlorhexidine trials found that use of chlorhexidine did not result in significant reduction in the incidence of nosocomial pneumonia, nor in alteration of the mortality rate.[12] The CDC (Centers for Disease Control and Prevention) guidelines recommend use of chlorhexidine only during the perioperative period for adult patients undergoing cardiac surgery; routine use in other critically ill populations is not recommended.[13]
- Several studies tested intervention bundles that included oral care as one of the interventions.[14–19] The studies demonstrate that bundled interventions decreased nosocomial respiratory infections; however, the contribution of oral care to the results could not be determined.
  - In addition to brushing, providing oral moisturization to oral mucosa and lips every 2 to 4 hours is often a component of the oral care protocols.[20]
- To date, data have been published from large, well-controlled clinical trials of oral care interventions in at-risk patients other than chlorhexidine studies. There are clinical reports of infection rates before and after changes in oral care procedures, but few have been published in referred journals. Some reports show a positive effect; however, the role of oral care in reducing nosocomial pneumonia is not clearly established by such projects, and it is possible that other changes in care occurred in the units and affected the results.[21–22]

AACN Evidence Leveling System

Level A Meta-analysis of quantitative studies or metasynthesis of qualitative studies with results that consistently support a specific action, intervention, or treatment

Level B Well-designed, controlled studies with results that consistently support a specific action, intervention, or treatment

Level C Qualitative studies, descriptive or correlational studies, integrative review, systematic reviews, or randomized controlled trials with inconsistent results

Level D Peer-reviewed professional organizational standards with clinical studies to support recommendations

Level E Multiple case reports, theory-based evidence from expert opinions, or peer-reviewed professional organizational standards without clinical studies to support recommendations

Level M Manufacturer's recommendations only

Excerpted from American Association of Critical-Care Nurses Practice Alert. Available online at http://aacn.org. All references cited in this alert are available with the associated resources related to this chapter. Visit: http://thepoint.lww.com

oral rinse, and materials to provide oral care for adult ventilated patients. Including the oral care guideline as part of VAP prevention may help reduce VAP by educating nurses and implementing practice changes, along with continuing quality improvement by monitoring the effectiveness of oral care and VAP protocols. Every ICU should either review its oral care guideline or create one with the current evidence-based research and available protocols. ICUs should follow the AACN Practice Alert on Oral Care in the critically ill.[28] Suggested guidelines may include the following:

1. Systematic assessment of the oral mucosa performed daily and with each cleaning
2. Handwashing before and after every nursing intervention
3. Routine brushing of teeth to remove dental plaque every 8 hours
4. Cleansing of the mouth every 2 hours and as needed
5. Use of an alcohol-free or antimicrobial (chlorhexidine) oral rinse every 8 or 12 hours to reduce oropharyngeal colonization
6. Suctioning the mouth and subglottic pharynx to minimize aspiration risk and provide a cover for the suction set with replacement every 8 or 24 hours
7. Applying a water-based mouth moisturizer to prevent mucosal drying and maintain integrity of the oral mucosa

Commercial kits are available that provide the suction catheters, covered tonsil-tip suction, toothbrushes with suction, and toothpaste for brushing in individually wrapped sets to prevent contamination.

Psychological Care

The ventilated patient is subjected to extreme physical and emotional stress in the ICU environment. Psychological distress can be caused by sleep deprivation, sensory overstimulation, sensory deprivation for familiar cues, pain, fear, inability to communicate, and commonly used pharmacological agents. Often, treatments can seem dehumanizing.

Feelings of helplessness and lack of control can be overwhelming. The patient may attempt to gain some element of control through constant demanding or exhibiting other "inappropriate" behavior. If the patient is incapable of dealing with stress through coping mechanisms, he or she may exhibit depression, apathy, and lack of emotional involvement. These reactions may be exacerbated in patients with a history of psychiatric problems or drug or alcohol abuse.

Assisted ventilation can precipitate a psychological dependence in those with primary respiratory disorders. If, for the first time in years, a patient is receiving enough oxygen to meet metabolic needs and does not have to struggle for air, he or she may be reluctant to give up the ventilator. Weaning can become even more stressful for this patient. Nursing interventions to improve sleep with quiet time, psychiatric consultation, and alternative therapy, such as music or massage, in addition to encouraging family support, will benefit the stressed patient. Taking the patient outside on the ventilator, sitting up in a chair, and ambulating on an MRB can improve the patient's mental health. Pet therapy, visiting with family and friends, and using a calendar for long-term patients can help keep them oriented. Resumption of home psychotropic medications, especially for depression or other disorders, is essential when compatible with the ongoing medical plan.

Facilitating Communication

A number of interventions can facilitate communication with the patient who has an endotracheal or tracheostomy tube. Before assessing the patient's ability to communicate, provide the patient with his or her eyeglasses or hearing aid (if applicable). Complete explanations from staff members regarding any procedures may help decrease the patient's stress. The care giver can use verbal and nonverbal communication skills. Nonverbal communication may include sign language, gestures, or lip reading. If the patient is unable to use these forms of nonverbal communication, helpful devices include pencil and paper, clipboard or dry erase boards, picture or alphabet boards, electronic communication boards, and even a computer.

Once the patient is off the ventilator and tolerating the tracheostomy collar, the tracheostomy patient can communicate by using a cap or speaking valves that occlude the tracheostomy tube. These allow for the passage of air around the tracheostomy to the vocal chords as long as the cuff is deflated. The tracheostomy may be capped for 24 to 48 hours before decannulation, and the patient breathes and speaks around the tracheostomy. The cap is the final test to ensure airway protection by the patient. Two other options to the cap are the Passy-Muir valve and the Shiley speaking valve. The Passy-Muir and Shiley speaking valves are one-way valves that allow air to enter during inspiration and then close to allow the air to flow over the vocal chords with exhalation. These valves each have a side port for oxygen tubing to be attached, providing oxygen support in addition to the humidified air from a tracheostomy collar. The tracheostomy collar should be used nearly continuously to prevent the accumulation of secretions and the drying of the airway mucosa. Neither speaking valve should be used during sleep to prevent aspiration with a deflated cuff. Patients with copious secretions are at risk for obstruction of these valves. They must be monitored very closely. In addition, patients at high risk for aspiration, especially those with laryngeal or pharyngeal dysfunction, should be carefully assessed before one of these devices is used. The nurse should store these valves in a container clearly identified with the patient's name for safekeeping because each type of valve is relatively costly. The patient should be taught to remove the valve with excessive sputum during cough and call for assistance to clean the valve before reuse. The tracheostomy patient with a speaking valve is at increased risk for aspiration because the cuff must be deflated for the patient to communicate.

Caring for the Family

Family members must deal with a strange environment, a critically ill loved one, and the financial strain imposed by the illness. Nursing support is given by familiarizing the family with the physical surroundings, supplying information about visitation policies, and providing frequent progress reports on the patient's condition.

Studies show improvement in patient outcomes from increased presence of loved ones during hospitalization. Based on these findings and on increased patient and family satisfaction, many ICUs have instituted open visitation policies and increased involvement of family members in patient care. Critical care patients and especially the elderly benefit from increased visitation, and the families benefit from improved communication when they are the decision makers.[29] Nurses increasingly recognize the importance of family visitation for the patient. The presence of family during invasive procedures or during a code has been associated with positive outcomes, and studies show family and care givers reported the benefit of being at the bedside.[30]

The nurse establishes open communication with the patient and family, proactively arranges for visits, and provides the family with information. Promoting spiritual and cultural support, scheduled family communication conferences, nursing education on visitation, and open communication assists the family with coping and reduces stress.[31] A system that includes liberal visitation policies and flexibility for individual patient and family needs promotes a healing environment and supports the family as partners in the care plan.

Weaning From Mechanical Ventilation

As soon as mechanical ventilation starts, plans begin for weaning the patient from mechanical support. The process to achieve this goal includes correcting the cause of respiratory failure, preventing complications, and restoring or maintaining physiological and psychological functional status. Patients can be categorized into two groups: those requiring

short-term ventilation (3 days or less) and those requiring long-term ventilation (more than 3 days).

Each patient is evaluated daily for readiness to wean by performing a spontaneous breathing trial. Boxes 25-18 and 25-19 present guidelines for weaning from short-term ventilation and long-term ventilation, respectively. It is important to perform this assessment and address weaning impediments before initiating weaning trials. Many weaning indices have been advocated for use in predicting weaning readiness. Some look exclusively at respiratory factors, such as muscle strength and endurance (eg, negative inspiratory pressure [NIP], PEP weaning index, or rapid shallow breathing index as the ratio of frequency to tidal volume). Others are integrated indices that look at a broad range of physiological factors that influence weaning readiness. Many of these factors individually lack predictability, and often several factors are assessed for weaning readiness.

In addition to controversy concerning weaning indices, there is disagreement and lack of evidence regarding which approach to weaning is best. Some clinicians maintain total ventilatory support up until the time of weaning trials; others use intermittent trials of increasing frequency and duration. The theoretical advantages of a gradual approach to weaning support include the following: (1) over time, the patient on partial rather than full ventilation is exposed to lower levels of pressure and volume, therefore reducing the risk for complications; and (2) a weaning approach that requires the patient to perform some level of work to breathe imposes an "exercise" regimen that should reduce deconditioning and atrophy of the muscles used in respiration.

The performance of the diaphragm, as well as accessory muscles of respiration, depends on both the endurance and strength of the muscles. The effectiveness of diaphragmatic contraction is a function of both the resting length of muscle

BOX 25-18 Guidelines for Weaning From Short-Term Ventilation

Patients are often intubated electively for surgical or other procedures or more urgently owing to respiratory distress related to underlying pulmonary disease or traumatic injury. The other common reason for intubation is the need for airway protection because of airway swelling (eg, as a result of acute inhalation injury) or significant change in mental status (eg, as with cerebrovascular accident or head injury). Once the procedure is completed or the patient is stabilized, the goal should be extubation as soon as the patient is able to protect the airway. The weaning process in this setting may proceed rapidly, based on individual patient response to reducing ventilatory support.

Readiness Criteria

- Hemodynamically stable, adequately resuscitated, and not requiring vasoactive support
- SaO_2 greater than 90% on FiO_2 40% or less, PEEP 5 cm H_2O or less
- Chest radiograph reviewed for correctable factors; treated as indicated
- Metabolic indicators (serum pH, major electrolytes) within normal range
- Hematocrit more than 25%
- Core temperature more than 36°C and less than 39°C
- Adequate management of pain/anxiety/agitation
- No residual neuromuscular blockade
- ABG values normalized or at patient's baseline

Weaning Intervention

- Reduce ventilator rate, then convert to pressure-support ventilation (PSV) only.
- Wean PSV as tolerated to 10 cm H_2O or less.
- If patient meets tolerance criteria for at least 2 hours on this level of support *and* meets extubation criteria (see later), may extubate.
- If patient fails tolerance criteria, increase PSV or add ventilator rate as needed to achieve "rest" settings (consistent respiratory rate <20 breaths/min) and review weaning criteria for correctable factors.
- Repeat wean attempt on PSV 10 cm after rest period (minimum, 2 hours). If patient fails second wean trial, return to rest settings and use "long-term" ventilation weaning approach.

Tolerance Criteria

If the patient displays any of the following, the weaning trial should be stopped and the patient returned to "rest" settings.

- Sustained respiratory rate greater than 35 breaths/min
- SaO_2 less than 90%
- Tidal volume 5 mL/kg or less
- Sustained minute ventilation greater than 200 mL/kg/min
- Evidence of respiratory or hemodynamic distress:
  - Labored respiratory pattern
  - Increased anxiety, diaphoresis, or both
  - Sustained heart rate greater than 20% higher or lower than baseline
  - Systolic blood pressure exceeding 180 mm Hg or less than 90 mm Hg

Extubation Criteria

- Mental status: alert and able to respond to commands
- Good cough and gag reflex and able to protect airway and clear secretions
- Able to move air around ETT with cuff deflated and end of tube occluded

Adapted from evidence-based practice guidelines used in the Surgical/Trauma Intensive Care Unit, University of Virginia Health System, Charlottesville, Virginia.

BOX 25-19 Guidelines for Weaning From Long-Term Ventilation

Patients on mechanical ventilation for longer than 72 hours or those having failed short-term weaning often display significant deconditioning as a result of acute or chronic complex illness, or both. These patients usually require a period of "exercising" respiratory muscles to regain the strength and endurance needed for successful return to spontaneous breathing. Goals for this process are:

- To have the patient tolerate two to three daily weaning trials of reduction in ventilatory support without exercising to the point of exhaustion
- To rest the patient between weaning trials and overnight on ventilator settings that provide diaphragmatic rest, with minimal or no work of breathing for the patient

Readiness Criteria

- Same as for short-term ventilation (see Box 25-18), with emphasis on hemodynamic stability, adequate analgesia/sedation (record scores on flow sheet), and normalizing volume status

Weaning Intervention

- Transfer to PSV mode, adjust support level to maintain patient's respiratory rate at less than 35 breaths/min.
- Observe for 30 minutes for signs of early failure (same tolerance criteria as with short-term ventilation; see Box 25-18).
- If tolerated, continue trial for 2 hours, then return patient to "rest" settings by adding ventilator breaths or increasing PSV to achieve a total respiratory rate of less than 20 breaths/min.
- After at least 2 hours of rest, repeat trial for 2 to 4 hours at same PSV level as previous trial. If the patient exceeds the tolerance criteria (listed in Box 25-18), stop the trial and return to "rest" settings. In this case, the next trial should be performed at a higher support level than the "failed" trial.
- Record the results of each weaning episode, including specific parameters and the time frame if "failure" observed, on the bedside flow sheet.
- The goal is to increase the length of the trials and reduce the PSV level needed on an incremental basis. With each successive trial, the PSV level may be decreased by 2 to 4 cm H_2O, the time interval may be increased by 1 to 2 hours, or both, while keeping the patient within tolerance parameters. The pace of weaning is patient specific, and tolerance may vary from day to day. Review readiness criteria for correctable factors daily *and* each time the patient "fails" a weaning trial.
- Ensure nocturnal ventilation at "rest" settings (with a respiratory rate of <20 breaths/min) for at least 6 hours each night until the patient's weaning trials demonstrate readiness to discontinue ventilatory support.

Discontinuing Mechanical Ventilation

The patient should be weaned until ventilator settings are FiO_2 40% or more, PSV 10 cm H_2O or less, and PEEP 5 cm H_2O or less. Once these settings are well tolerated, the patient should be placed on continuous positive airway pressure 5 cm H_2O or (if tracheostomy in place) on tracheostomy collar. If the patient meets tolerance criteria over the first 5 minutes, the trial should be continued for 1 to 2 hours. If clinical observation and ABG values indicate that the patient is maintaining adequate ventilation and oxygenation on this "minimal" support, the following options should be considered:

- If the patient meets extubation criteria (see Box 25-18), this step should be attempted.
- If the patient is on tracheostomy collar, the trials should be continued two to three times per day with daily increases in time on tracheostomy collar by 1 to 2 hours per trial until total time off the ventilator reaches 18 h/d. At this point, the patient may be ready to remain on tracheostomy collar for longer than 24 hours unless the tolerance criteria are exceeded.
- Ventilator weaning is considered successful once the patient achieves spontaneous ventilation (extubated or on tracheostomy collar) for at least 24 hours.

Adapted from evidence-based practice guidelines used in the Surgical/Trauma Intensive Care Unit, University of Virginia Health System, Charlottesville, Virginia.

fibers and the speed with which they contract. Both of these factors are affected by physiological changes that change the resting position of the diaphragm. With COPD, the resting length is shorter (weakening force of contraction), and with diaphragmatic distention, ascites, or morbid obesity, the diaphragm must push down abdominal contents as it contracts. Reactive airway disease increases the resistance to airflow, with increased workload for muscles of respiration. Any of these abnormalities can lead to significant fatigue of these muscles and respiratory distress.

Respiratory muscle fatigue impedes weaning. It may take as long as 24 hours of complete rest (the mechanical ventilator assumes all of the work of breathing for the patient) for recovery of fatigued respiratory muscles. Therefore, it is common practice to increase ventilatory support at night to ensure rest. This can be accomplished with any of the "resting" modes, so long as the patient's respiratory rate is less than 20 breaths/min. The intent here is to promote and simulate the normal decrease in rate and work of breathing that occurs during each person's sleep–rest cycle. Newer ventilator modes, such as APRV, may be useful to allow gradual reduction of support and increased patient work of breathing.[32]

Weaning trials are discontinued if signs of fatigue or respiratory distress develop. The weaning tolerance criteria and observations are summarized in Boxes 25-18 and 25-19. During physical therapy and activities, it is necessary to monitor the patient for fatigue with use of accessory muscles, increased respiratory rate, and decreased oxygen saturation that indicates respiratory muscle fatigue. The approach to prolonged mechanical ventilation includes aggressive physical therapy and activity to promote muscle conditioning and strength. The use of sedatives and narcotics during weaning should be limited to only the level of medication clearly needed to control pain or anxiety. Special considerations for weaning an older patient from a ventilator are given in Box 25-20.

Regardless of the mode or approach, certain factors have been found to influence weaning success positively. These include the use of collaborative, multidisciplinary teams to

BOX 25-20 CONSIDERATIONS FOR THE OLDER PATIENT

Overcoming Barriers to Ventilator Weaning

An elderly patient on mechanical ventilation poses a unique challenge to care givers. Successful weaning requires effective nursing interventions to address the patient's basic care needs.

- **Sleep Deprivation:** Learn the patient's normal sleep habits, establish a restful environment, and minimize interruptions for at least 6 hours each night and 2 hours of midday rest. Consult the pharmacist or doctor for sleep medication as needed at bedtime.
- **Imbalanced Nutrition: Less Than Body Requirements; and Risk for Fluid Volume Imbalance:** Consult the registered dietitian and begin delivery of recommended nutrition as soon as possible. Assess patient tolerance and increase to goal rate per order. Evaluate fluid balance each shift (input and output, weight, clinical examination) and discuss changes in intake and the possible need for diuretics with the physician.
- **Acute Pain; Anxiety; and Acute Confusion:** Administer analgesia per order, assessing need and effect of intervention by pain scale or physiological parameters. Carefully evaluate possible etiology of anxiety or agitation; when pharmacological intervention is indicated, titrate to achieve desired response using standardized sedation scale to limit sedation. Nonpharmacological interventions are essential in the elderly patient (eg, orientation to place, date, and time; spiritual; massage). Adjust care for hearing impairment or for other sensory limitations that may contribute to confusion or anxiety.
- **Risk for Constipation; and Diarrhea:** Learn the patient's "usual" elimination pattern if possible; start with similar intervention and add more aggressive bowel regimen as needed to establish regular bowel movements. Evaluate factors (eg, medications or narcotics) that may alter bowel function, and ensure adequate hydration by enteral route if possible.
- **Risk for Activity Intolerance; and Impaired Bed Mobility or Impaired Physical Mobility:** Consult the physical or occupational therapist to evaluate functional capacity and initiate appropriate therapy. Begin getting patient out of bed as soon as possible, and encourage active range-of-motion exercises and participation in activities of daily living.

formulate comprehensive plans of care based on assessment of individual patients, the use of standardized weaning protocols that are assigned to each patient based on individual assessment, and the use of critical pathways. The interplay of these strategies, all designed to promote consistency of and rationale for practice, truly leads to outcomes showing that the whole (process) is greater than the sum of its parts.

Short-Term Ventilation Weaning

Patients typically intubated for a short time include those who are intubated for surgical procedures, for an acute exacerbation of an underlying lung disease that can be easily reversed, and for airway protection during an acute neurological event (eg, drug overdose). It is important to evaluate the reason the patient was initially intubated to be certain that mechanical ventilation is no longer indicated. Weaning within a short period of time is desirable because physiological changes caused by the mechanical ventilation begin within 72 hours (see Box 25-18, p. 544).

Frequently used predictive criteria for short-term weaning success are an NIP of less than or equal to −20 cm H_2O (more negative as −30 cm H_2O), PEP of greater than or equal to +30 cm H_2O (more positive as +45 cm H_2O), and spontaneous minute volume of less than 12 L/min. NIP and PEP give an indication of respiratory muscle strength. The choice of weaning method does not appear to be important.

Weaning procedures may vary slightly from hospital to hospital, but general guidelines remain the same. For instance, weaning is generally initiated in the morning when the patient is rested. The patient is made comfortable, and the nurse elevates the HOB. Pharmacological agents for comfort, such as bronchodilators or sedatives, are administered as indicated. Sedation should be minimized to provide best odds for a successful weaning trial. By explaining the procedure, the nurse helps the patient through some of the discomfort and apprehension. Before a weaning trial, the nurse ensures a patent airway and provides suction if necessary.

Support and reassurance help the patient through the discomfort and apprehension as the nurse remains with the patient following initiation of the weaning process. The nurse also evaluates and documents the patient's response to weaning.

Long-Term Ventilation Weaning

The process of long-term weaning often takes weeks. It incorporates gradual and progressive conditioning for respiratory and body muscles using a multidisciplinary team approach. Success with whole body conditioning with emphasis on upper body strength and respiratory muscle function has improved ventilator liberation, and aggressive physiotherapy is necessary. Usually, the entire process is complicated, and it involves multiple delays and setbacks. During long-term weaning, the patient may fail a weaning trial and should then be rested on the ventilator up to 24 hours before another trial is attempted. The rest period allows for recovery of the respiratory muscles. Patients who fail a weaning trial often exhibit rapid, shallow breathing patterns consistent with their respiratory muscle weakness. Regular reevaluation of the weaning plan by the multidisciplinary team, coupled with continuous communication with the patient and family, is necessary (see Box 25-19).

Methods of Ventilator Weaning

Various methods have been studied for weaning from the ventilator. Controversies exist about which methods are best. Some of the most common weaning methods include T-piece, CPAP trials, and gradual PSV reduction to minimal settings. Comprehensive assessment of the patient's needs and progress toward weaning, monitoring of the weaning parameters, and following established goals promote successful weaning. Multidisciplinary and comprehensive approaches to weaning based on health care professional's monitoring and implementing a weaning plan with continuity promote positive outcomes.

T-Piece Trial

The T-piece is connected to the patient at the desired FiO_2 (usually slightly higher than the previous ventilator setting).

The patient's response and tolerance to the trial are continuously observed. The duration of T-piece trials is not standardized, and some clinicians extubate if an initial trial of 30 minutes ends with acceptable ABG values and patient response. Increasing frequency and duration of T-piece trials builds the patient's endurance, with periods of rest on the ventilator between extended trials. When the latter method is used, the patient is generally deemed ready to be extubated after 24 successive hours on a T-piece.

Synchronized Intermittent Mandatory Ventilation Mode

The SIMV mode was initially heralded as the optimal weaning mode, allowing some spontaneous breathing (to prevent respiratory muscle atrophy) while providing a backup rate. Weaning with the SIMV method entails a gradual reduction in the number of delivered breaths until a low rate is reached (usually 4 breaths/min). The patient is then extubated if all other weaning criteria are met. However, low levels of SIMV (fewer than 4 breaths/min) may result in a high level of work and fatigue. SIMV plus PSV, called synchronized pressure-support ventilation (SPSV), may be used to decrease the work of breathing associated with spontaneous breaths. Use of the SPSV mode can easily progress to PSV alone when the patient initiates all breaths by dialing down the ventilator breaths. As a result, PSV "stand-alone" mode is often preferred for weaning trials.

Continuous Positive Airway Pressure Method

CPAP entails breathing through the ventilator circuit with a small amount of (or zero) positive pressure. The use of CPAP instead of the use of a T-piece for weaning is controversial. Often, the decision to use one over the other is determined by observing the patient's response or is simply based on the clinician's preference.

Pressure-Support Ventilation Mode

Low levels of PSV decrease the work of breathing associated with ETTs and ventilator circuits. Weaning using the PSV mode entails a progressive decrease in IPL to 5 to 10 cm H_2O based on the patient maintaining an adequate tidal volume (6 to 12 mL/kg) and a respiratory rate of fewer than 25 breaths/min. PSV is associated with less work of breathing than with volume modes, so longer weaning trials may be tolerated. The 5-cm H_2O IPL is thought to overcome the work of breathing through the ETT and ventilator tubing. Typically, the IPL is reduced by 2 cm H_2O daily or twice daily following the patient's response to the ventilator change. Tolerance of PSV weaning is assessed as any weaning mode by assessing the patient's response to changes in respiratory rate, SaO_2, and heart rate, along with observing for fatigue (see Box 25-18, p. 544; Box 25-19, p. 545). There is support for use of CPAP, T-piece, or even PSV during a spontaneous breathing trial before extubation because each is an effective method for the weaning readiness trial.[33]

Adjuncts to Weaning

Several adjuncts to long-term weaning are used to improve weaning tolerance and patient comfort. The mode on newer ventilators that allows for the regulation of pressure support for the size and type of tube, endotracheal or tracheostomy, adjusts for the type of tube resistance to allow less work of breathing. Called automatic tube compensation (ATC) on some ventilators, this is another tool to consider during weaning. The fenestrated tracheostomy tube provides for communication during weaning periods, improving patient interaction. The fenestrated tracheostomy tube has an opening in the outer cannula but not the inner cannula. With the inner cannula in place and the cuff inflated, mechanical ventilation is easy. During the weaning process, the inner cannula is removed, the cuff deflated, the outer cannula capped, and supplemental oxygen supplied by nasal cannula. This system permits air to pass the vocal cords, allowing verbal communication by the patient. The cuff should never be inflated while the inner cannula is capped because the patient will be unable to breathe. The speaking valves provide communication during weaning periods for patients with nonfenestrated tracheostomy tubes, which are generally used more frequently. These valves provide less resistance than the fenestrated tracheostomy tube, and each type of speaking valve allows for supplemental oxygen through a side port. Humidified air with a tracheostomy collar is required to keep the airway moist and prevent secretions from drying, especially at night when speaking valves should be removed for sleep. Use of a large ETT (>7.0 mm) decreases resistance to breathing and decreases the work of breathing. A larger ETT also supports bronchoscopy and the removal of secretions when needed. Tracheostomy in many instances is more comfortable for patients and allows for improved oral care, better communication, and tracheostomy collar trials for weaning.

Extubation Criteria

Whichever mode or combination of modes is used for weaning, extubation cannot occur until several criteria are met based on short-term or long-term ventilation (see Box 25-18, p. 544; Box 25-19, p. 545). Before extubation, the patient must be able to maintain his or her own airway, as evidenced by an appropriate level of consciousness and the presence of cough and gag reflexes. In all patients, but especially in those with a history of difficult intubation or reactive airway disease, the cuff-leak test should be performed before extubation. This entails deflation of the tube cuff (after suctioning of the oropharynx) and a brief period of occluding the ETT in order to demonstrate an air leak with patient inspiration. Absence of a leak can indicate edema and may predict laryngeal stridor after extubation. If the cuff-leak test fails, the patient may be given corticosteroids to reduce edema for 24 to 48 hours and then be reassessed for cuff leak. A direct visualization of the trachea with a bronchoscope may be performed before extubation to determine whether the edema has resolved.

Extubation should never occur unless a qualified person is available to reintubate emergently if the patient does not tolerate extubation. After explaining the procedure and preparing the patient, the nurse or respiratory therapist suctions the patient's tube and posterior oropharynx. Equipment includes an MRB and mask at bedside. The ETT securing device or tape is loosened and the cuff is deflated. The ETT is removed quickly while having the patient cough. The patient's mouth

is suctioned, and humidified oxygen is applied immediately. The patient is evaluated for immediate signs of distress: stridor, dyspnea, and decrease in SaO_2. Treatment of stridor includes inhaled racemic epinephrine and sometimes administration of IV steroids (because steroids do not work immediately, they are given before extubation in those at risk). If these interventions fail, immediate reintubation may be necessary.

Home Care and Mechanical Ventilation

Certain patients requiring invasive mechanical ventilation may be candidates for home care. These patients may require full or partial invasive mechanical ventilation because of neuromuscular weakness, neurogenic hypoventilation, or cardiopulmonary diseases that result in ineffective gas exchange. Conditions that may warrant home care ventilator management include:

- Neurological disorders (eg, ALS, Guillain-Barré syndrome, multiple sclerosis, muscular dystrophy, myasthenia gravis, poliomyelitis, polymyositis, spinal cord injury)
- Restrictive disorders (eg, interstitial pulmonary fibrosis, kyphoscoliosis, obesity, sarcoidosis)
- Obstructive disorders (eg, bronchiectasis, bronchiolitis obliterans, bronchopulmonary dysplasia, chronic bronchitis and emphysema, cystic fibrosis, obesity, sleep apnea syndromes)

New Frontiers and Challenges for Ventilated Patients

Ventilated patients present major challenges to nurses because they are prone to iatrogenic complications with higher mortality and morbidity. It is important that nurses apply current evidence-based research and practice to patient care so that clinical and financial outcomes may improve. Research on the use of low-volume ventilation, high-frequency ventilation, and the appropriate fluid management in ARDS presents new challenges for nurses responsible for implementing the changes in clinical practice. Ventilator weaning by use of weaning teams, outcome managers, and weaning protocols shows promise for future improvement in outcomes, lowering hospital costs, and effective weaning from ventilators with decreased mortality.[34] These system approaches continue to demonstrate reduction of ventilator days, length of stay, and complications. They will continue to be popular initiatives for hospitals that are attempting to find the best outcomes, quality, and cost-effective solutions for complex patients.

Nurses can use current journals and the Internet to search for information about the most up-to-date information and research. The new nurse should seek access to a health science library database to obtain the most current research for his or her area of practice as well as subscribe to nursing journals. Three excellent sources for the new nurse for advanced topics are (1) the *AACN Procedure Manual for Critical Care*, (2) the *AACN Protocols for Practice: Care of the Mechanically Ventilated Patient*, and (3) the journal titled *AACN Advanced Critical Care*. These resources provide more in-depth coverage of topics related to ventilators, pathophysiology, and patient care.

The new frontiers of nursing and health care will continue to require that nurses keep abreast of rapidly changing practice by continually updating their knowledge. The key to excellent nursing care is evidence-based practice. The goal for new nurses is to integrate the best evidence-based practice, knowledge, therapies, and clinical skills of ventilator care into all aspects of clinical practice. The ever-changing technology and research of the 21st century will provide nurses with continued challenges to update their clinical practice with the latest evidence-based practice and research to provide the respiratory patient with the highest quality care.

▲ Clinical Applicability Challenges

CASE STUDY

Mr. S. is an 85-year-old man who developed a hospital-acquired pneumonia after cervical fusion surgery related to a motor vehicle crash. He has been hospitalized for 20 days. He developed hypoxia respiratory failure and was subsequently intubated. He has been receiving appropriate antibiotic coverage for 7 days. He has remained on mechanical ventilation for 7 days. His vital signs have remained stable. Current vital signs include temperature, 99.0°F; heart rate, 94 beats/min; respiratory rate, 22 breaths/min; and blood pressure, 147/68 mm Hg. Current ventilator settings include assist control, rate 15, tidal volume (TV) 350 mL, PEEP 5, FiO2 40%. He is in no acute distress. He has a small amount of thin white secretions. Current issues include hypoxia respiratory failure, bilateral pleural effusions, renal insufficiency, volume overload, deconditioning, and malnutrition. He has no family support. He is currently alert and able to make his own decisions concerning his care.

1. Explain the criteria that Mr. S. meets to perform ventilator weaning.
2. What factors will likely decrease Mr. S.'s chances of successful weaning?
3. What are the major priorities of nursing care for Mr. S.?

References

1. Ragavan AJ, Evrensel CA, Krumpe PK: Interactions of airflow oscillations, tracheal inclination, and mucus elasticity significantly improves simulated cough clearance. Chest 137(2):355–361, 2010
2. Pierce LNB: Management of the mechanically ventilated patient, 2nd ed. St. Louis, MO: Saunders, Elsevier, 2007
3. Staudinger T, Bojic A, Hozinger U, et al: Continuous lateral rotation therapy to prevent ventilator-associated pneumonia. Crit Care Med 38(2): 486–490, 2010
4. Daniels T: Physiotherapeutic management strategies for the treatment of cystic fibrosis in adults. J Mulitdiscip Healthc 3:201–212, 2010
5. Taccone P, Pesenti A, Latini R, et al: Prone positioning in patients with moderate and severe acute respiratory distress syndrome. JAMA 302(18):1977–1984, 2009
6. Al-Tawfiq JA, Abed MS: Decreasing ventilator-associated pneumonia in adult intensive care units using the Institute for Healthcare Improvement Bundle. Am J Infect Control 38(7):552–556, 2010
7. Swadener-Culpepper L: Continuous lateral rotation therapy. Crit Care Nurse 3(2):55–57, 2010
8. Preventing never events: Pressure ulcers. Joint Commission on Perspectives on Patient Safety (4):5–7, 2009
9. Bergstrom N, Braden BJ, Lagtuzza A, et al: The Braden scale for predicting pressure sore risk. Nurs Res 36:205–210, 1987
10. Halm MA, Krisko-Hagel K: Instilling normal saline with suctioning: Beneficial technique or potentially harmful sacred cow? Am J Crit Care 17(5):469–472, 2008
11. Lawrence DM: Procedure 18, chest tube placement (perform). In Lynn-McHale Wiegand DJ, Carlson KK (eds): AACN Procedure Manual for Critical Care, 6th ed. Philadelphia, PA: Elsevier, 2011
12. Lee TA, Schumock GT, Bartle B, et al: Mortality risk in patients receiving drug regimens with theophylline for chronic obstructive pulmonary disease. Pharmacotherapy 2(9):1039–1053, 2009
13. Gelinas C, Tousignant-Laflamme Y, Tanguay A, et al: Exploring the validity of the bispectral index, the critical-care pain observation tool and vital signs for the detection of pain in sedated and mechanically ventilated critically ill adults: A pilot study. Intensive Crit Care Nurs 27(1):46–52, 2011
14. Checkley W, Brower R, Korpak A, et al: Effects of a clinical trial on mechanical ventilation practices in patients with acute lung injury. Am J Respir Crit Care Med 177(11):1215–1222, 2008
15. Esan A, Hess DR, Raoof S, et al: Severe hypoxemic respiratory failure: Part 1 ventilator strategies. Chest 137(5):1203–1216, 2010
16. Modrykamien A, Chatburn R, Ashton RW: Airway pressure release ventilation: An alternative mode of mechanical ventilation in acute respiratory distress syndrome. Cleve Clin J Med 78(2):101–110, 2011
17. Griffiths MJ, Finney SJ: Small steps in the right direction for ventilator-induced lung injury: Prevention, prevention, prevention. Crit Care Med 3(1):196–197, 2010
18. Sawyer RG, Tache Leon C: Common complications in the surgical intensive care unit. Crit Care Med 38(9):S483–S493, 2010
19. Browne JA, Evans D, Christmas L, et al: Pursuing excellence: Development of an oral hygiene protocol for mechanically ventilated patients. Crit Care Nurs Q 34(1):25–30, 2011
20. Li Bassi G, Torres A: Ventilator-associated pneumonia: Role of positioning. Curr Opin Crit Care 17(1):57–63, 2011
21. Lacherade JC, De Jonghe B, Guezennec P, et al: Intermittent subglottic secretion drainage and ventilator-associated pneumonia. Am J Respir Crit Care Med 182:910–917, 2010
22. Wip C, Napolitano L: Bundles to prevent ventilator-associated pneumonia: How valuable are they? Curr Opin Infect Dis 22(2):159–166, 2009
23. Doig GS, Heighes PT, Simpson F, et al: Early enteral nutrition provided within 24 h of injury or intensive care unit admission, significantly reduces mortality in critically ill patients meta-analysis of randomized control trials. Intensive Care Med 35(12):2018–2027, 2009
24. Weber-Carstens S, Deja M, Koch S, et al: Risk factors in critical illness myopathy during the early course of critical illness: A prospective observational study. Crit Care 14(3):1–12, 2010
25. Zagli G, Linden M, Spina R, et al: Early tracheostomy in intensive care unit: A retrospective study of 506 cases of video-guided Ciaglia Blue Rhino tracheostomies. J Trauma 68(2):367–372, 2010
26. Munro CL, Grap MJ, Jones DJ, et al: Chlorhexidine, tooth brushing, and preventing ventilator-associated pneumonia in critically ill adults. Am J Crit Care 18(5):428–437, 2010
27. Tablan OC, Anderson LJ, Besser R, et al: Guidelines for preventing health-care associated pneumonia, 2003: recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee. MMWR Recomm Rep 53(RR03):1–36, 2004
28. AACN Practice Alert: Oral care for patients at risk for ventilator-associated pneumonia. 2010
29. Bailey JJ, Sabbagh M, Loiselle CG, et al: Supporting families in the ICU: A descriptive correlational study of informational support, anxiety, and satisfaction with care. Intensive Crit Care Nurs 226(2):114–122, 2010
30. Howlett MS, Alexander GA, Tsuchiya B: Health care providers attitudes regarding family presence during resuscitation of adults: An integrated review of the literature. Clin Nurse Spec 24(3):161–174, 2010
31. Henrich NJ, Dodek P, Heyland D, et al: Qualitative analysis of an intensive care unit family satisfaction survey. Crit Care Med 3(5):1–6, 2011
32. Kallet RH: Patient-ventilator interaction during acute lung injury, and the role of spontaneous breathing: Part 2 airway pressure support ventilation. Respir Care 56(2):190–206, 2011
33. Robertson TE, Mann HJ, Hyzy R, et al: Multicenter implementation of a consensus-developed, evidence-based, spontaneous breathing trial protocol. Crit Care Med 36(10):2753–2762, 2008
34. Girard TD, Kress JP, Fuchs BD, et al: Efficacy and Safety of a paired sedation and ventilator weaning protocol for mechanically ventilated patients in intensive care (Awakening and breathing controlled trial): A randomized control trial. Lancet 371(9607):126–134, 2008

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

26 Common Respiratory Disorders

Archana D. Patel, Sun-Ah Lee, Susan E. Anderson, and Debbi S. Spencer*

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Compare the etiology, pathophysiology, assessment, management, and prevention of community-acquired, hospital-acquired, and health care–associated pneumonia.
2. Describe the etiology, pathophysiology, assessment, and management of a patient with severe acute respiratory syndrome.
3. Discuss the pathophysiology, assessment, and management of pleural effusion.
4. Describe the pathophysiology, assessment, and management associated with pneumothorax.
5. Discuss the pathophysiology, assessment, management, and prevention of pulmonary embolism.
6. Explain the pathophysiology, assessment, management, and prevention of chronic obstructive pulmonary disease.
7. Compare the pathological processes, signs and symptoms, and management of chronic bronchitis and emphysema.
8. Describe the pathology, assessment, and management of a patient at various points on the asthma continuum, from mild attack to status asthmaticus.
9. Explain the key characteristics of hypoxemic acute respiratory failure and hypercapnic acute respiratory failure in terms of pathophysiology, assessment, and management.

Pneumonia remains a common infection found in both the community and hospital, even though there have been advances in identifying people at risk and implementing preventive measures. Critical care nurses encounter pneumonia when it complicates the course of a serious illness or leads to acute respiratory distress.

▲ Pneumonia

In the United States, pneumonia is the leading cause of death from infectious disease, the second most common nosocomial (hospital-acquired) infection, and the eighth leading cause of death.[1] In 2006, 56,326 people died as a result of pneumonia or influenza at a rate of 2.6% as compared to 2005 when pneumonia and influenza caused 63,001 deaths per 100,000 population.[1] Community-acquired pneumonia (CAP) is the leading cause of death from an infectious disease in the United States. CAP results in 500,000 hospitalizations and approximately 45,000 deaths in the United States. It is one of the most common causes of admissions to the intensive care unit (ICU). The mortality rate from severe CAP in the ICU can be as high as 58%. Little improvement has occurred in the mortality rate over the past several years.[2] According to some estimates, approximately 915,000 cases of CAP occur in individuals who are 65 years of age or older. The incidence of CAP requiring hospitalization is four times higher in elderly patients (over 65 years of age) than it is in those 45 to 64 years of age.[3]

CAP is defined by the Infectious Diseases Society of America (IDSA) as an acute infection of the pulmonary parenchyma that is associated with either an acute infiltration on chest x-ray or by auscultation of breath sounds that are consistent with pneumonia. CAP can be diagnosed only in individuals who have not been hospitalized or have not resided in a long-term care facility for more than 14 days before the onset of symptoms. There are no cardinal hallmarks of pneumonia.[2] In the outpatient setting, CAP has a low mortality rate (1% to 5%). However, the mortality rate climbs to 14% in patients requiring hospitalization and to 40% in those requiring admission to the ICU.[4]

Two sets of guidelines for treating patients with pneumonia have been issued. In the first set, issued by the Pneumonia Patient Outcomes Research Team (PORT), the initial site of treatment should be selected based on three criteria, which include (1)

*The views of the authors are their own and do not reflect the official policy or position of the Department of the Army, the Department of Defense, or the United States Government.

assessment of any preexisting conditions that compromise the safety of the patient being cared for at home, (2) calculation of the Pneumonia Severity Index (PSI), and (3) clinical judgment.[5] The PSI stratifies patients into five risk categories. The higher the score, the higher the risk for death, admission to the ICU, or readmission, and the longer the length of stay.[6] Patients in risk classes I, II, and III are at low risk for death and can most likely be treated safely in the outpatient setting. Patients in risk classes IV and V should be hospitalized, with those in class V being admitted to the ICU (Box 26-1).[6] In the second

BOX 26-1 Patient Outcomes Research Team (PORT) Pneumonia Severity Index

Pneumonia Patient Outcomes Research Team (PORT) Severity Index[44]

Step 1: Answer questions 1, 2, and 3. If the response is "no" to all three questions, assign patient to risk class I and institute outpatient treatment. If the response is "yes" to any one of the three questions, proceed to step 2 to determine the patient's risk class (II–V).

1. **Is the patient older than 50 years of age?**
2. **Does the patient have a history of any of the following coexisting conditions*?**
   Neoplastic disease, congestive heart failure (CHF), cerebrovascular disease, renal disease, liver disease
3. **Does the patient have any of the following abnormalities on physical examination?**
   Altered mental status†, pulse more than 125/min, respiratory rate more than 30 breaths/min, systolic blood pressure <90 mm Hg, temperature <35°C or >40°C

Step 2: Assess the following characteristics

Characteristic	Points assigned	Score
Demographic factor		
Age		
Men	Age (y)	
Women	Age (y) –10	
Nursing home resident	+10	
Coexisting illnesses*		
Neoplastic disease	+30	
Liver disease	+20	
Congestive heart failure	+10	
Cerebrovascular disease	+10	
Renal disease	+10	
Physical examination findings		
Altered mental status†	+20	
Respiratory rate >30 breaths/min	+20	
Systolic blood pressure <90 mm Hg	+20	
Temperature <35°C (95°F) or >40°C (104°F)	+15	
Pulse > 125 beat/min	+10	
Laboratory and radiographic findings (if study performed)		
Arterial blood pH < 7.35	+30	
Blood urea nitrogen (BUN) level >30 mg/dL	+20	
Sodium level <130 mmol/L	+20	
Glucose level >250 mg/dL	+10	
Hematocrit <30%	+10	
Partial pressure of arterial O_2 < 60 mm Hg Or Sat < 90%‡	+10	
Pleural effusion	+10	
Total score is the sum of points to identify the severity score and determine the patient's risk class.		

*Neoplastic disease is defined as any cancer—except basal- or squamous-cell cancer of the skin—active at time of presentation or diagnosed within 1 year of presentation. Liver disease is defined as a clinical or histologic diagnosis of cirrhosis or another form of chronic liver disease, such as chronic active hepatitis. CHF is defined as systolic or diastolic ventricular dysfunction documented by history, physical examination, and chest x-ray, echocardiogram, multiple-gated acquisition scan, or left ventriculogram. Cerebrovascular disease is defined as a clinical diagnosis of stroke or transient ischemic attack or stroke documented by magnetic resonance imaging or computed tomography. Renal disease is defined as a history of chronic renal disease or abnormal BUN or creatinine.

†Altered mental status is defined as disorientation with respect to person, place, or time that is not known to be chronic, stupor, or coma.

‡In the Pneumonia PORT cohort study, an oxygen saturation <90% on pulse oximetry or intubation prior to admission was also considered abnormal.

Step 3: Determine the patient's risk class

Total Score	Risk Class	Mortality*	Recommended Site of Treatment
None (see Step 1)	I	0.1%	Outpatient
<70	II	0.6%	Outpatient
71–90	III	0.9%	Outpatient
91–130	IV	9.3%	Inpatient
>130	V	27%	Inpatient

From Miskovich-Riddle L, Keresztes P: CAP management guidelines. Nurse Pract 31(1):43–53, 2006.

BOX 26-2 American Thoracic Society Criteria for Diagnosis of Severe Community-Acquired Pneumonia

Major Criteria
- Invasive mechanical ventilation
- Septic shock with the need for vasopressor

Minor Criteria
- Respiratory rate more than 30 breaths/min
- PaO_2/FiO_2 ratio less than 50
- Multilobar infiltrates
- Confusion/disorientation
- Uremia (BUN level 20 or more mg/dL)
- Leukopenia (WBC count <4,000 cells/mm^3)
- Hypothermia (core temperature <36°C)
- Hypotension requiring aggressive fluid resuscitation

BUN, blood urea nitrogen; PaO_2/FiO_2 ratio, arterial oxygen pressure/fraction of inspired oxygen; WBC, white blood cell.

From American Thoracic Society: Guidelines for the management of adults with community-acquired pneumonia. Am J Respir Crit Care Med 163:1730–1754, 2001, with permission; Mandell LA, et al. Infectious Disease Society of America/American Thoracic Society consensus guidelines on the management of community-acquired pneumonia in adults. Clin Infect Dis 44(Suppl 2) S27–S72, 2007.

set of guidelines developed by the American Thoracic Society (ATS), patients with severe CAP require admission to the ICU. Severe CAP is defined as the presence of one of two major criteria or the presence of two of three minor criteria (Box 26-2).[7]

Hospital-acquired pneumonia (HAP), ventilator-associated pneumonia (VAP), and health care–associated pneumonia (HCAP) continue to be important causes of morbidity and mortality despite advances in antimicrobial therapy and advanced supportive measures.[8] HAP is defined as pneumonia occurring more than 48 hours after admission to a hospital, which excludes infection that is incubating at the time of admission.[9] HAP occurs at a rate of 5 to 10 cases per 1,000 hospital admissions, and the incidence increases by 6- to 20-fold in patients receiving mechanical ventilation.[10] VAP is defined as the occurrence of pneumonia more than 48 to 72 hours after intubation. If a person experiences a severe episode of HAP requiring intubation and mechanical ventilation, that person should then be managed like a patient with VAP.[9] HCAP is defined as pneumonia in any patient who has been hospitalized in an acute care hospital for 2 or more days within 90 days of infection; has lived in a nursing home or long-term care facility; has received recent intravenous antibiotic therapy, chemotherapy, or wound care within the past 30 days of the current infection; or has received treatment at a hospital or hemodialysis clinic.[8]

The ATS HAP guidelines state that the criteria defining severe CAP can also be used to define severe HAP.[6] Severe HAP may occur in the ICU, with patients receiving mechanical ventilation being at the greatest risk, or it may precipitate admission to the ICU.[7] HAP independently contributes to mortality in critically ill patients; the attributable mortality rate is 33% to 50%.[9]

Etiology

Bacteria, viruses, mycoplasmas, other infectious agents such as fungi, and foreign material can all cause pneumonia. The specific etiology varies greatly depending on the type of pneumonia (CAP or HAP).[10] *Streptococcus pneumoniae* (pneumococcus) is the predominant pathogen associated with CAP, accounting for 30% to 60% of all occurrences of CAP. It is the most common cause of CAP identified in patients requiring hospitalization. Other organisms considered to be causative agents in CAP include *Haemophilus influenzae*, *Staphylococcus aureus*, and other Gram-negative bacilli.[5] Even in the approximately 50% of cases of CAP in which the causative organism is not identified, *S. pneumoniae* is believed to predominate.[5] Drug-resistant *S. pneumoniae* is frequently seen in individuals older than 65 years of age.[5] Pathogens that should be considered in severe CAP requiring admission to the ICU include *S. pneumoniae*, *Chlamydia pneumoniae*, *S. aureus*, *Mycobacterium tuberculosis*, *Legionella* species, respiratory viruses, and endemic fungi.[11]

Etiologic factors may be used to classify pneumonia as typical or atypical. Typical pneumonia, caused by pathogens such as *S. pneumoniae*, *Streptococcus pyrogenes*, and *Staphylococcus aureus*, is the result of the bacteria multiplying in the alveoli, causing inflammation and accumulation of fluid within the alveoli.[3] Atypical pneumonia is caused by pathogens such as *Mycoplasma pneumoniae*, *C. pneumoniae*, influenza virus, adenovirus, and *Legionella* species that give rise to inflammatory changes in the alveolar septa and lung interstitium.[3,10]

Time of onset of pneumonia is an important factor in determining the specific pathogens and outcomes in patients with HAP and VAP.[9] HAP and VAP occurring within 4 days of admission are more likely to be caused by bacteria sensitive to antibiotics. HAP and VAP occurring later on are more likely to be caused by resistant organisms.[9] HAP may be polymicrobial, and common causative pathogens include aerobic Gram-negative bacilli, such as *Escherichia coli*, *Klebsiella pneumoniae*, and *Pseudomonas aeruginosa*, as well as Gram-positive cocci such as *S. aureus*.[9] Polymicrobial HAP is particularly common (>50%) in patients receiving mechanical ventilation (VAP). Highly resistant Gram-negative organisms (eg, *P. aeruginosa*, *Acinetobacter* species) and methicillin-resistant *S. aureus* are frequently seen in late-onset HAP but may occur in early-onset HAP in patients with risk factors for these pathogens.[9,11] The spectrum of potential pathogens can be defined by assessment of a variety of factors, including the severity of the pneumonia, comorbidities, prior therapy (including antibiotics), and length of hospitalization.[9]

Pathophysiology

Pneumonia is an inflammatory response to inhaled or aspirated foreign material or the uncontrolled multiplication of microorganisms invading the lower respiratory tract. This response results in the accumulation of neutrophils and other proinflammatory cytokines in the peripheral bronchi and alveolar spaces.[11] The body's defense system, which includes anatomical, mechanical, humoral, and cellular defenses, is designed to repel and remove organisms entering the respiratory tract. Many systemic diseases increase the patient's risk for pneumonia by altering the respiratory defense mechanism. Pneumonia develops when normal pulmonary defense mechanisms are either impaired or overwhelmed, allowing microorganisms to multiply rapidly. The severity of pneumonia depends on the amount of material aspirated, the virulence of the organism, the amount of bacteria in the aspirate, and the host defenses.[11]

The means by which pathogens enter the lower respiratory tract include aspiration, inhalation, hematogenous spread from a distant site, and translocation. Risk factors that predispose an individual to one of these mechanisms may be categorized as (1) conditions that enhance colonization of the oropharynx, (2) conditions favoring aspiration, (3) conditions requiring prolonged intubation, and (4) host factors.[5] Colonization of the oropharynx (colonization is the presence of microorganisms other than the normal flora in the absence of clinical evidence of infection) has been identified as an independent factor in the development of HAP. Gram-positive bacteria and anaerobic bacteria normally live in the oropharynx, and they occupy bacterial binding sites in the oropharyngeal mucosa. When normal oropharyngeal flora is destroyed, these binding sites are susceptible to colonization by pathogenic bacteria. Risk factors associated with oropharyngeal colonization include previous antibiotic therapy, increased age, dental plaque, smoking, and chronic diseases, such as chronic obstructive pulmonary disease (COPD), gastroesophageal reflux disease, alcoholism, diabetes mellitus, and malnutrition.[11,12] The exact role the stomach plays in the development of pneumonia is controversial. In healthy individuals, the stomach is normally sterile because of the bactericidal activity of hydrochloric acid. However, when gastric pH increases above normal (pH > 4), as occurs with the use of histamine-2 antagonists and antacids for stress ulcer prophylaxis, microorganisms are able to multiply.[9] Gastric colonization increases retrograde colonization of the oropharynx and increases the risk for pneumonia. Individuals at risk for gastric colonization include the elderly; those with achlorhydria, ileus, or upper gastrointestinal disease; and those receiving antacids, histamine-2 antagonists, or enteral feedings.[2,5] The Gram-negative or pathogenic Gram-positive organisms that have colonized the oropharynx are readily available for aspiration into the tracheobronchial tree.

Aspiration occurs frequently in healthy individuals while they sleep. The risk for clinically significant aspiration is increased in individuals who are unable to protect their airways (eg, in patients with alcohol abuse, a depressed level of consciousness, or dysphagia; in those who have endotracheal or enteral tubes; or in those receiving enteral feedings). Aspiration of bacteria found in dental plaques is receiving increased attention as a significant source of pneumonia.

Inhalation of bacteria-laden aerosol devices from contaminated respiratory equipment is another potential source of pneumonia-causing bacteria. Condensate that collects in the ventilator tubing can become contaminated with secretions and serve as a reservoir for bacterial growth.[12] Inhalation is an effective entry mechanism for *Legionella* species, M. *tuberculosis*, certain viruses, and fungi. Organisms are carried through small inhaled droplets from the tracheobronchial tree into the lower respiratory tract.[12]

Hematogenous spread serves as a mechanism for the development of pneumonia; the pulmonary circulation provides a potential portal of entry for microbes. The pulmonary capillaries form a dense network in the walls of the alveoli that is ideal for gas exchange. Hematogenous microbes from distant sites of infection can migrate through this network and cause pneumonia. (Pneumonia can also cause bacteremia. Secondary bacteremia after pneumonia has been reported in 6% to 20% of pneumonia cases.) Finally, translocation of bacterial toxins from the gut lumen to the mesenteric lymph nodes and eventually to the lungs may possibly cause bacterial pneumonia. However, translocation has not yet been confirmed as a pathophysiological mechanism.[9]

Assessment

History

Knowledge of risk factors and symptoms can assist in identifying potential pathogens causing CAP and HAP. Hemoptysis implies tissue necrosis and is more common with pyogenic streptococcal pneumonia, anaerobic lung abscesses, *S. aureus*, necrotizing Gram-negative organisms, and invasive *Aspergillus* species.[11] Extrapulmonary symptoms may indicate specific pathogens; diarrhea and abdominal discomfort are present with *Legionella* species, and otitis media and pharyngitis are present with M. *pneumoniae*.[12] The clinical presentation in the older adult may vary somewhat from what is "typical" in a younger person (Box 26-3).

Historical information may also be extremely helpful in diagnosis of CAP and HAP. It is important to include information about contact with animals, especially birds, bats, rats, and rabbits, which can assist with the diagnosis of histoplasmosis, psittacosis, tularemia, and plague. In addition, a complete history that includes dental hygiene history and place of residence may assist in the differential diagnosis.[11] Diseases that may mimic pneumonia include heart failure, atelectasis, pulmonary thromboembolism, drug reactions, pulmonary hemorrhage, and acute respiratory distress syndrome (ARDS).

Physical Findings

A comprehensive cardiovascular and pulmonary assessment should be completed, with a focus on the ATS major and minor criteria (see Box 26-2, p. 552). The nurse assesses for signs of hypoxemia (duskiness or cyanosis) and dyspnea. Patients presenting with new-onset respiratory symptoms (eg, cough, sputum production, dyspnea, pleuritic chest pain) usually have an accompanying fever and chills. Inspection of

BOX 26-3 CONSIDERATIONS FOR THE OLDER PATIENT

Pneumonia

- **Presentation.** The usual symptoms (fever, chills, increased white blood count) may be absent. Confusion and tachypnea are common presenting symptoms in older patients with pneumonia. Other symptoms in the older patient include weakness, lethargy, failure to thrive, anorexia, abdominal pain, episodes of falling, incontinence, headache, delirium, and nonspecific deterioration.
- **Prevention.** People 65 years of age and older should receive both the pneumococcal vaccine (a one-time vaccination) and yearly influenza vaccines. The Health Care Financing Agency has approved the use of standing orders to give the vaccines to Medicare patients.

the chest includes assessing respiratory pattern and respiratory rate, observing the patient's posture and work of breathing, and inspecting for the presence of intercostal retractions. Percussion of the chest frequently reveals dullness with lobar pneumonia. Decreased breath sounds are heard on auscultation. Crackles or bronchial breath sounds are heard over the area of consolidation.

Extrapulmonary symptoms may include myalgia and gastrointestinal symptoms. Confusion may be a subtle symptom in elderly patients.[12,13]

Diagnostic Studies

The workup for severe CAP and HAP is similar. Diagnostic tests are ordered for two reasons: to determine whether the pneumonia is the cause of the patient's symptoms and to determine the pathogen when pneumonia is present.[9] Table 26-1 summarizes the current ATS recommendations. The diagnostic evaluation must be performed rapidly to prevent delays in initiation of antibiotic therapy.

All patients should have a chest radiograph (posteroanterior and lateral views) to identify both the presence and location of infiltrates. The chest radiograph is helpful in differentiating pneumonia from other conditions and identifying severe pneumonia, which is indicated by the presence of multilobular, rapidly spreading, or cavitary infiltrates.

The value of examining lower respiratory secretions with Gram stain and culturing sputum is controversial. The ATS does not recommend routine use of Gram stain and sputum culture and advises that results must be interpreted cautiously.[7,9] However, the IDSA recommends routine Gram stain and culture of deep-cough specimens.[5] Lower respiratory secretions can be easily obtained in intubated patients using endotracheal aspiration. Nonquantitative endotracheal aspiration cultures may assist in excluding certain pathogens and may be helpful in modifying initial empirical treatment.[5] Routine use of quantitative invasive diagnostic techniques (bronchoscopy with protected specimen brush [PSB] or bronchoalveolar lavage [BAL]) in severe pneumonia is not recommended by the ATS, the Centers for Disease Control and Prevention (CDC), or the IDSA.[5–7] Current guidelines suggest that BAL or PSB be used only in selected circumstances, such as in nonresponse to antimicrobial therapy, immunosuppression, suspected tuberculosis in the absence of a productive cough, pneumonia with suspected neoplasm or foreign body, or conditions that require lung biopsy.[5–7] The IDSA recommends HIV testing for people age 15 to 54 years as well.[5] Pneumococcal urinary antigen assay is being recommended as an addition to blood culture testing. The advantage of this test is the rapidity with which results are obtained (within 15 minutes).[5]

Table 26-1 Diagnostic Studies in Patients With Severe Community-Acquired Pneumonia or Severe Hospital-Acquired Pneumonia

Study	Rationale
Chest radiograph (anterior–posterior and lateral)	For evaluation of patient who are likely to have pneumonia, to aid in differentiating diagnosis To assess for pleural effusion
Pretreatment blood samples for culture (two sets of blood cultures from separate sites): Not necessary for community-acquired pneumonia (CAP), except for those with intensive care unit (ICU) admission, failure of outpatient antibiotic therapy, cavitary infiltrates, leukopenia, active alcohol abuse, severe liver disease, asplenia, positive pneumococcal urinary antigen test (UAT) result, and pleural effusion	To evaluate possible cause of severe CAP because of higher yield, great possibility of the presence of pathogen not covered by the usual empirical antibiotic therapy, and to narrow antibiotic treatment to treat the pathogen
Complete blood count Serum electrolytes Renal and liver function	To document the presence of multiple-organ dysfunction To evaluate for leukopenia that is associated with high incidence of bacteremia To help define severity of illness
Arterial blood gases (ABGs)	To define severity of illness To determine need for supplemental oxygen and mechanical ventilation
Thoracentesis (if pleural effusion is >10 mm identified on lateral decubitus film) Pleural fluid studies, including: White blood count with differential Gram stain and acid-fast stain Culture for bacteria, fungi, and mycobacteria	To rule out empyema
Pretreatment Gram stain and culture of expectorated sputum	Gram stain results will help broaden initial empirical coverage for less common pathogens and validate sputum culture results.
Obtain UAT for *Legionella pneumophila* and *Streptococcus pneumoniae*	To rule out *Legionella* and *Streptococcus*

From data in Mandell LA, et al: Infectious Disease Society of America/American Thoracic Society consensus guidelines on the management of community-acquired pneumonia in adults. Clin Infect Dis 44(Suppl 2):S27–S72, 2007.

Management

Antibiotic Therapy

Antibiotic therapy is the cornerstone of treatment for both CAP and HAP. Patients should initially be treated empirically, based on the severity of disease and the likely pathogens.[9] Table 26-2 presents ATS guidelines for treatment of severe CAP, and Table 26-3 presents guidelines for treatment of HAP and VAP. Initial therapy should be instituted rapidly. Data show that hospitalized patients with CAP who receive their first dose of antibiotic therapy within 8 hours of arrival at the hospital have reduced mortality at 30 days.[7] Double antibiotic coverage is necessary for people with severe CAP.[11] Initial therapy should not be changed within the first 48 to 72 hours unless progressive deterioration is evident or initial microbiological (blood or respiratory) cultures indicate a need to modify therapy.[7,9]

Factors to consider when determining the duration of therapy include concurrent illness, bacteremia, severity of pneumonia at the onset of antibiotic therapy, infecting pathogens, risk for multidrug resistance, and rapidity of clinical response.[7,9] Recommended duration of therapy is 7 to 10 days for *S. aureus* or *H. influenzae*; 10 to 14 days for *M. pneumoniae* or *C. pneumoniae*; and 8 to 14 days for *P. aeruginosa*, *Acinetobacter* species, multilobar involvement, malnutrition, or a necrotizing Gram-negative bacillus.[11]

Supportive Therapy

Common nursing diagnoses for a patient with a respiratory disorder (such as pneumonia) are given in Box 26-4. Oxygen therapy may be required to maintain adequate gas exchange. Mechanical ventilation to correct hypoxemia is frequently required in both severe CAP and HAP. Humidified oxygen should be administered by mask or endotracheal tube to promote adequate ventilation. Aggressive pulmonary toilet is indicated to mobilize secretions, open closed alveoli, and promote oxygenation. Adequate nutritional support is critical. In addition, a nutritional consult should be initiated with implementation of appropriate enteral or parenteral therapy.

Prevention

Because pneumonia is the seventh leading cause of death in the United States, prevention of both CAP and HAP is essential. Primary measures to prevent CAP include the use of influenza and pneumococcal vaccines.[7,9] All immunocompetent patients 50 years of age or older should receive the injected form of the inactivated influenza vaccine. The live attenuated intranasal influenza vaccine is an alternative for people 5 to 49 years of age without chronic disease. The pneumococcal vaccine is recommended for people older than 65 years of age and those with chronic illnesses, such as cardiovascular disease, COPD (but not asthma), diabetes mellitus, alcoholism, chronic liver disease, cerebrospinal fluid leaks, and functional or anatomic asplenia; those who belong to special populations, such as Alaska natives or other Native Americans; or those who live in special social settings, such as long-term care facilities.[7,9] The ATS recommends influenza vaccine for three target groups: persons at a high risk for influenza compli-

Table 26-2 Recommended Therapy for Community-Acquired Pneumonia, Inpatients*

Type of Patient	Therapy†,‡
Non-ICU inpatient	A respiratory fluoroquinolone A beta-lactam *plus* macrolide
ICU patient	A beta-lactam (cefotaxime, ceftriaxone, or ampicillin/sulbactam) *plus* either azithromycin or a respiratory fluoroquinolone (for penicillin-allergic patient, a fluoroquinolone and aztreonam are recommended)
At risk for *Pseudomonas aeruginosa*	Selected intravenous antipseudomonal beta-lactam (cefepime, imipenem, meropenem, piperacillin/tazobactam) plus intravenous antipseudomonal quinolone (ciprofloxacin or levofloxacin) or Selected intravenous antipseudomonal beta-lactam (cefepime, imipenem, meropenem, piperacillin/tazobactam) plus intravenous macrolide (azithromycin) and aminoglycoside or Selected intravenous antipseudomonal beta-lactam (cefepime, imipenem, meropenem, piperacillin/tazobactam) *plus* aminoglycoside and intravenous antipneumococcal fluoroquinolone (for penicillin-allergic patients, substitute aztreonam for above beta-lactam)
At risk for CA-methicillin-resistant *Staphylococcus aureus* (MRSA)	Add vancomycin or linezolid

*Excludes patients at risk for HIV.

†Combination therapy required.

‡In no particular order.

Reprinted with permission from the Infectious Disease Society of America/American Thoracic Society consensus guidelines on the management of community-acquired pneumonia in adults. Clin Infect Dis 44(Suppl 2):s27–s72, 2007.

Table 26-3 Antibiotic Therapy for Patients With Hospital-Acquired or Ventilator-Associated Pneumonia

Initial Empiric Antibiotic Therapy for Hospital-Acquired Pneumonia or Ventilator-Associated Pneumonia in Patients With No Known Risk Factors for Multidrug-Resistant Pathogens, Early Onset, and Any Disease Severity

Potential Pathogen	Recommended Antibiotic
*Streptococcus pneumoniae**	Ceftriaxone
Haemophilus influenzae	*or*
Methicillin-sensitive *Staphylococcus aureus*	Levofloxacin, moxifloxacin, or ciprofloxacin
Antibiotic-sensitive enteric Gram-negative bacilli	*or*
Escherichia coli	Ampicillin/sulbactam
Klebsiella pneumoniae	*or*
Enterobacter species	Ertapenem
Proteus species	
Serratia marcescens	

*The frequency of penicillin-resistant *S. pneumoniae* and multidrug-resistant *S. pneumoniae* is increasing; levofloxacin and moxifloxacin are preferred to ciprofloxacin, and the role of other new quinolones, such as gatifloxacin, has not been established.

Initial Empiric Therapy for Hospital-Acquired Pneumonia, Ventilator-Associated Pneumonia, and Health Care–Associated Pneumonia in Patients With Late-Onset Disease or Risk Factors for Multidrug-Resistant Pathogens and All Disease Severity

Potential Pathogens	Combination Antibiotic Therapy*
Pathogens listed in above table and MDR pathogens	Antipseudomonal cephalosporin (cefepime, ceftazidime)
Pseudomonas aeruginosa	*or*
Klebsiella pneumoniae (ESBL+)†	Antipseudomonal carbapenem (imipenem or meropenem)
Acinetobacter species‡	*or*
	β-Lactam/β-lactamase inhibitor (piperacillin/tazobactam) *plus*
	Antipseudomonal fluoroquinolone† (ciprofloxacin or levofloxacin)
	or
	Aminoglycoside (amikacin, gentamicin, or tobramycin) *plus*†
MRSA	Linezolid or vancomycin
Legionella pneumophila†	

*Initial antibiotic therapy should be adjusted or streamlined on the basis of microbiologic data and clinical response to therapy.

†If MRSA risk factors are present or there is a high incidence locally.

‡If an ESBL+ strain, such as *K. pneumoniae,* or an *Acinetobacter* species is suspected, a carbapenem is a reliable choice. If *L. pneumophila* is suspected, the combination antibiotic regimen should include a macrolide (eg, azithromycin) or a fluoroquinolone (eg, ciprofloxacin or levofloxacin) should be used rather than an aminoglycoside.

Reprinted with permission from the Infectious Diseases Society of America/American Thoracic Society consensus guidelines for the mangement of adults with hospital-acquired, ventilator-associated, and healthcare-associated pneumonia. Am J Respir Crit Care Med 171:388–416, 2005.

cations, persons who may transmit influenza to high-risk patients (eg, health care workers), and any person who wishes to decrease the chance of becoming infected with influenza.[9] High-risk patients include people older than 65 years of age, residents of long-term care facilities, patients with chronic cardiovascular or pulmonary disease, patients who required regular medical care or hospitalization during the preceding year, and pregnant women in the second or third trimester during influenza season. Because cigarette smoking is a risk factor for both HAP and CAP pneumonia, smoking cessation, particularly in patients who have previously had pneumonia, is an important preventive strategy.[9]

A complete understanding of the pathogenesis of HAP enables the critical care nurse to develop strategies about interventions that prevent the onset of pneumonia. The CDC, IDSA, and ATS consider education the cornerstone of an effective infection control program and the prevention of HAP.[5,9] Targets of opportunity in the prevention of HAP include strict infection control, handwashing using an alcohol-based disinfectant, surveillance for pathogens, and early removal of invasive lines.[9] The American Association of Critical-Care Nurses (AACN) has published guidelines for preventing VAP as part of their AACN Practice Alerts (see Chapter 25). According to the evidence-based directive from AACN, in all patients receiving mechanical ventilation as well as those at high risk for aspiration (1) the head of the bed should be elevated at 30 to 45 degrees unless medically contraindicated, (2) endotracheal tubes should have dorsal lumens above the cuff to allow drainage and continuous tracheal secretions, and (3) patient ventilator circuits should be changed based on need because of contamination rather than by routine.[14] The CDC has published comprehensive guidelines on the prevention of

BOX 26-4 EXAMPLES OF NURSING DIAGNOSES

For the Patient With a Respiratory Disorder

- Ineffective Airway Clearance related to pulmonary secretions
- Anxiety related to impaired breathing function
- Impaired Gas Exchange related to ventilation–perfusion imbalance
- Risk for Deficient Fluid Volume related to dyspnea and fever
- Activity Intolerance related to imbalanced oxygen supply and demand
- Ineffective Breathing Pattern related to respiratory muscle fatigue
- Deficient Knowledge related to disease process and treatment
- Imbalanced Nutrition: Less Than Body Requirements related to decrease in appetite secondary to medical treatment and fatigue
- Risk for Infection
- Disturbed Sleep Pattern related to dyspnea
- Ineffective Peripheral Tissue Perfusion related to decrease in cellular exchange
- Social Isolation related to change in physical well-being

HAP.[9] Internet access to the CDC guidelines is available at http://www.cdc.gov.

▲ Severe Acute Respiratory Syndrome

Severe acute respiratory syndrome (SARS), a viral illness of the lower respiratory tract, is caused by the SARS-associated coronavirus (SARS-CoV).[15] First cases were reported in Guangdong Province, China, in November 2002, and subsequently identified in Hong Kong, Vietnam, Thailand, Taiwan, Switzerland, Germany, Singapore, and Canada.[15,16] A total of 8,098 people who became sick with SARS, resulting in 774 deaths, were reported to the World Health Organization (WHO) between November 2002 and July 2003.[15] The illness has not been as prevalent in the United States, with only eight people having been hospitalized with evidence of the SARS-CoV.[15] What these people had in common was international travel to a SARS-affected region within 10 days of hospital admission.

The SARS virus is transmitted from person to person by droplets, direct or indirect contact, or viral shedding in feces and urine.[15,17] Droplets spread when an infected person coughs or sneezes and droplets land on the mucous membranes of the mouth, nose, or eyes of a person who is usually located within a distance of 3 ft. Direct or indirect contact results when a person touches a surface contaminated with droplets and then touches his or her eyes, nose, or mouth.[15] The virus spreads quickly, and medical personnel are among those commonly infected.[15,17]

The period from exposure to the onset of symptoms, or prodromal phase of SARS, is usually 2 to 7 days, although it may be as long as 10 to 14 days. The first symptoms are fever (>38°C), chills, and rigor.[15,17,18] Other symptoms include headache, malaise, and myalgias. Mild respiratory symptoms may be present. Inspiratory crackles may be heard at the bases of the lungs. Chest radiographs may be normal, or focal airspace infiltrates may be present in the peripheral lung zones (Fig. 26-1). Initial blood counts indicate lymphopenia, although the neutrophil count and monocyte count are often normal.[16,17] Sputum and blood cultures are obtained to rule out the other causes of atypical pneumonia to include *M. pneumoniae*, *C. pneumoniae*, human cytomegalovirus, adenoviruses, respiratory syncytial virus, and influenza viruses.[17]

After 3 to 7 days, the lower respiratory phase begins, as evidenced by a dry, nonproductive cough and dyspnea that may be accompanied by progressive hypoxemia.[17] In up to 20% of the cases, the respiratory illness is severe enough to require intubation and mechanical ventilation.[19] The focal infiltrates progress to more generalized, interstitial infiltrates, and significant consolidation is evident.[16] Findings of ground-glass opacification in the periphery are typical on thoracic computed tomography (CT) scan.[16] Laboratory findings include leukopenia, lymphopenia, and thrombocytopenia on the complete blood count. Serum alanine aminotransferase, creatinine kinase (CK), and lactate dehydrogenase (LDH) may be elevated, indicating extensive lung injury.[16,17] Advanced age, male gender, high peak CK, high LDH, high neutrophil count, and low serum sodium have been found to be predictive of significant illness, ICU admission, and death.[16,17]

Until SARS is specifically identified, the treatment plan for a patient with a suspected diagnosis of SARS includes empiric therapy using antibiotics effective against typical and atypical pathogens.[18] Antiviral agents such as ribavi-

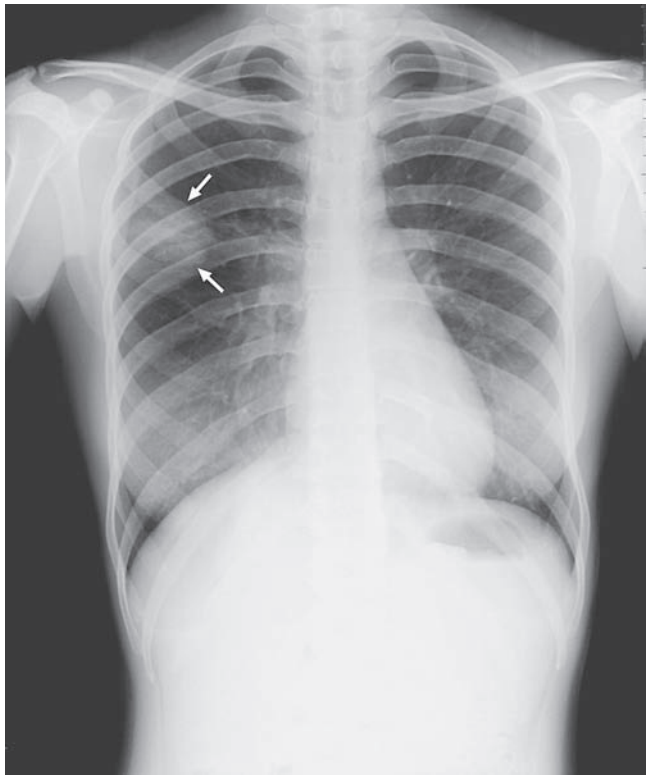


FIGURE 26-1 ▲ Frontal chest radiograph in a 25-year-old woman showing ill-defined airspace shadowing. (From Lee N, Hui D, Wu A, et al: A major outbreak of severe acute respiratory syndrome in Hong Kong. N Engl J Med 348(20):1986–1994, 2003. Copyright © 2003. Massachusetts Medical Society. All rights reserved.)

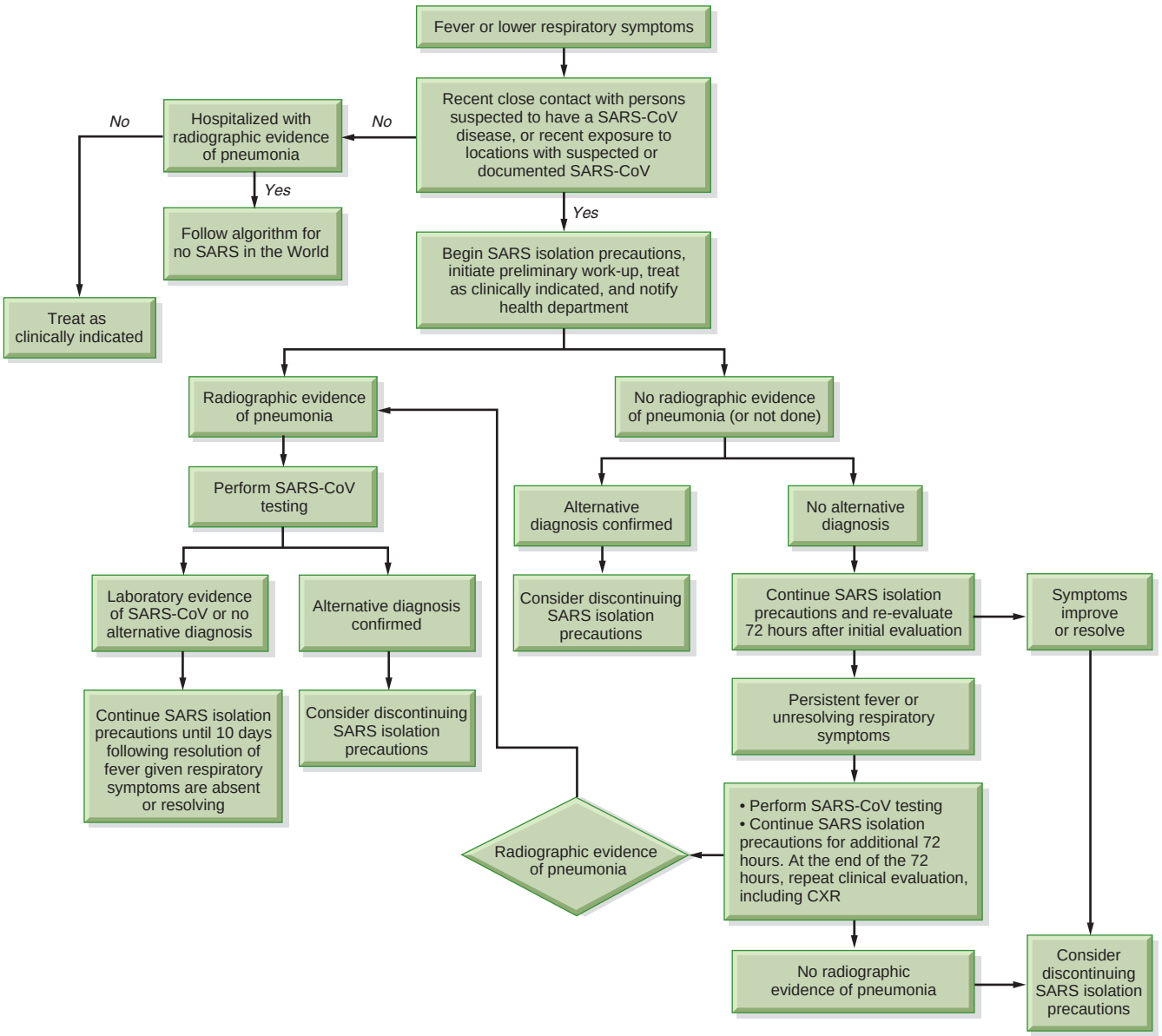


FIGURE 26-2 ▲ Algorithm for management of patients with fever or lower respiratory symptoms when person-to-person transmission of SARS-CoV is occurring in the world. (From Centers for Disease Control and Prevention: Clinical guidance on the identification and evaluation of possible SARS-CoV disease among persons presenting with community-acquired illness, Version 2, Supplement I: Infection control in health care, home, and community settings. Washington, DC: Department of Health and Human Services Centers for Disease Control and Prevention, 2004, pp 1–28.)

rin and corticosteroids may be administered as well.[20] In an analysis of 110 patients with SARS, 61% had an incident of hemolytic anemia, 57% hypocalcemia, and 46% hypomagnesemia.[20] Ventilatory support may be provided either through noninvasive positive-pressure ventilation or, as patient condition dictates, intubation with mechanical ventilation.

Because SARS is a virulent pathogen, a key part of the care of the patient is the institution of strict infection control measures. Droplet and contact precautions are a necessity. Personal protective equipment, including a gown, gloves, surgical mask or N95 disposable particulate respirator, and face shield, should be worn by those in contact with the patient.[17] A private room with negative air flow is recommended.[21] Strict handwashing and decontamination of any equipment used in patient care are a must (Fig. 26-2).[21] When patients are discharged, they should be instructed to limit their interactions outside the home and not go to any public places until 10 days after fever and respiratory symptoms resolve.[17]

▲ Pleural Effusion

The pleural space is a space between the visceral pleural that lines the lungs and parietal pleurae that lines the interior chest wall. This space is lubricated with 15 mL of serous

fluid.[22] The fluid originates from the parietal pleura and is eventually absorbed by the pulmonary lymphatics.

Pathophysiology

Pleural effusion is the accumulation of pleural fluid due to an increased rate of fluid formation, a decreased rate of fluid removal, or both.[23] This is caused by at least one of the five following mechanisms:[23]

- Increased pressure in pulmonary capillaries (eg, heart failure, massive PE)
- Increased capillary permeability (eg, pneumonia, malignancy, infection, pancreatitis)
- Decreased plasma osmotic pressure (eg, hypoalbuminemia, hypoproteinemia, cirrhosis)
- Increased intrapleural negative pressure (eg, atelectasis, trapped lung)
- Impaired lymphatic drainage of the pleural space (eg, pleural malignancy or infection)

Transudates

Transudative pleural effusions, unilateral or bilateral, are an ultrafiltrate of plasma, indicating that the pleural membranes are not diseased.[24] Systemic factors cause the fluid accumulation in transudative pleural effusion. Over 1 million cases of pleural effusion occur each year, half of which are caused by heart failure.[23] In heart failure, an increase in pulmonary venous pressure contributes to the formation of pleural effusions. Treatment focuses on reducing afterload and improving cardiac output with diuretics and/or inotropes.[24] Another cause of transudative pleural effusions is atelectasis, which may cause pleural fluid to accumulate because of a decrease in pleural pressure. The fluid continues to accumulate until the pleural–parietal pleural interstitial pressure gradient returns to normal.[25] Other causes of transudative pleural effusions include cirrhosis, nephrotic syndrome, malignancy, and peritoneal dialysis.

Exudates

The most common pleural effusion is exudative. Exudative pleural effusions result from local factors, such as pleural inflammation, increased pleural membrane permeability, or lymphatic obstruction.[26] Exudative pleural effusions satisfy any one of the following criteria, known as Light's criteria:[24,27]

- Pleural fluid-to-serum protein ratio greater than 0.5
- Pleural fluid-to-serum LDH ratio greater than 0.6
- Pleural fluid LDH that is two thirds of the upper normal limit for serum LDH

Four million Americans are affected by bacterial pneumonia, 20% of which require hospitalization. Forty percent of these hospitalized patients develop effusions.[26] Malignancies are the second most common cause of exudative pleural effusions. If a massive effusion opacifies, an entire hemithorax, metastatic disease, or chylothorax should be suspected. Other causes of exudative pleural effusion are tuberculosis, trauma, pancreatitis, mesotheliomas, and esophageal perforation.[24]

A hemothorax is a bloody exudative pleural effusion and is diagnosed by a pleural fluid/blood hematocrit ratio greater than 50%.[28] Trauma is the most common cause of a hemothorax (see Chapter 55). Other causes are invasive procedures (placement of central venous catheter, thoracentesis) and anticoagulation therapy. Empyema refers to gross pus in the pleural cavity and requires drainage with a chest tube or surgery. Chylothorax refers to the presence of chyle or a fatty substance in the pleural space.[28] The most common causes of chylothorax are malignancies, surgery, and trauma.[28]

Assessment

History and Physical Findings

Subjective findings include shortness of breath and pleuritic chest pain, depending on the amount of fluid accumulation. Objective findings include tachypnea and hypoxemia if ventilation is impaired, dullness to percussion, and decreased breath sounds over the involved area.

Diagnostic Studies

Diagnosis can be made by chest radiograph, ultrasound, or a CT scan; however, a lateral decubitus chest radiograph is the best demonstration of free pleural fluid. As little as 20 mL of pleural fluid can be seen in the decubitus position.[23] When a pleural effusion is suspected on the basis of physical examination and is confirmed radiologically, obtaining a sample of pleural fluid is necessary for diagnosis by aspiration of fluid from the pleural space. The laboratory tests performed on the pleural fluid obtained by thoracentesis are listed in Table 26-4. Evaluation of the pleural fluid is necessary to distinguish transudative from exudative effusions. When the distance between the pleural fluid line to the inside of the chest wall on lateral decubitus view is less than 1 cm, the pleural effusion is difficult to obtain by thoracentesis and not likely to be clinically significant.[23] In addition, the associated risk for pneumothorax outweighs the benefit of the thoracentesis.[29]

Management

Treatment of the underlying cause of the pleural effusion is necessary. Removal of the pleural effusion by thoracentesis, chest tube placement, or surgery may be indicated depending on the etiology and size of effusion. The primary indication for therapeutic thoracentesis is relief of dyspnea.

▲ Pneumothorax

A pneumothorax occurs when air enters the pleural space between the visceral and parietal pleurae, producing partial or complete lung collapse.

Pathophysiology

During spontaneous breathing, two opposing forces generate negative pleural pressures. Pressure in the airways is positive during expiration and negative during inspiration.[29] However, pleural pressure remains subatmospheric on both inspiration and expiration.[28] Therefore, airway

Table 26-4 Assessment of Pleural Fluid

Test	Comment
Red blood cell count < 100,000/mm^3	Trauma, malignancy, pulmonary embolism
Hematocrit >50% of peripheral blood	Hemothorax
White blood cell count (WBC)	
>50,000–100,000/mm^3	Grossly visible pus, otherwise total WBC less useful than WBC differential
>50% neutrophils	Acute inflammation or infection
>50% lymphocytes	Tuberculosis, malignancy
>10% Eosinophils	Most common: hemothorax, pneumothorax; also benign
>5 % Mesothelial cells	Asbestos effusions, drug reaction, paragonimiasis; tuberculosis *less likely*
Glucose < 60 mg/dL	Infection, malignancy, tuberculosis, rheumatoid, hemothorax, paragonimiasis, Churg-Strauss syndrome
Amylase >200 units/dL	Pleuritis, esophageal perforation, pancreatic disease, malignancy, ruptured ectopic pregnancy Isoenzyme profile: salivary–esophageal disease, malignancy (especially lung)
pH < 7.0	Complicated parapneumonic effusion
pH < 7.2	Systemic acidosis, esophageal rupture, rheumatoid pleuritis, tuberculous pleuritis, malignant pleural disease, hemothorax, paragonimiasis, or Churg-Strauss syndrome.
Triglyceride >110 mg/dL	Chylothorax
Microbiological studies	Etiology of infection
Cytology	Diagnostic of malignancy (adenocarcinoma, benign or malignant mesothelial cells)

Adapted from Light RW: Physiology of pleural fluid production. In Shield TW, LoCicero J, Reed CE, et al (eds): General Thoracic Surgery, 7th Ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, pp 763–770.

pressure remains higher than pleural pressure throughout the respiratory cycle. Sudden communication of the pleural space with either alveolar or external air allows gas to enter (Fig. 26-3). When the pleural pressure rises, the elasticity of the lung causes it to collapse. The lung continues to collapse until either the pressure gradient no longer exists or the pleural defect closes.[28] The main consequence of lung collapse is both decrease in vital capacity and arterial PO_2. In addition, patients with low arterial PO_2 also have an increase in the alveolar–arterial partial pressure of oxygen (PAO_2–PaO_2) gradient, a decreased ventilation–perfusion ratio, and an intrapulmonary shunt resulting in hypoxemia.[28]

There are two types of pneumothorax: spontaneous and traumatic. Spontaneous pneumothorax is any pneumothorax that develops without any trauma. There are two types of spontaneous pneumothorax: primary pneumothorax, which occurs in the absence of underlying lung disease, and secondary, which occurs as a result of underlying lung disease, such as COPD.[28] Primary spontaneous pneumothorax occurs primarily in young (early 20s to 30 years of age, rarely after age 40), tall, and thin men.[28] Family history (Birt-Hogg-Dube syndrome—an autosomal dominant disease) and cigarette smoking are important risk factors.[30,31] Secondary spontaneous pneumothorax most commonly occurs in patients who have COPD, *Pneumocystis carinii* pneumonia, cystic fibrosis, and tuberculosis.[32] Other less common causes include asthma, Marfan syndrome, lung cancer, necrotizing pneumonia, rheumatoid arthritis, sarcoidosis, lymphangioleiomyomatosis, and histiocytosis X.[28,33]

The most common causes of a traumatic pneumothorax in critically ill patients are invasive procedures and barotrauma.[28,34] (See Chapter 55 for a discussion of blunt and penetrating trauma as causes of pneumothorax.) Accidental entry of air into the pleural space during an invasive

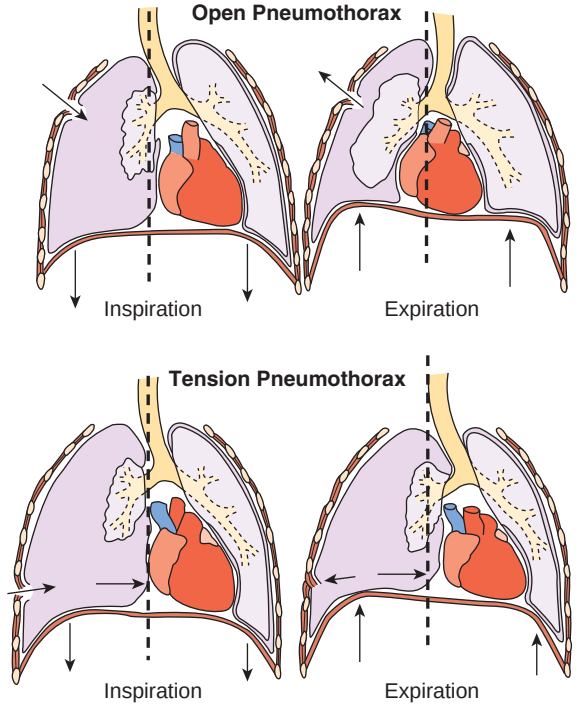


FIGURE 26-3 ▲ Open or communicating pneumothorax (**top**) and tension pneumothorax (**bottom**). In an open pneumothorax, air enters the chest during inspiration and exits during expiration. There may be slight inflation of the affected lung due to a decrease in pressure as air moves out of the chest. In tension pneumothorax, air can enter but not leave the chest. As the pressure in the chest increases, the heart and great vessels are compressed, and the mediastinal structures are shifted toward the opposite side of the chest. The trachea is pushed from its normal midline position toward the opposite side of the chest, and the unaffected lung is compressed. (From Porth C: Essentials of Pathophysiology, 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2011, p 571.)

procedure causes iatrogenic pneumothorax. Central line catheter insertions cause approximately 36,000 pneumothoraces per year, which affect 1% to 3.1% of the patients receiving central line catheters.[28,34] Pulmonary barotrauma occurs in approximately 1% to 15% of patients receiving positive-pressure mechanical ventilation and in 25% to 78% of patients with ARDS.[28] Barotrauma includes parenchymal interstitial gas, pneumomediastinum, subcutaneous emphysema, pneumoperitoneum, and pneumothorax.[28] Pulmonary interstitial gas or emphysema is the initial radiographic indication of barotrauma. The mechanically ventilated patient is at risk for development of a tension pneumothorax. A tension pneumothorax occurs when the pressure of air in the pleural space exceeds atmospheric pressure. As pressures in the thorax increase, the mediastinum shifts to the contralateral side, placing torsion on the inferior vena cava and decreasing venous return to the right side of the heart[28] (see Fig. 26-3).

Assessment

History and Physical Findings

The patient complains of sudden onset of acute pleuritic chest pain localized to the affected lung. The pleuritic chest pain is usually accompanied by shortness of breath, increased work of breathing, and dyspnea. Chest wall movement may be uneven because the affected side does not expand as much of the healthy side. Breath sounds are distant or absent. Chest percussion produces a hyperresonant sound. Tachycardia occurs frequently in all types of pneumothorax. Tension pneumothorax is a life-threatening condition manifested by respiratory distress (Box 26-5).

Diagnostic Studies

To detect a pneumothorax, a chest radiograph should be obtained with the patient in the upright or decubitus position. The chest film shows contralateral (opposite side) mediastinal shift, ipsilateral (same side) diaphragmatic depression, and ipsilateral chest wall expansion in the patient with tension pneumothorax.[29] To confirm the size of the pneumothorax, a chest CT maybe obtained.[29,35] When clinical symptoms of tension pneumothorax are present in a patient on mechanical ventilation, treatment should not be delayed to obtain radiographic confirmation. Arterial blood gas (ABG) findings are used to assess for hypoxemia and hypercapnia.

BOX 26-5 PATIENT SAFETY

Signs and Symptoms of Tension Pneumothorax

- Hypoxemia (early sign)
- Apprehension
- Respiratory distress (severe tachypnea)
- Increasing peak and mean airway pressures, decreasing compliance, and auto–positive end-expiratory pressure (auto-PEEP) in patients receiving mechanical ventilation
- Cardiovascular collapse (heart rate >140 beats/min with any of the following: peripheral cyanosis, hypotension, pulseless electrical activity)

Management

Supplemental oxygen should be administered to all patients with pneumothorax because oxygen accelerates the rate of air resorption from the pleural space.[28,35] If the pneumothorax is 15% to 20%, no medical intervention is required, and the patient is placed on bed rest or limited activity.[35] If the pneumothorax is greater than 20%, then a chest tube is placed in the apical and anterior aspect of the pleural space to assist air removal. Connecting the chest tube to underwater seal drainage alone is usually adequate to resolve the pneumothorax. Initially placing the chest tube to suction risks reexpansion pulmonary edema resulting from rapid reinflation of the collapsed lung.[28] If the pneumothorax persists after 12 to 24 hours of underwater seal drainage, 15 to 20 cm H_2O suction should be applied to facilitate closure.[35] In approximately one third of patients with COPD, persistent air leaks require multiple chest tubes to evacuate the pneumothorax.[35]

A tension pneumothorax is a life-threatening condition requiring immediate treatment; if untreated, it leads to cardiovascular collapse. If a chest tube is not immediately available, a large-bore (16- or 18-gauge) needle should be placed into the anterior second intercostal space. After needle insertion, a chest tube is placed and connected to underwater seal drainage. When the tension pneumothorax is relieved, the effect is rapid and occurs as an improvement in oxygenation, a decrease in heart rate, and an increase in blood pressure.

▲ Pulmonary Embolism

Most incidents of pulmonary embolism occur when a thrombus, which has broken loose and migrated to the pulmonary arteries, obstructs part of the pulmonary vascular tree (Fig. 26-4).[36] A pulmonary embolism usually results from a deep vein thrombosis (DVT) that has formed in the lower extremities. Other sites of clot formation include the right sight of the heart (as in untreated atrial fibrillation) and other deep vessels of the pelvic region.[37] In North America, venous thromboembolism occurs for the first time in approximately 100 of every 100,000 people.[38] Of these, approximately one third have a symptomatic pulmonary embolus. Major risk factors include age, recent surgery, cancer, and thrombophilia.[37] Risk factors that increase the incidence of venous thromboembolism are listed in Box 26-6. Nonthrombotic causes of pulmonary embolism include fat, air, and amniotic fluid but are much less common than thromboembolism.[37]

Pathophysiology

A cascade of events begins the process of thromboembolus development. Virchow's triad (venous stasis, hypercoagulability, vein wall damage) has long been acknowledged in the pathophysiology of venous thromboembolism. Venous return from the lower extremities is usually facilitated by contraction of the muscles of the lower extremities during activity. Conditions such as immobility, heart failure, dehydration, and varicose veins contribute to decreased venous return, increased retrograde pressure in the venous system, and stasis of blood with resultant thrombus formation.[39]

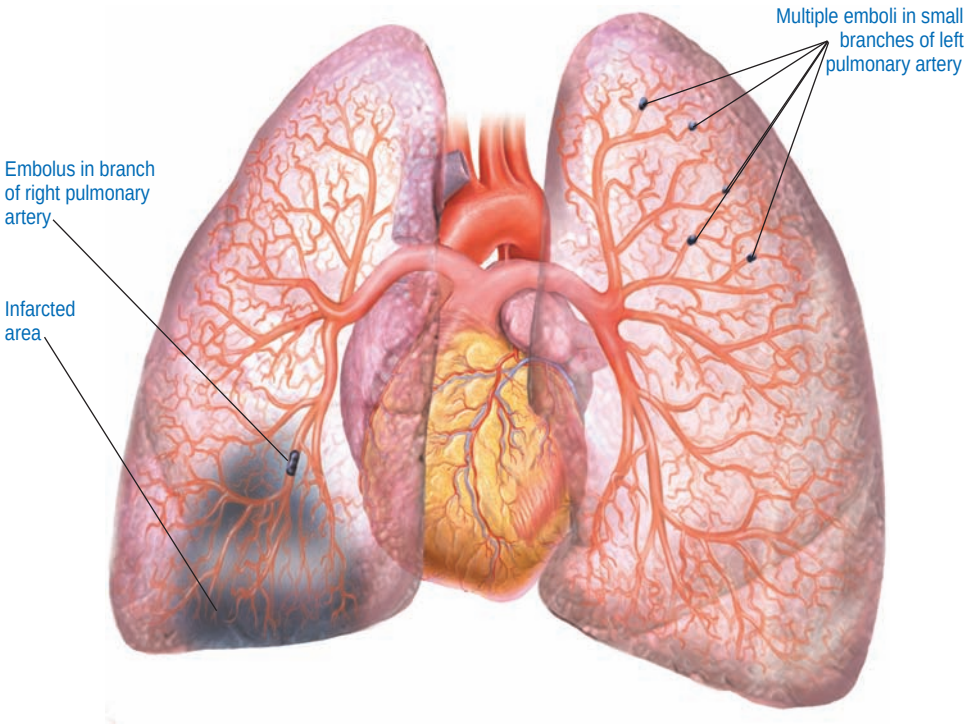


FIGURE 26-4 ▲ Sites of pulmonary emboli. (From Anatomical Chart Company: Atlas of Pathophysiology. Springhouse, PA: Lippincott Williams & Wilkins, 2010, p 107.)

Normally, there is a balance between activation of clotting factors and the fibrinolytic system that prevents thrombus formation. Hypercoagulability may occur in the presence of trauma, surgery, malignancy, pregnancy, or use of oral contraceptives. In these instances, coagulation may be initiated by contact of factor XII with collagen on exposed subendothelium of damaged vessels, by exposure of blood to tissue factor available as a result of vascular wall damage, by activated monocytes migrating to areas of vascular injury, and by release of activated platelets in response to injury. Factor X may be activated by substances from malignant cells and by substances released from hypoxic endothelial cells.[39]

BOX 26-6 Risk Factors for Thromboembolism

For Venous Thromboembolism

Strong Risk Factors
Fracture of the hip, pelvis, or leg
Hip or knee replacement
Major general surgery, major trauma
Spinal cord injury

Moderate Risk Factors
Arthroscopic knee surgery
Central venous lines
Malignancy
Heart or respiratory failure
Hormone replacement therapy, oral contraceptives
Paralytic stroke
Postpartum period
Previous venous thromboembolism
Thrombophilia

Weak Risk Factors
Bed rest for more than 3 days
Immobility due to sitting
Increasing age
Laparoscopic surgery
Obesity
Antepartum period
Varicose veins

Risk Stratification of Thromboemboli for Patients Undergoing Surgery

Low Risk
Uncomplicated surgery in patients aged less than 40 years with minimal immobility postoperatively and no risk factors

Moderate Risk
Any surgery in patients aged 40 to 60 years
Major surgery in patients aged less than 40 years and no other risk factors
Minor surgery in patients with one or more risk factors

High Risk
Major surgery in patients aged more than 60 years
Major surgery in patients aged 40 to 60 years with one or more risk factors

Very High Risk
Major surgery in patients aged more than 40 years with previous venous thromboembolism, cancer, or known hypercoagulable state
Major orthopedic surgery
Elective neurosurgery
Multiple trauma or acute spinal cord injury

From Blann AD, Lip GYH: Venous thromboembolism. BMJ 332(7535):215–219, 2006.

Damage to the vessel wall causes adhesion and aggregation of platelets and contributes to the activation of clotting factors.

Thrombus formation is frequently bilateral and often asymptomatic. Although most thrombi form in the calf, most pulmonary emboli (80% to 90%) arise from venous thrombi that extend into the proximal veins (popliteal and iliofemoral) of the lower extremities.[38] Proximal venous thrombi pose an approximately 50% risk for embolism.[38]

Occlusion of a pulmonary artery by an embolus produces both pulmonary and hemodynamic changes. Alveoli are ventilated but not perfused, producing areas of mismatched ventilation and perfusion. As a result, well-ventilated alveoli are underperfused, and gas exchange is compromised (increased respiratory dead space). Pulmonary vascular constriction resulting from a lack of carbon dioxide, which is normally present in pulmonary arterial blood, shifts ventilation from the underperfused alveoli. Accompanying physiological changes include increased minute ventilation, decreased vital capacity, increased airway resistance, and decreased diffusing capacity.[37]

The severity of hemodynamic change in pulmonary embolism depends on the size of the embolus and degree of pulmonary vascular obstruction as well as on the preexisting status of the cardiopulmonary system. In patients with no previous cardiopulmonary disease, there is a relationship between the degree of pulmonary artery obstruction and the pulmonary artery pressure. Increased right ventricular afterload results from obstruction of the pulmonary vascular bed by embolism. In patients with no preexisting cardiopulmonary disease, obstruction of less than 20% of the pulmonary vascular bed produces compensatory events that minimize adverse hemodynamic consequences.[37] Cardiac output is maintained by increases in both right ventricular stroke volume and heart rate, and recruitment and distention of pulmonary vessels occur, producing normal or near-normal pulmonary artery pressure and pulmonary vascular resistance.[37] When the degree of pulmonary vascular obstruction exceeds 30% to 40%, increases in pulmonary artery pressure occur, followed by modest increases in right atrial pressure.[37] As the degree of pulmonary artery obstruction exceeds 50% to 60%, compensatory mechanisms are overcome, producing a decrease in cardiac output and dramatic increases in right atrial pressure.[37] Patients with preexisting cardiopulmonary disease have degrees of pulmonary hypertension that are disproportionate to the degree of embolic obstruction.[37] Severe pulmonary hypertension may develop from a relatively small reduction of pulmonary blood flow.

Assessment

Pulmonary embolism is difficult to diagnose because of its nonspecific signs and symptoms. Pulmonary embolism should be suspected with patient with new worsening dyspnea or sustained hypotension without other explanation. However, it is also common that a DVT or embolus produces no significant symptoms and may be an incidental finding when the patient undergoes imaging for other reasons.[37]

Clinical evaluation may indicate a need for further studies but is not reliable for confirmation or exclusion of the diagnosis of pulmonary embolism.[37] A diagnostic algorithm for the evaluation of patients with suspected pulmonary embolism is presented in Figure 26-5. When a patient has either renal failure or allergy to contrast dye, ventilation–perfusion (VQ) scans are often used instead of CT angiogram of the chest. Transthoracic echocardiogram is often used to confirm diagnosis of a pulmonary embolism in the main pulmonary artery. Signs and symptoms of pulmonary embolism are listed in Box 26-7. Dyspnea is the most frequent symptom in patients with angiographically confirmed pulmonary embolism. Other signs and symptoms (in order of frequency) are pleuritic chest pain, cough, apprehension, leg swelling, and pain.[36]

Management

Heparin and thrombolytic agents are used to treat pulmonary embolism. Guidelines developed by the American College of Chest Physicians (ACCP) for the treatment of venous thromboembolism are shown in Table 26-5.[40]

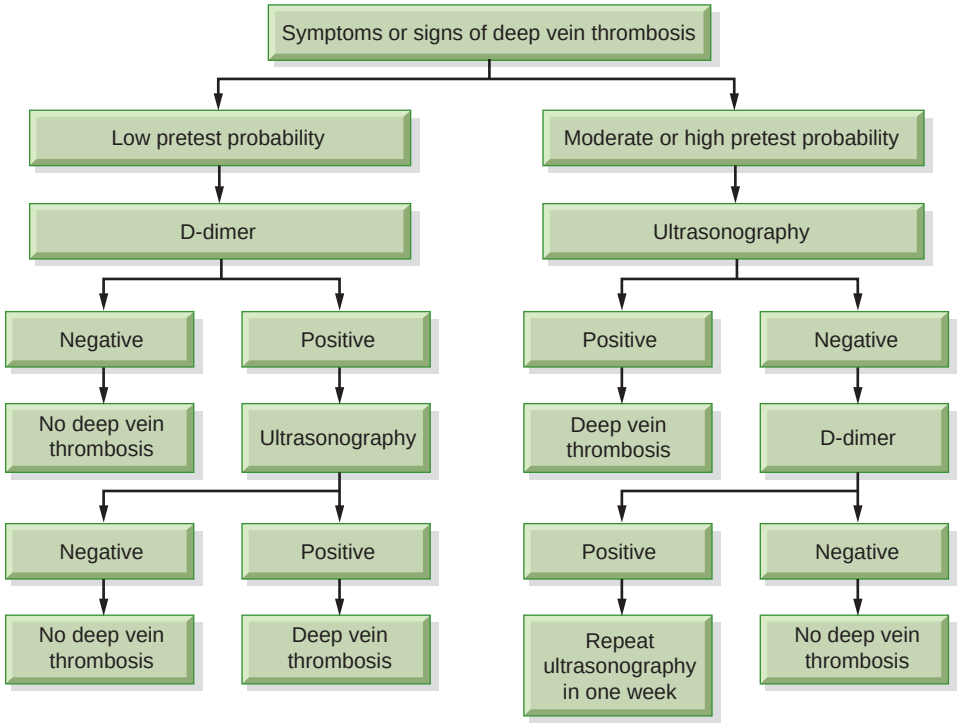
Patients with DVT or pulmonary embolism should be treated with unfractionated intravenous heparin or adjusted-dose subcutaneous heparin. Though adjusted-dose subcutaneous heparin is an option for treatment of PE, it is not commonly used due to the decreased bioavailability of heparin when given subcutaneously.

Low-molecular-weight heparin (LMWH) can be substituted for unfractionated heparin in patients with DVT and in stable patients with pulmonary embolism. Treatment with heparin or LMWH should continue for at least 5 days, overlapped with oral anticoagulation for at least 4 to 5 days (grade A1).[40]

The recommended length of anticoagulation therapy varies, depending on the patient's age, comorbidities, and the likelihood of recurrence of pulmonary embolism or DVT. In most patients, anticoagulation therapy with warfarin should be continued for 3 to 6 months.[40] The first episode of idiopathic DVT should be treated for at least 6 months.[40] Patients with new-onset DVT and a risk factor (eg, cancer, inhibitor deficiency state) or recurrent venous thrombosis should be treated indefinitely.[40] Patients with massive pulmonary embolism or severe iliofemoral thrombosis may require a longer period of heparin therapy.[40] Anticoagulation using full-dose subcutaneous heparin for several months is effective in patients who have contraindications to warfarin (eg, pregnant women) but who can safely take heparin.[40]

Thrombolytic therapy is only recommended for patients with acute massive pulmonary embolism who are hemodynamically unstable and not prone to bleeding.[40] All thrombolytic agents act systemically and have the potential to lyse a fresh platelet–fibrin clot anywhere and cause bleeding at that site.[40] Intracranial disease, recent surgery, trauma, and hemorrhagic disease are contraindications to thrombolytic therapy. Urokinase, streptokinase, and recombinant tissue plasminogen activator are the thrombolytic agents approved for treating pulmonary embolism and venous thromboembolism. Heparin therapy is not administered concurrently with thrombolytics; however, thrombolytic therapy is followed by administration of heparin, then warfarin.

An inferior vena cava filter is recommended to prevent pulmonary embolism in patients with contraindications to heparin therapy (risk for major bleeding or drug sensitivity).[40] Placement of an inferior vena cava filter is also recommended in patients with recurring thromboembolism despite adequate anticoagulation, chronic recurrent embolism and pulmonary hypertension, and concurrent surgical pulmonary embolectomy or pulmonary endarterectomy procedures.



For assessment of pretest probability of suspected deep vein thrombosis:

- Score 1 point each for following: tenderness along entire deep vein system; swelling of entire leg, >3 cm difference in calf circumference; pitting edema; collateral superficial veins; risk factors present (active cancer, prolonged immobility or paralysis, recent surgery, or major medical illness)
- Subtract 2 points for alternative diagnosis likely (for example, ruptured Baker's cyst in rheumatoid arthritis, superficial thrombophlebitis, or infective cellulitis)

Result >3 = high probability; 1-2 = moderate probability; ≤0 = low probability

FIGURE 26-5 ▲ Diagnostic algorithm for evaluation of patients with symptoms suggestive of acute pulmonary embolism. (From Blann AD, Lip GYH: Venous thromboembolism. BMJ 332(7535):215–219, 2006.)

BOX 26-7 PATIENT SAFETY

Signs and Symptoms of Pulmonary Embolism

Small to Moderate Embolus

- Dyspnea
- Tachypnea
- Tachycardia
- Chest pain
- Mild fever
- Hypoxemia
- Apprehension
- Cough
- Diaphoresis
- Decreased breath sounds over affected area
- Rales
- Wheezing

Massive Embolus

A more pronounced manifestation of the above signs and symptoms, plus the following:

- Cyanosis
- Restlessness
- Anxiety
- Confusion
- Hypotension
- Cool, clammy skin
- Decreased urinary output
- Pleuritic chest pain: associated with pulmonary infarction
- Hemoptysis: associated with pulmonary infarction

Signs of Pulmonary Embolism in Intensive Care Patients

- Worsening hypoxemia or hypocapnia in a patient on spontaneous ventilation
- Worsening hypoxemia and hypercapnia in a sedate patient on controlled mechanical ventilation
- Worsening dyspnea, hypoxemia, and a reduction in $PaCO_2$ in a patient with chronic lung disease and known carbon dioxide retention
- Unexplained fever
- Sudden elevation in pulmonary artery pressure or central venous pressure in a hemodynamically monitored patient

Table 26-5 American College of Chest Physicians Recommendations for Treatment of Venous Thromboembolism

Anticoagulation Guidelines for	Recommended Therapy
Unfractionated Heparin (UFH)	
Suspected VTE	• Obtain baseline aPTT, PT, CBC. • Check for contraindications to heparin therapy. • Give heparin 5,000 units IV. • Order imaging study.
Confirmed VTE	• Re-bolus with heparin 80 units/kg IV or 5,000 units, and start maintenance infusion at 18 units/kg/h or 1,300 units/h. • Check aPTT at 6 h; maintain a range corresponding to a therapeutic heparin level. • Start warfarin therapy on day 1 at 5 mg; adjust subsequent daily dose according to INR. • Stop heparin after 4–5 d of combined therapy, when INR is >2.0 (2.0–3.0) for 24 h. • Anticoagulate with warfarin for at least 3 mo (target INR 2.5; 2.0–3.0).
Low-Molecular-Weight Heparin (LMWH)	
Suspected VTE	• Obtain baseline aPTT, PT, CBC. • Check for contraindication to heparin therapy. • Give UFH, 5,000 units IV. • Order imaging study.
Confirmed VTE	• Give LMWH (enoxaparin), 1 mg/kg subcutaneously every 12 h. • Start warfarin therapy on day 1 at 5 mg; adjust subsequent daily dose according to the INR. • Consider checking platelet count between days 3 and 5. • Stop LMWH after at least 4–5 d of combined therapy, when INR is >2.0 on 2 consecutive days. • Anticoagulate with warfarin for at least 3 mo (goal INR 2.5; 2.0–3.0), then it is recommended to continue with low intensity therapy (INR range 1.5–1.9) with less frequent monitoring over stopping treatment.

VTE, venous thromboembolism; aPTT, activated partial thromboplastin time; PT, prothrombin time; INR, international normalized ratio; CBC, complete blood count.

From American College of Chest Physicians: Eighth ACCP consensus conference on Antithrombotic and Thrombolytic Therapy. Chest 133:454S–545S, 2008.

Prevention

Prevention of venous thromboembolism is essential to decreasing the morbidity and mortality associated with pulmonary embolism. Prophylactic measures are based on the patient's specific risk factors.[41] See Table 26-6 for preventive measures recommended by the ACCP.

▲ Chronic Obstructive Pulmonary Disease

COPD is a disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases (primarily cigarette smoke) or an inherited deficiency of α_1-antitrypsin (see Spotlight on Genetics 26-1).[42] COPD includes two diseases: emphysema and chronic bronchitis.[42] COPD is a major cause of chronic morbidity and mortality, ranking as the fourth leading cause of death in the United States.[42]

The WHO estimates that COPD shares fourth place as a single cause of death (behind coronary heart disease, cerebrovascular disease, and lower respiratory infection), and unlike some of the other diseases, the death rates from COPD have increased. The WHO estimates that COPD will be the third leading cause of death by 2020 in the burden of disease caused worldwide.[43]

Pathophysiology

In COPD, pathologic changes occur in the central airways, peripheral airways, lung parenchyma, and pulmonary vasculature.[43] As the disease progresses, pathophysiological changes usually occur in the following order: mucous hypersecretion, ciliary dysfunction, airflow limitation, pulmonary hyperinflation, gas exchange abnormalities, pulmonary hypertension, and cor pulmonale.[43] The peripheral airways become the major site of obstruction in patients with COPD. The structural changes in the airway wall are the most important cause of the increase in peripheral airway resistance. Inflammatory changes, such as airway edema and hypersecretion of mucus, also contribute to narrowing of the peripheral airways.[43] Mucal hypersecretion is caused by the stimulation of the enlarged mucus-secreting glands and the increased number of goblet cells by inflammatory mediators such as leukotrienes, interleukins, and tumor necrosis factor.[43] Ciliated epithelial cells undergo squamous metaplasia, leading to impaired mucociliary clearance, which is usually the first physiological abnormality to occur in COPD.[43] This abnormality may be evident for many years before any other abnormalities develop.[43]

Table 26-6 American College of Chest Physicians Recommendations for Prevention of Venous Thromboembolism

Patient Population	Recommended Therapy	Grade*
Low-risk general surgery	Early and frequent ambulation	A1
Moderate-risk general surgery	LMWH, LDUH, or fondaparinux	A1
Higher-risk general surgery	LMWH, LDUH three times a day, or fondaparinux	A1
High-risk general surgery with multiple risk factors	LDUH three times a day, LMWH, fondaparinux combined with graduated compression stockings, and/or intermittent pneumatic compression	C1
Total hip replacement surgery	LMWH, started 12 h before surgery or 12–24 h after surgery, or 4–6 h after surgery at half usual high-risk dose and then increasing to usual high-risk dose of following day or Fondaparinux (2.5 mg started 6–24 h after surgery) or Adjusted-dose warfarin, started preoperatively or the evening of the surgical day (INR target 2.5; INR range, 2.0–3.0)	A1
Total knee replacement surgery	LMWH (at the usual high-risk dose), fondaparinux, warfarin (INR target, 2.5; INR range, 2.0–3.0); Intermittent pneumatic compression	A1 B1
Acute spinal cord injury	LMWH Elastic stockings and intermittent pneumatic compression and either LDUH or LWMH	B1 B1 C1
Trauma patients with identifiable risk factor for thromboembolism	LMWH, as soon as safe or LMWH and Intermittent pneumatic compression. For high-risk patients with suboptimal prophylaxis, mechanical thromboprophylaxis with intermittent pneumatic compression or possibly with graduated compression stocking alone can be used.	A1 B1 B1
Myocardial infarction	UFH or LMWH or bivalirudin or fondaparinux Intermittent pneumatic compression or elastic stockings when heparin is contraindicated	A1 C1
Ischemic stroke and lower extremity paralysis	Low-dose SC heparin or LMWH For patients with contraindication for anticoagulants, intermittent pneumatic compression, or elastic stockings	A1 B1
Medical patients with risk factors for VTE (including heart failure and chest infections)	LDUH, LMWH, or fondaparinux	A1
Patients with long-term indwelling central vein catheters	Recommended that clinicians NOT use either prophylactic doses of LMWH or minidose warfarin to try to prevent catheter-related thrombosis	B1
Patients receiving a spinal puncture or epidural catheter placement	Use appropriate patient selection and caution when using anticoagulant thromboprophylaxis	A1

*A1: Methods strong, results consistent—randomized clinical trials (RCTs), no heterogeneity, effect clear that benefits do (or do not) outweigh risks. A2: Methods strong, results consistent—RCTs, no heterogeneity, effect equivocal—uncertain whether benefits outweigh risks. B1: Methods strong, results inconsistent—RCTs, heterogeneity present, effect clear that benefits do (or do not) outweigh risks. B2: Methods strong, results inconsistent—RCTs, heterogeneity present, effect equivocal—uncertain whether benefits outweigh risks. C1: Methods weak—observational studies, effect clear that benefits do (or do not) outweigh risks. C2: Methods weak—observational studies, effect equivocal—uncertain whether benefits outweigh risks.

LDUH, low-dose unfractionated heparin; LMWH, low-molecular-weight heparin.

From American College of Chest Physicians: Eighth ACCP Conference on Antithrombotic and Thrombolytic Therapy. Prevention of venous thromboembolism. Chest 133(6 Suppl):381S–453S, 2008.

SPOTLIGHT ON GENETICS 26-1

ALPHA-1 ANTITRYPSIN DEFICIENCY

- This disorder affects about 1 in 1,500 to 3,500 individuals with European ancestry. It is uncommon in people of Asian descent but is commonly observed in COPD patients.
- Mutations in the *SERPINA1* gene cause alpha-1 antitrypsin deficiency. This gene provides instructions for making a protein called alpha-1 antitrypsin, which protects the body from a powerful enzyme called neutrophil elastase.
- Leads to a deficiency of alpha-1 antitrypsin or an abnormal form of the protein that cannot control neutrophil elastase. Without enough functional alpha-1 antitrypsin, neutrophil elastase destroys alveoli and causes lung disease.
- There are numerous genetic tests available for the detection of alpha-1 antitrypsin deficiency.

Genetic Home Reference—Available at http://ghr.nlm.nih.gov. Accessed July 14, 2011.

Janciauskiene SM, Bals R, Koczulla R, et al: The discovery of α1-antitrypsin and its role in health and disease. Respir Med 105(8):1129–1139, 2011.

Expiratory airflow limitation is an essential finding in COPD. As the disease process progresses, forced expiratory volume in 1 second (FEV_1) and forced vital capacity (FVC) decrease; this is related to the increased thickness of the airway wall, loss of alveolar attachments, and loss of lung elastic recoil. Frequently, the first sign of developing airflow limitation is a decrease in the FEV_1/FVC ratio.[43] According to the 2009 Global Initiative for Chronic Obstructive Lung Disease (GOLD), the presence of a postbronchodilator FEV_1 less than 80% of the predicted value, in combination with an FEV_1/FVC ratio less than 70%, confirms the presence of airflow limitation that is not fully reversible (Table 26-7).[43] In severe COPD, air is trapped in the lungs during forced expiration, leading to an abnormally high functional residual capacity (FRC). Increasing FRC leads to pulmonary hyperinflation.[44]

In advanced COPD, peripheral airway obstruction, parenchymal destruction, and pulmonary vascular irregularities reduce the lung's capacity for gas exchange, resulting in hypoxemia (low blood oxygen) and hypercapnia (high blood carbon dioxide).[43] A VQ ratio mismatch is the driving force behind hypoxemia in patients with COPD, regardless of the stage of the disease. Chronic hypercapnia usually indicates inspiratory muscle dysfunction and alveolar hypoventilation.[43] As hypoxemia and hypercapnia progress late in COPD, pulmonary hypertension often develops, which causes hypertrophy of the right ventricle, better known as cor pulmonale.[43] Right-sided heart failure leads to further venous stasis and thrombosis that may potentially result in pulmonary embolism and further compromise the pulmonary circulation. Last, COPD is associated with systemic inflammation and skeletal muscle dysfunction that may result in limitation of exercise capacity and decline of health status.[43]

Table 26-7 Spirometric Classification of COPD Severity Based on Postbronchodilator FEV_1 and the Recommended Treatment

Stage	Characteristics	Recommended Treatment
I: Mild COPD	• FEV_1/FVC < 70% • FEV_1 80% or more predicted • With or without symptoms	• Active reduction of risk factors: influenza vaccination • Short-acting bronchodilator when needed
II: Moderate COPD	• FEV_1/FVC < 70% • 50% or less FEV_1 > 80% predicted • With or without symptoms	• Active reduction of risk factors: influenza vaccination • Short-acting bronchodilator when needed • Add regular treatment with one or more long-acting bronchodilators • Add rehabilitation
III: Severe COPD	• FEV_1/FVC < 70% • 30% FEV_1or less more than 50% predicted	• Active reduction of risk factors: influenza vaccination • Short-acting bronchodilator when needed • Regular treatment with one or more long-acting bronchodilators • Rehabilitation • Inhaled glucocorticosteroids if significant symptoms and lung function response or if repeated exacerbations
Stage IV: Very Severe	• FEV_1 /FVC < 70% • FEV_1 < 30% predicted or • FEV_1 < 50% predicted plus chronic respiratory failure	• Active reduction of risk factors: influenza vaccination • Short-acting bronchodilator when needed • Regular treatment with one or more long-acting bronchodilators • Rehabilitation • Inhaled glucocorticosteroids if significant symptoms and lung function response or if repeated exacerbations • Add long-term oxygen therapy if respiratory failure • Consider surgical treatments

Patients must be taught how and when to use their treatment, and treatments being prescribed for other conditions should be reviewed. Beta-blocking agents (including eye-drop formulations) should be avoided.

FEV_1, forced expiratory volume in 1 s; FVC, forced vital capacity.

From Rodriguez-Roisin R, et al: Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease (Updated 2009): National Heart, Lung, and Blood Institute and World Health Organization global initiative for chronic obstructive lung disease (GOLD). Available at http://www.goldcopd.com/download.asp?intId=556.

Assessment

History

A detailed medical history of a new patient with known or suspected COPD should include the following:

- Exposure to risk factors, such as smoking, and occupational or environmental exposures to pollutants
- Past medical history, including asthma, allergy, sinusitis or nasal polyps, respiratory infections in childhood, and other respiratory diseases
- Family history of COPD or other chronic respiratory disease
- Pattern of symptom development. COPD typically develops in adults, and most patients are aware of the occurrence of increased breathlessness, increased frequency of winter "colds," and some social restriction for a number of years before seeking medical attention.
- History of exacerbations or previous hospitalizations for respiratory disorder. Patients may be conscious of periodic worsening of symptoms even if these episodes have not been identified as acute exacerbations of COPD. Indications for hospital assessment or admission for acute exacerbation of COPD are listed in Box 26-8.
- Comorbidities such as heart disease and rheumatic disease, which may also contribute to restriction of activity
- Appropriateness of current medical treatments, such as beta blockers, commonly prescribed for heart disease. Beta blockers are usually contraindicated in COPD.
- Impact of disease on patient's life, including limitation of activity, missed work and economic consequences, effect on family routines, or feelings of depression or anxiety
- Social and family support
- Possibilities for reducing risk factors, especially smoking cessation[42,43]

Physical Findings

Two patterns of disease are evident in advanced COPD (Table 26-8). These patterns may become increasingly apparent as the disease progresses.[43] A physical examination is rarely diagnostic in COPD, although it remains an important aspect of patient care.[43] The physical examination should include inspection, palpation, percussion, and auscultation.

Table 26-8 Patterns of Disease in Advanced Chronic Obstructive Pulmonary Disease

Feature	Type A	Type B
Commonly used name	Pink puffer	Blue bloater
Disease association	Predominant emphysema	Predominant bronchitis
Major symptom	Dyspnea	Cough and sputum
Appearance	Thin wasted, not cyanotic	Obese, cyanotic
PO_2	↓	↓↓
PCO_2	Normal or ↓	Normal or ↑
Elastic recoil of lung	↓	Normal
Diffusing capacity	↓	Normal
Hematocrit	Normal	Often ↑
Cor pulmonale	Infrequent	Common

Adapted from Toung D: COPD: General Anesthesia. In Modak RJ, et al (eds): Anesthesiology Keyword Review, 1st ed. Philadelphia, PA: Lippincott William & Wilkins, 2008, p 115.

BOX 26-8 Indications for Hospital Assessment or Admission for Acute Exacerbation of Chronic Obstructive Pulmonary Disease*

Marked increase in intensity of symptoms, such as sudden development of resting dyspnea
Severe background COPD
Onset of new physical signs (eg, cyanosis, peripheral edema)
Failure of exacerbation to respond to initial medical management
Significant comorbidities
Frequent exacerbations
Newly occurring dysrhythmias
Diagnostic uncertainty
Older age
Insufficient home support

*Local resources need to be considered.

From RR Roisin, et al: Global Initiative for Chronic Obstructive Lung Disease. Executive Summary: Global strategy for the diagnosis, management and prevention of COPD: updated 2009. Available at http://www.goldcopd.com/Guidelineitem.asp?l1=2&l2=1&intId=2180. Accessed October 2010.

Inspection

- Central cyanosis or bluish discoloration of the mucosal membranes. This feature may be present but is difficult to detect in artificial light and in many racial groups.
- Common chest wall abnormalities, which reflect the pulmonary hyperinflation seen in COPD, including relatively horizontal ribs, barrel-shaped chest, and protruding abdomen
- Flattening of the hemidiaphragms, which may be associated with paradoxical indrawing of the lower rib cage on inspiration, reduced cardiac dullness, and widening xiphisternal angle
- Resting respiratory rate, which is often increased to more than 20 breaths/min; breathing may be shallow.
- Pursed-lip breathing, which may serve to slow expiratory flow and permit more efficient lung emptying
- Resting muscle activation, which may be indicative of respiratory distress. While lying supine, patients with COPD often use the scalene and sternocleidomastoid muscles.
- Ankle or lower leg edema, which may be an indication of right-sided heart failure

Palpation and Percussion

- Palpation and percussion, which are often unhelpful in COPD
- Heart apex beat, which may be difficult to detect due to pulmonary hyperinflation
- Pulmonary hyperinflation, which also leads to downward displacement of the liver and an increase in the ability to palpate this organ without it actually being enlarged

BOX 26-9 Indications for ICU Admission for Patients With Acute Exacerbation of Chronic Obstructive Pulmonary Disease*

Severe dyspnea that responds inadequately to initial emergency therapy
Confusion, lethargy, coma
Persistence of worsening hypoxemia (PaO_2 < 6.7 kPa, 50 mm Hg), or severe/worsening hypercapnia ($PaCO_2$ > 9.3 kPa, 70 mm Hg), or severe/worsening respiratory acidosis (pH < 7.30) despite supplemental oxygen and noninvasive positive-pressure ventilation

*Local resources need to be considered.

From Roisin RR, et al: Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease (Updated 2009). Available at http://www.goldcopd.com/download.asp?intId=552.

Auscultation

- Reduced breath sounds. Patients with COPD often have reduced breath sounds.
- Wheezing. Occurrence during quiet breathing is a useful indicator of airflow limitation. However, wheezing heard only after forced expiration is of no diagnostic significance.
- Inspiratory crackles, which occur in some patients with COPD but are of little assistance diagnostically
- Heart sounds, which are best heard over the xiphoid area

Symptoms of COPD include cough, sputum production, and shortness of breath on exertion. Indications for ICU admission for patients with acute exacerbation of COPD are listed in Box 26-9.[42]

Diagnostic Studies

Laboratory and diagnostic tests in COPD are summarized in Table 26-9.

SPIROMETRY Expiratory airflow limitation is the hallmark diagnostic sign of COPD. Because spirometry is the most reproducible and objective measure of airflow limitation, it remains the gold standard for diagnosing COPD and monitoring its progression.[43,44] Spirometry is performed in patients with chronic cough and sputum production even without dyspnea. Spirometry measures the maximal volume of air forcibly exhaled from the point of maximal inspiration (FVC) and the volume of air exhaled during the first second of this exercise (FEV_1). The ratio of these two measurements (FEV_1/FVC ratio) is then calculated.[43,44] Another measurement of significant value from a pulmonary function test is the DLCO, or the diffusion capacity of the lung for carbon monoxide. This value is indicative of the amount of gas exchange that is occurring in the lung. When one takes the data from the FEV_1 and the DLCO, one can determine the true respiratory status of a patient. Spirometry measurements are evaluated by comparison of the results with appropriate reference values based on age, height, sex, and race.

Figure 26-6 demonstrates a normal spirogram and a spirogram characteristic of a patient with COPD with mild to moderate airflow limitation. Patients with COPD have decreased FEV_1 and FVC, and the degree of spirometric abnormality generally reflects the severity of the disease[43,44] (see Table 26-8). By itself, the FEV_1/FVC ratio is the most sensitive measure of airflow limitation, and an FEV_1/FVC ratio less than 70% is considered an early sign of airflow limitation in patients whose FEV_1 remains normal (80% or more of the predicted value).

Table 26.9 Laboratory and Diagnostic Tests for Patients With Chronic Obstructive Pulmonary Disease

Test	Rationale
Spirometry	Measures FVC and FEV_1; gold standard for diagnosing disease and monitoring progression
Bronchodilator reversibility	Performed once during diagnosis stage and useful for the following reasons: • To rule out an asthma diagnosis (If FEV_1 returns to predicted normal range after administration of bronchodilator, airflow limitation is likely due to asthma.) • To establish a patient's best attainable lung function at that point in time
Chest radiography	Bullous disease may be evident Radiological changes seen include: • Flattened diaphragm on lateral chest film • Increased volume of retrosternal air space (signs of hyperinflation) • Hyperlucency of the lungs • Rapid tapering of vascular markings
Computed tomography (CT) and ventilation–perfusion scanning	Assessment of surgical patient to visualize airway and parenchymal disease May assist with differential diagnosis
ABGs	Performed if FEV_1 is <50% predicted or if signs of respiratory failure or right-sided heart failure are present
$Alpha_1$-antitrypsin deficiency screening	Indicated for patients in whom COPD develops at <45 years of age or who have a strong family predisposition ($alpha_1$-antitrypsin serum level below 15%–20% of normal value is highly suggestive of homozygous $alpha_1$-antitrypsin deficiency)

FEV_1, forced expiratory volume in 1 s; FVC, forced vital capacity.

Adapted from Roisin RR, et al: Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease (Updated 2009) Available at http://www.goldcopd.com/download.asp?intId=552.

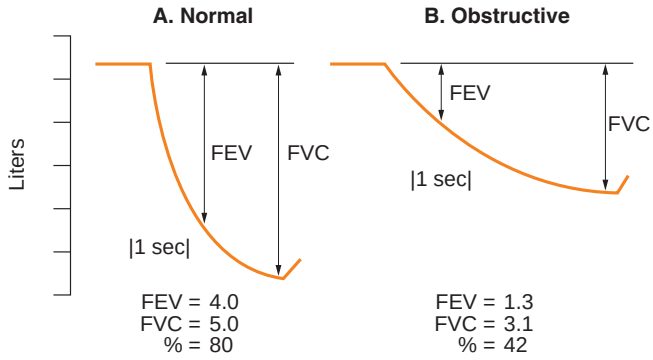


FIGURE 26-6 ▲ Normal, obstructive, and restrictive patterns of a forced expiration. (FVC, forced vital capacity; FEV, forced expiratory volume.) (From West JB: Pulmonary Pathophysiology: The Essentials, 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008.)

ARTERIAL BLOOD GASES. ABG measurements should be performed in all patients in moderate and severe stages of the disease (FEV_1 < 40% predicted) or when clinical signs of respiratory failure or right-sided heart failure are present (ie, central cyanosis, ankle swelling, increase in jugular venous pressure).[42] Respiratory failure is indicated by a partial pressure of arterial oxygen (PaO_2) of 60 mm Hg with or without a partial pressure of arterial carbon dioxide ($PaCO_2$) of 45 mm Hg while breathing air at sea level.[42] Several precautions must be taken to ensure accurate results. First, it should be noted if the patient is currently receiving an oxygen source and the amount of oxygen delivered to the patient during the blood gas sample time. Second, if the fraction of inspired oxygen (FiO_2) has been changed, a period of 20 to 30 minutes should elapse before gas tensions are rechecked.[43]

Management

Several different treatment modalities, ranging from exercise training, nutrition counseling, and education, to drug therapy, oxygen use, and surgery, may be effective in COPD treatment. See Table 26-7 on page 567 for therapeutic guidelines for the various stages of COPD. Box 26-10 provides a collaborative care guide for the patient with COPD.

Nonpharmacological Therapy

The main goals of pulmonary rehabilitation are to decrease symptoms, improve quality of life, and increase physical and emotional participation in day-to-day activities.[43] The 2009

BOX 26–10 *COLLABORATIVE CARE GUIDE For the Patient With Chronic Obstructive Pulmonary Disease*

Outcomes	Interventions
Oxygenation/Ventilation	
Patient has arterial blood gases (ABGs) within normal limits and pulse oximeter value >90%.	• Assess respiratory rate, effort, and breath sounds every 2–4 h. • Obtain ABGs per order or signs of respiratory distress. • Monitor arterial saturation by pulse oximeter. • Provide supplemental oxygen by nasal cannula or face mask using lowest possible FiO_2 and flow rate. • Provide humidification with oxygen. • Provide intubation and mechanical ventilation as necessary (refer to Collaborative Care Guide for the Patient on Mechanical Ventilation, Chapter 25 on pages 538–539).
Patient maintains normal rate and depth of respiration.	• Monitor respiratory rate, pattern, and effort (eg, use of accessory muscles). • Assess respirations during sleep; note sleep apnea or Cheyne-Stokes patterns.
Patient has clear chest x-ray.	• Obtain chest x-ray daily
Patient has clear breath sounds.	• Monitor breath sounds for crackles, wheezes, or rhonchi every 2–4 h. • Administer diuretics per order. • Administer bronchodilators and mucolytics as indicated.
There is no evidence of atelectasis or pneumonia.	• Encourage nonintubated patients to use incentive spirometer, cough, and deep breathe every 2–4 h and PRN. • Assess quantity, color, and consistency of secretions. • Turn side to side every 2 h. • Mobilize out of bed to chair.
Circulation/Perfusion	
Blood pressure, heart rate, and hemodynamic parameters are within normal limits.	• Monitor vital signs every 1–2 h. • Monitor pulmonary artery pressures and right atrial pressure every 1 h and cardiac output, systemic venous resistance, and peripheral venous resistance every 6–12 h if pulmonary artery catheter is in place. • Assess for signs of right ventricular dysfunction (eg, increased central venous pressure, neck vein distention, peripheral edema).
Patient is free of dysrhythmias.	• Maintain patent intravenous access. • Monitor for atrial dysrhythmias due to right atrial dilation and ventricular dysrhythmias due to hypoxemia and hypoxia.
Serum lactate will be within normal limits.	• Monitor lactate daily until it is within normal limits. • Administer red blood cells, positive inotropic agents, colloid infusion as ordered to increase oxygen delivery.

(*continued on page 571*)

BOX 26–10 *COLLABORATIVE CARE GUIDE For the Patient With Chronic Obstructive Pulmonary Disease (continued)*

Outcomes	Interventions
Fluids/Electrolytes	
Renal function is maintained as evidenced by urine output more than 30 mL/h, normal laboratory values.	• Monitor intake and output every 1–2 h. • Monitor blood urea nitrogen, creatinine, electrolytes, Mg, PO_4. • Replace potassium, magnesium, and phosphorus per order or protocol. • Weigh daily.
Patient is euvolemic.	• Administer fluid volume and diuretics based on vital signs, physical assessment, secretion viscosity as ordered.
Mobility/Safety	
There is no evidence of loss of muscle tone or strength.	• Promote standing at bedside, sitting up in chair, ambulating with assistance as soon as possible. • Establish activity program. • Monitor response to activity.
Patient maintains joint flexibility.	• Consult with physical therapist. • Use passive and active range of motion every 4 h while awake.
There is no evidence of infection. WBC counts are within normal limits.	• Monitor systemic inflammatory response syndrome criteria: increased WBC count, increased temperature, tachypnea, tachycardia. • Use strict aseptic technique during procedures and monitor others. • Maintain invasive catheter tube sterility. • Per hospital protocol, change invasive catheters, culture blood, line tips, or fluids.
There is no evidence of deep vein thrombosis (DVT).	• Initiate DVT prophylaxis within 24 h of admission. • Monitor for leg pain, redness, or swelling.
Skin Integrity	
There is no evidence of skin breakdown.	• Turn side to side every 2 h. • Remove self-protective devices from wrists, and monitor skin per hospital policy. • Assess risk for skin breakdown using objective tool (eg, Braden scale). Consider pressure relief/reduction mattress.
Nutrition	
Caloric and nutrient intake meets metabolic requirements per calculation (eg, Basal Energy Expenditure).	• Provide parenteral, enteral, or oral nutrition within 48 h. • Consult dietitian or nutritional support service. • Avoid high-carbohydrate load if patient retains CO_2. • Monitor albumin, prealbumin, transferrin, cholesterol, triglycerides, glucose.
Comfort/Pain Control	
Patient is comfortable and evaluates pain as <4 on the pain scale.	• Assess pain/comfort every 4 h. • Administer analgesics and sedatives cautiously, closely monitoring respiratory rate, depth, and pattern. • Differentiate between agitation caused by discomfort or caused by hypoxia before medication administration. • Elevate head of bed to improve breathing comfort.
Psychosocial	
Patient demonstrates decreased anxiety.	• Assess vital signs during treatments, discussions, and so forth. • Cautiously administer sedatives. • Consult social services, clergy as appropriate. • Provide for adequate rest and sleep. • Provide support during periods of dyspnea.
Teaching/Discharge Planning	
Patient/significant others understand procedures and tests needed for treatment.	• Prepare patient/significant others for procedures such as chest physical therapy, bronchoscopy, pulmonary artery catheter insertion, or laboratory studies.
Significant others understand the severity of the illness, ask appropriate questions, anticipate potential complications.	• Explain the causes and effects of COPD and the potential for complications, such as pneumonia or cardiac dysfunction. • Encourage significant others to ask questions related to the ventilator, pathophysiology, monitoring, treatments, and so forth.
In preparation for discharge to home, patient understands activity levels, dietary restrictions, medication regimen, metered inhaler.	• Make appropriate referrals and consults early during hospitalization. • Initiate family education regarding proper use of metered inhaler, signs and symptoms of respiratory failure, and appropriate actions.

GOLD guidelines for the diagnosis, management, and prevention of COPD recommend a comprehensive pulmonary rehabilitation program.

PULMONARY REHABILITATION. In recent years, the value of pulmonary rehabilitation in managing COPD is being realized. Studies have shown muscle fatigue rather than dyspnea is likely the primary cause of deconditioning in patients with COPD.[43] Pulmonary rehabilitation does not improve lung function but rather helps patients optimize their functional status as well as their quality of life within the limitations of their pulmonary disease process. Pulmonary rehabilitation has been shown to increase exercise tolerance, reduce dyspnea, decrease anxiety and depression, improve quality of life, improve cognitive function, and provide the patient with a feeling of empowerment, meaning that they have control over their disease process.[43,44] Pulmonary rehabilitation programs consist of three to four hourly supervised sessions per week lasting for 6 to 12 weeks.

NUTRITIONAL COUNSELING. Malnutrition is a common problem in patients with COPD and is present in more than 50% of patients with COPD admitted to the hospital. The incidence of malnutrition varies with the degree of gas exchange abnormality. Malnutrition results in wasting of respiratory muscles and further respiratory muscle weakness.[43] A complete nutritional assessment should be conducted to identify strategies to maximize the patient's nutritional status. Improving the nutritional state of weight-losing patients with COPD can lead to increased respiratory muscle strength.[43]

SMOKING CESSATION. Smoking cessation is the single most effective method of reducing the risk for development of COPD. The value of smoking cessation cannot be underestimated in slowing the progression of COPD. In addition, it is the most cost-effective method. A brief (3-minute) counseling session to encourage a smoker to quit results in smoking cessation rates of 5% to 10%. Every smoker should have such a counseling session at every visit to a health care provider.[43] The effectiveness of smoking cessation is gained only when the patient is mentally ready to stop smoking. Numerous effective pharmacotherapies exist today for smoking cessation. These therapies consist of nicotine replacement products in various forms (inhalation, oral, sublingual, or transdermal). One of the most effective pharmacotherapies that have proved effective in smoking cessation is varenicline, which is an alpha-4-beta-2 neuronal nicotinic acetylcholine receptor partial agonist. Many clinical trials are ongoing in regard to varenicline. However, studies show that varenicline therapy in conjunction with counseling is very effective in diminishing withdrawal symptoms and helping patients achieve smoking cessation and that this therapy is superior to therapy with nicotine replacement products or bupropion (Wellbutrin).[43] The current treatment guidelines consist of a 12-week course of therapy.

Pharmacological Therapy

The goal of pharmacotherapy for COPD is to prevent and decrease symptoms, to reduce the frequency and severity of exacerbations, as well as to improve exercise tolerance and improve health status. According to the 2009 GOLD guidelines, pharmacological treatment for stable patients with COPD consists of bronchodilators, beta agonists, anticholinergics, and inhaled glucocorticosteroids.[43] These medications can be administered alone or in combination depending on the response to therapy as well as the severity of the disease. A combination of agents may produce greater effects than single-agent therapy, and the inhaled route is the preferred route of delivery.[43] Other pharmacological treatments that have been used in the past, such as systemic glucocorticoid, mucoactive agents, and chronic antibiotic therapy, are no longer being used for treating stable COPD patients.[43]

BRONCHODILATORS. Bronchodilators, primarily beta agonists and anticholinergics, along with inhaled glucocorticoids[43,44]are the mainstay of pharmacological management of stable COPD. These medications are sometimes used as single agents or in combination. Bronchodilators offer long-term improvement in symptoms, exercise capacity, and quality of life. However, these medications, as well as any other pharmacological agents, have not been shown to reverse the progression of COPD. Patients are usually on a combination of long-acting bronchodilator, such as tiotropium bromide (Spiriva), which is a long-acting anticholinergic, along with a short-acting bronchodilator, such as albuterol (Ventolin), which is a beta agonist, for rescue inhaler.[43,45] Long-acting anticholinergics are preferred over long-acting beta agonists because they have been shown to decrease hyperinflation, decrease COPD exacerbation, and improve dyspnea.[46] They increase the FEV_1 by widening the smooth muscle tone of the airways rather than by altering the elastic recoil properties of the lung.[46] Long-acting bronchodilators are the most convenient. The choice of the particular form of bronchodilator therapy depends on availability and the patient's response in terms of symptom relief and side effects. Combination therapy, rather than an increased dose of a single agent, may lead to improved efficacy and a decreased risk for side effects.

GLUCOCORTICOIDS. COPD is a disease that is characterized by systemic and airway inflammation.[42] The aim of inhaled glucocorticoids is to reduce the inflammation.[42] Studies have shown that inhaled glucocorticoids decrease COPD exacerbations but have little to no impact on overall mortality or improvement of lung function.[42] Inhaled glucocorticoids should always be used in conjunction with long-acting bronchodilators and never as a single agent.[43,45] Regular treatment with inhaled glucocorticosteroids for COPD is appropriate only for patients with GOLD stage III–IV who have FEV_1/FVC less than 70% of predicted value and with increased symptoms and repeated exacerbations on optimal bronchodilator therapy.[43]

OTHER PHARMACOLOGICAL AGENTS. Several other drugs may be useful but are not universally recommended. One such medication is theophylline. Studies have shown that theophylline decreases dyspnea and improves gas exchange.[45] However, several problems exist with using theophylline.[43] Theophylline is a medication that requires monitoring of blood levels. Theophylline is metabolized by the liver, so if there is anything that interferes with an individual's liver function, then the individual can have changes in the theophylline blood level, which leads to toxicity. Theophylline also interacts with a lot of other medications. It is for these reasons that using theophylline in the standard

management of COPD has lost favor.[43] Antibiotics should not be used in COPD except for treating infectious exacerbations and other bacterial infections.[43] Patients with COPD are more susceptible to seasonal flu and pneumonia; therefore, they should be routinely immunized with the seasonal flu vaccine and the pneumonia vaccine. Current research indicates that mucolytic agents have minimal overall benefits, and their widespread use is not recommended. *N*-acetylcysteine, a drug with mucolytic and antioxidant properties, has been shown to cause bronchoconstriction when inhaled. The antioxidant properties were not able to be demonstrated in studies; therefore, it is no longer used in the management of COPD.[43]

Oxygen Therapy

Oxygen therapy is one of the principal nonpharmacological treatments for patients with severe COPD. Oxygen therapy improves the quality of life, cognitive performance, as well as long-term survival of patients in a hypoxic state.[43] Oxygen therapy can be administered as long-term continuous therapy, during exercise, nocturnally, and in the relief of acute dyspnea. The goal of long-term oxygen therapy is to produce an oxygen saturation as measured by pulse oximetry (SpO_2) of at least 90% at rest, sleep, and exercise.[43] Supplemental oxygen therapy is recommended for individuals who are hypoxic as a result of COPD progression or in individuals recovering from an exacerbation with a PaO_2 at or below 60 mm Hg or an SaO_2 at or less than 90%, with or without hypercapnia.[43] Supplemental oxygen therapy is also indicated in individuals when PaO_2 is between 55 and 60 mm Hg or when SaO_2 is below 90% and there is evidence of cor pulmonale or polycythemia despite optimal medical management.[43]

Historically, there has been hesitation to administer oxygen to patients with COPD. However, in the face of acute exacerbation, prevention of tissue hypoxia overrules any concern regarding CO_2 retention.[43] If successful oxygenation (SaO_2 of 90% or more) is not obtained without a progression of respiratory acidosis, intubation and mechanical ventilation are usually performed.[43]

Surgical Therapy

Surgical intervention may include lung volume reduction surgery (LVRS), bullectomy, and lung transplantation. Hopefully, the benefit of these procedures for a select group of patients with COPD is improvement in lung volumes, exercise tolerance, dyspnea, pulmonary function tests, quality of life, and survival.[43]

LUNG VOLUME REDUCTION SURGERY. LVRS is a surgical procedure in which parts of the nonfunctioning or scarred lung, usually the upper lobes, are resected, thereby reducing the lung volume. LVRS can be performed via a median sternotomy or video-assisted thoracoscopic surgery. A rationale as to why LVRS works is that removal of the nonfunctioning or scarred part of the upper lung lobes improves the mechanical functioning of the diaphragm and intercostal muscles. This is achieved by decreasing the FRC and returning the diaphragm to its more normal curvature and lengthening configuration.[43] Therefore, this surgery improves the mechanics of breathing and hopefully decreases the work of breathing.

In the United States in the late 1990s, a multicenter national clinical trial called the National Emphysema Treatment Trial (NETT) compared LVRS to medical management of severe emphysema. An analysis of the data upon conclusion of this trial revealed several things regarding which individuals with emphysema should have LVRS. The data identified five groups. The group that was considered as poor candidates for this surgery had extremely poor pulmonary function (FEV_1 of 20% or less of predicted value and DLCO, or diffusion capacity, of 20% or less of predicted value) and homogenous emphysema. This group was at very high risk for LVRS. The second group had nonhomogenous upper lobe predominant emphysema with low exercise capacity after preoperative pulmonary rehabilitation. The third group had predominately upper lobe emphysema with a high exercise capacity after preoperative pulmonary rehabilitation. The fourth group consisted of individuals with homogenous emphysema and low postrehabilitation exercise capacity. The last group had homogenous emphysema and low postpulmonary rehabilitation exercise capacity.[47] Analysis of the data revealed that the individuals who gained the most benefit from LVRS when compared to medical management were the individuals who were in the group that had nonhomogenous emphysema with upper lobe predominance and limited exercise tolerance after preoperative pulmonary rehabilitation. These individuals had a lower mortality rate, better exercise tolerance, and improved health status. The individuals with predominantly upper lobe emphysema and good postrehabilitation exercise tolerance along with the individuals with homogenous emphysema and low postpulmonary rehabilitation exercise tolerance did gain some benefit in terms of improvement in exercise tolerance and health status but not to the extent as the abovementioned group.[47]

The NETT trial revealed the importance of pulmonary rehabilitation in the management of emphysema. The NETT trial also concluded that LVRS improves the quality of life of the patient with emphysema but does not improve overall survival advantage over medical management.[47] For patients awaiting eventual lung transplantation, LVRS provides a means of managing their symptoms while awaiting transplantation.

OTHER SURGICAL PROCEDURES. Patients with severe COPD (stage III) may also consider bullectomy and lung transplantation.[42,43] Bullectomy is a surgical procedure for bullous emphysema, which is effective in alleviating dyspnea and improving overall lung function.[42,43] Patients with very advanced COPD are candidates for lung transplantation. Lung transplantation has been shown to improve quality of life, exercise capacity, and functional capacity; however, long-term survival is still questionable.[47]

Prevention

Influenza vaccines reduce serious illness and death by about 50% in patients with COPD.[43] Vaccines containing killed or live, inactivated viruses are recommended because they are more efficacious in elderly patients with COPD. Vaccines are administered either once (autumn) or twice (autumn and winter) each year. Some experts do recommend pneumococcal vaccine administration once each year for

patients with COPD and chronic bronchitis and every other year for asplenic patients or patients at risk for decreased antibody levels (eg, in transplantation or in chronic renal failure).[42,43]

Chronic Bronchitis

Chronic bronchitis is defined as the presence of a productive cough for at least 3 months per year over 2 consecutive years, in the absence of other medical causes.[48] During an exacerbation or episode of acute infection, a patient with chronic bronchitis may present with airflow obstruction similar to that of a patient with asthma. However, the difference lies in the fact that, with chronic bronchitis, the primary cause of airflow obstruction is not due to airway hyperreactivity, and there are residual clinical symptoms even when the patient returns to baseline function.[12]

Pathophysiology

Airway obstruction is caused by inflammation of the major and small airways in chronic bronchitis (Fig. 26-7). Subsequently, edema and hyperplasia of submucosal glands and excess mucous excretion into the bronchial tree occur, resulting in a chronic productive cough.[12] Cigarette smoking is the major causal factor in the development of chronic bronchitis.[12] Other causes of chronic airway irritation include air pollutants and occupational exposure to nitrogen, sulfur oxides, or endotoxin.[12] Nonspecific pathological changes in the lung, including infiltration of airway mucosa and submucosa with neutrophils and mononuclear cells, smooth muscle hypertrophy, and enlargement of the submucosal secretory glands, may also contribute to the development of chronic bronchitis.[12]

Once the airway lumen is occluded by secretions and narrowed by a thickened wall, patients develop airflow obstruction and COPD. Acute bacterial or viral infection in patients with existing chronic bronchitis can increase airway and parenchymal damage, impair mucociliary clearance, obstruct bronchioles, and contribute to chronic epithelial damage and bacterial colonization that further exacerbate symptoms and airway obstruction.[12] Common bacteria isolated from the secretions of patients with chronic bronchitis include *H. influenzae*, *Haemophilus parainfluenzae*, *S. pneumoniae*, and *Moraxella catarrhalis*.[12] Even in nonsmoking patients, acute viral infection may lead to the chronic airway inflammation and chronic sputum production characteristic of chronic bronchitis.[12] In contrast to emphysema, chronic bronchitis may have a reversible component if the source of chronic infection or irritation is treated. These patients normally do not have hyperinflation or abnormal diffusion test results.

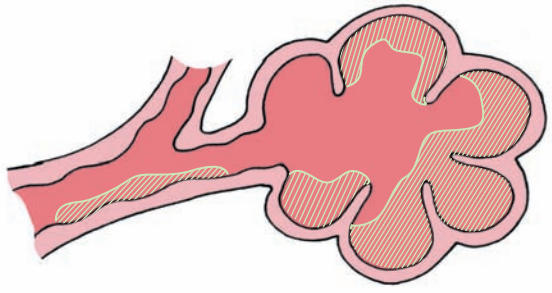


FIGURE 26-7 ▲ Bronchitis: inflammation and thickening produce narrowing of airways. *Lined areas* indicate secretions.

Assessment

Excessive bronchial secretions and subsequent airway obstruction and vasoconstriction lead to ventilation–perfusion mismatching. Patients do not compensate by increasing their ventilation and, therefore, develop hypoxemia, cyanosis, and eventually cor pulmonale with peripheral edema.[43] Common physical signs and symptoms may include:[11]

- Copious sputum expectoration arising from sleep
- Sputum that is usually mucoid, often with brownish discoloration
- Increased sputum volume or changes in color from whitish to yellow or green (signs of an endobronchial infection)
- Hemoptysis occurring during acute exacerbations
- Decreased breath sounds, wheezes, or rhonchi
- Resting respiratory rate greater than 16 breaths/min
- Prolonged forced expiratory time (greater than the normal 4 seconds)

Often, patients wait to seek medical treatment until they are in severe distress. Manifestations of severe exacerbations of chronic bronchitis are listed in Box 26-11.

Management

Patients with chronic bronchitis without airflow obstruction require no specific pharmacological treatment. An

BOX 26-11 Manifestations of Severe Exacerbations of Chronic Bronchitis

Constitutional Signs
Temperature frequently subnormal
White blood cell count varies—may be slightly ↑, normal, or ↓

Central Nervous System Disturbances
Headache
Confusion
Hallucinations
Depression
Drowsiness
Somnolence
Coma
Papilledema

Cardiovascular Signs
Diaphoresis
Tachycardia
Blood pressure varies: normal, ↑, or ↓
Vasoconstriction initially followed by vasodilation

Neuromuscular Signs
Fine tremors
Asterixis
Flaccidity
Convulsions

essential prophylactic measure involves rigorous bronchial hygiene to promote the clearance of secretions, which provide an ideal medium for bacterial growth in peripheral airways. It is important to prevent the development of an acute inflammatory process to avoid exacerbations. Other preventive measures include smoking cessation, immunization against influenza virus and *S. pneumoniae*, and prompt antibiotic treatment for acute exacerbations caused by bacterial tracheobronchitis.[12]

Chronic bronchitis with airflow obstruction does require pharmacological treatment. The major goals of drug therapy in chronic bronchitis with COPD are to reverse or slow the progression of airway obstruction and mucosal edema, decrease secretion volume, alleviate bronchial smooth muscle spasm, and decrease airway inflammation.[42,43] Primary agents include inhaled bronchodilators (beta$_2$-adrenergic agents, anticholinergic agents, corticosteroids) and theophylline.[42,43] An effective treatment regimen may include using a combination of drugs. By combining drugs with different mechanisms of action and duration of effect (long acting, short acting), more effective bronchodilation may occur with less incidence of side effects, such as tachycardia, restlessness, or toxicity. Although excessive cough and mucous production are common symptoms, regular use of antitussives and mucolytic agents is not indicated.[45]

Emphysema

The ATS defines emphysema as a loss of lung elasticity and abnormal, permanent enlargement of the airspaces distal to the terminal bronchioles with destruction of the alveolar walls and capillary beds without obvious fibrosis (Fig. 26-8).[42] Most patients with COPD have a combination of chronic bronchitis (mucous hypersecretion) and emphysema rather than "pure" bronchitis or emphysema.[42]

There are three types of emphysema: centrilobular emphysema, panacinar emphysema, and paraseptal emphysema (Fig. 26-9). Centrilobular emphysema is most common in smokers and often localizes in the upper lung zones. Panacinar emphysema is most frequently found in patients with alpha$_1$-protease inhibitor deficiency and is often localized in the lower lobes. Paraseptal emphysema is also most common in smokers and is localized peripherally, with possible formation of large bullae.[12]

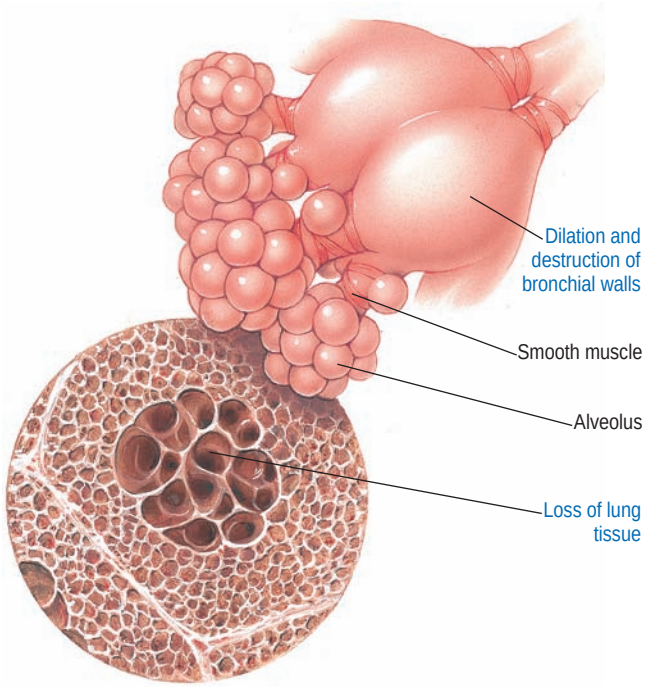


FIGURE 26-8 ▲ Lung changes in emphysema. Airspaces are enlarged in the emphysematous lung. (From Anatomical Chart Company: Atlas of Pathophysiology. Springhouse, PA: Lippincott Williams & Wilkins, 2010, p 91.)

Pathophysiology

The enlargement of the airspaces in emphysema results in hyperinflation of the lungs and increased total lung capacity.[12] Emphysema is believed to result from the breakdown of elastin by enzymes, called proteases, which digest proteins. These proteases, especially elastase, are released from neutrophils, alveolar macrophages, and other inflammatory cells.[44] Two recognized conditions that cause emphysema are smoking and inherited alpha$_1$-antitrypsin deficiency. Smoking contributes to increased inflammatory cells in the alveoli, enhanced release of elastase from neutrophils, increased elastase activity in macrophages, and activation of mast cells that release mast cell elastases.[44] Alpha$_1$-antitrypsin usually protects the lung from the destructive inflammatory cells; however, the elastic tissue–destructive process continues unabated in patients with an inherited alpha$_1$-antitrypsin deficiency.[44]

Almost all people who develop emphysema before 40 years of age have an alpha$_1$-antitrypsin deficiency. Evidence has shown that cigarette smoking decreases levels of alpha$_1$-antitrypsin and increases the number of macrophages in the alveolar walls. This vicious cycle promotes increased numbers of neutrophils. A hereditary deficiency in alpha$_1$-antitrypsin is responsible for about 1% of all cases of COPD.[44] Smoking and repeated respiratory tract infections further decrease alpha$_1$-antitrypsin levels, adding to the risk for emphysema in people with low alpha$_1$-antitrypsin levels.[44]

A common phenomenon in emphysema is spontaneous pneumothorax related to rupture of thinned parenchyma.[12] Patients may experience acute severe dyspnea and respiratory failure depending on the amount of pulmonary reserve (see the discussion of barotrauma in the section on Pneumothorax).

Assessment

Patients with pulmonary emphysema are referred to as "pink puffers" because their oxygen levels are usually satisfactory, and their skin remains pink.[12] There is a proportionate loss of ventilation and perfusion area in the lung. In severe COPD, air is trapped in the lungs during forced expiration, leading to abnormally high residual volume.[12] These patients develop a puffing style of breathing. Common physical examination findings in emphysema include increased basal respiratory rate; barrel-shaped chest; decreased breath sounds; soft, dry crackles in lung bases; right-sided S_3 gallop auscultated substernally; supraclavicular wasting and nasal flaring; and

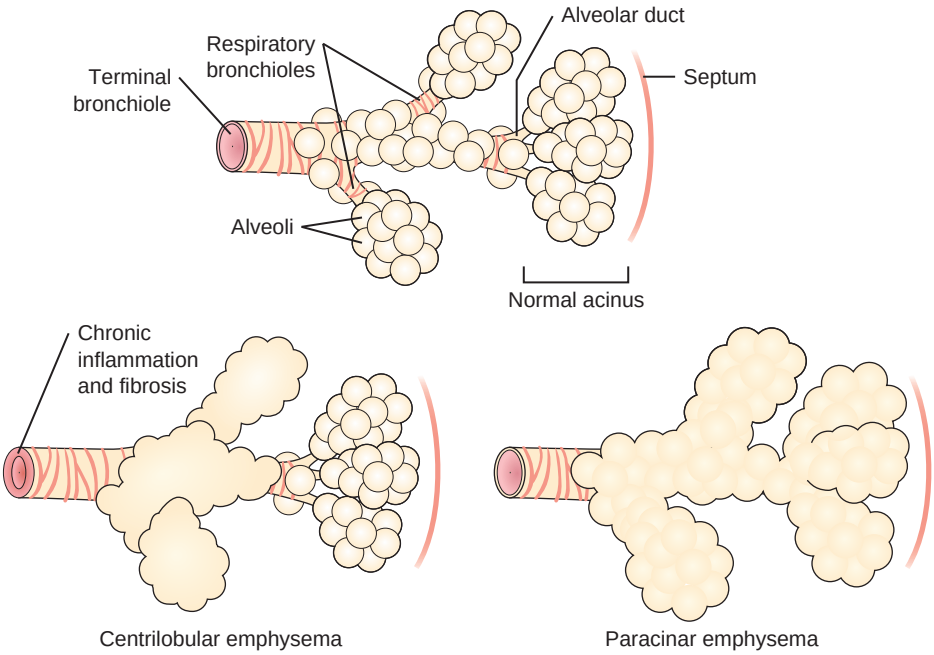


FIGURE 26-9 ▲ Types of emphysema. The acinus, the gas-exchanging structure of the lung distal to the terminal bronchiole, consists of the terminal bronchiole, respiratory bronchioles, alveolar ducts, alveolar sacs, and alveoli. In centrilobular (proximal acinar) emphysema, the respiratory bronchioles are mainly involved. In paraseptal (distal acinar) emphysema, the alveolar ducts are mainly affected. In panacinar (panlobular) emphysema, the acinus is uniformly damaged.

evidence of pulmonary hypertension, including an increased second heart sound, jugular venous distention, and right ventricular heave.[12] Cyanosis may be present in advanced cases of emphysema.

Management

The medical and surgical therapies described for COPD also pertain to patients with emphysema (see section on COPD, Management, pp. 570–573). Preventive measures include an annual influenza vaccine and a pneumococcal vaccine every 5 to 10 years. Medical therapy involves smoking cessation, pulmonary rehabilitation, and oxygen therapy in all patients who are hypoxemic (PaO_2 < 55 mm Hg or SaO_2 < 88%).[44] Pharmacological therapy includes bronchodilators ($beta_2$-agonists, anticholinergics, and theophylline), possibly mucolytics, and $alpha_1$-protease inhibitor replacement in young patients with homozygous disease. In addition, adequate nutrition is important. Patients with advanced emphysema (FEV_1 < 750 mL) may experience significant weight loss related to a variety of factors, including increased energy expended during breathing secondary to the use of accessory muscles, performing activities of daily living, and reduced caloric intake.[42] Patients who are overweight and hypercapnic should strive to lose weight to diminish the respiratory workload.[44]

Three surgical treatments available for patients with emphysema are LVRS, bullectomy, and lung transplantation. Currently, LVRS is the only recognized therapy that can increase respiratory function (FEV_1, FVC, ABG values, and exercise capacity) in moderate to severe emphysema.[47] For end-stage emphysema, the only definitive surgical treatment is single-lung transplantation. Because of the short supply of donor lungs, lung transplantation is usually reserved for younger patients (<60 years of age) with $alpha_1$-protease inhibitor deficiency.[43] Studies have indicated improvements in exercise performance and ABG values after lung transplantation.[44]

▲ Thoracic Surgery

Thoracic surgery is indicated as part of the management plan for a variety of diseases involving the lungs and the associated structures. Specific surgeries involving removal of part of the lung include wedge resection, lobectomy, pneumonectomy, lung volume reduction, and lung transplantation. Wedge resection or segmentectomy is performed for the removal of benign or malignant lesions; segmentectomy is the preferred method when patients are a poor risk with limited pulmonary reserve.[44,48] Bleeding may be extensive following the surgery, and two chest drains are usually in place to drain air or blood. Lobectomy may be performed as a treatment for malignant or benign tumors and infections such as bronchiectasis, tuberculosis, or fungal infection. Pneumonectomy is performed to remove one lung, usually because of primary carcinoma or significant infection. LVRS involves the excision of 20% to 30% of the volume of each lung to improve elastic recoil and diaphragmatic function in the patient with moderate to severe emphysema.[44,48] Lung transplantation may involve one lung or both lungs, or it may be inclusive as part of a heart–lung transplantation. To be considered a viable candidate for lung transplantation, a patient should

have advanced lung disease that is unresponsive to other medical therapy and minimal comorbidities. Significant areas of concern involved in this surgery include primary graft failure, lifelong immunosuppressive therapy, and organ rejection.[44]

▲ Acute Asthma

Asthma is a chronic inflammatory disease of the airways characterized by airway hyperresponsiveness to a variety of stimuli, reversible airflow limitation, and chronic inflammation of the airway submucosa.[49] It is manifested as variable airway obstruction that resolves either spontaneously or after bronchodilator administration.[49] Based on symptoms and lung function, the National Asthma Education and Prevention Program (NAEPP) has classified asthma as mild intermittent, mild persistent, moderate persistent, or severe persistent (Table 26-10). According to the CDC, asthma is a worldwide epidemic. Asthma affects 7% of the population or 17 million adults and children in the United States.[50] Asthma accounts for 2 million emergency department visits, 500,000 hospital admissions, and 5,000 deaths annually in the United States. Most cases are believed to be preventable.[50]

Pathophysiology

Inflammation may be present throughout the bronchial tree, from large airways to the alveoli. This inflammation is characterized by mast cell activation, inflammatory cell infiltration, edema, denudation and disruption of the bronchial epithelium, collagen deposition beneath the basement membrane, goblet cell hyperplasia (which contributes to mucous hypersecretion), and smooth muscle thickening (Fig. 26-10). This inflammatory process contributes to airway hyperresponsiveness, airflow limitation, pathological damage, and associated respiratory symptoms (ie, wheezing, shortness of breath, and chest tightness).[12]

Factors contributing to the airflow limitation in asthma include acute bronchoconstriction, airway mucosal edema, chronic formation of mucous plugs, and airway remodeling.[12]

The T lymphocytes (helper T [Th] cells) are believed to play a crucial role in the inflammation process.[51] Th1 cells serve a protective role against airway inflammation, and Th2 cells promote development of chronic airway inflammation. Recent studies suggest that possible early childhood viral and bacterial infections may contribute to Th2 cell stimulation and result in asthma pathogenesis.[51]

The etiology and pathogenesis of asthma are not fully understood. Inhaled irritants, such as cigarette smoke, inorganic dusts, and environmental pollutants, are common precipitants. These irritants stimulate the irritant receptors in the walls of the larynx and large bronchi, which initiate a reflex arc that travels to the central nervous system and back through the vagus nerve. This in turn induces bronchoconstriction.[12] The most common precipitant of an acute asthmatic exacerbation is an upper respiratory tract viral infection. The proposed mechanism of action is through epithelial damage and airway inflammation.[12] Other potential infectious causal factors include infection with *C. pneumoniae*, tracheobronchitis related to herpes simplex, and exposure to aspirin or other nonsteroidal anti-inflammatory cyclooxygenase-1 inhibitors, which can lead to life-threatening asthmatic reactions in selected patients.[49] The mechanism of action in exercise-induced asthma is less clear but thought to be related to hyperemia and stimulation of irritant receptors following the rewarming of cooled airways. Common triggers of asthma exacerbations are given in Box 26-12.

Assessment

History and Physical Findings

The medical history should address the following areas:[49,51]

- Symptoms and symptom patterns
- Precipitating and aggravating factors
- Development of disease

Table 26-10 Classification of Asthma Severity by Clinical Features Before Treatment

	Symptoms	Nighttime Symptoms	Lung Function
Intermittent	Symptoms once a week or less Brief exacerbations	Two or less times a month	FEV_1 or PEF 80% or more predicted PEF or FEV_1 variability 20% or less.
Mild persistent	Symptoms once a week but less than once a day Exacerbations may affect activity and sleep	More than two times a month	FEV_1 or PEF > 80% predicted PEF or FEV_1 variability 20%–30%
Moderate persistent	Symptoms daily Daily use of inhaled short-acting β_2-agonist Exacerbations affect activity and sleep	More than once a week	FEV_1 or PEF, or >60% to <80% predicted PEF or FEV_1 variability >30%
Severe persistent	Symptoms daily Frequent exacerbations Limitation of physical activities	Frequent nocturnal asthma symptoms	FEV_1 or PEF 60% or less predicted PEF or FEV_1 variability >30%

FEV_1, forced expiratory volume in 1 s; PEF, peak expiratory flow.

Adapted from 2007 Update: Global Strategy for Asthma Management and Prevention, Global Initiative for Asthma (GINA) 2007. Available at http://www.ginasthma.org.

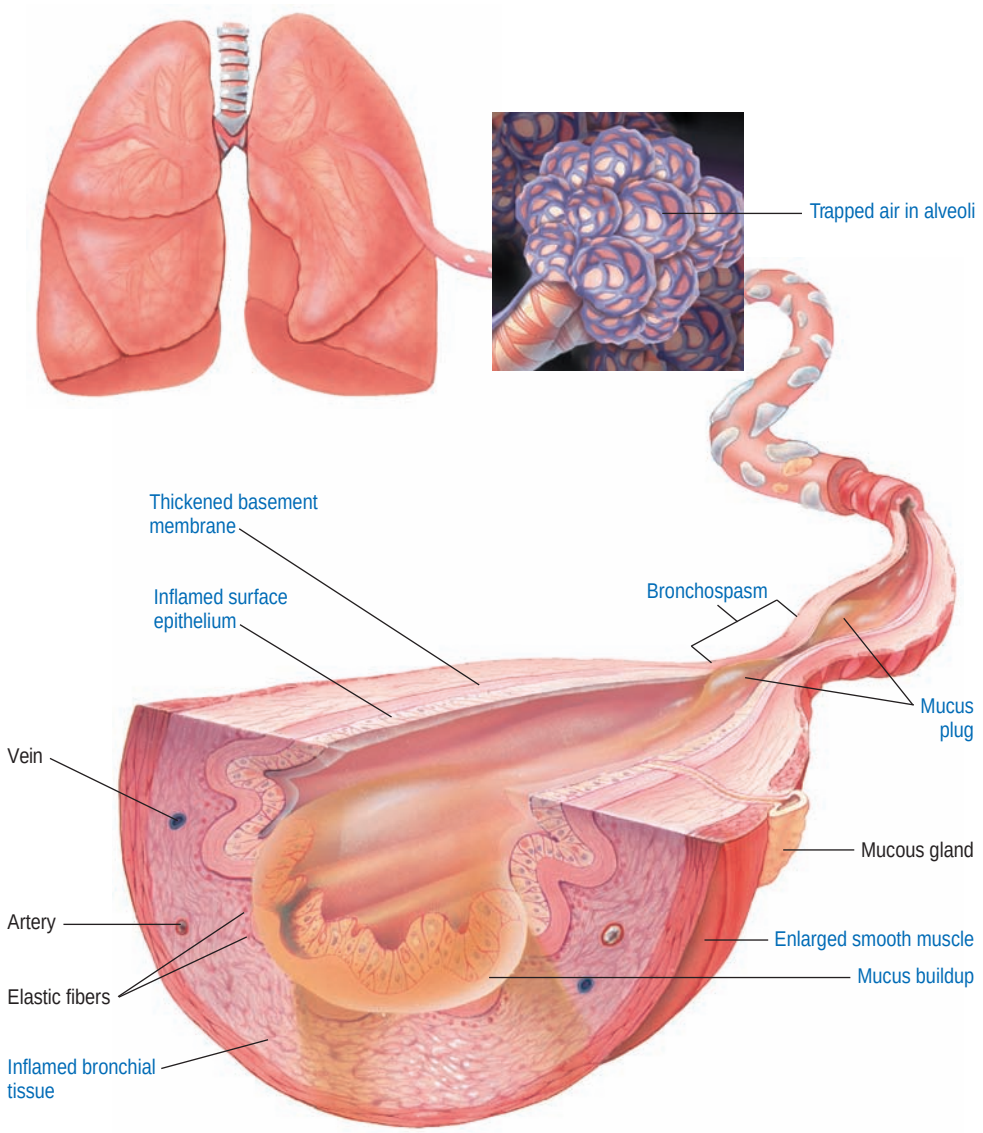


FIGURE 26-10 ▲ Asthmatic bronchus. (From Anatomical Chart Company: Atlas of Pathophysiology. Springhouse, PA: Lippincott Williams & Wilkins, 2010, p 85.)

BOX 26-12 Common Triggers of Asthma

Factors Influencing the Development and Expression of Asthma,

Host Factors

Genetic, for example,
- Genes predisposing to atopy
- Genes predisposing to airway hyperresponsiveness

Obesity
Sex

Environmental Factors

Allergens
- Indoor: Domestic mites, furred animals (dogs, cats, mice), cockroach, allergen, fungi, molds, yeasts
- Outdoor: Pollens, fungi, molds, yeast

From Global Initiatives for Asthma (GINA) Guidelines: Global strategy for asthma management and prevention. Available at www.ginasthma.org. Accessed December 2010.

- Current treatment
- Effect of symptoms on activities of daily living
- Impact of asthma on the patient and family
- Perceptions of the disease by the patient and family (parent, if appropriate)

The physical examination should focus on the following areas:[49,51]

- Vital signs
- Height, weight, and a comparison of normal values for age
- Inspection of skin for evidence of atopic dermatitis or eczema
- Mouth breathing
- Dark discoloration beneath the lower eyelids ("allergic shiners")
- Edematous or pale nasal mucosa
- Clear nasal discharge
- Hypertrophy of tonsils and adenoids

- Presence of tearing and periorbital edema
- Lung auscultation for wheezing
- Hyperexpansion of the thorax
- Use of accessory muscles
- Presence of tachypnea

Signs and symptoms vary with the degree of bronchospasm. Patients may complain of shortness of breath associated with wheezing, especially during the late night and early morning hours, along with disruption of sleep.[51] According to the NAEPP guidelines, asthma symptoms may be classified as mild intermittent, mild persistent, moderate persistent, or severe persistent depending on frequency, duration, timing, and associated lung function level (see Table 26-10). Additional findings, including tachycardia, retractions, restlessness, anxiety, inspiratory or expiratory wheezing, hypoxemia, hypercapnia, cough, sputum production, expiratory prolongation, cyanosis, and an elevated pulsus paradoxus (systolic blood pressure in expiration exceeding that in inspiration by >10 mm Hg), may be observed in patients who have severe attacks.[50] The severity of an acute asthma exacerbation is evaluated using patient symptoms, physiologic signs, and lung function results and can be classified as mild, moderate, severe, and impending failure (Table 26-11).

Diagnostic Studies

Objective measures in the diagnosis and measurement of asthma severity consist of spirometry and pulmonary function testing. Allergy testing may be performed to ascertain precipitating allergens.[51,52] Spirometry measurements of the FVC, FEV_1, and FEV_1/FVC ratio are performed before and after the patient inhales a short-acting bronchodilator, which determines whether airflow obstruction is present and whether it is reversible. An increase of at least 12% and 200 mL in FEV_1 after inhaling a short-acting bronchodilator indicates significant reversibility and confirms the presence of asthma.[51,52] The FEV_1/FVC ratio is less than 65% predicted. Airway resistance is increased, so FEV is reduced out of proportion to FVC reduction. Portable peak flow meters are used to monitor ongoing lung function. Patients are instructed how to measure peak expiratory flow, an indicator of the degree of airflow obstruction in the large airways, by using the peak flow meter on a regular basis.[51,52]

Management

The level of treatment is based on the patient's level of asthma severity, which changes with time, age, and compliance with

Table 26-11 Classification of Severity of Asthma Exacerbations

	Mild	Moderate	Severe	Impending Respiratory Failure
Symptoms				
Breathlessness	Walking Can lie down	Talking Prefers sitting	At rest Hunched forward	At rest
Talks in	Sentences	Phrases	Words	
Alertness	May be agitated	Usually agitated	Usually agitated	Confused or drowsy
Signs				
Respiratory rate	Increased	Increased	Often more than 30 breaths/min	
Use of accessory respiratory muscles	Usually not	Commonly	Usually	Paradoxical thoracoabdominal movement
Breath sounds	Moderate wheezing, often end expiratory	Loud wheezes	Usually loud wheezes	Absence of wheezes
Heart rate (beats/min)	<100	100–120	>120	Bradycardia
Pulsus paradoxus (mm Hg)	Absent or <10	May be present, 10–25	Often present, >25	Often absent
Functional Assessment				
PEF (% predicted or personal best)	>80%	Approximately 60%–80%	<60% predicted or personal best or response to therapy lasts <2 h	
SaO_2 (%, room air)	>95	91–95	<90	
PaO_2 (mm Hg, room air)	Normal	>60	<60	
$PaCO_2$ (mm Hg)	<45	<45	>45	

Adapted from 2007 Update: Global Strategy for Asthma Management and Prevention, Global Initiative for Asthma (GINA), 2007. Available at http://www.ginasthma.org.

treatment.[51,52] Frequent reassessments of the level of severity are necessary to provide adequate therapy. The overall goals of therapy are to prevent chronic and troublesome symptoms, prevent exacerbations of symptoms, maintain normal activity levels, maintain normal pulmonary function, optimize pharmacotherapy and minimize side effects, and satisfy the patient's and the family's expectations and goals for asthma care.[52] A stepwise pharmacological approach is recommended in treating patients with asthma. The main goal is to gain control quickly and to "step down" to the lowest medication level required to maintain asthma control.[52] A list of quick-relief asthma medications is presented in Table 26-12. A flow chart outlining the stepwise approach is shown in Figure 26-11.

Asthma education and self-management training are critical to helping the asthmatic patient control airway inflammation. The 2009 Global Initiatives for Asthma guidelines describe the critical components of asthmatic patient education.[52] An important aspect of asthmatic patient education programs is training in the necessary management skills. These skills include proper medication administration, understanding the need for maintenance medications, knowing the early

Table 26-12 Drug Therapy in Asthma

	Examples	Possible Routes of Administration	Mechanism of Action
Bronchodilators			
$Beta_2$ agonist Short acting	Fenoterol Levalbuterol Salbutamol (albuterol) Terbutaline reproterol	Inhaled, oral, parenteral (depending on particular drug)	↑ cAMP via stimulation of adenylate cyclase, which results both in bronchodilation and inhibition of mediator release in immediate hypersensitivity reaction
Long acting	Terbutaline Salbutamol Bambuterol Formoterol		
Xanthines	Theophylline	Oral	Adenosine antagonism and induction on histone deacetylase, which decreases proinflammatory cytokines
Short-acting anticholinergics	Ipratropium	Inhaled	Blockade of cholinergic (bronchoconstrictor) effect on airways by diminishing cyclic guanosine monophosphate concentration and inhibit vagal efferent pathway
Anti-Inflammatory Drugs			
Corticosteroids	Prednisone Prednisolone Hydrocortisone Methylprednisolone	Systemic (oral or parenteral, depending on particular drug)	Direct and indirect inhibitory effects on multiple inflammatory genes
	Beclomethasone Triamcinolone Flunisolide Fluticasone Budesonide Ciclesonide	Inhaled	
Cromones	Sodium cromoglycate Nedocromil	Inhaled	Inhibition of mediator release from mast cells
Drugs Directed at Specific Targets			
5-Lipoxygenase inhibitors	Zileuton	Oral	Decreased production of leukotrienes
Leukotriene antagonists	Zafirlukast Montelukast Pranlukast	Oral	Cysteinyl-leukotriene 1 receptor antagonism
Anti-IgE antibody	Omalizumab	Parenteral	Binds circulating IgE

cAMP, cyclic adenosine monophosphate.

Adapted from Goroll AH, Mulley AG: Primary Care Medicine: Office Evaluation and Management of the Adult Patient, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, pp 390–403.

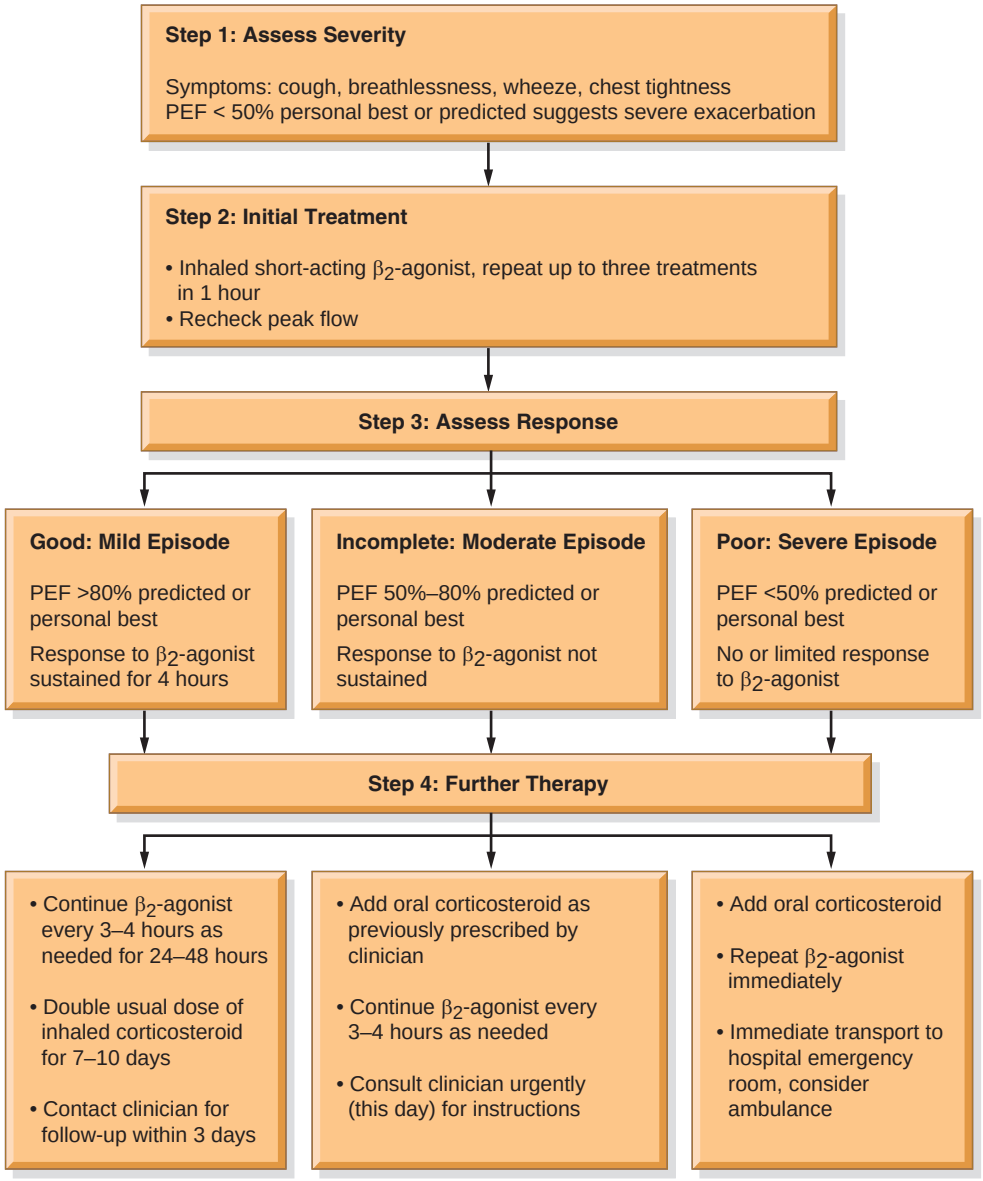


FIGURE 26-11 ▲ Management of Asthma Exacerbation in Acute Care Settings. PEF, peak expiratory flow. (From GINA report, Global Initiatives for Asthma (GINA) Guidelines: Global Strategy for Asthma Management and Prevention. Available at www.ginasthma.org. Accessed December 2010.)

warning signs of an asthma attack, making decisions on the basis of self-monitoring of symptoms and peak flow results, and maintaining control of environmental asthma triggers (ie, dust mites, fur-bearing animals).[52] Recent studies have confirmed that appropriate therapy, coupled with structured asthma education, significantly improves short-term compliance with therapy and decreases asthma morbidity.[52] Long-term asthma management requires regular follow-up care with a clinician experienced in long-term asthma to maintain optimal asthma control and avoid preventable complications.[52]

Status Asthmaticus

Status asthmaticus is a medical emergency. It is an acute refractory asthma attack that has not responded to rigorous therapy with beta$_2$-adrenergic compounds or intravenous theophylline. Patients present with a dramatic picture of acute anxiety, markedly labored breathing, tachycardia, and diaphoresis. Deterioration of pulmonary function results in alveolar hypoventilation with subsequent hypoxemia, hypercapnia, and acidemia. A rising $PaCO_2$ in a patient with an acute asthmatic attack is often the first objective indication of status asthmaticus.[50]

The treatment of status asthmaticus involves the institution of multiple therapeutic modalities. All patients with status asthmaticus demonstrate hypoxemia and require oxygen therapy. Patients are also usually dehydrated and require fluid resuscitation. Pharmacological agents include methylxanthines, sympathomimetic amines, and corticosteroids.[50,52] If pulmonary function cannot be improved and respiratory

failure ensues, patients may require intubation and assisted ventilation (see Chapter 25). A spontaneous pneumothorax may occur during severe acute asthmatic attacks, as well as during positive-pressure mechanical ventilation (see section on Pneumothorax, Management, p. 561).

▲ Acute Respiratory Failure

Acute respiratory failure is a sudden and life-threatening deterioration in pulmonary gas exchange, resulting in carbon dioxide retention and inadequate oxygenation.[53] Acute respiratory failure remains a major cause of morbidity and mortality in the intensive care setting, despite the technological advances in diagnosis, monitoring, and management that have been made in the past four decades.[54] A study of more than 1,400 patients found that 44% of patients diagnosed with acute respiratory failure who required admission to the ICU died in the hospital; in the past 20 years, this statistic appears not to have changed significantly.[55,56] However, a recent analysis of past studies on the mortality of patients with acute respiratory failure showed that there have been improvements due to more lung protective ventilatory therapies.[57] Acute respiratory failure may be responsible for as many as 10% to 15% of admissions to medical ICUs and for as many as 50% to 75% of those patients who require ICU hospital stays longer than 7 days.[54]

Pathophysiology

Acute respiratory failure is defined as a PaO_2 of 55 mm Hg or less, a $PaCO_2$ greater than 50 mm Hg, and an arterial pH less than 7.35.[56,58] This definition is valid only in cases in which baseline ABG values are assumed to be normal.[58] In patients with established chronic hypoxemia or hypercapnia, acute respiratory failure is indicated by the acute deterioration of blood gases relative to their previous levels rather than their absolute values.[58] In patients with chronic lung disease, ABG values associated with classic acute respiratory failure may not be present because these patients have adapted to blood gas levels outside this range, consistent with their disease process.[58]

Acute respiratory failure may be caused by a variety of pulmonary and nonpulmonary diseases (Box 26-13). Respiratory failure may result from malfunction of the respiratory center, abnormal respiratory neuromuscular system, chest wall diseases, airway obstruction, or parenchymal lung disorders.[59] Many factors may precipitate or exacerbate acute respiratory failure (Box 26-14).

A vicious positive feedback mechanism characterizes the deleterious effects of continued hypoxemia and hypercapnia. Hypoxemia affects every organ and tissue, and hypercapnia impairs cellular functions.[58] Hypoxemia in respiratory failure may be caused by any of these conditions, separately or in various combinations; Table 26-13 lists the

BOX 26-13 Causes of Acute Respiratory Failure

Intrinsic Lung/Airway Diseases

Large Airway Obstruction
- Congenital deformities
- Acute laryngitis, epiglottitis
- Foreign bodies
- Intrinsic tumors
- Extrinsic pressure
- Traumatic injury
- Enlarged tonsils and adenoids
- Obstructive sleep apnea

Bronchial Diseases
- Chronic bronchitis
- Asthma
- Acute bronchiolitis

Parenchymal Diseases
- Pulmonary emphysema
- Pulmonary fibrosis and other chronic diffuse infiltrative diseases
- Severe pneumonia
- Acute lung injury from various causes (acute respiratory distress syndrome)

Cardiovascular Disease
- Cardiac pulmonary edema
- Massive or recurrent pulmonary embolism
- Pulmonary vasculitis

Extrapulmonary Disorders

Diseases of the Pleura and the Chest Wall
- Pneumothorax
- Pleural effusion
- Fibrothorax
- Thoracic wall deformity
- Traumatic injury to the chest wall: flail chest
- Obesity

Disorders of the Respiratory Muscles and the Neuromuscular Junction
- Myasthenia gravis and myasthenia-like disorders
- Muscular dystrophies
- Polymyositis
- Botulism
- Muscle-paralyzing drugs
- Severe hypokalemia and hypophosphatemia

Disorders of the Peripheral Nerves and Spinal Cord
- Poliomyelitis
- Guillain-Barré syndrome
- Spinal cord trauma (quadriplegia)
- Amyotrophic lateral sclerosis
- Tetanus
- Multiple sclerosis

Disorders of the Central Nervous System
- Sedative and narcotic drug overdose
- Head trauma
- Cerebral hypoxia
- Cerebrovascular accident
- Central nervous system infection
- Epileptic seizure: status epilepticus
- Metabolic and endocrine disorders
- Bulbar poliomyelitis
- Primary alveolar hypoventilation
- Sleep apnea syndrome

BOX 26-14 PATIENT SAFETY

Precipitating Factors Leading to Acute Respiratory Failure

- Changes of tracheobronchial secretions
- Infection: viral or bacterial
- Disturbance of tracheobronchial clearance
- Drugs: sedatives, narcotics, anesthesia, oxygen
- Inhalation or aspiration of irritants, fumes, smoke, vomitus, foreign body
- Cardiovascular disorders: heart failure, pulmonary embolism, shock
- Mechanical factors: pneumothorax, pleural effusion, abdominal distention
- Trauma, including surgery
- Neuromuscular abnormalities
- Allergic disorders: bronchospasm
- Increased oxygen demand: fever, infection
- Inspiratory muscle fatigue

From Farzan S: Respiratory failure. In Farzan S (ed): A Concise Handbook of Respiratory Diseases, 4th ed. Stamford, CT: Appleton & Lange, 1997, pp 371–386, with permission.

causes of hypoxemia.[56,58,59] Hypercapnia results from alveolar hypoventilation and ventilation–perfusion mismatching when there is no compensation by increased ventilation of well-perfused regions.[59] In acute hypercapnia, arterial blood pH is decreased, indicating acute respiratory acidosis. Patients with advanced COPD and chronic hypercapnia may exhibit an acute rise of $PaCO_2$ to a high level, a decrease of blood pH, and a significant increase in serum bicarbonate during the onset of acute respiratory failure.[59]

Hypoxemia and hypercapnia may have precipitous effects, which include the following:[55]

- Increased pulmonary vascular resistance
- Cor pulmonale
- Right-sided heart failure
- Impaired left ventricular function
- Reduced cardiac output
- Cardiogenic pulmonary edema
- Diaphragmatic fatigue from increased workload of respiratory muscles

Classification

Acute respiratory failure is classified as acute hypoxemic respiratory failure, acute hypercapnic respiratory failure, or combined hypoxemic and hypercapnic respiratory failure.[59] Acute hypoxemic respiratory failure is a direct defect in oxygenation. Acute hypercapnic respiratory failure is a direct defect in ventilation. Historically, acute hypoxemia respiratory failure and acute hypercapnia were discussed in literature as type I and type II, respectfully, respiratory failure. Current literature discusses these pathologies as ARDS.

Acute Hypoxemic Respiratory Failure

Acute hypoxemic respiratory failure is the result of abnormal oxygen transport secondary to pulmonary parenchymal disease, with increased alveolar ventilation resulting in a low $PaCO_2$.[60] The principal problem in this respiratory failure is the inability to achieve adequate oxygenation, as evidenced by a PaO_2 of 60 mm Hg. The most common cause of hypoxemia is decreased FiO_2, hypoventilation, diffusion impairment (low DLCO), ventilation–perfusion mismatch, and shunt. Careful analysis of the ABGs and calculation of an alveolar–arterial gradient (*A-a*) will assist in evaluating the severity of the hypoxemia.[60] The major causes of this type of respiratory failure are listed in Table 26-14.

Acute Hypercapnic Respiratory Failure

Acute hypercapnic respiratory failure, or ventilatory failure, is the result of inadequate alveolar ventilation and is characterized by marked elevation of carbon dioxide with relative preservation of oxygenation.[56] Hypoxemia results from

Table 26-13 Causes of Hypoxemia in Acute Respiratory Failure

Mechanism	Predominant Gas Exchange Abnormality
Controller dysfunction—nervous system: sedative medications, chronic obstructive or interstitial lung disease, toxic overdoses, hypothermia postoperatively, brainstem strokes	Hypoxemia and hypercarbia
Musculature (pump) dysfunction: medications—paralytics, aminoglycosides, steroids, botulism; myopathy; myositis; metabolic abnormalities (hypothyroidism, hypophosphatemia); myasthenia gravis	Hypercapnia
Airway dysfunction: asthma, emphysema/chronic bronchitis, bronchiolitis, endobronchial tumor, mass or stricture	Mild: hypoxemia and hypocarbia Severe: hypoxemia and hypercarbia
Alveolar dysfunction: pneumonia, pulmonary edema, pulmonary hemorrhage, ARDS, drug reaction, pulmonary contusion	Hypoxemia and hypercarbia
Pulmonary vasculature dysfunction: acute pulmonary embolism, pulmonary hypertension, AVM or intracardiac shunt	New-onset hypoxemia with or without hypercarbia Exertional hypoxemia Hypoxemia that is refractory to oxygen therapy

From Lilly C, Ingenito EP, Shapiro SD: Respiratory failure. In Kasper DL, Fauchi AS (eds): Harrison's Principles of Internal Medicine, 17th ed. New York, NY: McGraw-Hill, 2008, with permission.

Table 26-14 Evaluation and Management of Causes of Acute Respiratory Failure

Etiology	Key Clinical Findings	Key Diagnostic Tests	Specific Therapy
Respiratory Failure Caused by Central Nervous System Dysfunction*			
CNS-depressant drugs	History of drug overdose, head trauma, or anoxic encephalopathy Pupillary changes, needle marks	Response to naloxone Toxicology screen Electrocardiogram	Antidotes for the drugs taken Neurological evaluation
Hypothyroidism	Myxedema	Thyroid function test	Cautious thyroid replacement
Starvation	Cachexia Diarrhea	↓Albumin ↓Cholesterol	Nutrition
Metabolic alkalosis	Lethargy Confusion	ABGs Serum electrolytes	Treat underlying causes
Structural brain stem damage	Localizing neurologic findings	CT, MRI, cerebrospinal fluid cytology	Radiation, chemotherapy
Neoplasm	Headache		
Infection	Headache, fever	CT, MRI, cardiac echo	Antimicrobial therapy
Primary alveolar hypoventilation (Ondine's curse)	Daytime hypersomnolence, headache, rarely dyspneic, polycythemia, cor pulmonale	Blunted or absent ventilatory response to ↑CO_2, ↓O_2 in inspired gas Normal pulmonary function test	Nighttime ventilatory support Electrophrenic pacing Medroxyprogesterone acetate Supplemental oxygen
Central sleep apnea	Same as primary alveolar hypoventilation	Polysomnography: apnea without respiratory effort Normal CO_2, O_2 response curves while awake	Nighttime ventilatory support Electrophrenic pacing Supplemental oxygen
Respiratory Failure Caused by Peripheral Nervous System Dysfunction*			
Spinal cord disease	Above C5, diaphragm, intercostal, and abdominal activity abolished	Spinal x-ray film, CT, MRI	Supportive, vital capacity tends to improve more than 3 mo in traumatic lesions C5 and below
Traumatic	Below C5, diaphragm preserved, intercostal and abdominal activity abolished		Phrenic nerve pacing for high cervical cord lesions with intact phrenic nerve
Strychnine	Intense muscle spasms Apnea Metabolic acidosis	Toxicology screen Clinical picture	Supportive Gastric lavage, charcoal
Hyperthyroidism	Thyrotoxicosis heat intolerance, tachycardia, hyperreflexia	TSH, TFT	Propylthiouracil, methimazole
Hypothyroidism	Myxedema, cold intolerance Hyporeflexia, bradycardia	TSH, TFTs	Replace thyroid hormone
Respiratory Failure Caused by Respiratory Muscle Dysfunction*			
Muscle dystrophies	Proximal muscle weakness and atrophy Hereditary	Muscle biopsy Elevated CPK Genetic analysis	Supportive Duchenne: prednisone
Periodic paralyses	Hypokalemic, hyperkalemic, or normokalemic Genetic Muscle weakness associated with exercise, emotional upset, cold, alcohol	Serum potassium Family history	Avoid precipitating factors Carbonic anhydrase inhibitor

(*continued on page 585*)

Table 26-14 Evaluation and Management of Causes of Acute Respiratory Failure (continued)

Etiology	Key Clinical Findings	Key Diagnostic Tests	Specific Therapy
Respiratory Failure Caused by Chest Wall, Pleural, and Upper Airway Diseases*			
Kyphoscoliosis	Spinal curvature ≥120 degrees Progressive dyspnea on exertion over several years	Spinal x-ray films Restriction on PFTs	Nighttime ventilatory support
Flail chest	Multiple rib fractures, paradoxical respiration ± pleuritic chest pain	Chest film	Mechanical positive-pressure ventilation
Ankylosing spondylitis	Limited chest expansion Apical pulmonary fibrosis Limited lumbar mobility Chronic lower back pain	PFTs (↑functional residual capacity, ↓total lung capacity) HLA-B27 Spine and sacroiliac x-ray films	Anti-inflammatory agents Flexibility exercises
Angioedema/anaphylaxis	Stridor in setting of Hymenoptera sting, contrast media, or drug administration	Other evidence of angioedema/anaphylaxis; complement levels	Epinephrine parenterally Cricothyroidotomy
Foreign body aspiration	Unable to speak Stridor or apnea	X-ray film helpful when foreign body below vocal cords	Heimlich maneuver Bronchoscopy Cricothyroidotomy
Respiratory Failure by Intrapulmonary Causes			
Cardiogenic pulmonary edema†	Rales, diaphoresis	Chest x-ray: pulmonary edema Echocardiogram	Fluid management for adequate peripheral perfusion (diuresis/fluid) Reduce LVEDP
Adult respiratory distress syndrome†	Rales PaO_2 < 55 mm Hg with FiO_2 more than 60% PaO_2/FiO_2 200 mm Hg or less (regardless of PEEP) Fever	CXR: bilateral infiltrates CBC Pulmonary artery catheter: PAOP 18 mm Hg or less when measured OR no clinical evidence of left atrial hypertension	Treat underlying cause Pulmonary vasodilators Corticosteroids Mechanical ventilation Surfactant replacement
Acute lung injury (caused by sepsis, blood transfusion, pneumonia, aspiration, and/or multiple traumas)†	Dyspnea PaO_2/FiO_2 ≤ 300 mm Hg (regardless of PEEP)	CXR: Bilateral airspace disease, CT: Pulmonary edema from increased permeability ABG Pulmonary artery catheter: PAOP 18 mm Hg or less when measured OR no clinical evidence of left atrial hypertension	Increase FiO_2 PEEP Bronchodilation Inhaled nitric oxide Antibiotics
COPD‡	Dyspnea on exertion Prolonged forced expiratory time Wheezing Decrease in breath sounds Hyperinflation New onset of paradoxical respiratory motion or respiratory alternans	PFTs ABG CXR CT scan	Oxygen therapy Bronchodilators Antibiotics Corticosteroids Nutritional support Smoking cessation

*Adapted from Hollingsworth HM, Pratter MR, Irwin RS. Respiratory failure Part V: Extrapulmonary causes of respiratory failure. In Irwin RS, Rippe JM (eds): Irwin and Rippe's Intensive Care Medicine, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, pp 541–555.

†Adapted from Allen GB, Parsons PE: Respiratory failure Part II: Acute respiratory failure due to acute respiratory distress syndrome and pulmonary edema. In Irwin RS, Rippe JM (eds): Irwin and Rippe's Intensive Care Medicine, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, pp 497–515.

‡Adapted from Balter MS, Grossman RF. Respiratory failure Part IV: Chronic obstructive pulmonary disease. In Irwin RS, Rippe JM (eds): Irwin and Rippe's Intensive Care Medicine 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, pp 531–540.

CNS, central nervous system; CBC, complete blood cell count; CT, computed tomography; CPK, creatinine phosphokinase; CXR, chest x-ray; HLA-B27, human leukocyte antigen-B27; LVEDP, left ventricular end-diastolic pressure; MRI, magnetic resonance imaging; PAOP, pulmonary artery occlusion pressure; PFTs, pulmonary function tests; TFT, thyroid function text; TSH, thyroid stimulating hormone.

reduced alveolar pressure of oxygen (PaO_2) and is proportionate to hypercapnia.[60] Three factors contribute to hypercapnia: increased exercise, overfeeding, hyperthyroidism, burns, fever, sepsis, and decreased elimination of carbon dioxide (lack of respiratory drive, muscular disorder, and increased respiratory compliance).[60] Increased work of breathing most commonly occurs in COPD (increased dead space) or asthma (elevated airway resistance), and it may also result from thoracic abnormalities (restriction on lungs), such as pneumothorax, or pleural effusions.[60] The major causes of this type of respiratory failure are listed in Table 26-14.[60]

Assessment

History

A complete medical and social history should be obtained from the patient or a family member to determine the patient's baseline respiratory status on admission (see Chapter 24, Box 24-1, p. 486). Data from a comprehensive assessment are used in goal setting, intervention, and evaluation to ensure that patients receive high-quality health care.[61] Self-management categories address the patient's capacity for self-care (physical, cognitive, psychosocial, socioeconomic, and environmental). The health care professional will find this a valuable tool in the continual assessment process of patients with pulmonary disorders.

Physical Findings

Presentation of acute respiratory failure may vary, depending on the underlying disease, precipitating factors, and degree of hypoxemia, hypercapnia, or acidosis.[58–60] It is essential to determine whether intubation and positive-pressure ventilation are required as emergency measures; this is the most critical assessment objective.[54] Typically, intubation and ventilation are necessary in patients with depressed mental status or coma, severe respiratory distress, extremely low or agonal respiratory rate, obvious respiratory muscle fatigue, peripheral cyanosis, or impending cardiopulmonary arrest.[54,60] Patients with altered mental status are at risk for aspiration of gastric contents. In any of these situations, immediate intervention is vital and should not be postponed pending the results of ABG studies or chest radiography.[54,60]

The classic symptom of hypoxemia is dyspnea, although this may be completely absent in ventilatory failure resulting from depression of the respiratory center.[60] Other presenting symptoms of hypoxemia include cyanosis, restlessness, confusion, anxiety, delirium, tachypnea, tachycardia, hypertension, cardiac dysrhythmias, and tremor.[60] Peripheral cyanosis of the skin, lips, or nail beds suggests the presence of profound arterial hypoxemia, usually with a PaO_2 less than 50 mm Hg.[54]

The cardinal symptoms of hypercapnia are dyspnea and headache.[60] Other clinical manifestations of hypercapnia include peripheral and conjunctival hyperemia, hypertension, tachycardia, tachypnea, impaired consciousness, papilledema, and asterixis.[60] Uncorrected carbon dioxide narcosis leads to diminished alertness, disorientation, increased intracranial pressure, and ultimately unconsciousness.[54] Other physical findings on examination may include use of accessory muscles of respiration, intercostal or supraclavicular retraction, and paradoxical abdominal movement if diaphragmatic weakness or fatigue is present.[56]

Diagnostic Studies

Because the signs and symptoms of acute respiratory failure are nonspecific and insensitive, the physician must request an ABG analysis to determine the exact level of PaO_2, $PaCO_2$, and blood pH in cases of suspected acute respiratory failure. Only determination of the blood gases and pH can confirm the diagnosis.[60] Other diagnostic tests necessary to determine the etiology of acute hypoxemic respiratory failure include chest radiography, sputum examination, pulmonary function testing, angiography, ventilation–perfusion scanning, CT, toxicology screen, complete blood count, serum electrolytes, cytology, urinalysis, bronchogram, bronchoscopy, electrocardiography, echocardiography, and thoracentesis.[60] See Table 26-14 for more details about the use of these diagnostic tests in acute respiratory failure.

Management

Treatment of acute respiratory failure warrants immediate intervention to correct or compensate for the gas exchange abnormality and identify the cause.[60] Therapy is directed toward correcting the cause and alleviating the hypoxia and hypercapnia.[60] Although the recommended therapeutic intervention may vary according to the specific disease's pathological process, general management principles are applicable to every patient with acute respiratory failure. See Table 26-15 for specific therapies for managing common causes of acute respiratory failure.[60]

If alveolar ventilation is inadequate to maintain PaO_2 or $PaCO_2$ levels related to respiratory or neurological failure, endotracheal intubation and mechanical ventilation may be lifesaving.[58,60] The initial assessment and the decision to initiate mechanical ventilation should be performed rapidly to minimize the life-threatening complications associated with extended hypoxemia (eg, cardiac dysrhythmias, anoxic encephalopathy).[60] Controlled oxygen therapy and mechanical ventilation are used to increase PaO_2 by increasing FiO_2 and to normalize pH by increasing minute ventilation.[60] See Chapter 25 for further information on airway management and care of the patient on a ventilator.

Patients with acute hypoxemic respiratory failure should receive immediate treatment with rapidly increased FiO_2 and continuous pulse oximetry monitoring until an SaO_2 of 90% or higher is obtained.[60] Correction of hypoxemia in the acute setting takes precedence over possible attenuation of hypoxic respiratory drive.[60] Therefore, once hypoxemia is reversed, oxygen is titrated to the minimum level necessary for correction of hypoxemia and prevention of significant carbon dioxide retention.[60]

Patients with acute hypercapnic respiratory failure should be immediately assessed for either an impaired central respiratory drive associated with sedative or narcotic therapy or for underlying bronchospasm secondary to an asthma exacerbation or COPD.[60] Reversal agents (opiate antagonists, eg, naloxone) are used in the case of impaired central respiratory drive, and inhaled bronchodilators and systemic corticosteroids are used in the case of underlying bronchospasm.[60]

Table 26-15 Management of Respiratory Failure

Management Principle	Therapeutic Intervention
Establishment and maintenance of an adequate airway	• Use oropharyngeal or nasopharyngeal tubes for upper airway obstruction during transient loss of consciousness. • Tracheal intubation may be necessary to prevent aspiration, maintain airway patency, and provide effective suctioning. • Strictly adhere to adequate tracheobronchial toilet (ie, deep breathing, coughing, tracheobronchial suctioning).
Oxygenation	• Increase the inspired oxygen (FiO_2) concentration by administration of oxygen via a Venturi mask or nasal cannula. • Improve cardiac output, correct anemia, and reduce metabolic rates (fever) to improve tissue oxygenation. • Consider continuous positive airway pressure or expiratory positive airway pressure via a nasal or facial mask for alert and cooperative patients. • Mechanical ventilatory support may be needed in more severe cases with refractory and progressive hypoxemia.
Correction of acid–base disturbance	• Correct pH disturbances: in acute hypercapnia with acidosis, improve alveolar ventilation by providing mechanical ventilatory support, establishing and maintaining an adequate airway, treating bronchospasm, and controlling heart failure, fever, and sepsis. • Consider bicarbonate administration in acute respiratory acidosis or metabolic acidosis.
Restoration of fluid and electrolyte balance	• Prevent excessive intravenous fluid administration and, conversely, poor fluid intake. • Monitor fluid intake and output closely. • Perform daily body weight measurement. • Prevent and treat promptly hypokalemia and hypophosphatemia.
Optimization of cardiac function	• Maintain adequate cardiac output. • Consider use of pulmonary artery catheter for accurate hemodynamic monitoring.
Identification and treatment of underlying correctable conditions and precipitating causes	• Prevent or treat respiratory tract infections (viral, bacterial, or fungal). • Prevent potential airway obstruction by maintenance of proper tracheobronchial hygiene; recognize increased tracheobronchial secretions, changes in their characteristics, or difficulty in their elimination due to various factors. • Identify and treat heart failure appropriately. • Recognize and treat bronchospasm with bronchodilators and corticosteroids. • Assess for organic or metabolic disorder affecting the central nervous system or neuromuscular function. • Assess tolerance to sedative, hypnotic, and narcotic drugs in patients with chronic ventilatory insufficiency. In case of a narcotic drug overdose, a proper antidote may be administered. • Avoid indiscriminate use of oxygen; it may potentiate carbon dioxide retention or result in carbon dioxide narcosis. • Remove air or fluid in the pleural cavity. • Prevent and treat abdominal distention by insertion of a nasogastric tube. • For trauma and surgical patients, assess limitation of the thoracic wall movement, ineffective cough, immobility, and lack of deep breathing. • Control fever and other causes of increased metabolism. • Assess diaphragmatic fatigue; if present, mechanical ventilatory support is indicated to rest these muscles and restore their contractility. • Promptly identify and adequately treat hypophosphatemia, hypokalemia, and hypocalcemia.
Prevention and early detection of potential complications	• Most of these complications occur in mechanically ventilated patients (see Chapter 25).
Nutritional support	• Enteral alimentation is preferred over parenteral feeding because bowel wall integrity is maintained. • Recommend high-lipid formulas over high carbohydrates to limit carbon dioxide production.
Periodic assessment of the course, progress, and response to therapy	• Perform frequent ABG measurements. • Monitor arterial oxygen saturation by pulse oximetry.
Determination of a need for mechanical ventilatory support	• Continuously assess the patient's respiratory status and need for ventilator support (see Chapter 25).

From Farzan S: Respiratory failure. In Farzan S (ed): A Concise Handbook of Respiratory Diseases, 4th ed. Stamford, CT, Appleton & Lange, 1997, pp 371–386, with permission.

▲ Clinical Applicability Challenges

CASE STUDY

A 42-year-old Caucasian female, Ms. R., presents to the emergency room 4 days after being knocked down by a wave at the beach. When she was knocked down, she did not lose consciousness. However, she did develop some back pain as well as some dyspnea on exertion. These symptoms did not worsen over the past 4 days nor did they improve. For this reason, she presented to the emergency room.

Upon further discussion with the patient, we learn that Ms. R. had a pneumothorax in 2008 when she had a very severe pneumonia. Her past medical history consists of hypertension, allergy-related asthma, and recurrent right-sided pneumothoraces. In terms of her medication, she takes hyzaar/hydrochlorothiazide. She is allergic to ACE inhibitors that cause facial numbness.

Her social history consists of no alcohol or illicit drug use. She did smoke a pack of cigarettes a day from the age of 17 to age of 32; therefore, she smoked for 15 years. She has the following vital signs: temperature of 99.4°F, heart rate of 110, respiratory rate of 30 breaths/min, blood pressure of 164/84 mm Hg, height of 168 cm, weight of 88.7 kg, and pulse oximetry of 88% on room air. Her physical examination findings are benign with the exception of her respiratory examination. She has some increased work of breathing. Her breath sounds are clear upon auscultation on the left lung fields. On the right lung fields, her breath sounds are clear in the right lower and middle lobes and somewhat diminished in her right upper lobe. She also complains of pleuritic pain with deep inspiration.

A posteroanterior (PA) and lateral chest x-ray was performed that revealed no pulmonary infiltrates but did show slight white-out in the right upper lung field on the PA view. In the lateral view, the x-ray film showed an air level in the upper lung field.

1. What additional diagnostic tests would be useful?
2. What medical treatment options are there for Ms. R.?
3. Is there a role for surgical treatment? If so, what are the options?

References

1. Heron M: National Vital Statistics Reports. 58(14):1–100, 2010
2. Restrepo MI, Anzueto A: Severe community acquired pneumonia. Infect Dis Clin North Am 23(3), 2009
3. Miskovich-Riddle L, Keresztes P: CAP management guidelines. Nurse Pract 31(1):43–53, 2006
4. Nilsson KR, Piccini J: The Osler Medical Handbook, 2nd ed. St. Louis, MO: MD Consult, 2006
5. Mandell LA, Wenderink RG, et al: Infectious Disease Society of America/American Thoracic Society consensus guidelines on the management of community acquired pneumonia in adults. Clin Infect Dis 44:S27–S72, 2007
6. Halm EA, Teirstein AS: Management of community-acquired pneumonia. N Engl J Med 347(25):2039–2045, 2002
7. American Thoracic Society: Guidelines for the management of adults with community-acquired pneumonia. Am J Respir Crit Care Med 163(7):1730–1745, 2001
8. Leeper KV, Moss M: Bacterial pneumonia. In Hanley ME, Walsh CH (eds): Current Diagnosis and Treatment in Pulmonary Medicine. New York, NY: McGraw-Hill, 2003, pp 361–371
9. American Thoracic Society: Guidelines for the management of adults with hospital-acquired, ventilator-associated, and healthcare-associated pneumonia. Am J Respir Crit Care Med 171(4):388–416, 2005
10. Lutfiyya MN, Henley E, Chang LF, et al: Diagnosis and treatment of community-acquired pneumonia. Am Fam Physician 73(3):442–450, 2006
11. Tablan OC, Anderson LJ, Besser R, et al: Guidelines for preventing health-care–associated pneumonia, 2003. MMWR Morb Mortal Wkly Rep 53(RR03):1–36, 2004
12. Weinberger SE, Cockrill BA, et al: Principles of Pulmonary Medicine, 5th ed. Philadelphia, PA: Elsevier Saunders, 2008
13. Ramsdell J, Narsavage GL, Fink JB; for the American College of Chest Physicians' Home Care Network Working Group: Management of community-acquired pneumonia in the home: An American College of Chest Physicians clinical position statement. Chest 127(5):1752–1763, 2005
14. McKay CA, Speers M: Watch for AACN practice alerts. AACN News 21(2):1–4, 2004. Available at: http://www.aacn.org
15. Centers for Disease Control and Prevention: Frequently Asked Questions about SARS. Washington, DC: Department of Health and Human Services, 2004, pp 1–4. Available at: http://www.cdc.gov/ncidod/sars/sars-faq.pdf
16. Centers for Disease Control and Prevention: Update: Outbreak of severe acute respiratory syndrome—worldwide, 2003. MMWR Morb Mortal Wkly Rep 52(12):241–248, 2003
17. Lee N, Hui D, Wu A, et al: A major outbreak of severe acute respiratory syndrome in Hong Kong. N Engl J Med 348(20):1986–1994, 2003
18. Centers for Disease Control and Prevention: Update: Outbreak of severe acute respiratory syndrome—worldwide, 2003. MMWR Morb Mortal Wkly Rep 52(13):269–272, 2003
19. Drosten C, Gunther S, Preiser W, et al: Identification of a novel coronavirus in patients with severe acute respiratory syndrome. N Engl J Med 348(20):1967–1976, 2003
20. World Health Organization: Preliminary clinical description of severe acute respiratory syndrome: Epidemic and pandemic alert and response, 2006. Available at: http://www.who.int/csr/sars/clinical/en
21. Booth CM, Stewart TE: Severe acute respiratory syndrome and critical care medicine: The Toronto experience. Crit Care Med 33(1):S53–S60, 2003
22. El-Masri MM, Williamson KM, Fox-Wasylyshyn SM: Severe acute respiratory syndrome: Another challenge for critical care nurses. AACN Clin Issues 15(1):150–159, 2004
23. Centers for Disease Control and Prevention: Clinical guidance on the identification and evaluation of possible SARS-CoV disease among persons presenting with community-acquired illness, Version 2, Supplement I: Infection control in healthcare, home, and community settings. Washington, DC: Department of Health and Human Services Centers for Disease Control and Prevention, 2004, pp 1–28. Available at: http://www.cdc.gov/sars
24. Juergens RA, Spira AI, Brahmer JR: Effusions. In: Abeloff MD, Armitage JO, Niederhuber JE, et al (eds). Abeloff's Clinical Oncology. 4th ed. Philadelphia, PA: Churchill Livingstone Elsevier, 2008, pp 925–944
25. Broaddus VC, Light RW: Pleural effusion. In Mason RJ, et al (eds): Murray & Nadel's Textbook of Respiratory Care, 5th ed. Philadelphia, PA: Elsevier Saunders, 2010, pp 1719–1764

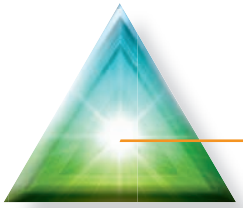
26. Haydel M, DeBlieux PMC: Pleural effusions. In Wolfson AB, Hendey GW, Ling LJ, et al (eds): Harwood-Nuss' Clinical Practice of Emergency Medicine, 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, pp 425–431
27. Porcel JM, Light RW: Diagnostic approach to pleural effusion in adults. Am Fam Physician 73(7):1211–1220, 2006
28. Blok BK: Thoracentesis. In: Roberts JR, Hedges JR (eds): Clinical Procedures in Emergency Medicine, 4th ed. Philadelphia, PA: Saunders Elsevier, 2004, pp 171–186
29. Light RW, Lee YCG: Pneumothorax, chylothorax, hemothorax, and fibrothorax. In Mason RJ, Broaddus VC, et al (eds): Murray and Nadel's Textbook of Respiratory Medicine, 5th ed. Philadelphia, PA: Elsevier Saunders, 2010, pp 1764–1791
30. De Hoyos A, Fry WA: Pneumothothorax. In Shield TW, LoCicero J, Reed CE, et al (eds): General Thoracic Surgery, 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, pp 739–762
31. Chesnutt MS, Prendergast TJ: Lung. In Tierney L, McPhee SJ, Papadakis MA (eds): Current Medical Diagnosis and Medical Treatment, 46th ed. New York, NY: McGraw-Hill, 2007, pp 222–315
32. Toro JR, Pautler SE, Stewart L, et al. Lung cysts, spontaneous pneumothorax, and genetic associations in 89 families with Birt-Hogg-Dubé syndrome. Am J Respir Crit Care Med 175(10):1044–1053, 2007
33. MacDuff A, Arnold A, Harvey J: Management of spontaneous pneumothorax: British Thoracic Society Pleural Disease guideline 2010. Thorax 65(Suppl 2):ii18–ii31, 2010
34. Noppen M, De Keukeleire T: Pneumothorax. Respiration 76(2):121–127, 2008
35. McGee DC, Could MK: Preventing complication of central venous catheterization. N Engl J Med 348:1123–1133, 2003
36. Des Jardin T, Burton GG: Pneumothorax in Clinical Manifestations and Assessments of Respiratory Diseases, 6th ed. Philadelphia, PA: Elsevier Mosby, 2011, pp 303–312
37. Robinson GV: Pulmonary embolism in hospital practice. BMJ 332(7534):156–160, 2006
38. West JB: Vascular disease. In Pulmonary Pathophysiology: The Essentials, 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2007, pp 99–119
39. Blann AD, Lip GYH: Venous thromboembolism. BMJ 332(7535): 215–219, 2006
40. Kearon C, Kahn SR, Agnelli G, et al: Antithrombotic therapy for venous thromboembolic disease: ACCP evidence-based clinical practice guidelines. Chest 133(6S):454S–545S, 2008
41. Geerts WH, Bergqvist D, Pineo GF, et al: Prevention of venous thromboembolism: ACCP evidence-based clinical practice guidelines. Chest 133(6S):381S–453S, 2008
42. Global Initiative for Chronic Obstructive Lung Disease. Executive Summary: Global strategy for the diagnosis, management and prevention of COPD: updated 2009. Available at: http://www.goldcopd.com/Guidelineitem.asp?l1=2&l2=1&intId=2180. Accessed October 2010.
43. Global Strategy for Diagnosis, Management, and Prevention of COPD. Available at http://www.goldcopd.com/download.asp?intId=554. Accessed October 2010
44. Anthonisen N: Chronic obstructive pulmonary disease. In Goldman (eds): Cecil Medicine, 23rd ed. Philadelphia, PA: Saunders Elsevier, 2007, pp 619–926
45. Hanaia NA, Sharafkhaneh A: Update on pharmacologic therapy for chronic obstructive pulmonary disease. Clin Chest Med 28(3):589–607, 2007
46. O'Donnell DE, Fluge T, et al: Effects of tiotropium on lung hyperinflation, dyspnea and exercise tolerance in COPD. Eur Respir J 23:832–840, 2004
47. National Emphysema Treatment Trial Research Group: A randomized trial comparing lung-volume-reduction surgery with medical therapy for severe emphysema. N Engl J Med 348(21):2059–2073, 2003
48. Slinger PD, Campos JH. Anesthesia for thoracic surgery. In Miller, et al (eds): Miller's Anesthesia, 7th ed. Philadelphia, PA: Churchill Livingstone, Elsevier, 2010, pp 1819–1888
49. Kaminshy DA: Asthma. In Hanley ME, Walsh CH (eds): Current Diagnosis and Treatment in Pulmonary Medicine. New York, NY: McGraw-Hill, 2004, pp 67–81
50. John J, Idell S: Managing severe exacerbations of asthma. Emerg Med 38(4):20–32, 2006
51. National Heart, Lung, and Blood Institute: Expert Panel Report 3: Guidelines for the Diagnosis and Management of Asthma. 2007, pp 1–416. Available at: http://www.nhlbi.nih.gov/guidelines/asthma/asthgdln.pdf. Accessed December 2010
52. Global Initiatives for Asthma (GINA) Guidelines: Global strategy for Asthma Management and Prevention. Available at: www.ginasthma.org. Accessed December 2010
53. Markou NK, Myrianthefs PM, Baltopoulos GJ, et al: Respiratory failure: An overview. Crit Care Nurs Q 27(4):353–379, 2004
54. Van Hoozen B, Albertson TE: Acute respiratory failure. In Burton GG, Hodgkin JE, Ward JJ (eds): Respiratory Care: A Guide to Clinical Practice, 4th ed. Philadelphia, PA: JB Lippincott, 1997, pp 1107–1132
55. Vasileyev S, Schaap RN, Mortensen JD: Hospital survival rates of patients with acute respiratory failure in modern respiratory intensive care units. Chest 107(4):1083–1088, 1995
56. Reardon C, et al: Acute respiratory failure. In Crapo JD, Glassroth J, Karlinsky J, et al (eds): Baum's Textbook of Pulmonary Diseases, 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2004, pp 1049–1071
57. Zambon M, Vincent JL: Mortality Rates for patients with acute lung injury/ARDS have decreased over time. Chest 133(5):1120–1127, 2008
58. Hudson LD, Slutsky AS: Acute Respiratory Failure. In Goldman (ed): Cecil Medicine, 23rd ed. Philadelphia, PA: Saunders Elsevier, 2007, pp 723–733
59. Del Sorbo L, Martin EL, Ranieri VM: Hypoxemic respiratory failure. In Mason RJ (eds): Murray and Nadel's Textbook of Respiratory Medicine, 5th ed. Philadelphia, PA: Saunders Elsevier, 2010, pp 2130–2138
60. D'Alessio F, Nilsson Jr KR, Wittine L, et al: Acute respiratory failure. In Piccini JP, Nilsson KR (eds): The Osler Medical Handbook, 2nd ed. Philadelphia, PA: Saunders Elsevier, 2006, Chapter 18
61. Krider SJ: Interview and respiratory history. In Wilkins RL, Sheldon RL, Kidder SJ (eds): Clinical Assessment in Respiratory Care, 4th ed. St. Louis, MO: Mosby, 2000, pp 11–50

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!



Acute Lung Injury and Acute Respiratory Distress Syndrome

Paul A. Thurman, Mary van Soeren, and Christina Hurlock-Chorostecki

27

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Compare and contrast the causes, definition, assessment findings, and outcomes between acute lung injury (ALI) and acute respiratory distress syndrome (ARDS).
2. Relate the assessment and diagnostic findings of ARDS to the pathophysiological processes.
3. Describe mechanical ventilation strategies used to prevent ventilator-associated lung injury (VALI).
4. Explain the management of patients with ARDS and rationales for the interventions.
5. Review the use of critical care "bundles" as they relate to the care and treatment of patients with ARDS.
6. Discuss potential complications of ARDS and the related interventions.

Acute respiratory distress syndrome (ARDS) represents a complex clinical syndrome (rather than a single disease process) and carries a high risk for mortality. The severity of the clinical course, the uncertainty of the outcome, and the reliance on the full spectrum of critical care resources for treatment mean that the entire health care team is challenged. Since the 1960s, researchers and clinicians have investigated the nature of the pathological process and explored treatment options with the goal of improving outcome. Through this application of research to practice, we know that some previous strategies have been ineffective, and innovations in mechanical ventilation, sedation, nutrition, and pharmacological intervention remain important research initiatives. A key role for the critical care nurse is early detection and prevention of ARDS. Therefore, with respect to ARDS, it is essential that critical care nurses be knowledgeable about risk factors, assessment tools and protocols, and prevention strategies.

ARDS was first described in 1967 and was termed *adult* (rather than *acute*) respiratory distress syndrome because of a misconception that the syndrome occurred only in adults. Recognition of the prevalence of this syndrome in younger patients led to the current terminology. ARDS is at the extreme end of a continuum of hypoxic acute lung injury (ALI) that results in respiratory failure. In 1994, the American-European Consensus Conference members issued definitions of ALI and ARDS that are widely used by researchers today[1] (Table 27-1).

▲ Etiology, Diagnostic Criteria, and Incidence

The causes of ARDS are many and diverse. The syndrome may be precipitated by direct or indirect pulmonary injury, possibly in previously healthy people who are exposed to an insult (Box 27-1). ARDS is acute in onset, and symptoms typically develop over 4 to 48 hours after the inciting insult, making a cause-and-effect association somewhat difficult. Recently, several related respiratory disorders have been found presenting with clinical signs of ALI. These disorders may ultimately lead to development of ARDS and include severe acute respiratory syndrome (SARS), discussed in Chapter 26, and transfusion-related acute lung injury (TRALI).

TRALI is the leading cause of transfusion-related mortality in the United States.[2] It is theorized that an interaction occurs between the recipient's blood and the donor's, among the bioactive compounds produced during blood storage, or a combination of both.[3] Clinical presentation is the sudden onset of respiratory distress within 1 to 2 hours after a transfusion of red blood cells or thawed plasma.[3] Peak airway pressures are elevated, airway secretions may be frothy, and the chest radiograph shows patchy infiltrates. Management of the patient is supportive and involves the same principles of mechanical ventilation used in ARDS and avoidance of aggressive diuresis. The blood bank should be notified of a TRALI case, and the patient should not receive further blood products from that donor.

Diagnostic criteria for ARDS have been difficult to define because ARDS closely resembles other conditions. Diagnostic testing is used to "rule out" these conditions, yet the final diagnosis of ARDS is largely based on clinical presentation. No one test, including radiographic evidence, a plasma brain natriuretic peptide less than 500 pg/mL, or a pulmonary artery occlusion pressure (PAOP, formerly

Table 27-1 Comparison of Acute Lung Injury (ALI) and Acute Respiratory Distress Syndrome (ARDS)

Criterion	ALI	ARDS
PaO_2:FiO_2 ratio,* regardless of PEEP level	<300	<200
Chest x-ray	Bilateral infiltrates	Bilateral infiltrates
Pulmonary artery occlusion pressure	<18 mm Hg or no indication of left atrial hypertension	<18 mm Hg or no indication of left atrial hypertension

*Ratio of arterial oxygen to inspired oxygen.

PEEP, positive end-expiratory pressure.

Adapted from Bernard GR, Artigas A, Brigham KL, et al: The American-European Consensus conference on ARDS: Definitions, mechanisms, relevant outcomes, and clinical trials co-ordination. Am J Respir Crit Care Med 149:818–824, 1994.

known as pulmonary artery wedge pressure [PAWP]) less than 18 cm H_2O, is truly indicative of ARDS. However, an early feature of ARDS is diffuse alveolar damage (DAD), and recent research findings have shown promise in diagnosing DAD with cytological examination of bronchoalveolar fluid.[4]

Approximately 190,600 cases of ARDS occur each year in the United States, of which 74,500 result in death.[5] The patients most at risk for development of ARDS are older than 65 years, with a severe acute illness on presentation, such as sepsis or a preexisting chronic disorder. Although sepsis is the most common cause of ARDS, any person with one of the potential precipitating causes of ARDS is susceptible, and nurses need to be vigilant for early warning signs (Box 27-1). Most patients with ARDS require a period of mechanical ventilation support for days to weeks.

▲ Pathophysiology

In 1967, Ashbaugh and others described ARDS in case reports of 12 patients presenting with acute tachypnea, decreased lung compliance, diffuse pulmonary infiltrates on chest radiograph, and hypoxemia.[1] Later researchers used histological examination of lungs of patients with ARDS to show lung fibrosis that was unlike other diseases. This led to new understanding that the pathological process was not limited to the lung endothelium but was a result of alterations of lung epithelium and vascular tissue as well as the development of hyaline membranes. Pathological changes in lung vascular tissue, increased lung edema, and impaired gas exchange are hallmarks of the pathophysiology. The pathological pulmonary alterations of ARDS are directly related to a cascade of events resulting from release of cellular and biochemical mediators. The activation, interactions, and multisystem actions of biological mediators are extremely complex.

Pathological Changes in ARDS

Mediators released as a result of either direct or indirect injury can precipitate acute respiratory distress syndrome (ARDS), including lipopolysaccharide in gram-negative bacterial sepsis. There is a relationship between clinical presentation (severe acute hypoxemia resistant to improvement with supplemental oxygen, tachypnea, and dyspnea),

BOX 27-1 Causes and Predisposing Conditions for Acute Respiratory Distress Syndrome (ARDS)

Genetic predisposition

Direct injury
- Aspiration (gastric fluids, near-drowning)
- Infectious pneumonia
- Lung contusions with trauma
- Toxic inhalation
- Upper airway obstruction (relieved)
- Severe acute respiratory syndrome (SARS) coronavirus
- Neurogenic pulmonary edema
- Acute eosinophilic pneumonia*
- Bronchiolitis obliterans with organizing pneumonia (BOOP)*
- Miliary tuberculosis*

Indirect pulmonary injury
- Sepsis
- Burns
- Trauma
- Blood transfusion (TRALI)
- Lung or bone marrow transplantation
- Drug or alcohol overdose
- Drug reaction
- Cardiopulmonary bypass
- Acute pancreatitis
- Multiple fractures
- Venous air embolism
- Amniotic fluid embolism
- Pancreatitis

Systemic Inflammatory Response Syndrome (SIRS) Criteria

SIRS is manifested by two or more of the following:
- Temperature greater than 100.4°F (38°C) or less than 96.8°F (36°C)
- Heart rate greater than 90 beats/min
- Respiratory rate greater than 20 breaths/min or an arterial carbon dioxide tension ($PaCO_2$) less than 32 mm Hg
- White blood cell count greater than 12,000 cells/mm^3 or less than 4,000 cells/mm^3 OR more than 10% immature (band) forms.

*Specific treatment required.
TRALI, transfusion-related acute lung injury.

Table 27-2 Examples of Pathological Responses to Biological Mediators

Response	Biological Mediators
Persistent inflammatory response	Cytokines: interleukins (IL-1, IL-6), interferon-γ (INF-γ), tumor necrosis factor-α (TNF-α), complement, thromboxane
Endothelial membrane disruption	Complement, thromboxane, kinins, TNF-α, toxic oxygen metabolites, leukotrienes, prostaglandins (PGE_1 and PGE_2)
Selective vasoconstriction	Thromboxane, TNF-α, platelet-activating factor (PAF), toxic oxygen metabolites
Systemic vasodilation	Complement, prostaglandins, TNF-α, IL-1, IL-6
Myocardial depression	Complement, leukotrienes, TNF-α, myocardial depressant factor
Bronchoconstriction	Complement, thromboxane, leukotrienes, PAF

mediator release (interleukins [ILs], tumor necrosis factor-α [TNF-α], and platelet-activating factor [PAF]), and pathological changes (microvascular permeability, pulmonary hypertension, and pulmonary endothelial damage). Some primary mediators responsible for lung damage in ARDS and their major actions as they relate to ARDS are listed in Table 27-2.[6,7]

Adequate pulmonary gas exchange depends on open, air-filled alveoli, intact alveolar–capillary membranes, and normal blood flow through the pulmonary vasculature. The pathogenesis of ARDS is illustrated in Figure 27-1. Diffuse alveolar–capillary membrane damage occurs and increases membrane permeability, thus allowing fluids to move from the vascular space into the interstitial and alveolar space. Air spaces fill with bloody proteinaceous fluid and debris from degenerating cells, causing interstitial and alveolar edema. As a result, oxygenation is impaired. Inflammatory mediators cause vasoconstriction of the pulmonary vascular bed. Pulmonary hypertension and reduced blood flow to portions of the lung result. Because of the reduced blood flow and decreased hemoglobin in capillaries, there is a decrease in oxygen available for diffusion and transport, further impairing oxygenation.

The pathological changes affect pulmonary blood vessels, gas exchange, and lung and bronchial mechanics (Fig. 27-2). Ventilation is impaired because of a decrease in lung compliance and an increase in airway resistance. Lung compliance is reduced as a result of the stiffness of fluid-filled, nonaerated lung. The presence of these fluid-filled alveoli alongside collapsed alveoli gives the chest radiograph the classic "patchy" or "ground glass" appearance. Surfactant, a substance that normally decreases the surface tension of alveoli, is lost, resulting in alveolar collapse. Mediator-induced bronchoconstriction causes airway narrowing and increased airway resistance, restricting the flow of air into the lungs.

Systemic Inflammatory Response Syndrome

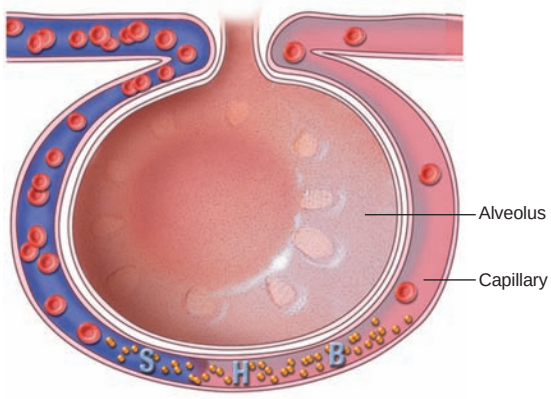
Systemic inflammatory response syndrome (SIRS) describes the inflammatory response occurring throughout the body as a result of some systemic insult. Most patients with ARDS manifest the symptoms that define SIRS (see Box 27-1), and the respiratory system may be the earliest and most common organ system to be involved in the systemic response. Thus, an understanding of the pathophysiology of SIRS and knowledge of the interventions used for SIRS are important in relation to ARDS. Often, patients with SIRS develop multisystem organ dysfunction (MODS), primarily in the liver and kidney. As endothelial damage progresses and tissue hypoxia ensues from the severely impaired gas exchange, the inflammatory response is perpetuated, and the SIRS cascade intensifies (up-regulates) with the release of more mediators. ARDS and MODS are therefore part of a vicious cycle and the continuum of SIRS. Determination of the triggers for SIRS and ARDS that are present in some individuals but not others and investigations of how to stop the cascade pathways are the subjects of ongoing research. For a more detailed discussion of SIRS and MODS, see Chapter 54.

Stages of ARDS

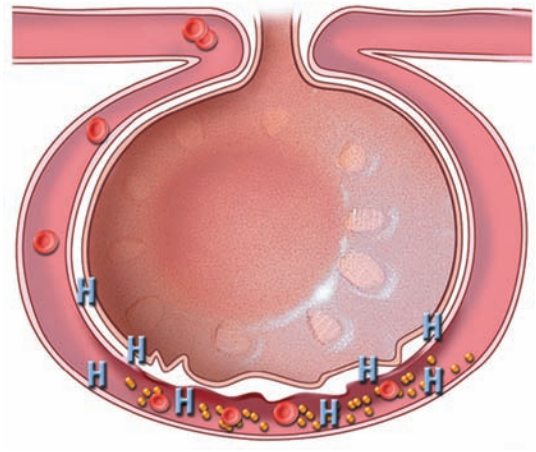
There is progression in the pathological changes associated with ARDS, starting with increasing pulmonary edema in the early stages and progressing to inflammation, fibrosis, and impaired healing in the later stages (Table 27-3, p. 595). Recognizing the dynamic nature of the morphological changes involved with ARDS enables the nurse to understand the changes in physical assessment, mechanical ventilation strategies, treatment, and management that occur throughout the patient's critical care stay.

In stage 1, diagnosis is difficult because the signs of impending ARDS are subtle. Clinically, the patient exhibits increased dyspnea and tachypnea, but there are few radiographic changes. At this point, neutrophils are sequestering; however, there is no evidence of cellular damage. Within 24 hours (a critical time for early treatment), the symptoms of respiratory distress increase in severity, with cyanosis, coarse bilateral crackles on auscultation, and radiographic changes consistent with patchy infiltrates. A dry cough or chest pain may be present. It is at this point (stage 2) that the mediator-induced disruption of the vascular bed results in increased interstitial and alveolar edema. The endothelial and epithelial beds are increasingly permeable to proteins. This is referred to as the "exudative" stage. The hypoxemia is resistant to supplemental oxygen administration, and mechanical ventilation will most likely be commenced in response to a worsening ratio of arterial oxygen to fraction of inspired oxygen (PaO_2:FiO_2 ratio).

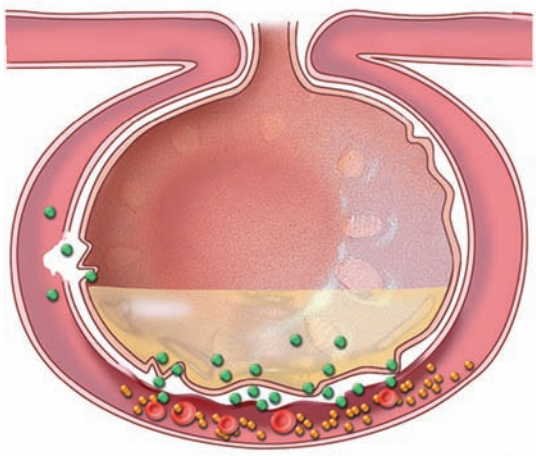
Stage 3, the "proliferative" stage, develops from the 2nd to the 10th day after injury. Evidence of SIRS is now present, with hemodynamic instability, generalized edema, possible



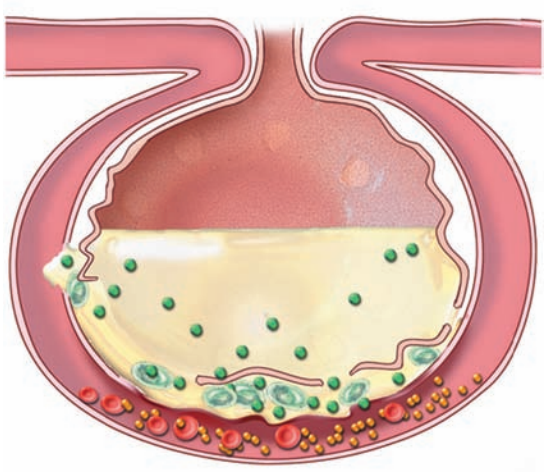
Phase 1. Injury reduces normal blood flow to the lungs. Platelets aggregate and release histamine (H), serotonin (S), and bradykinin (B).



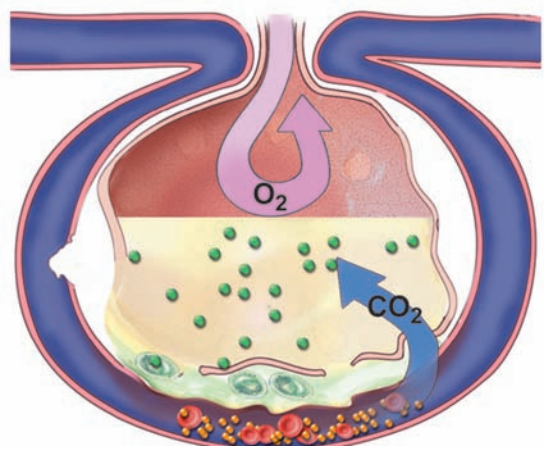
Phase 2. Those substances, especially histamine, inflame and damage the alveolar–capillary membrane, increasing capillary permeability. Fluids then shift into the interstitial space.



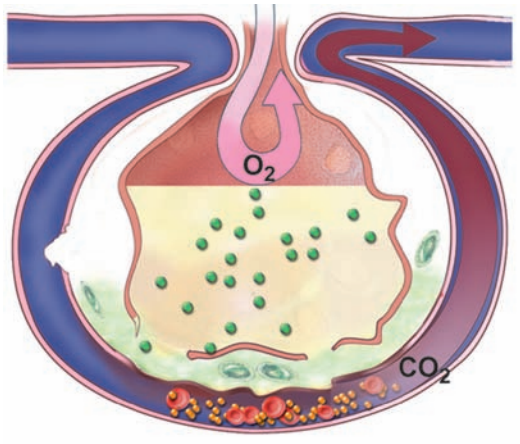
Phase 3. As capillary permeability increases, proteins and fluids leak out, increasing interstitial osmotic pressure and causing pulmonary edema.



Phase 4. Decreased blood flow and fluids in the alveoli damage surfactant and impair the cell's ability to produce more. As a result, alveoli collapse, impeding gas exchange and decreasing lung compliance.



Phase 5. Sufficient oxygen cannot cross the alveolar–capillary membrane, but carbon dioxide (CO_2) can and is lost with every exhalation. Oxygen (O_2) and CO_2 levels decrease in the blood.



Phase 6. Pulmonary edema worsens, inflammation leads to fibrosis, and gas exchange is further impeded.

FIGURE 27-1 ▲ Pathogenesis of acute respiratory distress syndrome (ARDS). Changes in lung epithelium and vascular endothelium result in fluid and protein movement, changes in lung compliance, and disruption of the alveoli with accompanying hypoxia. (From Anatomical Chart Company: Atlas of Pathophysiology, 3rd ed. Ambler, PA: Lippincott Williams & Wilkins, 2010, pp 81, 83.)

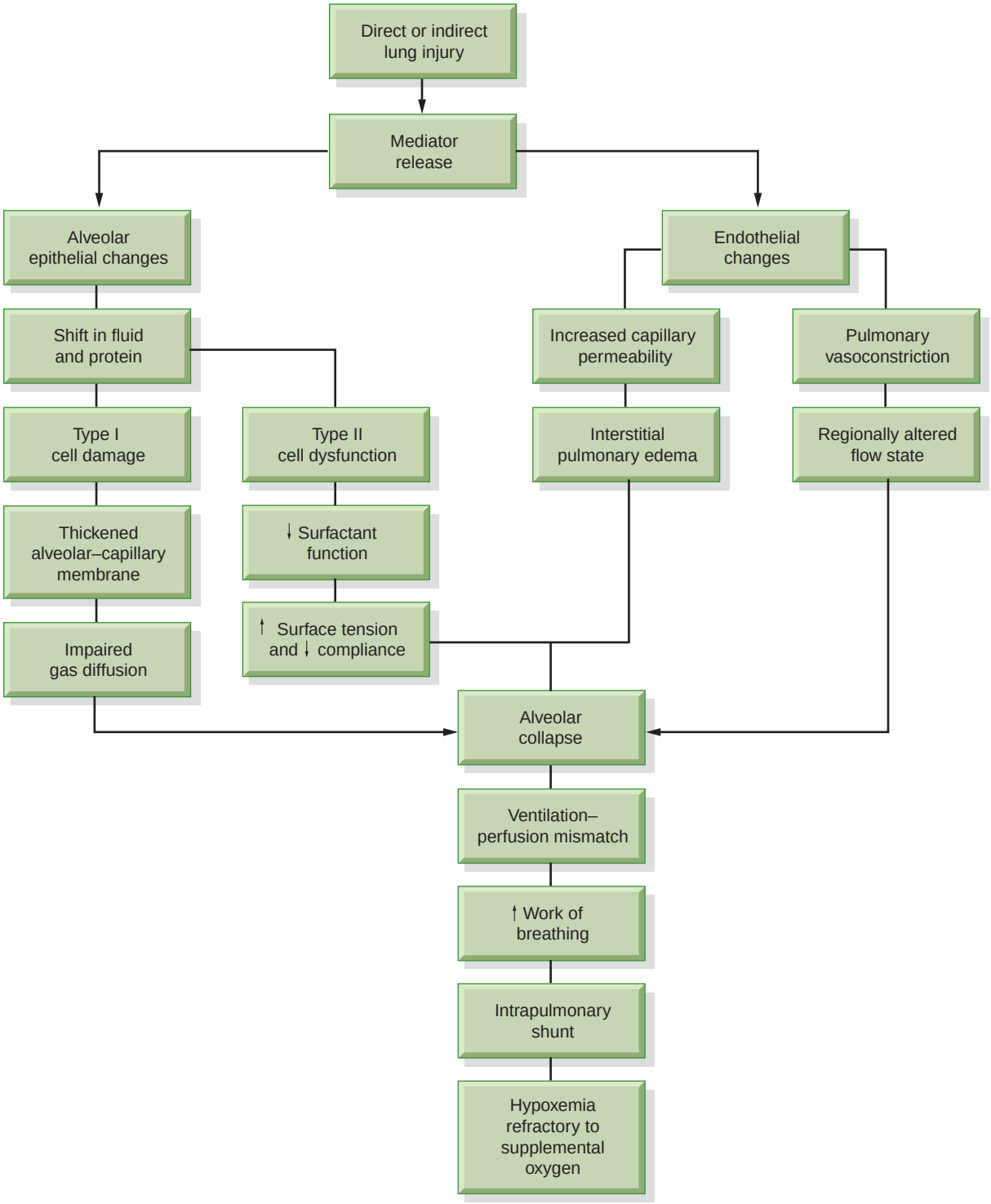


FIGURE 27-2 ▲ Pathophysiological cascade is initiated by injury resulting in mediator release. The multiple effects result in changes to the alveoli, vascular tissue, and bronchi. The ultimate effect is ventilation–perfusion mismatching and refractory hypoxemia.

onset of nosocomial infections, increased hypoxemia, and lung involvement. Air bronchograms may be evident on chest radiography as well as decreased lung volumes and diffuse interstitial markings.

Stage 4, the "fibrotic" stage, develops after 10 days and is typified by few additional radiographic changes. There is increasing multiorgan involvement, SIRS, and increases in the arterial carbon dioxide tension ($PaCO_2$) as progressive lung fibrosis and emphysematous changes result in increased dead space. Fibrotic lung changes result in ventilation management difficulties, with increased airway pressure and development of pneumothoraces.

Table 27-3 Clinical Presentation and Pathological Changes During Acute Respiratory Distress Syndrome

Radiographic Change	Clinical Presentation	Pathological Change
Stage 1 (first 12 h): Normal chest x-ray	Dyspnea, tachypnea	Neutrophil sequestration, no evidence of cellular damage
Stage 2–Exudative (24 h): Patchy alveolar infiltrate, primarily in dependent lung areas; normal heart size	Dyspnea, tachypnea, cyanosis, tachycardia, coarse crackles, hypoxemia	Neutrophil infiltration, vascular congestion, fibrin strands, increased interstitial and alveolar edema
Stage 3–Proliferative (2–10 d): Diffuse alveolar infiltrates, possibly air bronchograms, decreased lung volume, normal heart size	Hyperdynamic hemodynamic parameters, SIRS presentation	Type II cell proliferation, microemboli formation, increased interstitial and alveolar inflammatory exudate, early deposition of collagen
Stage 4–Fibrotic (more than 10 d): Persistent infiltrates, new pneumonic infiltrates, recurrent pneumothorax	Multiple organ involvement, difficulty maintaining adequate oxygenation, sepsis, pneumonia	Type II cell hyperplasia, thickening of interstitial wall with fibrosis, macrophages, fibroblasts, remodeling of arterioles, cyst formation

Adapted from van Soeren MH, Diehl-Jones WL, Maykut RJ, et al: Pathophysiology and implications for treatment of acute respiratory distress syndrome. AACN Clin Issues 11(2):179–197, 2000.

▲ Assessment

History

The need for a complete and accurate history for the patient presenting with ARDS is important. The history may provide information that allows for removal of the precipitating cause, interrupting the ensuing mediator response. However, a thorough history may be difficult to obtain because of the critical presentation of the patient and problems associating a remote event with the ALI. Because the outcome is uncertain and often involves a long critical care admission, the health care team plays a large role in providing support to both the patient and the family. Developing a relationship early (eg, by taking the time to obtain a thorough history) may assist with care throughout the course of admission.

All health care team members contribute information to the history. Information about past relevant incidents (medications, blood transfusions, radiographic contrast agents), the use of medical and complementary therapies, and social factors may be helpful for the person's care. Items of importance include assessment of risk factors for the development of ARDS (see Box 27-1), a social history to assess risk behaviors (eg, human immunodeficiency virus status, smoking, substance abuse), medications (including over-the-counter medications), and complementary therapies (all exogenous substances, including inhalations). This information is obtained in addition to the history of the present illness and presenting signs and symptoms.

Physical Examination

Acute respiratory failure initially may present within a few hours to several days, depending on the initial insult, and does not always progress to ARDS. Monitoring patients who meet the SIRS criteria (see Box 27-1, p. 591) may aid identification of those who are at risk for development of ARDS. No unexplained change in respiratory rate should be taken lightly because there are few reliable early indicators of impending ALI or ARDS. Vital signs throughout the progression of ARDS vary, but the general trend is hypotension, tachycardia, and hyperthermia or hypothermia. Respiration, initially rapid and labored, varies once mechanical ventilation is instituted.

Early signs and symptoms of respiratory failure include tachypnea, dyspnea, and tachycardia. Breath sounds often are clear in this phase (Table 27-4). Patients with acute respiratory failure may exhibit neurological changes, such as restlessness and agitation associated with impaired oxygenation and decreased perfusion to the brain. Use of accessory respiratory muscles is evident. The cardiovascular response is tachycardia to improve cardiac output as compensation for poor tissue oxygenation. These attempts to reduce hypoxia represent an adaptive sympathetic nervous system response. In both ALI and ARDS, these attempts to reduce hypoxia are likely to be ineffective because mediators are already circulating and triggering a cascade of systemic responses.

As the pathological changes progress, lung auscultation may reveal crackles secondary to an increase in secretions and narrowed airways; however, the bubbling crackles of cardiogenic pulmonary edema may be minimal. Assessment must be considered in the context of the presenting or initiating disease. For example, pneumonia, one risk factor for ARDS, may confound the ability to diagnose early-stage lung sound changes. The patient may be increasingly restless and confused secondary to hypoxia. Decreases in arterial oxygen saturation (SaO_2) are early signs of impending decompensation.

The ability to compensate decreases with increasing pathological changes to the lung, pulmonary vasculature, and bronchi. Dependent lung fields have decreased breath sounds as fluid accumulates and alveoli collapse. Agitation may give way to lethargy, an ominous sign in which interventions to

Table 27-4 Integrated Assessment of the Patient With Acute Respiratory Distress Syndrome

Stage	Physical Examination	Diagnostic Test Results
Stage 1 (first 12 h)	• Restlessness, dyspnea, tachypnea • Moderate to extensive use of accessory respiratory muscles	• *ABG:* Respiratory alkalosis • *CXR:* No radiographic changes • *Chemistry:* Blood results may vary depending on precipitating cause (eg, elevated white blood cell count, changes in hemoglobin) • *Hemodynamics:* Elevated PAP, normal or low PAOP
Stage 2 (24 h)	• Severe dyspnea, tachypnea, cyanosis, tachycardia • Coarse bilateral crackles • Decreased air entry to dependent lung fields • Increased agitation and restlessness	• *ABG*: Decreased Sa_{O_2} despite supplemental oxygen administration • *CXR:* Patchy bilateral infiltrates • *Chemistry:* Increasing acidosis (metabolic) depending on severity of onset • *Hemodynamics:* Increasingly elevated PAP, normal or low PAOP
Stage 3 (2–10 d)	• Decreased air entry bilaterally • Impaired responsiveness (may be related to sedation necessary to maintain mechanical ventilation) • Decreased gut motility • Generalized edema • Poor skin integrity and breakdown	• *ABG:* Worsening hypoxemia • *CXR:* Air bronchograms, decreased lung volumes • *Chemistry:* Signs of other organ involvement: decreased platelets and hemoglobin, increased white blood cell count, abnormal clotting factors • *Hemodynamics:* Unchanged or becoming increasingly worse
Stage 4 (more than 10 d)	• Symptoms of MODS, including decreased urine output, poor gastric motility, symptoms of impaired coagulation **OR** • Single-system involvement of the respiratory system with gradual improvement over time	• *ABG:* Worsening hypoxemia and hypercapnia • *CXR:* Air bronchograms, pneumothoraces • *Chemistry:* Persistent signs of other organ involvement: decreased platelets and hemoglobin, increased white blood cell count, abnormal clotting factors • *Hemodynamics:* Unchanged or becoming increasingly worse

ABG, arterial blood gas; CXR, chest radiograph; MODS, multisystem organ dysfunction syndrome; PAP, pulmonary artery pressure; PAOP, pulmonary artery occlusion pressure.

support ventilation and oxygenation are required quickly. Other later stages of progression result from tissue hypoxia and include dysrhythmias, chest pain, decreased renal function, and decreased bowel sounds. These are indications of multisystem involvement as highly perfused organ systems respond to decreased oxygen delivery with diminished function.

In the later stages of ARDS, mechanical ventilation support is required. Consolidation of the lungs with fluid reduces breath sounds. Lung compliance decreases, and increasing difficulties maintaining ventilation in the face of increasing resistance ensue. Unexplained changes in ventilation (such as decreased PaO_2 or increased peak inspiratory pressure) cannot be minimized because development of spontaneous pneumothoraces is a frequent complication of ARDS in the later stages. Transmitted sounds, poor air entry throughout all lung fields, and diffuse crackles coupled with ventilation make breath sounds difficult to assess. A further complicating event is myocardial depression, a mediated response. Therefore, despite persistent tachycardia, cardiac output decreases, and hypotension results.

Diagnostic Studies

Throughout the stages of ARDS, the reliance on diagnostic tests is important (see Table 27-4). In the early stages, the need to establish cause may require specific tests, such as blood cultures, bronchoalveolar lavage cultures, and computed tomography (CT) for abscess (eg, abdominal abscesses). In later stages, further vigilance is required to intervene for early management of any nosocomial infections. Ongoing monitoring of routine blood gas values, chemistry, and hematology is performed to ensure stability in metabolic parameters and optimization of existing function. Other laboratory studies are generally nonspecific and may include leukocytosis and lactic acidosis.

Blood Gas Analysis

Deterioration of arterial blood gas (ABG) values, despite interventions, is a hallmark of ARDS. Initially, hypoxemia (an arterial oxygen tension, or PaO_2, of <60 mm Hg) may improve with supplemental oxygen; however, refractory hypoxemia (no improvement of PaO_2 with supplemental oxygen) and a persistently low SaO_2 eventually develop. Early in acute respiratory failure, dyspnea and tachypnea are associated with a decreased $PaCO_2$. Hypercarbia develops as gas exchange, and ventilation becomes increasingly impaired. Arterial pH in the early phase may be high (>7.45), a finding that is consistent with respiratory alkalosis secondary to rapid respirations and a low $PaCO_2$. The arterial pH measurements in ARDS are typically lower because of respiratory and ventilatory failure and tissue hypoxia, anaerobic metabolism, and

subsequent metabolic acidosis. Base excess and deficit follow a similar trend, depending on the degree of tissue and organ hypoxia.

Measurement of arterial lactate is commonly ordered as an indication of tissue hypoxia and anaerobic metabolism. An elevated blood lactate concentration is common in early ARDS and resolves as oxygenation improves. Lactate measurement is not performed routinely once adequate, although perhaps not optimal, oxygenation has been achieved.

Radiographic Studies

In the early phase of ARDS, the chest radiographic changes are usually negligible. Within a few days, the chest radiographic findings show patchy bilateral alveolar infiltrates, usually in the dependent lung fields. This may be mistaken for cardiogenic pulmonary edema. Over time, these patchy infiltrates progress to diffuse infiltrates, consolidation, and air bronchograms. CT of the chest also shows areas of infiltrates and consolidation of lung tissue. Daily chest radiographs are important in the continuing evaluation of the progression and resolution of ARDS and for ongoing assessment of potential complications, especially pneumothoraces.

Intrapulmonary Shunt Measurement

An intrapulmonary shunt is a type of ventilation–perfusion mismatch. It may be defined as the percentage of cardiac output that is not oxygenated owing to pulmonary blood flowing past collapsed or fluid-filled and nonventilated alveoli (a physiological shunt), absence of blood flow to ventilated alveoli (alveolar dead space), or a combination of both of these conditions (silent unit [alveoli with no ventilation and no perfusion]; see Chapter 23, Fig. 23-16). Normally, an intrapulmonary shunt of 3% to 5% is present in all people. Advanced respiratory failure and ARDS are associated with a shunt of 15% or more because of the pathological changes in blood flow, endothelial disruption, and alveolar collapse. As the intrapulmonary shunt increases to 15% and greater, more aggressive interventions, including mechanical ventilation, are required because this level of shunt is associated with profound hypoxemia and may be life threatening.

The intrapulmonary shunt fraction (Qs/Qt) is calculated using the arterial oxygen content (CaO_2), the mixed venous oxygen content (CvO_2), and the capillary oxygen content (CcO_2). Oxygen content is determined by hemoglobin (Hgb), oxygen saturation (SO_2), and partial pressure of oxygen (PO_2), measured by calculating the oxygen content in the pulmonary capillary bed, in the systemic arterial system, and in the mixed venous blood from the pulmonary artery. The intrapulmonary shunt fraction may be estimated using a simple calculation, the ratio of arterial oxygen to inspired oxygen (ie, the PaO_2:FiO_2 ratio). In general, a PaO_2:FiO_2 ratio greater than 300 is normal, a value of 200 is associated with an intrapulmonary shunt of 15% to 20%, and a value of 100 is associated with an intrapulmonary shunt of more than 20%.

Lung Compliance, Airway Resistance, and Pressures

Lung mechanics are altered in ARDS, resulting in reduced alveolar ventilation and pulmonary gas exchange. Lung compliance, or distensibility, decreases as the alveoli fill with fluid or collapse. More effort and greater pressure are required to move air into the lungs as they become increasingly "stiff." In addition, the resistance to airflow into and out of the lungs increases with the accumulation of secretions and mediator-induced bronchoconstriction. Because the patient with ARDS requires mechanical ventilation to support oxygenation and ventilation, lung compliance and airway resistance can be evaluated by assessing ventilator pressures and tidal volume changes.

Close monitoring of airway pressures, including the mean airway pressure, the peak inspiratory pressure, and the plateau pressure, is an important component of patient assessment in ARDS. Increases in these pressures as tidal volumes are maintained to achieve a normal $PaCO_2$ indicate reduced compliance and increased resistance to airflow. As airway pressures rise, the lung epithelium is traumatized, resulting in further lung tissue damage. Volutrauma (lung epithelial damage) from persistently elevated airway pressures thus has additional deleterious effects on ventilation and oxygenation.

Possible nursing diagnoses for a patient with ARDS are given in Box 27-2.

BOX 27-2 EXAMPLES OF NURSING DIAGNOSES

For the Patient with Acute Respiratory Distress Syndrome

- Impaired Gas Exchange related to refractory hypoxemia and pulmonary interstitial/alveolar leaks found in alveolar capillary injury states
- Ineffective Airway Clearance related to increased secretion production and decreased ciliary motion
- Ineffective Breathing Patterns related to inadequate gas exchange, increased secretions, decreased ability to oxygenate adequately, fear, or exhaustion
- Anxiety related to critical illness, fear of death, role changes, or permanent disability
- Risk for Infection related to invasive monitoring devices and endotracheal tube

▲ Management

Therapeutic modalities to actually treat ARDS have remained elusive. Although there are multiple potential causes of ARDS, management principles are similar. Treatment is supportive; that is, contributing factors are corrected or reversed, and while the lungs heal, care is taken so that treatment does no further damage.

In addition, extensive work has gone into creating "bundles," which are elements of care considered core to the management and treatment of specific critical illnesses in intensive care units (ICUs). Box 27-3 lists essential critical care bundles that apply to managing ARDS. These treatments span prevention at early stages of disease onset, such as early goal-directed fluid resuscitation and longer-term prevention of complications, such as sedation protocols. Regardless, one of the most important roles for critical care nurses is ensuring attention to all these elements to prevent mortality and to promote recovery.

BOX 27-3 Care "Bundles" in Critical Care

Ventilator: ventilator-associated pneumonia (VAP) "bundle" basics
- Elevated head of the bed 30 to 45 degrees
- Daily weaning assessment (spontaneous breathing trials)
- Daily sedation withholding
- Weaning protocol
- Deep vein thrombosis (DVT) prophylaxis
- Peptic ulcer prophylaxis

Sepsis "bundle" basics
- Appropriate antibiotic therapy
- Early goal-directed fluid resuscitation
- Steroid administration
- Activated protein C
- DVT prophylaxis
- Peptic ulcer prophylaxis

Other protocols that may be added
- Tight glucose control
- Postpyloric feeding
- Subglottic suctioning
- Electrolyte replacement

Oxygenation and Ventilation

Oxygen Delivery

Refractory hypoxemia is one of the hallmarks of ARDS; therefore, attention to improving oxygen delivery is paramount. Strategies have attempted to optimize normal oxygen delivery parameters, including hemoglobin, cardiac output, and oxygen saturation. Oxygen delivery (DaO_2) is the amount of oxygen delivered to the tissues and organs every minute and depends on the flow of oxygenated blood through the tissue beds. Parameters that determine DaO_2 are hemoglobin, arterial oxygenation, and cardiac output.

Adequate DaO_2 (>800 mL O_2/min) is essential to meet tissue requirements for oxygen, thereby preventing anaerobic metabolism and hypoxia, which can trigger and perpetuate SIRS. Critically ill patients with ARDS have high demands for oxygen to maintain organ function.

Hemoglobin combines with oxygen to form oxyhemoglobin; therefore, sufficient amounts of hemoglobin are necessary to carry oxygen to the cells. There is little research to support the intuitive concept that normal or increased hemoglobin is required to promote oxygen delivery in patients with SIRS or ARDS. Studies on transfusion requirements indicate that values of approximately 8.0 g/dL are sufficient for critically ill patients, except for those with cardiac disease.

Cardiac output is typically altered in ARDS because of the SIRS, the effect of hypoxemia on the myocardium, and the decrease in venous return induced by mechanical ventilation. Evaluation of the cardiac output is important so that oxygen delivery can be assessed and appropriate interventions initiated. Therapies to optimize cardiac output are directed toward enhancing preload and contractility and normalizing afterload. Routine use of a thermodilution pulmonary artery catheter to assess oxygen delivery and consumption for patients with ARDS has decreased over the last decade, but may be used to ensure that appropriate interventions are instituted. There are other less-invasive methods to measure cardiac output utilizing the patient's transduced arterial blood pressure, as well as utilizing the patient's central venous catheter to measure the venous oxygen content. These techniques are less accurate, but able to be used for trending.

Fluid management has been used for many years in an effort to balance the type of fluid needed to manage the hallmark edema and decompensation associated with ARDS. At the onset of the disease, early goal-directed fluid resuscitation is recommended. Diuretics and reduced fluid administration have been studied to reduce lung edema. Conservative fluid management with diuresis, plus albumin for hypoproteinemic patients, is associated with modest improvements in oxygenation.[8]

Positive inotropic agents, such as dobutamine, are used to enhance contractility and increase cardiac output; however, care must be taken with these agents as they may cause systemic vasodilation worsening hypotension. Vasoconstrictors, such as norepinephrine, may be added to the therapies to counteract the vasodilation induced by SIRS. Vasoconstricting agents must be administered cautiously because many vascular beds, especially in the lungs, are constricted, also as a result of SIRS mediators and hypoxia. Patients receiving inotropic or vasoactive medications require continuous arterial blood pressure monitoring and may require evaluation of cardiac output and other hemodynamic measures.

Mechanical Ventilation

The goal of therapy is to improve tissue oxygenation and ventilation. Methods to deliver appropriate levels of oxygen and allow for removal of carbon dioxide include types of mechanical ventilation and positioning. Lung-protective ventilation strategies limit ventilator-associated (or ventilator-induced) lung injury (VALI); these include low tidal volumes (<6 mL/kg predicted body weight), the use of adequate positive end-expiratory pressure (PEEP) to reduce the risk of using a high FiO_2 and precipitating oxygen toxicity, and limiting plateau pressures to 30 cm H_2O.[9,10] All other ventilation therapies, including high-frequency oscillation ventilation (HFOV),[11] and extracorporeal lung-assist technology,[8] have not demonstrated consistent improvements in patient outcomes in ARDS, but may be life saving in refractory hypoxemia.

Multiple modes of mechanical ventilation are available to support the patient with respiratory failure. (See Chapter 25 for a complete discussion of mechanical ventilation.) In general, the principle of "do no harm" includes use of the lowest FiO_2 to achieve adequate oxygenation and use of small tidal volumes to minimize airway pressures, thus preventing or reducing lung damage (volutrauma). Permissive hypercapnia may be necessary to prevent an increased respiratory rate in the face of lower tidal volumes. PEEP prevents collapse and recruits alveoli, allowing diffusion of gases across the alveolar–capillary membrane. Recommended values for PEEP are 10 to 15 cm H_2O, but values in excess of 20 cm H_2O are acceptable to reduce inspired oxygen requirements or maintain adequate oxygenation.[9,10,12]

Permissive hypercapnia is a strategy that allows the $PaCO_2$ to rise slowly above normal through reduction of tidal volume, therefore limiting the plateau and peak airway pressures. A $PaCO_2$ between 55 and 60 mm Hg and a pH of 7.25 to 7.35 are tolerated when achieved gradually. It is necessary

to monitor the increase in $PaCO_2$ to prevent too rapid a rise, and overall values should be no greater than 80 to 100 mm Hg because of the potential effects on cardiopulmonary function. These techniques are not used for patients with cardiac or neurological involvement.

Several modes of mechanical ventilation are directed toward minimizing airway pressures and iatrogenic lung injury, associated with conventional volume-controlled mechanical ventilation. Pressure-controlled ventilation limits the peak inspiratory pressure to a set level (as opposed to volume-controlled ventilation, which delivers a set tidal volume despite the pressure required to move the set volume into the lungs). Pressure-controlled ventilation also uses a decelerating inspiratory airflow pattern to minimize the peak pressure while delivering the necessary tidal volume. Patients on pressure-controlled ventilation mode may require sedation to prevent dyssynchrony with the ventilator. Inverse-ratio ventilation is another strategy thought to improve alveolar recruitment. Reversal of the normal inspiratory–expiratory ratio (I:E ratio) to 2:1 or 3:1 prolongs inspiration time, preventing complete exhalation. An inverse I:E ratio is achieved through manipulation of the mechanical ventilator. This increased end-expiratory volume creates auto-PEEP (intrinsic PEEP) that is added to the applied extrinsic PEEP. The theoretical advantages include reduced alveolar pressures and overall PEEP levels. This therapy requires patients to be sedated and given paralytics to improve tolerance. Airway pressure release ventilation similarly inverses the I:E ratio but with the advantage of allowing the patient to initiate breaths. These patients do not require the same level of sedation or paralysis to achieve pressure-limited ventilation and may have improved recruitment of alveoli.

Novel Ventilation Strategies

HFOV uses very low tidal volumes (1 to 4 mL/kg) delivered at rates of 3 to 15 Hz or cycles/second rather than breaths/minute, resulting in lower airway pressures and reduced volutrauma. Deleterious effects of HFOV include increased trapping of air in the alveoli (auto-PEEP) and increased mean airway pressures to high levels in some patients. HFOV requires sedation and paralysis as any change in airway pressure will cause oscillation to cease.

Extracorporeal lung-assist technology involves the use of large vascular cannulas to remove blood from the patient. A pumping device and circuit circulate the blood, and one or two "artificial lungs" remove carbon dioxide and oxygenate the blood. Extracorporeal membrane oxygenation and extracorporeal carbon dioxide removal ($ECCO_2R$) may potentially be effective in managing ARDS, but at present their use is controversial.[8] These highly invasive, high-risk technologies allow the lungs to "rest" because near-apneic ventilation or ventilation with small tidal volumes and slow respiratory rates greatly reduces airway pressures while gas exchange takes place in the artificial membrane lungs. The need for intensive resources and personnel with a high degree of expertise, coupled with the potential for devastating complications (particularly intracranial hemorrhage) and a lack of conclusive benefits for patients with ARDS, make extracorporeal lung-assist technology of limited use.

Positioning

Frequent position change is well established as a means to prevent and reverse atelectasis and to facilitate removal of secretions from the airways. Although not a treatment for ARDS, elevating the head of the bed greater than 30 degrees is considered necessary care for preventing ventilator-associated pneumonia (VAP).

Prone positioning, either in the patient's bed, using a Stryker frame or Roto-Prone™ therapy system, improves pulmonary gas exchange, facilitates pulmonary drainage in the dorsal lung regions, and aids resolution of consolidated dependent alveoli (in the supine position), particularly in the dorsal lung regions. The evidence for the effectiveness of prone positioning, now a common intervention with ARDS, is variable.[13] There are alternative explanations for the improved oxygenation associated with prone positioning, and the question of whether the improvement in oxygenation persists beyond a short time remains controversial. The associated risks include loss of airway control through accidental extubation, loss of vascular access, facial edema and development of pressure areas, and difficulties with cardiopulmonary resuscitation. Recommendations on the steps involved in prone positioning appear in Box 27-4.

Pharmacological Therapy

Antibiotic therapy is appropriate in the presence of a known microorganism but should not be used prophylactically. The signs of SIRS are similar to those of infection

BOX 27-4 Summary of Key Steps to Consider for Prone Positioning

1. Evaluate with the interdisciplinary team the patient's condition and determine whether a trial of prone positioning is warranted.
2. Organize the team to ensure familiarity with the procedure and patient care while prone.
   - Use your hospital's evidence-based procedure
   - Equipment on site
   - Assign and clarify team roles during prone positioning
3. Prepare the patient for the procedure.
   - Provide explanation to patient and family
   - Consider insertion of feeding tube, nasogastric tube, or both as necessary
4. Assess and document the patient's preprone positioning status.
   - Hemodynamic and ventilatory parameters, skin or wound condition, and so forth
5. Protect and maintain the patient's airway.
   - Secure endotracheal tube
   - Apply in-line suction if not already in place
6. Use safety precautions to ensure body position will be maintained during the prone positioning procedure.
7. Administer adequate sedation and analgesic medication.
8. Complete the procedure as per protocol. Note: Risks for inadvertent extubation or line displacement are high during the procedure.
9. Assess, evaluate, and monitor the patient's condition.
10. Implement preventive care for pressure areas, eyes, and skin.

(ie, tachycardia, fever, increased white blood cell count), thus creating the temptation to treat the patient with SIRS with antimicrobial therapy. It is essential to identify a source of infection (isolation of specific bacteria through blood, wound, pulmonary, and other cultures) before initiating antibiotics. Prophylactic antibiotic therapy has not been shown to improve outcome. Emphasis is on prevention of infection, especially nosocomial infection related to the use of invasive vascular catheters and ventilators (eg, VAP).

Bronchodilators and mucolytics are useful in ARDS to assist in maintaining airway patency and reducing the inflammatory reaction and accumulation of secretions in the airways. The response to therapy is evaluated by monitoring airway resistance and pressures and lung compliance.

Administration of intravenous corticosteroids in low-to-moderate doses for prolonged duration has been associated with improved oxygenation when initiated, less than 14 days after onset of ARDS; this has been recommended by an international group of experts.[8] In a meta-analysis, the use of low-dose corticosteroids was associated with improved mortality and morbidity outcomes without increased adverse reactions. The consistency of results in both study designs and all outcomes suggests that they are an effective treatment for ALI or ARDS.[14]

Nitric oxide is an inhaled gas that causes selective pulmonary vasodilation and therefore reduces the deleterious effects of pulmonary hypertension. To date, nitric oxide has not been shown to improve mortality or oxygenation beyond the first 24 hours of therapy.[8] Nitric oxide should be reserved for those patients with life-threatening refractory hypoxemia after mechanical ventilation has been maximized. Inhaled prostacyclin has also produced pulmonary vasodilation similar to nitric oxide and may be considered.[8]

Sedation

Effective use of sedation to promote comfort and reduce respiratory effort, thus decreasing oxygen demand, is an important consideration for nurses dealing with patients with ARDS. Neuromuscular blocking agents and general anesthetics, such as propofol, although not sedatives, are all used to decrease the work of breathing and facilitate ventilation for patients with ARDS. Neuromuscular blocking agents require concurrent use of sedation to prevent patients who are chemically paralyzed from being alert but unable to move. Frequent assessment of adequacy of both neuromuscular blockade and sedation is an important nursing intervention. Neuromuscular blocking agents have been associated with critical illness polyneuropathy and polymyopathy, especially when concurrently administered with corticosteroids.

Pain, anxiety, and delirium are all possible reasons for needing pharmacological treatment, and it is important to distinguish between them because each requires a different pharmacological intervention. It is vital to understand why each is being given, what the goals of therapy are, and what the long-term implications of overuse can be. These considerations are balanced with the need to decrease oxygen demand and provide comfort for patients requiring intensive ventilation management and undergoing potentially uncomfortable procedures.

Nutritional Support

Early initiation of nutritional support is essential for patients with ARDS because nutrition plays an active therapeutic role in recovery from critical illness. There are two major theoretical reasons to use early enteral feeding as a therapeutic intervention in SIRS and ARDS. Mediators (TNF-α and IL-1 in particular) stimulate release of proteolytic enzymes that stimulate protein catabolism from skeletal muscle. Persistent protein loss is compounded by interstitial loss through capillary leak and down-regulation of messenger RNA production of intravascular proteins, such as albumin. Earlier in this chapter, reference was made to changes in circulatory patterns resulting from hypoxic sympathetic nervous system reactions. In this way, there is decreased perfusion to the gut. After resuscitation, increases in neutrophil release further damage the injured, reperfused colon through increased vascular endothelial permeability, thus releasing normal gut bacteria into the systemic circulation and leading to increases in the incidence of peritonitis, pneumonia, and sepsis. The mechanism through which enteral feeding improves outcome remains unproved, but the reduction in mortality in the critically ill who receive enteral feedings indicates that this practice is of general benefit.

A diet with a balanced caloric, protein, carbohydrate, and fat intake is calculated based on metabolic needs, with particular attention paid to specific amino acids, lipid, and carbohydrate intake. Patients with SIRS or ARDS usually require 35 to 45 kcal/kg/d. High-carbohydrate solutions are avoided to prevent excess carbon dioxide production. Recent innovations in amino acid supplementation are being reviewed because of the role of amino acids in the immune response. The role of antioxidants and omega-3 fatty acids is still being investigated as to their use in improving outcomes in ARDS patients.[8]

The problem facing the practitioner is the ability to deliver enteral nutrition in the face of decreased gut motility. Insertion of small bowel feeding tubes may be considered. The role of total parenteral nutrition is controversial, and some clinicians rarely use it, either alone or in combination with enteral nutrition.[8] The risk for aspiration associated with enteral feeding needs to be appreciated, and careful monitoring of absorption and gut function is essential.

A collaborative care guide for the patient with ARDS is given in Box 27-5.

▲ Prevention of Complications

Complications of ARDS are primarily related to SIRS, VALI, and immobility imposed by critical illness. The most serious of these is the development of MODS due to hypoxemia, hypoxia, and the persistent inflammatory response. An entire spectrum of potential complications exists for the critically ill patient. Critical care forums have compiled evidence-based protocols into bundles for two major critical care situations: VAP and

BOX 27–5 COLLABORATIVE CARE GUIDE for the Patient With Acute Respiratory Distress Syndrome

Outcomes	Interventions
Oxygenation/Ventilation	
Patent airway will be maintained. A PaO_2:FIO_2 ratio of 200 to 300 or more will be maintained, if possible.	• Auscultate breath sounds every 2–4 h and as required. • Intubate to maintain oxygenation and ventilation and to decrease work of breathing. • Suction endotracheal airway when appropriate (see Chapter 25, Box 25-16, Collaborative Care Guide for the Patient on Mechanical Ventilation). • Hyperoxygenate before and after each suction pass.
Lung-protective ventilation strategies will be used. Maintain a low tidal volume (<6 mL/kg), a plateau pressure ≤30 cm H_2O, and positive end-expiratory pressure (PEEP) levels titrated to pressure–volume curve.	• Monitor airway pressures every 1–2 h. • Monitor airway pressures after suctioning. • Administer bronchodilators and mucolytics. • Obtain a PEEP study to determine optimal oxygen delivery. • Consider a change in ventilator mode to prevent volutrauma.
The risk for atelectasis, ventilator-associated pneumonia (VAP), and volutrauma will be reduced and oxygenation will be improved.	• Turn patient side-to-side every 2 h. • Perform chest physiotherapy every 4 h, if tolerated. • Elevate head of bed 30 degrees. • Monitor chest x-ray daily.
Oxygenation will be maximized (a PaO_2 of 55 to 80 mm Hg or a SaO_2 of 88% to 95%).	• Monitor pulse oximetry and end-tidal carbon dioxide. • Monitor arterial blood gas values as indicated by changes in noninvasive parameters. • Monitor intrapulmonary shunt (Qs/Qt and PaO_2:FIO_2 ratio). • Increase PEEP and FIO_2 to decrease intrapulmonary shunting, using lowest possible FIO_2. • Consider permissive hypercapnia to maximize oxygenation. • Monitor for signs of volutrauma, especially pneumothorax. • Consider risk for prolonged hyperoxia and decrease FIO_2 to <65% as soon as able.
Circulation/Perfusion	
Blood pressure, cardiac output, central venous pressure (CVP), and pulmonary artery pressures remain stable related to mechanical ventilation.	• Assess hemodynamic effects of initiation of mechanical ventilation (eg, decreased venous return and cardiac output). • Monitor electrocardiogram (ECG) for dysrhythmias related to hypoxemia. • Assess hemodynamic effects of changes in inspiratory pressure settings, tidal volume, PEEP, and ventilatory modes. • Assess effects of ventilator setting changes on cardiac output and oxygen delivery. • Administer intravascular volume to maintain preload.
Blood pressure, heart rate, and hemodynamic parameters are optimized to therapeutic goals.	• Monitor vital signs every 1–2 h. • Monitor pulmonary artery pressures and right atrial pressure every hour and cardiac output, systemic vascular resistance, peripheral vascular resistance, DaO_2, and oxygen consumption (VO_2) every 6–12 h, if pulmonary artery catheter is in place. • Administer intravascular volume agents as indicated by real or relative hypovolemia, and evaluate response.
Serum lactate level will be within normal limits.	• Monitor lactate level as required until it is within normal limits. • Administer red blood cells, positive inotropic agents, and colloid infusion as ordered to increase oxygen delivery.
Fluids/Electrolytes	
Patient is euvolemic. Urine output is >0.5 mL/kg/h.	• Monitor hydration status to reduce viscosity of lung secretions. • Monitor intake and output. • Avoid use of nephrotoxic substances and overuse of diuretics. • Administer fluids and diuretics to maintain intravascular volume and renal function.
There is no evidence of electrolyte imbalance or renal dysfunction.	• Replace electrolytes as ordered. • Monitor urine output, blood urea nitrogen (BUN), creatinine, creatinine clearance, serum osmolarity, and urine electrolytes as required.

(continued on page 602)

BOX 27–5 *COLLABORATIVE CARE GUIDE for the Patient With Acute Respiratory Distress Syndrome (continued)*

Mobility/Safety	
There is no evidence of complications related to bed rest and immobility.	• Initiate deep venous thrombosis (DVT) prophylaxis. • Reposition patient frequently. • Mobilize patient to chair when hemodynamic stability and hemostasis are achieved. • Consult physiotherapist. • Conduct range-of-motion and strengthening exercises when able.
Physiological changes are detected and treated without delay.	• Monitor mechanical ventilator alarms and settings and patient parameters (eg, tidal volume) every 1–2 h. • Ensure appropriate settings and narrow limits for hemodynamic, heart rate, and pulse oximetry alarms.
There is no evidence of infection; white blood cell count is within normal limits.	• Monitor for systemic inflammatory response syndrome (SIRS) criteria (increased white blood cell count, increased temperature, tachypnea, tachycardia). • Use strict aseptic technique during procedures, and monitor others. • Maintain sterility of invasive catheters and tubes. • Change chest tube and other dressings and invasive catheters. • Culture blood and other fluids and line tips when they are changed.
Skin Integrity	
Skin will remain intact.	• Assess skin every 4 h and each time patient is repositioned. • Turn patient every 2 h. • Consider pressure relief/reduction mattress, kinetic therapy bed, or prone positioning. • Use Braden Scale to assess risk for skin breakdown.
Nutrition	
Caloric and nutrient intake will meet metabolic requirements per calculation (eg, basal energy expenditure).	• Provide enteral nutrition within 24 h. • Consult dietitian or nutritional support service. • Consider small bowel feeding tube if gastrointestinal motility is an issue for enteral feeding. • Monitor lipid intake. • Monitor albumin, prealbumin, transferrin, cholesterol, triglyceride, and glucose levels.
Comfort/Pain Control	
Patient will be as comfortable as possible as evidenced by stable vital signs or cooperation with treatments or procedures.	• Objectively assess comfort/pain using a pain scale. • Provide analgesia and sedation as indicated by assessment. • Monitor patient cardiopulmonary and pain response to medication. • If patient is receiving neuromuscular blockade for ventilatory control: • Use peripheral nerve stimulator to assess pharmacological paralysis. • Provide continuous or routine (every 1–2 h) intravenous sedation and analgesia.
Psychosocial	
Patient demonstrates decreased anxiety.	• Assess vital signs during treatments, discussions, and the like. • Cautiously administer sedatives. • Consult social services, clergy, as appropriate. • Provide for adequate rest and sleep.
Teaching/Discharge Planning	
Patient/significant others understand procedures and tests needed for treatment.	• Prepare patient/significant others for procedures, such as bronchoscopy, pulmonary artery catheter insertion, or laboratory studies. • Explain the causes and effects of ARDS and the potential for complications, such as sepsis, volutrauma, or renal failure.
Significant others understand the severity of the illness, ask appropriate questions, and anticipate potential complications.	• Encourage significant others to ask questions related to the ventilator, the pathophysiology of ARDS, monitoring, and treatments.

BOX 27-6 CONSIDERATIONS FOR THE OLDER PATIENT

Acute Respiratory Distress Syndrome

- People who are 65 years of age or older are at increased risk for multisystem organ involvement with less chance of recovering from ARDS; therefore, the mortality rate rises in this population.
- Because of increased immunosuppression with aging, the elderly are at greater risk for infection; therefore, nosocomial infections, such as urinary tract infections and ventilator-associated pneumonia, are more common.
- Hemodynamic instability adds metabolic insults to already-decreased renal function, thus predisposing this group to renal failure.
- Decreased stroke volume; possible coronary artery disease (CAD), atherosclerosis, or both; and increased systolic blood pressure and peripheral vascular resistance alter hemodynamic recovery.
- Decreased maximal oxygen uptake associated with decreased lung volumes puts elderly patients at greater risk for ventilator-associated lung injury.
- Decreased muscle mass associated with aging makes recovery from prolonged immobility more difficult. Therefore, an elderly person with ARDS may require prolonged rehabilitation.
- Generalized peripheral edema, multiple invasive tests, and prolonged bed rest, combined with the decreased skin integrity associated with old age, increase the elderly patient's potential for development of pressure ulcers and skin tears.
- Elderly patients with ARDS are at risk for not receiving the same quality and quantity of treatment and care as younger patients, due to the effects of ageism. The patient's age is one factor to consider in outcome and prognosis, but not the only one.
- The incidence of comorbid conditions, especially non–insulin-dependent diabetes mellitus and CAD, increases with age. Research findings indicate that comorbid conditions increase the risk for death in patients with ARDS.
- Based on previously expressed wishes, the patient and family may request no initiation of, or early removal from, life support. A person's life experience or vision of risk related to prolonged illness with high possibility of mortality may influence this decision, and these wishes should be respected.

sepsis (see Box 27-3, p. 598). The introduction of care bundles into critical care supports application of evidence to reduce major complications. The implementation of care bundles has been shown effective in reducing length of stay and reducing ventilator days, but consistent application requires teamwork and monitoring.

Mechanical ventilation with high levels of PEEP, high tidal volumes, and volume-controlled modes predisposes the patient with ARDS to volutrauma, as previously described. Volutrauma may present as a pneumothorax, pneumomediastinum, or subcutaneous or interstitial emphysema. Prompt chest tube insertion is required for a pneumothorax. Prevention of volutrauma by maintaining the lowest possible airway pressures, PEEP, and tidal volumes may be achieved through the use of pressure-limiting modes of mechanical ventilation.

Prevention or reduction in the incidence of VAP can be accomplished through using in-line suction catheters. The use of endotracheal tubes that allow for the removal of pooled subglottic secretions have been shown to reduce VAP. Sinusitis is also associated with VAP. The critical care nurse needs to monitor for nasal secretions and ensure that devices such as nasotracheal or feeding tubes are removed from the nose when these occur. Oral care is an essential component in the prevention of VAP, as it decreases the amount of organisms in the mouth, which may migrate to the lungs (see Evidence-Based Practice Highlight 25-2, p. 542). Elevating the head of the bed 30 degrees and feeding the critically ill patient with a postpyloric feeding tube have been shown to reduce microaspiration and VAP.

Immobility caused by bed rest, sedation, or pharmacological paralysis has multisystem effects. Not infrequently, nosocomial pneumonia is acquired from accumulated secretions in the airways and atelectasis secondary to immobilization, with bacterial access through and around the endotracheal tube. As discussed, frequent repositioning accompanied by chest physiotherapy will help to reduce stasis of secretions and facilitate removal.

Deep venous thrombosis (DVT) and subsequent pulmonary embolus may be life-threatening complications of immobility. Initiation of DVT prophylaxis within 48 hours of admission minimizes the risk for development of DVT. Low-dose heparin, graded elastic stockings, external pneumatic compression devices, frequent mobilization, and ambulation have been useful in reducing DVT formation.

The physiological aging process compounds the severity of the metabolic insults and complications of ARDS (Box 27-6).

▲ Clinical Applicability Challenges

CASE STUDY

P.F., a 52-year-old woman with a history of cholelithiasis, is admitted to the hospital for acute pancreatitis. She progresses to necrotizing pancreatitis and develops shock. She is transferred to the intensive care unit (ICU).

After arrival in the ICU, the patient develops cardiorespiratory failure and is intubated and placed on vasoactive support of vasopressin, 0.04 units/min, and norepinephrine, 10 mcg/min. She is having multiple episodes

(continued on page 604)

CASE STUDY *(Continued)*

of oxygen desaturation requiring an FiO_2 of 1.00. She is placed on assist-control ventilation of 20 breaths/min, a tidal volume of 6 mL/kg with positive end-expiratory pressure (PEEP) of 10 cm H_2O. Her breath sounds are clear with no crackles or wheezes. Her temperature is 101.1°F (38.4°C). A bronchial lavage is performed, and no pus, fluid, or blood is seen in the airways. As the day progresses, P.F. becomes difficult to ventilate and is sedated with a fentanyl infusion and midazolam (Versed) infusion. She also requires neuromuscular blockade with cisatracurium (Nimbex). Her cardiac index is 4.0, heart rate is 120 beats/min, central venous pressure is 8 mm Hg, PAOP is 10 mm Hg, and systemic vascular resistance index is 900 dynes/s/m^2/cm^5. Arterial blood gas (ABG) values show PaO_2 of 68 mm Hg, $PaCO_2$ of 39 mm Hg, pH of 7.36, HCO_3 of 18 mEq/L, and O_2 saturation of 95.5%.

The following day, P.F. continues to decline on 100% FiO_2. She has developed a mild to moderate bilateral wheeze in his chest. Chest radiography shows small bilateral effusions but is otherwise clear. PaO_2 remains low at 65 mm Hg. Blood, sputum, and urine specimens are sent to the laboratory for culture. Empiric broad-spectrum antibiotics are started. With no improvement in the ABGs and the rising pulmonary arterial pressures (47/16 mm Hg), nitric oxide is started at 10 ppm.

Computed tomography (CT) of the thorax is performed the following day to rule out pulmonary embolus and shows bilateral airspace opacity. This is visible on a chest radiograph the following day. A postpyloric feeding tube is inserted, and enteral feedings are started while P.F.'s lipase levels are monitored.

Seven days after admission, FiO_2 remains at 100%, and nitric oxide continues at 10 ppm with no improvement in oxygenation. Temperature remains elevated at 102°F (39.1°C) with a white blood cell count of 12,000/mm^3. All cultures remain negative. Chest radiography shows fluffy white opacities bilaterally and a collapsed right upper lung lobe. A bronchoscopy is performed to examine the right upper lobe, and copious secretions are removed. Several short attempts at lung recruitment to expand the lungs are performed, and because there is still no improvement, P.F. is placed in the prone position. Once in the prone position, her oxygenation improves.

Over the next few days, P.F. makes small steps forward with her oxygenation yet continues to require high levels of oxygen. A transbronchial biopsy is performed and demonstrates DAD and organizing pneumonia without evidence of infection. At this time, low-dose corticosteroids are started intravenously and continued for 3 weeks. The dose is tapered off over a 3-month period.

P.F. does well after corticosteroids are initiated. However, she has several episodes of VAP before becoming free of the ventilator.

After almost 4 months in hospital, P.F. is discharged home. On a follow-up examination, she had recovered to about 60% of her prehospital status. Her pulmonary function test shows only mild impairment. CT of her thorax shows scarring and fibrosis in nondependent lung zones and some bronchiectasis in dependent lung zones. This is interpreted as ventilator-acquired (induced) lung injury with stretch injury in the nondependent areas and airway trauma in the dependent airways.

1. What symptoms were presented early in P.F. that indicated the onset of ARDS?

2. Early enteral feeding was used to support P.F. For what other care should the nurse advocate to reduce complications in Ms P.F.?

3. What is the evidence that corticosteroids have a positive benefit in treating a patient with ARDS?

References

1. Bernard GR, Artigas A, Brigham KL, et al: The American-European Consensus Conference on ARDS: Definitions, mechanisms, relevant outcomes, and clinical trials coordination. Am J Respir Crit Care Med 149:818–824, 1994
2. http://www.fda.gov/downloads/BiologicsBloodVaccines/SafetyAvailability/ReportaProblem/TransfusionDonationFatalities/UCM205620.pdf. Accessed July 17, 2010
3. Triulzi DJ: Transfusion-related acute lung injury: current concepts for the clinician. Anesth Analg 108(3):770–776, 2009
4. Zhang H, Damas P, Preiser JC: The long way of biomarkers: From bench to bedside. Intensive Care Med 36(4):565–566, 2010
5. Rubenfeld GD, Caldwell E, Peabody E, et al: Incidence and outcomes of acute lung injury. N Engl J Med 353:1685–1693, 2005
6. Maniatis NA, Orfanos SE: The endothelium in acute lung injury/acute respiratory distress syndrome. Curr Opin Crit Care 14(1):22–30, 2008
7. Gropper MA, Wiener-Kronish J: The epithelium in acute lung injury/acute respiratory distress syndrome. Curr Opin Crit Care 14(1):11–5, 2008
8. Raoof S, Goulet K, Esan A, et al: Severe hypoxemic respiratory failure: Part 2—nonventilatory strategies. Chest 137(6):1437–1448, 2010
9. Putensen C, Theuerkauf N, Zinserling J, et al: Meta-analysis: Ventilation strategies and outcomes of the acute respiratory distress syndrome and acute lung injury. Ann Intern Med 151(8):566–576, 2010
10. Meade MO, Cook DJ, Guyatt GH, et al; Lung Open Ventilation Study Investigators: Ventilation strategy using low tidal volumes, recruitment maneuvers, and high positive end-expiratory pressure for acute lung injury and acute respiratory distress syndrome: A randomized controlled trial. JAMA 299(6):637–645, 2008
11. Sud S, Sud M, Friedrich JO, et al: High frequency oscillation in patients with acute lung injury and acute respiratory distress syndrome (ARDS): Systematic review and meta-analysis. BMJ 340:c2327, 2010

12. Oba Y, Thameem DM, Zaza T: High levels of PEEP may improve survival in acute respiratory distress syndrome: A meta-analysis. Respir Med 103(8):1174–1181, 2009
13. Taccone P, Pesenti A, Latini R, et al; Prone-Supine II Study Group: Prone positioning in patients with moderate and severe acute respiratory distress syndrome: A randomized controlled trial. JAMA 302(18): 1977–1984, 2009
14. Tang BM, Craig JC, Eslick GD, et al: Use of corticosteroids in acute lung injury and acute respiratory distress syndrome: A systematic review and meta-analysis. Crit Care Med 37(5):1594–1603, 2009

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

PART 7

RENAL SYSTEM

28

Anatomy and Physiology of the Renal System

Kara Adams Snyder and Kimberli Haas

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Describe the impact of afferent and efferent blood supply on renal function.
2. Discuss the structures comprising the nephron: glomerulus, proximal tubule, loop of Henle, and distal and collecting tubules.
3. Differentiate the functions of the nephron, including glomerular filtration, active and passive transport, and tubular secretion.
4. Compare normal fluid pressures in the nephron and how they affect glomerular filtration rate.
5. Explain the relationship of antidiuretic hormone, renin, and aldosterone to fluid regulation by the kidneys.
6. Explain the mechanisms used by the kidneys to help achieve clearance of substances and maintain homeostasis.
7. Describe the physiological roles of the predominant electrolytes.

With each contraction of the heart, the kidneys receive 21% of the cardiac output. This means that approximately 1.2 liters of blood passes through the kidneys each minute, and the body's entire blood volume is filtered through the kidneys 340 times per day.[1] Given this large volume of blood, the kidneys have a dominant role in filtration and a minor role in metabolism. Therefore, the kidneys have a large requirement for pressure and a relatively smaller requirement for oxygen. The regulation and maintenance of the concentration of solutes in the extracellular fluid (ECF) of the body are the primary functions of the kidney. The kidneys remove metabolic waste products and excess concentrations of constituents and conserve substances present in normal or low quantities.

▲ Macroscopic Anatomy of the Renal System

The kidneys are bean-shaped organs that lie in a retroperitoneal position in the abdomen, one on each side of the vertebral column (Fig. 28-1). The kidneys are partially protected by the last pair of ribs, with the right kidney slightly lower than the left because of the location of the liver. A tough, fibrous coat, known as the renal capsule, surrounds each kidney. The adrenal glands, which are discussed in more detail in Chapter 42, cap the kidneys.

The adult kidneys are approximately 12 cm long, 6 cm wide, and 2.5 cm thick. The kidney weighs about 150 g, the

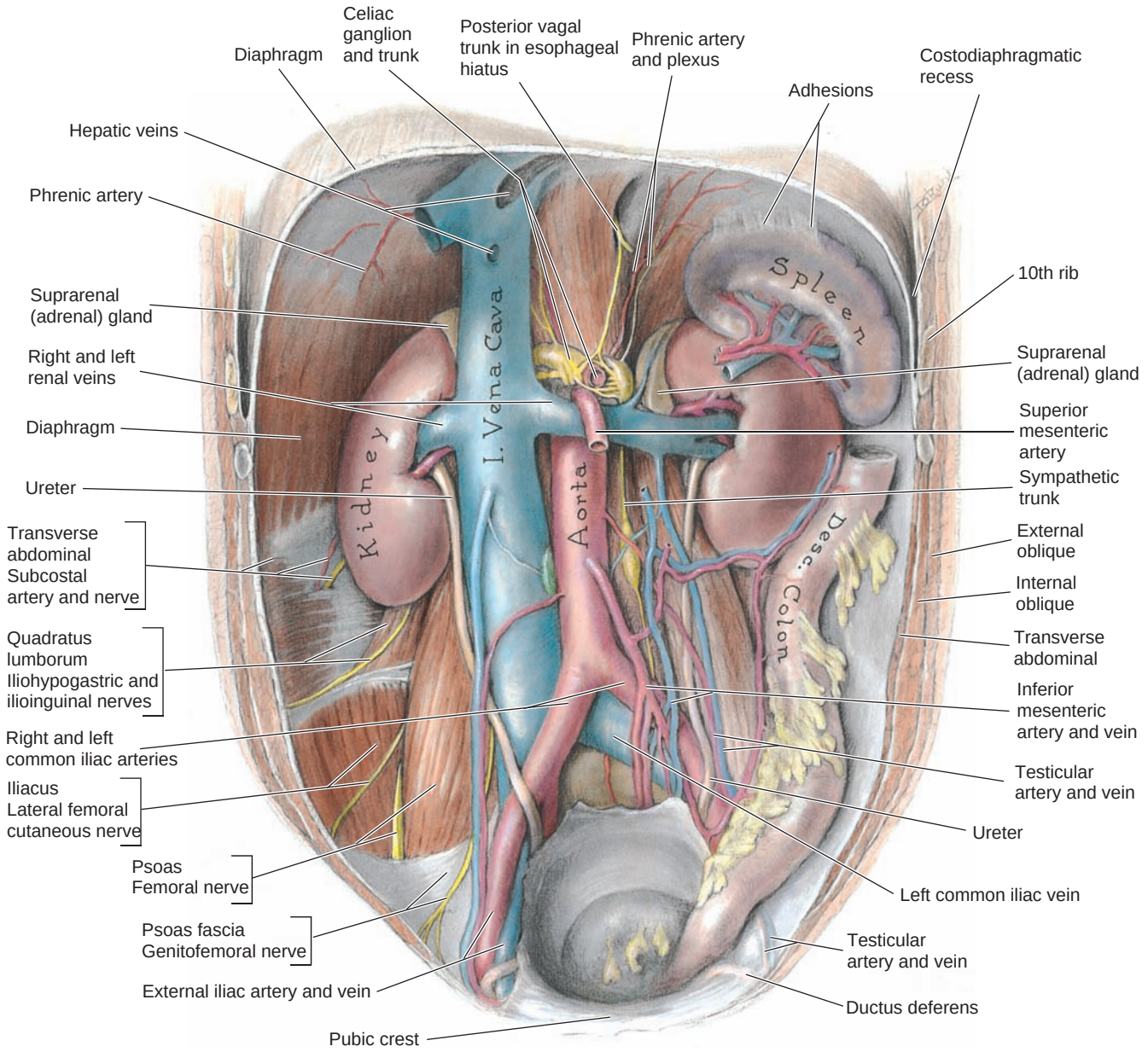


FIGURE 28-1 ▲ Anatomy of the kidneys and urinary system. (From Moore KL, Dalley AF, Agur AMR: Clinically Oriented Anatomy, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 290.)

size of a clenched fist. The size and weight of the kidneys are clinically valuable indicators in ultrasound-guided differential diagnosis of renal failure (see Chapter 29).

There are two distinct layers of the kidney: the renal cortex and the renal medulla (Fig. 28-2). The renal cortex is the outer portion of the kidney and has two regions: the cortical region and the juxtamedullary ("next to the medulla") region. The cortex contains the glomeruli, the proximal tubules, the cortical loops of Henle, the distal tubules, and the cortical collecting ducts. The inner layer, the medulla, in addition to the cortical structures, contains the renal pyramids. The renal cortex receives 90% of the total renal blood flow, and only 5% to 10% reaches the outer medulla. The pyramids contain the medullary loops of Henle and the medullary portions of the collecting ducts, which join to form a minor calyx. Minor calyces come together to form a major calyx. The renal calyces further join to become the conduit for directing urine into the ureter.

Urine exits the kidney at an oblique angle through a fibromuscular structure, the ureter. Peristalsis helps maintain the flow of urine through the ureter. The ureter enters the bladder in the trigone region. The trigone region of the bladder is so called for the three structures that form the shape of a triangle: the two ureters and the urethra. The peristaltic actions in the ureter and the angle of entry at the bladder help prevent the reflux of urine. Urine exits the bladder through the urethral orifice via the urethra. The male urethra is about 20 cm long; the female urethra is about 3 to 5 cm long.[1]

On the medial aspect of each kidney, there is an indentation known as the hilum. It is through this indentation

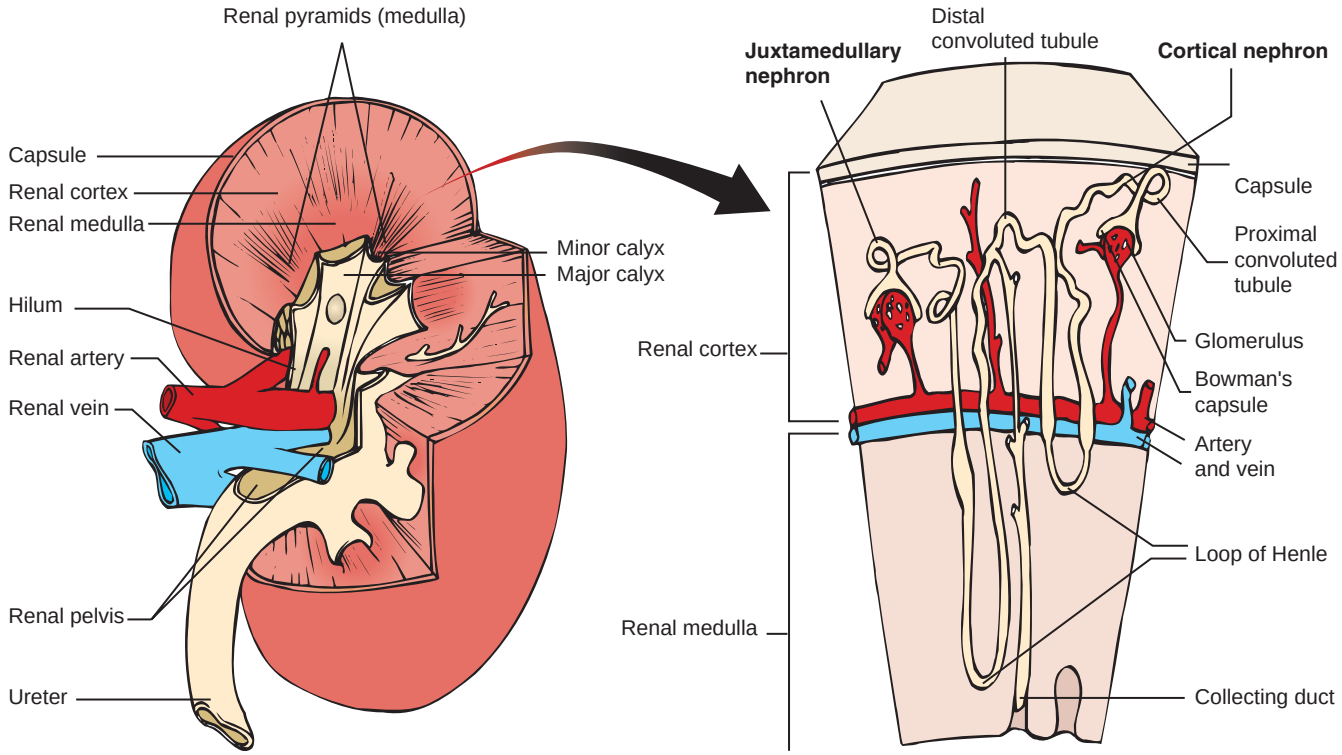


FIGURE 28-2 ▲ Macroscopic anatomy of the kidney.

that the renal arteries and nerves enter and the renal veins, lymphatics, and ureters exit (see Fig. 28-2).

The kidneys receive their blood supply from the renal artery, a branch of the descending aorta. The renal artery divides into several smaller branches known as the interlobular arteries (Fig. 28-3). Further branching produces numerous afferent arterioles. Each afferent arteriole forms a tuft of capillaries, known as the glomerulus, where blood is filtered. Leaving the glomerulus is the efferent arteriole. The efferent arteriole branches to form a second capillary bed, known as the peritubular capillaries (see Fig. 28-3). The peritubular capillaries surround the loop of Henle to reabsorb more water and solutes as needed for homeostasis. Reconnecting, this vast network of vessels eventually returns to the central circulation through the renal veins. The renal blood flow per weight unit is higher than any other major organ in the body.

The glomerular filtration rate (GFR) is relatively stable over a wide range of arterial blood pressures. The concept of "autoregulation" offers the kidneys such stability: the afferent arterioles adjust their diameter in response to the pressure of blood coming to them. If the blood pressure decreases, the smooth muscles of the afferent arterioles relax, vasodilation occurs, and perfusion increases, thereby maintaining the GFR at its normal rate. Conversely, with an increase in blood pressure, these vessels constrict. In healthy persons, autoregulation maintains homeostasis quite nicely when mean blood pressure falls approximately within a range of 80 to 180 mm Hg. Outside of this range, autoregulation is limited and GFR is proportional to renal perfusion. For example, if the systemic blood pressure falls greatly, such as in shock, the GFR falls to near zero, thereby producing near anuria.

▲ Microscopic Anatomy of the Renal System and Normal Renal Physiology

Urine, the end product of kidney function, is formed from the blood by the smallest unit of the kidney, the nephron (see Fig. 28-3). Each human kidney consists of about 1 million nephrons, all of which function identically; therefore, kidney function can be explained by describing the function of one nephron (Table 28-1, p. 611). A nephron is composed of a glomerulus, a proximal tubule, a loop of Henle, and a distal tubule. Several distal tubules drain into a collecting duct.

Approximately 80% of the filtrate is returned to the bloodstream by reabsorption in the proximal tubule.[1] In a healthy person, all the filtered glucose and amino acids, much sodium, chloride, hydrogen, and other electrolytes, uric acid, and urea are all reabsorbed here. The proximal tubule cells also secrete substances (eg, some drugs, organic acids, and organic bases) into the filtrate.

In the loop of Henle, the filtrate (urine) becomes highly concentrated. This part of the nephron is composed of a thin-walled descending portion and a thick-walled ascending portion. Loops of Henle belonging to juxtamedullary nephrons dip into the medulla of the kidney, which contains a highly concentrated interstitial fluid (ISF). The thin walls of the descending portion are quite permeable. This permeability, together with the high concentration of the ISF at this point, causes water to move by osmosis from the filtrate into the ISF. This makes the filtrate quite concentrated by the time it reaches the ascending limb of the loop.

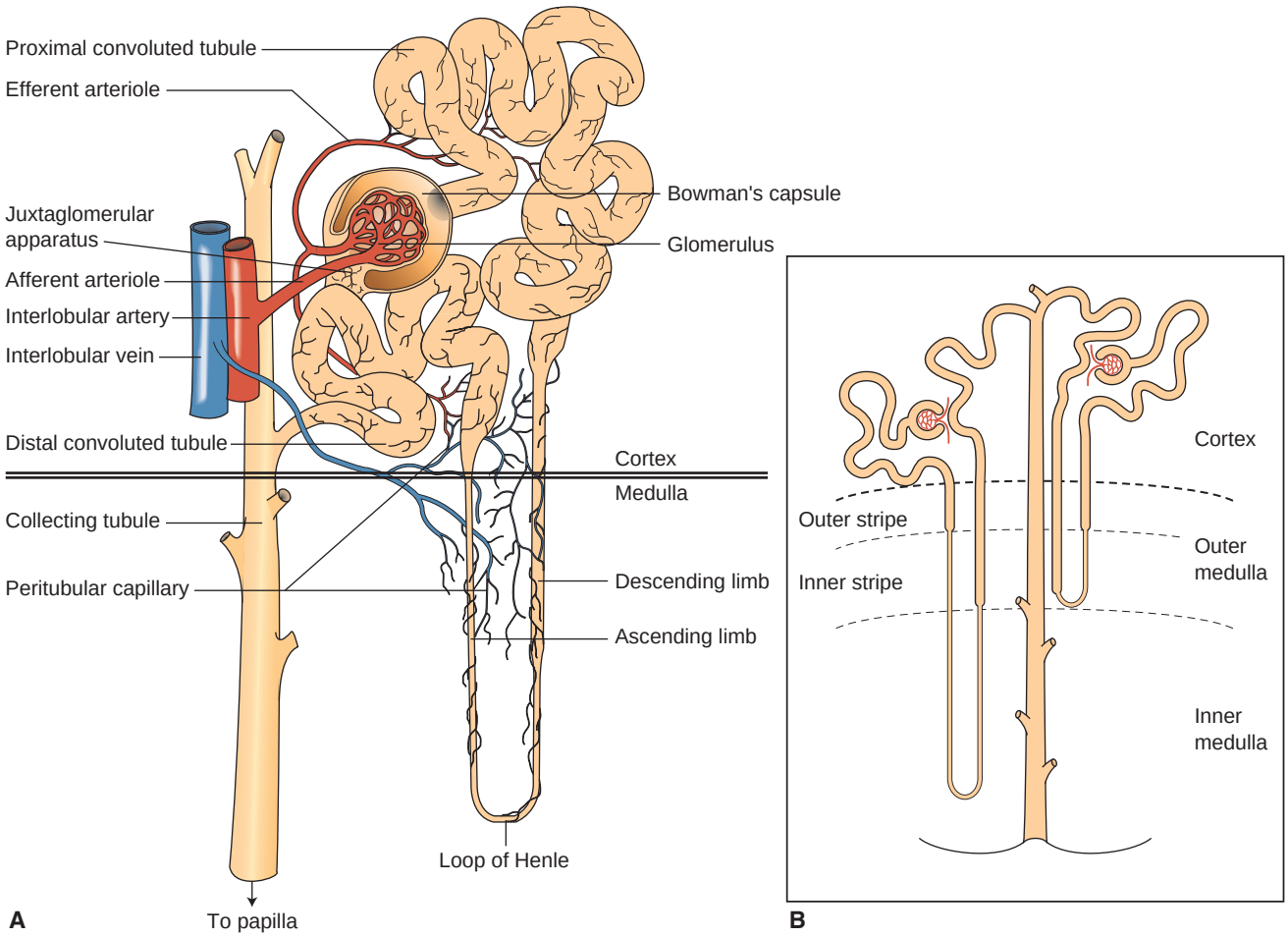


FIGURE 28-3 ▲ **A:** Nephron, showing the glomerular and tubular structures along with the blood supply. **B:** Comparison of differences in location of tubular structures of the cortical and juxtamedullary nephrons. (From Porth CM: Pathophysiology: Concepts of Altered Health States, 9th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 743.)

The thicker-walled ascending limb is relatively impermeable to water. It contains ion carriers that actively transport chloride ions out of the filtrate, which creates an electrochemical gradient that "pulls" the positively charged sodium ions from the filtrate. This exit of electrolytes without water now makes the filtrate more dilute than before.

In the distal tubule, sodium again is reabsorbed by active transport, and hydrogen, potassium, and uric acid can be added to the urine by tubular secretion.

The collecting ducts receive the contents from many distal tubules. At this point, there is no further electrolyte reabsorption or secretion. In the well-hydrated person, there is also no further water reabsorption. Water reabsorption without electrolyte reabsorption can occur in the collecting ducts under the stimulus of antidiuretic hormone (ADH).

Juxtaglomerular Apparatus

The nephron is arranged so that the initial portion of the distal tubule lies at the juncture of the afferent and efferent arterioles, which is very near the glomerulus. Here, macula densa cells of the distal tubule lie in approximation to the juxtaglomerular cells of the wall of the afferent arteriole. Both these cell types (juxtaglomerular and macula densa cells) plus some connective tissue cells constitute the juxtaglomerular apparatus (Fig. 28-4). A major function of the juxtaglomerular cells is to secrete renin, which thereby initiates the renin–angiotensin–aldosterone system.[2] When a decrease in sodium chloride concentration is sensed, the macula densa cells initiate two signals: one signal to reduce afferent arteriole tone (increasing afferent arteriole hydrostatic pressure) and a second to increase renin release from the juxtaglomerular cells.[2] In this manner, the juxtaglomerular apparatus helps maintain and promote glomerular filtration.

Glomerulus

The glomerulus consists of a tuft of capillaries fed by the afferent arteriole and drained by the efferent arteriole. The glomerulus is surrounded by Bowman's capsule. High hydrostatic pressure in the afferent arteriole causes rapid filtration. Fluid that is filtered from the capillaries into this capsule then flows into the tubular system, which is divided into four

Table 28-1 Nephron Functions

Nephron Structure	Function	Concentration of Filtrate Along the Nephron
Glomerulus	Free filtration of blood through Bowman's capsule to produce filtrate Hydrostatic and osmotic pressure forces create net filtration pressure	Isosmotic
Proximal convoluted tubule	Reabsorbs sodium, potassium, calcium, glucose, ketone bodies, and amino acids by active transport Reabsorbs chloride and bicarbonate by electromechanical gradient Reabsorbs water by osmosis Reabsorbs urea by diffusion	Isosmotic
Loop of Henle		
Thin descending limb	Reabsorbs sodium by active transport Further reabsorbs chloride by electromechanical gradient Reabsorbs water by osmosis Reabsorbs urea by diffusion	Isosmotic
Thick ascending limb	Reabsorbs sodium and chloride by active transport Blocks water reabsorption at thick ascending limb Reabsorbs bicarbonate by electromechanical gradient	Hypo-osmotic
Distal tubule	Reabsorbs sodium by active transport and aldosterone Reabsorbs water by osmosis Reabsorbs phosphorus, chloride, and bicarbonate by electromechanical gradient	Hypo-osmotic
Collecting tubule	Antidiuretic hormone promotes selective water reabsorption Secretes or reabsorbs bicarbonate and hydrogen ions to maintain pH Secretes potassium and hydrogen ions depending on body requirements or effects of drugs Secretes some creatinine Actively reabsorbs potassium	Depends on body requirements for fluid

sections: the proximal tubule, the loop of Henle, the distal tubule, and the collecting duct (see Fig. 28-3). Lower hydrostatic pressure in the efferent circulation allows reabsorption. Most of the water and electrolytes are reabsorbed into the blood in the peritubular capillaries that surround the tubular structures. The end products of metabolism remaining in the tubules pass into the urine.

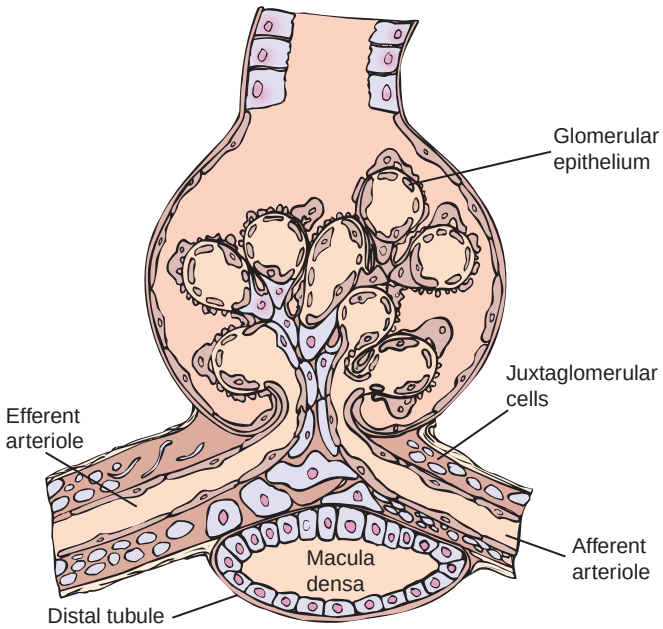


FIGURE 28-4 ▲ The juxtaglomerular apparatus. The macula densa cells lie in close proximity to the afferent and efferent arterioles, which help to regulate nephron functions.

Glomerular filtration is determined by net filtration pressure. Hydrostatic pressure and osmotic pressure forces are major factors. Hydrostatic pressure is driving or "pushing" pressure. Osmotic pressure is the pressure exerted by water (or any solvent) on a semipermeable membrane as it attempts to cross the membrane into an area containing more molecules that cannot cross the semipermeable membrane. The pores in the glomerular capillary make it a semipermeable membrane that permits smaller molecules and water to cross but prevents larger molecules (eg, plasma proteins) from crossing. Protein concentrations are the greatest factors in determining an osmotic pressure, and therefore, osmotic pressure is often referred to as colloid osmotic pressure. Four forces are considered when determining net filtration of fluid. (Fig. 28-5).

The rate at which the filtrate is formed is the GFR. Major clinical factors that influence the GFR are the blood hydrostatic pressure and the filtrate osmotic pressure. Hypoproteinemia, as in starvation, lowers filtrate osmotic pressure and increases the GFR.[3] The GFR decreases with severe hypotension because of a drop in blood hydrostatic pressure, when autoregulatory control may be lost. Other factors that decrease the hydrostatic pressure (and therefore the GFR) are afferent arteriole constriction and renal artery stenosis.

From the 20% to 25% of the cardiac output that goes to the kidneys in a resting adult, about 125 mL of filtrate is pro-

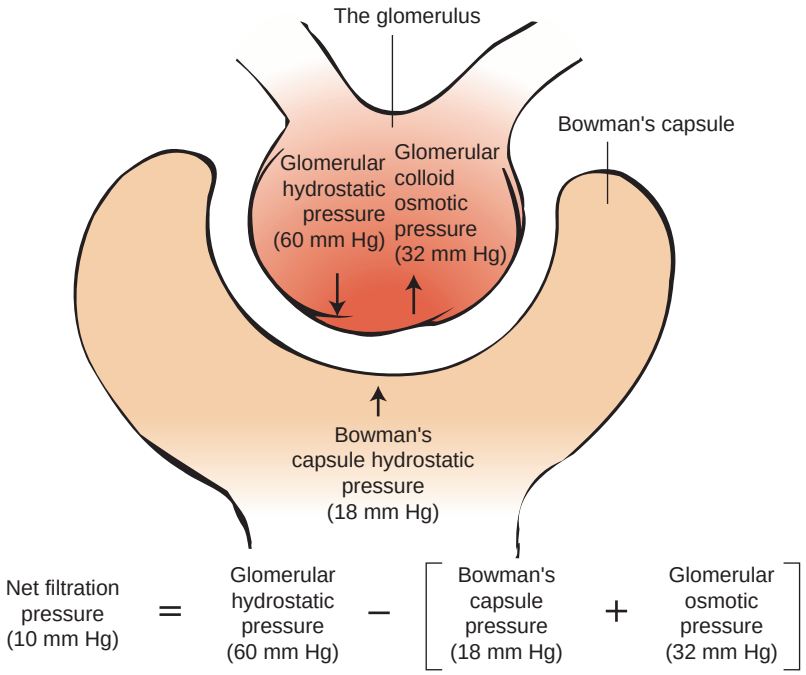


FIGURE 28-5 ▲ Interaction of hydrostatic and osmotic forces for glomerular filtration at Bowman's capsule.

duced each minute. This totals 180 L/d and is about 4.5 times the total amount of fluid in the body. Obviously, not all this filtrate can be excreted as urine. As this filtrate passes from Bowman's capsule through the remainder of the nephrons, all but about 1.5 L/d returns to the bloodstream. Similarly, at plasma glucose levels of less than 200 mg/dL, none of the filtered glucose is found in the urine when it enters the collecting tubules. The volume and content of the urine are the result of tubular reabsorption and tubular secretion.

Tubules

Tubular Reabsorption

Reabsorption is accomplished by active transport, osmosis, and diffusion. It occurs in all parts of the nephron as substances moving from the lumen into the peritubular capillaries.

ACTIVE TRANSPORT. Active transport involves the binding of a molecule of a substance to a carrier, which then moves the molecule from one side of the membrane to the other against the concentration gradient of that substance. Because it helps molecules to move in a direction opposite the one they would move by simple diffusion, the carrier acts like a pump. Many processes for active transport use the sodium–potassium pump. Therefore, the small oxygen requirements of the kidneys are closely linked to the active transport processes that occur in the nephron.

In tubular cells, the carrier is located in the cell membrane nearest the peritubular capillaries, and it transports material out of the tubular cell into the peritubular fluid. This lowers the intracellular concentration of the type of molecule being transported. The decreased concentration enables more of those molecules to diffuse from the urine (filtrate) into the tubule cell. These molecules, in turn, exit the cell and enter the peritubular fluid by active transport. The movement of molecules increases the peritubular fluid concentration of the molecule, and this increase stimulates the diffusion of the molecule into the peritubular capillaries. In the nephrons, reabsorption by active transport removes molecules from the filtrate (urine) back to the bloodstream.

Because active transport involves carrier molecules and energy exchanges, there is an upper limit to the number of molecules of a substance that can be transported at one time. This maximal limit for reabsorption rates is called T_{max}. Glucose is an example of a molecule that appears in the same concentrations that it appears in the blood. As serum glucose rises, filtrate glucose also rises. The renal tubules reabsorb the filtered glucose at faster and faster rates, until all of this molecule's active transport mechanisms are being used. At this T_{max}, more glucose is appearing in the filtrate than can be reabsorbed, and glucose is excreted in the urine. This "spilling" of glucose into the urine indicates serum levels higher than T_{max}.

OSMOSIS. The active transport of sodium is responsible for the osmotic reabsorption of water from the filtrate in the proximal (and later, in the distal) tubule. As sodium ions are actively transported out of the cell and into the peritubular fluid, they make the osmotic pressure of this peritubular fluid higher than that of the cellular or tubular fluid. Water is thereby osmotically "pulled out" of the tubular fluid. Both water and sodium then diffuse into peritubular capillaries and are returned to the bloodstream. This movement of positively charged sodium ions also creates an electrochemical gradient that draws negatively charged ions (especially chloride) out of the tubular fluid and back into the bloodstream.

DIFFUSION. Urea is an example of a molecule that is reabsorbed by diffusion. Under the high pressures in the glomerular capillaries, urea is filtered. In the tubules, as water is reabsorbed into the bloodstream, urea follows by simple diffusion. No selective permeability prevents its return to the bloodstream, and no transport mechanism is required. The

reabsorption rates of urea range from 40% to 60% of what is filtered and depend entirely on water reabsorption rates.

Tubular Secretion

Secretion involves active transport and is performed only by distal tubule cells. Substances move from the peritubular capillaries through tubule cells into the filtrate. Many substances that are secreted do not occur naturally in the body (eg, penicillin). Naturally occurring bodily substances that are secreted include uric acid, potassium, and hydrogen ions.

In the distal tubule, the active transport of sodium uses a carrier system that is also involved in the tubular secretion of hydrogen and potassium ions. In this relationship, every time the carrier transports sodium out of the tubular fluid, it also carries either a hydrogen ion or a potassium ion into the tubular fluid on its "return trip." Thus, for every sodium ion reabsorbed, a hydrogen or potassium ion must be secreted, and vice versa. The choice of cation to be secreted depends on the ECF concentration of these ions (hydrogen and potassium).

This cation exchange system in the distal tubule helps explain some of the relationships that these electrolytes have with one another. For example, it is clear why an aldosterone blocker (such as spironolactone) may cause hyperkalemia. The aldosterone blocker reduces sodium reabsorption. Such reduced reabsorption of sodium also reduces the tubular secretion of either hydrogen or potassium. The hydrogen excess can be buffered by bicarbonate, but the potassium simply rises to above-normal levels, leading to hyperkalemia. Similarly, the cation exchange system helps explain why there can be an initial decrease in plasma potassium in alkalosis or as severe acidosis is corrected therapeutically. In severe acidosis, the nephrons attempt to compensate by increasing hydrogen ion secretion. But as acidosis is therapeutically corrected (eg, by sodium bicarbonate administration), potassium ions are secreted. As hydrogen ions no longer need to be secreted, potassium ions become the sole exchange for sodium ions, leading, it is thought, to a reduction in plasma potassium.

▲ Hormonal Influences

Through the reabsorption of sodium and the passive "following" of water and chloride, it is possible to make urine of the same osmolality as blood. However, under conditions of dehydration, urine is very concentrated, whereas if a great deal of water is consumed, urine is more dilute than blood. This final regulation of urine (and, therefore, serum osmolality and volume) is under the influence of three hormones: ADH, renin, and aldosterone.

Antidiuretic Hormone

Osmoreceptors in the hypothalamus are sensitive to serum osmolality. During dehydration, when serum osmolality rises, osmoreceptors in the hypothalamus respond by stimulating the hypothalamus to secrete ADH, which increases the permeability of collecting tubule cells to water. This permits the reabsorption of water alone (without electrolytes), which in turn decreases the concentration of the ECF. Negative feedback loops regulate ADH secretion. This means that as the concentration of the ECF returns to normal, the stimulus for ADH secretion disappears, and ADH secretion stops.

Renin

Another hormone that influences urine concentration is renin. When the GFR falls because of dehydration or blood loss, the juxtaglomerular apparatus secretes renin.[1] Subnormal sodium levels in the filtrate also stimulate renin secretion. Renin converts angiotensinogen, which is secreted by the liver, into angiotensin I. Pulmonary capillary cells in turn convert angiotensin I into angiotensin II with angiotensin converting enzyme (ACE) (see Chapter 42, Fig. 42-9). Angiotensin II constricts the smooth muscle surrounding the arterioles. This increases blood pressure, which increases the GFR.

Aldosterone

Angiotensin II also triggers the secretion of aldosterone by the adrenal cortex (see Chapter 42, Fig. 42-9). Aldosterone is the third substance that influences urine osmolality. By increasing sodium reabsorption in distal tubule cells, aldosterone causes an increase in renal water reabsorption. This increases blood pressure and decreases serum osmolality. Simultaneously, potassium is excreted in the urine in exchange for the sodium reabsorption. Therefore, aldosterone also is secreted in response to subnormal serum sodium and elevated potassium levels.

▲ Functions of the Renal System

Renal Clearance

From the previous discussion, an important concept in renal function emerges: clearance. As the filtrate moves along the nephron, it contains a large proportion of metabolic end products. These products are removed (cleared) from the blood and exit the body in the urine. Indeed, of each 125 mL of glomerular filtrate formed per minute, about one half, or 60 mL, returns to the blood without urea, and about one half is excreted with urea. Stated another way, 60 mL of plasma is "cleared" of urea each minute in normally functioning kidneys. In the same way, the 125 mL of plasma is also cleared of creatinine, uric acid, potassium, sulfate, phosphate, and so forth each minute. It is possible to calculate renal clearance by simultaneously sampling urine and plasma. By dividing the quantity of substance found in each milliliter of plasma into the quantity found in the urine, the milliliters cleared per minute can be calculated. This method is used as one means of testing kidney function.

Other methods of assessing renal function involve chemicals that are known to be filtered only, or both filtered and secreted. The polysaccharide, inulin, for example, is filtered only and neither absorbed nor secreted. Therefore, the clearance of inulin provides a measure of glomerular filtration. The sodium concentration in the urine can also serve as an index of tubular health in certain situations. For example,

in acute renal failure, an increased clearance of sodium can indicate acute tubular necrosis. Accordingly, supernormal blood levels of filtered substances (creatinine and other nitrogenous wastes) indicate a decrease in glomerular filtration and therefore in nephron health.

Regulation

In addition to excreting nitrogenous wastes as urea and other by-products of metabolism, the kidneys help regulate the electrolyte concentration and the pH of the ECF (ie, the blood and ISF of the body).

Electrolyte Concentration

Electrolytes are substances that, when in water, disassociate and become charged. When charged, the solution is capable of carrying an electrical current. Positively charged electrolytes are cations; negatively charged electrolytes are anions. Most electrolytes are dissolved in body fluids, although some are bound to proteins or deposited as solids to form bones and teeth.

Despite the complex physiology associated with electrolytes, they have four main functions in homeostasis:

1. Cell metabolism and contribution to body structures
2. Facilitation of water movement between body compartments
3. Help in the maintenance of acid–base balance
4. Maintenance and production of membrane potentials in nerve and muscle cells

The functions of individual electrolytes are given in Table 28-2. For normal functions to occur, the concentration of electrolytes must be carefully maintained. Energy, usually in the form of adenosine triphosphate (ATP), is often required to maintain this balance. As described earlier in this chapter, the kidneys play a crucial role in electrolyte balance. In addition to being lost in the urine, electrolytes are lost from the gastrointestinal tract in the stool and emesis and through the skin in sweat.

SODIUM. The sodium content of a normal adult is approximately 142 mEq/L of ECF. As the most abundant extracellular electrolyte, sodium exerts an extracellular osmolality, thereby regulating movement of body fluids. Sodium plays a role in nerve impulses through active transport and the sodium–potassium pump. The balance of sodium is carefully regulated by the kidneys, with influences of aldosterone and ADH. Regulation occurs primarily through reabsorption (or excretion) in the proximal tubule under the influence of aldosterone.

POTASSIUM. In contrast to sodium, potassium is the most abundant intracellular electrolyte, with an approximate plasma concentration of only 4.5 mEq/L. Potassium, among other substances and factors, is critical in the maintenance of nervous and impulse conduction in the heart. Because of the small plasma concentration, potassium plays little role in osmotic regulation. Although some potassium may be lost in sweat and feces, the kidneys excrete approximately 80% to 90% of the potassium lost by the body. In cases of hyperkalemia, aldosterone release facilitates increased potassium excretion. Potassium also assists with acid–base regulation through the cellular exchange with hydrogen ions.

Table 28-2 Electrolyte Functions

Electrolyte	Normal Range	Functions
Sodium (Na^+)	135–145 mEq/L	Exerts an extracellular osmolality, thereby regulating movement of body fluids Facilitates nerve impulses through active transport and the sodium–potassium pump
Potassium (K^+)	3.5–5 mEq/L	Maintains nervous impulse conduction in the heart Promotes skeletal muscle function Plays small role in osmotic regulation Assists with acid–base regulation
Chloride (Cl^-)	100–110 mEq/L	Maintains electroneutrality by passively following the positively charged ions Helps regulate osmotic pressure differences between intracellular and extracellular fluid compartments Regulates body water balance with sodium Combines with H^+ in gastric mucosal cells to make hydrochloric acid
Calcium (Ca^{2+})	8.5–10.0 mg/dL (total) 4.4–5.4 mg/dL (ionized)	Major structural component of bones and teeth Plays role in blood coagulation Promotes muscle contraction and nervous impulse transmission Decreases neuromuscular irritability
Phosphorus (PO_4^-)	2.5–4.5 mg/dL	A structural component of bones and teeth Helps maintain acid–base balance Energy production (adenosine triphosphate) Delivery of oxygen to tissues as a component of 2,3-diphosphoglycerate
Magnesium (Mg^{2+})	1.8–2.5 mEq/L	Ensures the cross-membrane transport of sodium and potassium in the sodium–potassium pump Promotes neuromuscular excitability Plays role in heart contraction Facilitates transmission of central nervous system impulses Part of many enzymatic reactions for carbohydrate and protein metabolism

CHLORIDE. Chloride is the most abundant extracellular anion. Negatively charged, chloride passively follows the positively charged sodium to maintain electroneutrality. Chloride plays an important role in electroneutrality because all positive charges and negative charges must be in equilibrium. Therefore, chloride passively follows the secretion or reabsorption of the predominant cations, sodium and potassium. A large amount of chloride is also found in the gastric mucosal cells in the form of hydrochloric acid.

CALCIUM. Calcium has both structural and functional roles in homeostasis. In the form of calcium phosphate, it is the major structural component of bones and teeth. In the free, plasma form, calcium has a function in clotting, muscle contraction, and nervous impulse transmission. In the ionized form, about half is bound to plasma proteins, such as albumin.

Unlike the other electrolytes, calcium is absorbed from the small intestine under the influence of vitamin D, with the remaining ingested calcium lost in the feces. Excretion also occurs in the proximal convoluted tubule of the kidneys.

Parathyroid hormone (PTH) is produced and released by the parathyroid glands. A low calcium concentration stimulates its release. PTH facilitates the shift of calcium in its solid form (calcium phosphate, found in the bones) to its ionized form. PTH also increases the calcium absorbed from the intestine by signaling the kidneys to activate vitamin D. Reabsorption of calcium at the renal tubules is also increased under the influence of PTH. Calcitonin, secreted by the thyroid gland, is another hormone that plays a comparatively small role in calcium regulation. Calcitonin acts in opposition to PTH in an effort to reduce plasma calcium levels.

PHOSPHORUS. Like calcium, phosphorus has both structural and functional roles. Approximately 85% is found in the organic form in bones and teeth. The remaining phosphorus is in the inorganic, ionized forms, HPO_4^{2-} and $H_2PO_4^-$. Carried in two forms, phosphate is able to accept or donate an ion, thereby assisting with acid–base balance. The intracellular ionized form plays a role in many critical metabolic processes, with its primary function being the formation of ATP. Phosphorus is required for the delivery of oxygen to tissues because it is the primary substrate for 2,3-diphosphoglycerate.

PTH regulates phosphorus, with effects directly opposite those of calcium. PTH causes an increase in calcium plasma concentration and promotes excretion of phosphorus. PTH also causes release of phosphorus in the bones and shifts it to the ECF. Presumably, this would cause an increase in phosphorus; however, PTH also decreases the transport of phosphate ions by the kidney tubules, so more phosphate ions are lost in the urine.

MAGNESIUM. Magnesium is a predominantly intracellular ion. Magnesium ensures the cross-membrane transport of sodium and potassium in the sodium–potassium pump. In addition, magnesium functions in the maintenance of neuromuscular excitability and in the transmission of central nervous system impulses. It also plays a role in enzymatic reactions for carbohydrate and protein metabolism. Often, reactions requiring calcium require magnesium as well.

pH. If respiratory buffers for pH regulation are insufficient, the kidneys begin to take part, although much more slowly than the lungs. Although respiratory control of carbon dioxide, and therefore hydrogen ion levels, can take only seconds to achieve, 48 to 72 hours may pass before the renal system can change the serum acid–base balance significantly.

Alkalosis occurs as a result of too few hydrogen ions or too many bicarbonate ions. To compensate, the body must conserve hydrogen ions. In renal compensation for alkalosis, tubular reabsorption of hydrogen ions is increased and secretion is decreased. This increases the hydrogen ion concentration of the ECF and thereby decreases the alkalosis.

Acidosis occurs as a result of too many hydrogen ions or too few bicarbonate ions. To compensate, the body must secrete hydrogen ions. Renal compensation for acidosis involves increasing the hydrogen ion secretion of the tubule cells, especially in the distal tubule cells. In this case, bicarbonate and sodium ions are continually being filtered from the glomerulus. Also, hydrogen ion secretion by distal tubule cells causes an increase in sodium reabsorption. Such sodium reabsorption can increase bicarbonate reabsorption electrochemically. Therefore, as hydrogen ions are being eliminated from the ECF, sodium and bicarbonate ions are being added to it. Both serve to decrease the acidosis (Fig. 28-6).

Urine can be acidified (by hydrogen ion secretion) only to a pH level of 4.0 to 4.5. If the tubular secretion of hydrogen ions was the only mechanism operating, only a few hydrogen ions could be secreted before the critical shut-off level of 4.0 was reached. This would occur because hydrogen would combine with urinary chloride to make hydrochloric acid. Not many of these strong hydrochloric acid molecules are needed to make the urine pH 4.0. The formation of hydrochloric acid would then stop tubular hydrogen ion secretion before sufficient compensation for acidosis could be obtained. This does not occur because tubule cells deaminate certain amino acids and secrete the nitrogenous component as ammonia. This ammonia combines with hydrogen in the urine to form ammonium. Because tubule membranes are not permeable to ammonium, much of it is secreted in this form. Some ammonia combines with chloride to form ammonium chloride.

Fluid Balance

The body contains about 60% water in most individuals.[4] This percentage may vary between 50% and 70%, depending on a person's fat content. Adipose tissue has very low water, and therefore people with more fat have a lower percentage of body weight as water. Women have a tendency to have higher body fat percentages than men and therefore have lower total-body water (usually around 50%).[4]

Water is distributed among the two main compartments in the body: intracellular fluid (ICF) and ECF. The ICF is the amount of volume within the cell and makes up about two thirds of the total-body water, or about 40% of the body weight. Primarily, the ICF is a solution of ions, including potassium, proteins, and organic ions. The ECF represents the remaining one third of the body water, or about 20% of the body weight. The solution primarily contains sodium chloride and bicarbonate. Figure 28-7 illustrates the different body water compartments.

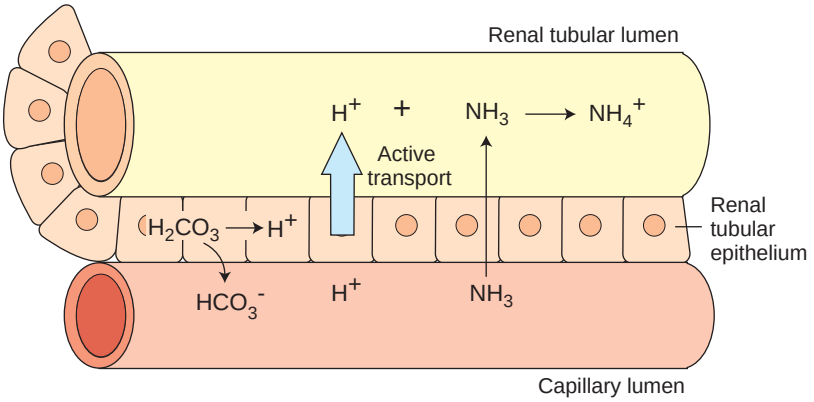


FIGURE 28-6 ▲ Renal compensation for acidosis. Hydrogen (H^+) is moved from blood into the filtrate by active transport and exits in the urine as ammonium (NH_4^+). HCO_3^-, bicarbonate; NH_3, ammonia.

There are three subcompartments to the ECF: the ISF, the plasma, and the transcellular fluid. ISF surrounds the cells but does not circulate. This subcompartment makes up about three fourths of the ECF. The second subcompartment of the ECF is the plasma, which circulates as the extracellular component of blood. Plasma makes up about one fourth of the ECF.

The third subcompartment of ECF is called transcellular fluid. This fluid is neither in the plasma nor in the interstitium; instead, it is the fluid that makes up the digestive juices, cerebrospinal fluid, synovial fluid, pericardial fluid, and mucus. Although the transcellular fluid is only about 1 to 2 L in total (<1 pound), it plays a very important role in homeostasis. Transcellular fluid helps cushion the heart with each beat, makes joint movement smooth, carries critical oxygen and glucose to the brain, and removes bacteria and antigens from the respiratory tract.

There is a constant movement of water between body compartments. For example, in diseases in which there is a lack of plasma oncotic pressure (eg, liver disease), there may be excessive movement of fluid from the plasma to the interstitium. This additional ISF is reabsorbed by the lymph for recirculation; however, the volume that the lymph system is capable of holding becomes overwhelmed. This then causes edema formation. During times of dehydration, hormonal influences are recruited to pull additional ICF, ISF, and transcellular water into the plasma to maintain effective circulating volume.

Several factors may influence body water. Body water moves between compartments and is regulated by hormones, such as ADH, aldosterone, and atrial natriuretic peptide. Approximately 2.5 L of water are lost each day through normal bodily functions, such as urination, defecation, respiration, and sweating. This volume of water lost must be replaced. As people age, there is a decrease in total-body water as the ratio of muscle to fat changes. As fat increases, total-body water decreases. In part, this physiological change accounts for the older adult's propensity for dehydration.

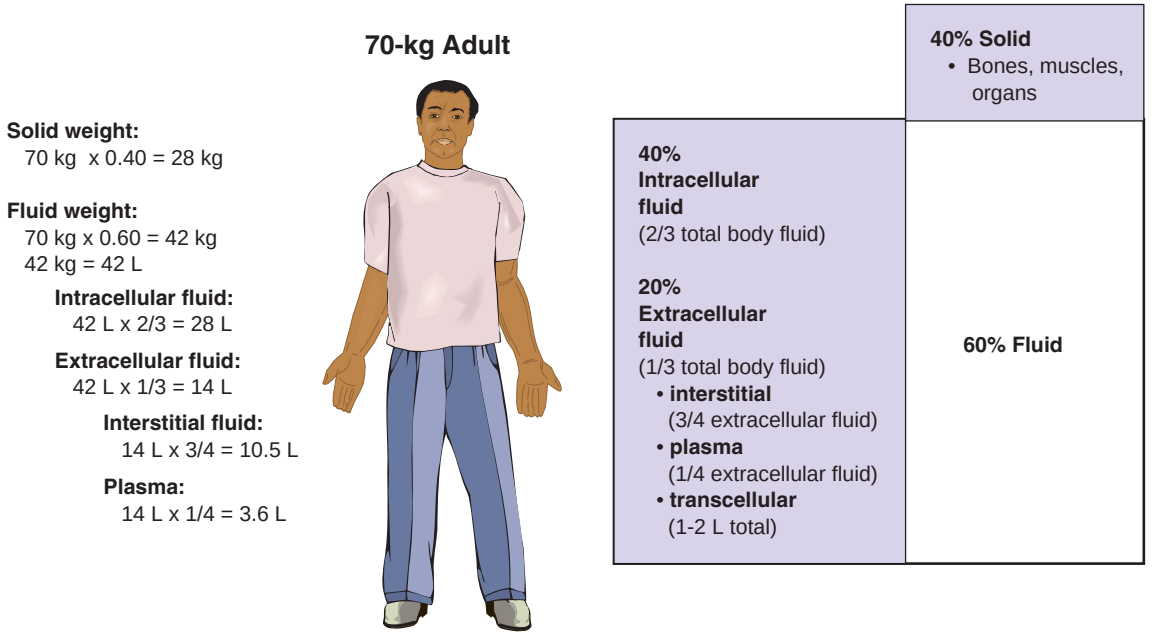


FIGURE 28-7 ▲ Body water compartments.

Other Renal Functions

Renal interstitial (not nephron) cells manufacture and secrete two hormones, calcitriol (vitamin D) and erythropoietin, the actions of which are unrelated to urine formation. Calcitriol is a hormone that increases plasma calcium concentration by increasing intestinal absorption of calcium, promoting bone resorption, and stimulating the renal tubular reabsorption of calcium. Erythropoietin is a glycoprotein hormone that stimulates the bone marrow to produce red blood cells. Any process that decreases the oxygen content in the blood, such as bleeding or hypoxemia, is sensed by the kidney and initiates the release of erythropoietin. This increases the arterial oxygen content required to maintain cell integrity.

The kidneys also activate vitamin D. Ingested with food, vitamin D is absorbed in an inert form. The kidneys activate vitamin D so that it may assist in the absorption of calcium, which occurs in the intestines. Calcium has many functions as discussed earlier in this chapter. If renal failure occurs, there is a marked reduction in vitamin D and subsequently bioavailable calcium, thereby putting the patient at risk for bone diseases (such as osteoporosis) and bleeding.

▲ Clinical Applicability Challenges

SHORT ANSWER QUESTIONS

1. Describe the role of the macula densa in autoregulation.
2. A 65-year-old patient is admitted to your unit. On your health history, you note a history of elevated blood glucose levels. The patient states that his primary provider has him checking his urine for "sugar" on office visits, but he cannot understand why he needs to do that since he was told that he had elevated sugar levels in the blood. Describe the physiological processes involved in using the urine glucose level as an estimate for serum glucose level.

References

1. Guyton AC, Hall JE: Textbook of Medical Physiology, 12th ed. Philadelphia, PA: WB Saunders, 2010
2. Gradman AK, Kad R: Renin inhibition in hypertension. J Am Coll Card 51(5):519–528, 2008
3. Valencia E, Marin A, Hardy G: Nutrition therapy for acute renal failure: A new approach based on 'risk, injury, failure, loss and end-stage kidney' classification (RIFLE). Curr Opin Clin Nutr Metab Care 12(3): 241–244, 2009
4. Ricci Z, Ronco C: Year in review: Nephrology. Crit Care 13(5): 227–233, 2008

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

29 Patient Assessment: Renal System

Kara Adams Snyder and Kelley Caldwell Crusius

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Formulate a plan for collecting history and physical examination data for patients with renal disorders and fluid and electrolyte imbalance.
2. Describe diagnostic and laboratory blood tests used to evaluate renal and electrolyte status.
3. Discuss methods to evaluate fluid balance.

Assessment of the renal system involves determining how well the kidneys perform their many functions. It also includes gathering information about other systems. A careful assessment of the history and physical findings, with interpretation of laboratory and diagnostic test results, provides early clues to the diagnosis of disorders of water and volume imbalance and other complications of renal dysfunction in the critically ill patient.

▲ History

The patient history provides important information that helps determine the cause, severity, treatment, and management of renal dysfunction. It involves gathering information about the present illness, past health history, family history, personal history, and social history. In addition, relevant information about the status of other body systems is gathered through a review of systems. A guide for renal assessment is summarized in Box 29-1.

▲ Physical Examination

The physical examination provides objective data that are used to substantiate and clarify the history. The nurse begins the physical examination by observing the patient's overall appearance, including facial expression, height and weight, position in bed, grooming, personal hygiene, and signs of distress. The nurse observes the patient's level of responsiveness, cognition, and interaction with people, including positive, negative, or unusual responses.

Because patients with renal problems usually have significant problems with fluid and electrolyte balance, the nurse evaluates the patient's volume status throughout the examination. The nurse begins by taking the vital signs. Particular attention is paid to the blood pressure, noting pulse pressure and presence of a positive pulse paradoxus. An elevated temperature may indicate an infection.

Throughout the physical examination, the nurse inspects the skin on the extremities and trunk for color and evidence of excoriation, bruising, or bleeding; palpates for moistness, dryness, temperature (using the back of the fingers), and edema; and checks mobility and turgor by lifting a fold of skin and noting the ease (mobility) and speed with which it returns into place (turgor). To assess hydration further, the nurse inspects the tongue and mucous membranes in the mouth and looks for a saliva pool under the tongue. Additional volume status assessment is done when examining the neck, as the nurse observes for jugular vein distention and determines the need to measure jugular venous pressure.

The anterior and posterior chest is inspected for respiratory rate, rhythm, depth, and effort. Deformities of the thorax, shape of chest, or bulging of interspaces during expiration are noted. The precordial area is observed and palpated for heaves, pulsations, and thrills. The nurse listens for heart rate and rhythm, extra heart sounds, murmurs, clicks, and pericardial friction rub. Fluid overload often results in the presence of a third or fourth heart sound.

Anterior and posterior lung fields are auscultated. The nurse notes the quality of vesicular breath sounds and the presence of adventitious breath sounds (crackles, wheezes, rubs), which may indicate volume overload.

After auscultating the posterior chest, the nurse can assess kidney tenderness. To do this, the nurse places one hand over the posterior costovertebral angle (CVA). Then, using the fist of the second hand, the nurse gently percusses the CVA and notes whether the patient has discomfort (Fig. 29-1), which is known as CVA tenderness (CVAT).

The nurse inspects the abdomen and then listens for bowel sounds. In addition to auscultating bowel sounds, the nurse auscultates the renal arteries for bruits by placing the stethoscope above and to the left and right of the umbilicus (Fig. 29-2). A bruit is an abnormal sound that resembles a blowing or swishing noise, similar to the sound of a cardiac

BOX 29-1 HEALTH HISTORY for Renal Assessment

Chief Complaint
- Patient's description of the problem

History of the Present Illness
- Complete analysis of the following signs and symptoms (using the NOPQRST format, see Chapter 17, Box 17-1, p. 207).
- Frequency
- Urgency
- Hesitancy
- Burning
- Dysuria
- Hematuria
- Incontinence
- Lower back pain
- Pain with urination
- Change in color, odor, or amount of urine
- Thirst
- Change in weight
- Edema

Past Health History
- Relevant antenatal history and immunizations: prematurity; antenatal use of angiotensin-converting enzyme inhibitors, angiotensin-receptor blockers, or nonsteroidal antiinflammatory drugs (NSAIDs; eg, ibuprofen); ensuring antenatal vaccination against rubella; screening for cytomegalovirus or toxoplasmosis
- Past acute and chronic medical problems, including treatments and hospitalizations: renal failure; renal calculi; renal cancer; glomerulonephritis; Wegener's granulomatosis; polycystic kidney disease; dialysis, including type, frequency, and duration; urinary tract infections; systemic lupus erythematosus; sickle cell anemia; cancer; AIDS; hepatitis C; heart failure; diabetes; hypertension
- Risk factors: age; trauma; heavy use of ibuprofen, naproxen, or acetaminophen; use of heroin or cocaine
- Past surgeries: kidney transplantation, placement of dialysis fistula
- Past diagnostic tests and interventions: urinalysis, cystoscopy, intravenous pyelography, ultrasound of kidneys, renal biopsy, magnetic resonance imaging, diagnostic tests that have used contrast dyes
- Medications: diuretics, aminoglycosides, antibiotics, NSAIDs
- Allergies and reactions: radiographic contrast media
- Transfusions

Family History
- Health status or cause of death of parents and siblings: hereditary nephritis, polycystic kidney disease, diabetes, high blood pressure

Personal and Social History
- Tobacco, alcohol, and substance use
- Family composition
- Occupation and work environment: exposure to nephrotoxic substances such as organic acids, pesticides, lead, and mercury
- Living environment: exposure to nephrotoxic substances such as organic acids, pesticides, lead, mercury
- Diet
- Sleep patterns
- Exercise
- Cultural beliefs
- Spiritual and religious beliefs
- Coping patterns and social support systems
- Leisure activities
- Sexual activity
- Recent travel

Review of Systems
- Skin: dryness, itching
- HEENT: periorbital edema
- Cardiovascular: hypertension, heart failure, vascular disease
- Respiratory: Goodpasture's syndrome
- Gastrointestinal (GI): hepatitis, cirrhosis
- Endocrine: diabetes mellitus
- Neurological: numbness, tingling, burning, tremors, memory loss
- Hematological: sickle cell anemia
- Immune: systemic lupus erythematosus
- Musculoskeletal: rhabdomyolysis, muscle weakness

murmur. The presence of a renal bruit may indicate renal artery stenosis, which means there may be diminished blood flow to the kidney. This diminished blood flow may result in acute or chronic renal dysfunction.

Next, the nurse percusses and palpates the abdomen and then palpates the liver border to determine enlargement. If ascites is suspected, the nurse measures abdominal girth and may check for a fluid wave or shifting dullness. During the abdominal examination, the right and left kidneys are palpated by placing one hand under the patient's flank and placing the other examining hand in the quadrant just below the costal margin at the mid-clavicular line. The kidneys are normally not palpable. An enlarged kidney may be palpable, and the enlargement may be due to a cyst, tumor, or hydronephrosis (Fig. 29-3). If indicated by history, the nurse may palpate and percuss the bladder. The bladder cannot be palpated unless it is distended above the symphysis pubis. When palpated, the dome of the distended bladder feels smooth and round. For palpation and percussion of the bladder, the nurse begins at the symphysis pubis and moves upward and outward to estimate the bladder size. A full bladder is dull to percussion (Fig. 29-4).

If the patient is at risk for excess vascular volume, the nurse looks for hypertension; pulmonary edema; crackles; engorged, elevated neck veins; liver congestion and enlargement; heart failure; and shortness of breath. Signs and symptoms related to excess extravascular volume include pitting edema of feet, ankles, hands, and fingers; periorbital edema; sacral edema; and ascites. Table 29-1 presents a scale used to document levels of pitting edema.

While examining the extremities, the nurse can check the quality of the peripheral pulses; observe for tremors; test for paresthesia, numbness, and weakness; and palpate fingernails and toenails, checking for color, shape, and capillary refill time.

If the patient has an arteriovenous graft or fistula for dialysis, the nurse assesses it for patency and for adequate circulation to the extremity distal to the access. Palpating for a thrill and auscultating for a bruit help to assess patency of the graft. If assessment reveals a change, the physician or practitioner must be notified urgently because the graft may be saved through radiological or surgical intervention. If the patient has temporary dialysis access, the exit site is inspected for signs of inflammation or infection. Often the lumens of the

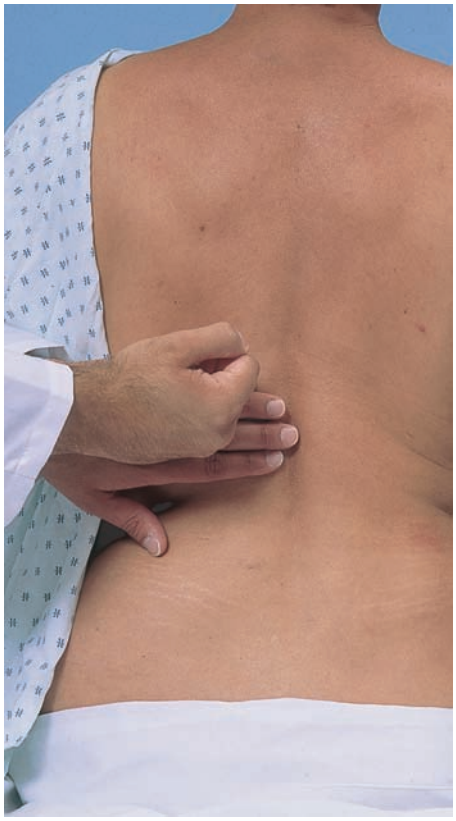


FIGURE 29-1 ▲ Assessing CVA tenderness. (From Bickley LS: Bates' Guide to Physical Examination, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 446.)

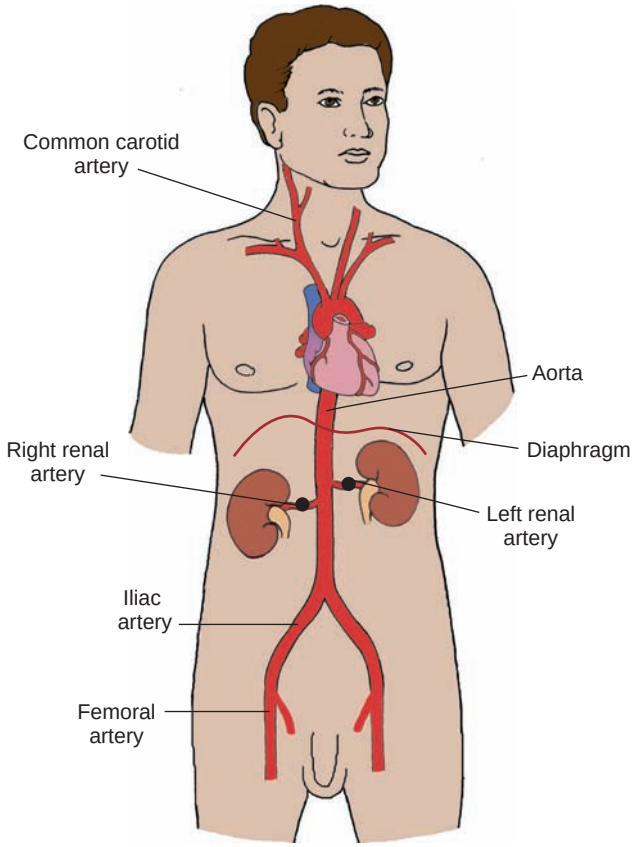


FIGURE 29-2 ▲ Sites for auscultation of renal bruits.

temporary access have high doses of heparin to maintain patency, so flushing or use of the device is clarified with the physician or practitioner before any manipulation or use of the catheter.

Patients with renal impairment may be at risk for hypocalcemia, hypomagnesemia, or both. Physical assessment of these electrolyte changes can be achieved by checking for Chvostek's and Trousseau's signs. Chvostek's sign occurs when there is facial irritability after tapping the facial nerve in front of the auditory meatus with the finger. Trousseau's sign occurs when there is spasm of the hands and feet (carpopedal spasm) in response to arm compression (eg, as with a blood pressure cuff).

During the history and examination, the critical care nurse continuously observes the patient's level of consciousness and mental status. If more data are needed, the nurse may use tools such as the Glasgow Coma Scale and Folstein Mini-Mental Examination.

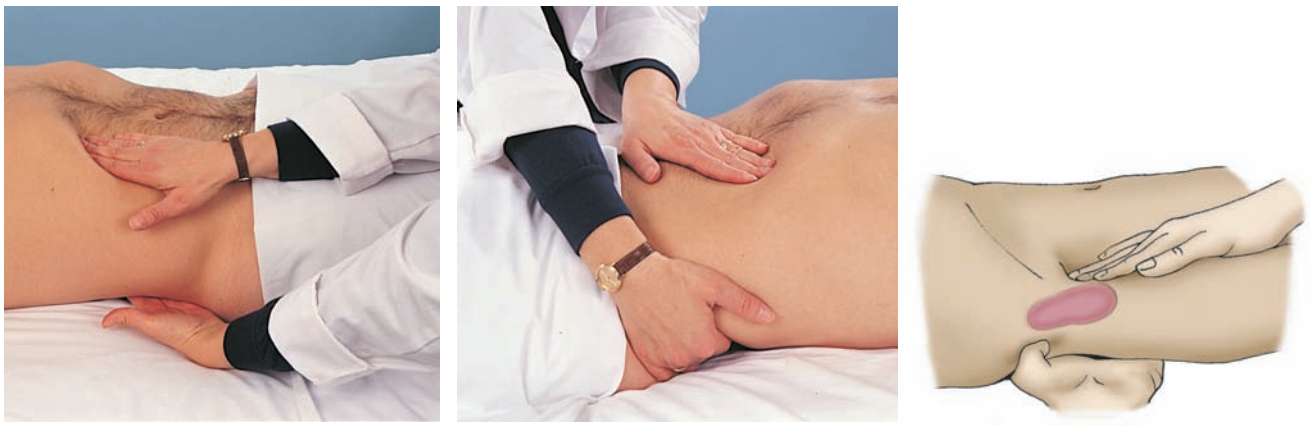


FIGURE 29-3 ▲ Palpating the right and left kidney. (Adapted from Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 437.)

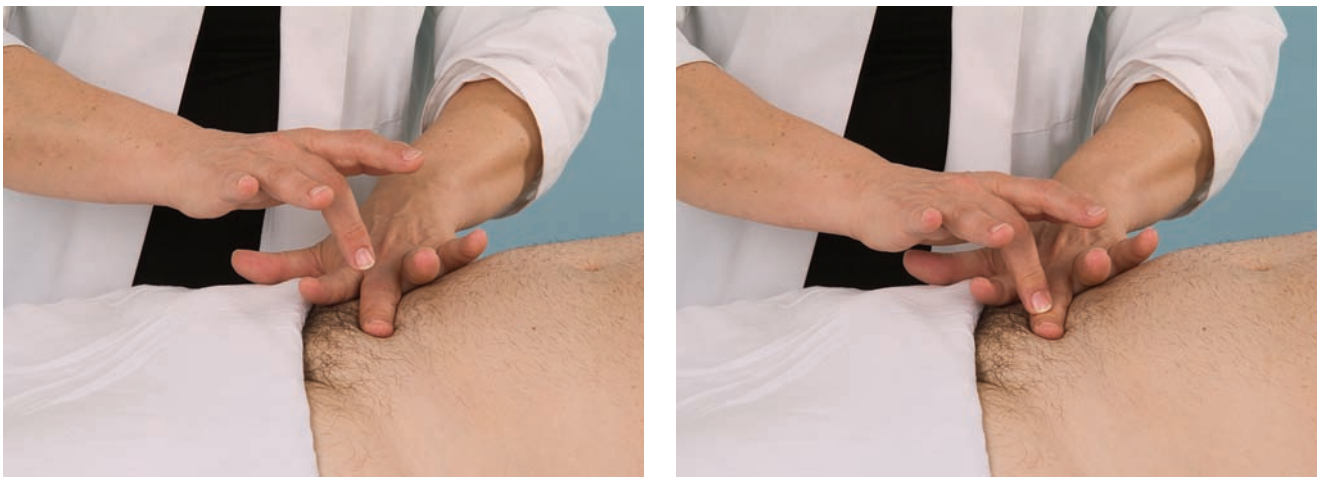


FIGURE 29-4 ▲ Percussing the bladder. (From Rhoads J: Advanced Health Assessment and Diagnostic Reasoning. Philadelphia, PA: Lippincott Williams & Wilkins, 2006, p 305.)

▲ Assessment of Renal Function

Laboratory Studies

Urine Studies

URINALYSIS. The nurse inspects the urine for color, clarity, and odor. Normally, the urine is clear and yellow to straw-colored and smells of ammonia. Changes in the characteristics of the urine can indicate kidney damage, infection, excretion of drugs, or the kidney's compensation for systemic homeostatic imbalance. Cloudy urine may indicate infection, whereas very clear and colorless urine may be a sign of diuresis, either induced pharmacologically or by diabetes insipidus. Blood in the urine may appear bright red or dark brown. If hematuria is present, additional evaluation and follow-up with specialists may be considered to investigate the presence of a malignancy.[1] Urinalysis is used to identify more specifically the components of the urine. Table 29-2 summarizes the components of the urinalysis.

URINE VOLUME. The difference between the glomerular filtration rate (GFR) and the amount of water reabsorbed determines the urine volume. A patient with normal renal function, filters 180 L daily and must reabsorb approximately 179 L. This is the equivalent of roughly greater than 99% of filtered volume. Patients with renal disease and impairment may actually excrete an appropriate amount of urine. For example, a patient with severe renal disease may have a GFR of 10 L but still filter 1 liter or 90% reabsorption. Thus, urine volume is of little diagnostic importance in this setting. However, urine volume is important in the setting of acute anuria. In acute anuria, a patient may be making normal volume and experience an abrupt change in pattern. Causes of acute anuria include:

- Complete bilateral obstruction (ie, abdominal compartment syndrome)
- Glomerulonephritis
- Bilateral vascular occlusion

However, trends in urine production can provide important clues to the body's recruitment of important compensatory responses, as in hypovolemia. The body initiates the renin–angiotensin–aldosterone system to maintain the crucial water balance (see Chapter 28).

URINE pH. Normal urinary pH is acidic, with a range between 5.0 and 6.5, depending primarily on dietary intake. The kidneys play a tremendous role in acid–base balance (see Chapter 28). Clinically, urinary pH is important in two settings. First, an alkaline urinary pH (above 7.5) suggests the presence of a urinary tract infection. Second, a low, or acidic, pH indicates that the kidney may be compensating for a serum acidosis. Physiologically, in this state, the kidneys reabsorb more bicarbonate and excrete more hydrogen ions to buffer the serum acidosis. The urine becomes increasingly acidic (lower pH) when the body is attempting to conserve sodium, as in states of dehydration.

URINE PROTEIN. Most proteins are large molecules and under normal conditions should not penetrate Bowman's capsule. Normal urinary protein levels, therefore, are zero to trace. Proteinuria usually indicates damage to the capillary structures, as in the case of glomerular diseases (glomerulonephritis) and intrarenal acute kidney injury. For diagnostic purposes, a 24-hour sample of urine is used to assess for proteinuria. Single dipstick measurements are not as sensitive and may lead to false-positive values.

Table 29-1 Assessing Pitting Edema

Scale	Description	Depth of Indentation (mm)	Return to Baseline
4+	Severe	8	2–5 min
3+	Moderate	6	1–2 min
2+	Mild	4	10–15 s
1+	Trace	2	Disappears rapidly

From Rhoads J: Advanced Health Assessment and Diagnostic Reasoning. Philadelphia, PA: Lippincott Williams & Wilkins, 2006, p 253.

Table 29-2 What Urinalysis Findings Mean

Test	Normal Values or Findings	Abnormal Findings	Possible Causes of Abnormal Findings
Color and odor	• Straw color	Clear to black	Dietary changes; use of certain drugs, metabolic inflammatory, or infectious disease
	• Slightly aromatic odor	Fruity odor	Diabetes mellitus, starvation, dehydration
	• Clear appearance	Turbid appearance	Renal infection
Specific gravity	• Between 1.005 and 1.030, with slight variations from one specimen to the next	Below-normal specific gravity	Diabetes insipidus, glomerulonephritis, pyelonephritis, acute renal failure (ARF), alkalosis
		Above-normal specific gravity	Dehydration, nephrosis
		Fixed specific gravity	Severe renal damage
pH	• Between 4.5 and 8.0	Alkaline pH (above 8.0)	Fanconi's syndrome (chronic renal disease) urinary tract infection, metabolic or respiratory alkalosis
		Acidic pH (below 4.5)	Renal tuberculosis, phenylketonuria, acidosis
Protein	• No protein	Proteinuria	Renal disease (such as glomerulosclerosis, acute or chronic glomerulonephritis, nephrolithiasis, polycystic kidney disease, and acute or chronic renal failure)
Ketones	• No ketones	Ketonuria	Diabetes mellitus, starvation, conditions causing acutely increased metabolic demands and decreased food intake (such as vomiting and diarrhea)
Glucose	• No glucose	Glycosuria	Diabetes mellitus
Red blood cells (RBCs)	• 0–3 RBCs/high-power field	Numerous RBCs	Urinary tract infection, obstruction, inflammation trauma, or tumor; glomerulonephritis; renal hypertension; lupus nephritis; renal tuberculosis; renal vein thrombosis; hydronephrosis; pyelonephritis; parasitic bladder infection; polyarteritis nodosa; hemorrhagic disorder
Epithelial cells	• Few epithelial cells	Excessive epithelial cells	Renal tubular degeneration
White blood cells (WBCs)	• 0–4 WBCs/high-power field	Numerous WBCs	Urinary tract inflammation, especially cystitis or pyelonephritis
		Numerous WBCs and WBC casts	Renal infection (such as acute pyelonephritis and glomerulonephritis, nephrotic syndrome, pyogenic infection, and lupus nephritis)
Casts	• No casts (except occasional hyaline casts)	Excessive casts	Renal disease
		Excessive hyaline casts	Renal parenchymal disease, inflammation, glomerular capillary membrane trauma
		Epithelial casts	Renal tubular damage, nephrosis, eclampsia, chronic lead intoxication
		Fatty, waxy casts	Nephrotic syndrome, chronic renal disease, diabetes mellitus
		RBC casts	Renal parenchymal disease (especially glomerulonephritis), renal infarction, subacute bacterial endocarditis, sickle cell anemia, blood dyscrasias, malignant hypertension, collagen disease
Crystals	• Some crystals	Numerous calcium oxalate crystals	Hypercalcemia
		Cystine crystals (cystinuria)	Inborn metabolic error
Yeast cells	• No yeast crystals	Yeast cells in sediment	External genitalia contamination, vaginitis, urethritis, prostatovesiculitis
Parasites	• No parasites	Parasites in sediment	External genitalia contamination
Creatinine clearance	• Male (age 20): 90 mg/min/173 m^2 of body surface	Above-normal creatinine clearance	Little diagnostic significance
	• Female (age 20): 84 mL/min/1.73 m^2 of body surface	Below-normal creatinine clearance	Reduced renal blood flow (associated with shock or renal artery obstruction), acute tubular necrosis, acute or chronic glomerulonephritis, advanced bilateral renal lesions (as in polycystic kidney disease, renal tuberculosis, and cancer), nephrosclerosis, heart failure, severe dehydration
	• Older patients: normally decreased concentrations by 6 mL/min/decade		

From Critical Care Nursing Made Incredibly Easy, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, pp 505–506.

URINE GLUCOSE AND KETONES. Glucose, like most proteins, is not present in the urine under normal conditions. Unlike proteins, however, glucose is freely filtered but is reabsorbed in the proximal tubule. Glucose becomes detectable if the serum glucose is elevated (>200 mg/dL) as the filtered load exceeds the kidney's reabsorptive abilities. Findings of glucosuria should be confirmed with serum or capillary blood glucose measurement.

Ketone bodies are byproducts of fat metabolism and are formed in states of insulin deficiency. Three ketone bodies are formed: β-hydroxybutyric acid (the primary ketone formed), acetoacetic acid, and acetone. The latter two ketone bodies are detected in the urine. Acetone may be measured in the serum. A urine sample that is positive for ketones may indicate diabetic ketoacidosis.

URINARY SEDIMENT. Sediment is particulate matter that, when examined, can reveal certain physiological conditions in the renal system. Sediment in general refers to casts, red cells, white cells, epithelial cells, and crystals. Casts are the breakdown products of cellular material formed in the collecting tubules. Urinary stasis, as in prerenal disease, may promote cast formation. Casts can be made up of different types of cells, and thus, the shape, composition, and size of the casts can help in identifying the presence and etiology of a disease.

Red Blood Cells. Red blood cells (RBCs) may be microscopic (microscopic hematuria) or grossly visible (macroscopic hematuria). RBCs enter the urine anywhere along the urinary tract. Any injury or damage to the structures making up the urinary tract can cause hematuria. Kidney stones, trauma, and prostatic disease are examples of extrarenal causes of hematuria (ie, not related to the kidneys).

Microscopic bleeding can be present in glomerular diseases, such as glomerulonephritis. When assessing the results of the urinalysis, take note of the presence of RBC casts and the RBC morphology. Glomerular bleeding is often associated with some type of fragmentation of the RBC, whereas extrarenal bleeding often leaves the cell intact. The presence of RBC casts is virtually diagnostic of glomerulonephritis.

Myoglobin in the urine makes the urine appear red; however, when the urine is inspected under the microscope, there is no evidence of RBCs. Myoglobin is a component of skeletal muscle breakdown, or rhabdomyolysis. Crush injuries or protracted down times are the greatest predictors of this disease. When muscle begins to break down, it releases the myoglobin, which is similar in chemical structure to hemoglobin. Because of its large molecular size, myoglobin blocks the renal tubules, placing patients at very high risk for intrarenal acute kidney injury.

White Blood Cells. White blood cells (WBCs) in the urine (pyuria) usually indicate infection anywhere along the urinary tract. Leukocyte esterase is an enzyme produced by WBCs along the urinary tract that can be detected in the urine. This enzyme is present along the urinary tract as a component of the local immune response. High levels of this enzyme can indicate infection. The presence of nitrites may also aid in the diagnosis of a bacterial infection along the urinary tract.

SPECIFIC GRAVITY AND OSMOLALITY. The specific gravity of the urine tests the kidneys' ability to concentrate and dilute the urine. The specific gravity measures the buoyancy of a solution compared with water and depends on the number of particles in the solution and their size and weight. Three methods are used to obtain this measurement in clinical practice: a multiple-test dipstick that has a reagent area for specific gravity, the urinometer, and the refractometer (TS meter). The multiple-test dipstick has a poor sensitivity and can therefore miss problems that are actually present. The urinometer has been in clinical use for many years and requires enough urine to float the urinometer. Its results are questionable. The refractometer gives highly reproducible results and requires only one drop of urine for the measurement. In addition, this instrument can be used to measure the total solids in plasma (hence the name, TS meter), which is a good indicator of the plasma protein concentration and can be a useful indicator of a patient's fluid balance (especially if serial determinations are done).

The normal kidney has the capacity to dilute the urine to a specific gravity of 1.001 and to concentrate the urine to at least 1.022. For reference, the specific gravity of water is 1.000. Normally, a person's water balance determines whether the urine is concentrated or dilute; dilute urine is an indicator of water excess, and concentrated urine indicates water deficit. In many renal diseases, the ability of the kidneys to form concentrated urine is lost, and the specific gravity can become "fixed" at approximately 1.010. Often, this finding is seen in acute tubular necrosis, acute nephritis, and chronic renal disease. A falsely high specific gravity can be seen when high-molecular-weight substances, such as protein, glucose, mannitol, and radiographic contrast material, are present in the urine. Therefore, a greater degree of accuracy can be obtained by checking the urine osmolality in these cases.

Osmolality measures the osmoles of solute particles present per kilogram of solvent. The main determinants of osmolality are the sodium, urea, and glucose. In states of volume depletion or excess, several neuroendocrine responses interact to maintain homeostasis, thereby affecting the urinary osmolality. Because of this dynamic interaction, particularly in critical illness, single measurements of the osmolality are of little diagnostic importance. The urinary osmolality is often followed for the evaluation of patients with hyponatremia.

Normal urine osmolality ranges from 300 to 900 mOsm/kg/24 h. Because of this wide range, more information about renal function is obtained when simultaneous serum and urine samples are collected and interpreted. In renal disease, one of the first functions to be lost is the ability to concentrate urine. This can result in the urine osmolality becoming fixed within 150 mOsm of the simultaneously determined serum osmolality.

URINARY SODIUM CONCENTRATION. The urinary sodium excretion is used as an indicator of renal function in differentiating the oliguria associated with acute kidney injury from other prerenal causes. States of poor kidney perfusion are usually associated with a decrease in urinary sodium concentration (usually <10 mEq/L). This is a compensatory reaction generated by the activation of the renin–angiotensin–aldosterone system. Activation of this neuroendocrine response allows for increased reabsorption of sodium (reduced excretion) with a subsequent increase in water reabsorption. The root cause of kidney hypoperfusion can be anything that causes a reduction in effective circulating volume: Volume depletion and heart failure are two examples. Acute kidney injury may develop if hypoperfusion persists. In acute kidney injury, urine sodium concentration usually is greater than 30 to 40 mEq/L despite oliguria because of damage to the

tubular transport mechanisms. However, when the urine pH is alkaline, urine sodium concentration does not reflect sodium balance accurately, and the chloride concentration becomes a better indicator of volume status.

FRACTIONAL EXCRETION OF SODIUM TEST. The fractional excretion of sodium (FE_{Na}) test gives a more precise estimation of the amount of filtered sodium that remains in the urine and is more accurate in predicting tubular injury than the urinary sodium concentration.[2] One benefit of the FE_{Na} compared with the urinary sodium is that it removes the confounding effect of water. It can be calculated by using the following formula:

$$FE_{Na} = \frac{U_{Na} \times P_{Cr}}{P_{Na} \times U_{Cr}}$$

where U and P are the urinary and plasma concentrations of sodium and creatinine, respectively. (Although volume measurements are necessary to derive the absolute urinary excretion of both sodium and creatinine, these cancel out in deriving the formula.)

The FE_{Na} test requires the determination of both serum and urinary sodium and creatinine concentrations on simultaneously obtained samples. Values less than 1% indicate transient acute kidney injury, typically caused by underperfusion. Values greater than 1% (and frequently >3%) are indicative persistent acute kidney injury. Some situations render a falsely low (<1%) FE_{Na}, including glomerulonephritis, myoglobinuric renal failure, contrast nephropathy, renal transplant rejection, acute interstitial nephritis, and acute urinary tract obstruction.[2] FE_{Na} is also a poor indicator of renal function in patients who have received diuretic therapy.[2]

Blood Studies

CREATININE AND CREATININE CLEARANCE. Creatinine is a byproduct of normal muscle metabolism and is excreted in the urine primarily as the result of glomerular filtration, with a small percentage secreted into the urine by the kidney tubules. Therefore, creatinine is currently the most useful indicator of GFR. The amount of creatinine excreted in the urine is directly related to muscle mass and normally remains constant unless significant muscle wasting (a catabolic state) occurs. Normal serum values for creatinine are 0.6 to 1.2 mg/dL.

The creatinine clearance can be defined as the amount of blood that is cleared of creatinine in 1 minute and is an excellent clinical indicator of renal function. As renal function diminishes, creatinine clearance decreases. To obtain an accurate creatinine clearance, the nurse collects all urine made in a 24-hour period and obtains a blood specimen at some point during the urine collection. Thus, it is essential for the nurse to communicate to other team members that a 24-hour collection is in progress. For consistency, the blood sample is usually collected at the midpoint of the urine collection. It is important to note the exact beginning and ending times of the urine collection.

The actual creatinine clearance is calculated by the following formula:

$$CrCl = \frac{U_{Cr} \times V}{P_{Cr}}$$

where U is the urine creatinine concentration, V the urine volume, and P the plasma creatinine concentration.

The product U multiplied by V tells how much creatinine appears in the urine during the period of collection. This can be converted readily to milligrams per minute, which is the standard reference point. Dividing this value by the plasma creatinine concentration (which must be converted from milligrams per 100 mL to milligrams per milliliter) tells the minimal number of milliliters of plasma that must have been filtered by the glomeruli to produce the measured amount of creatinine in the urine. The final result is usually expressed in milliliters per minute. The normal range varies between 80 and 120 mL/min, depending on the person's size, age, and sex. The results can be adjusted to a standard body size of 1.73 m^2 (body surface area [BSA]), which can be derived from standard tables if the patient's height and weight are known; it averages between 120 and 125 mL/min/1.73 m^2 BSA. After age 40 years, normal creatinine clearance values generally decrease 6.5 mL/min per decade because of a decline in GFR.

There are also formulas that estimate creatinine clearance based on a single serum creatinine level. An estimate may be made when there is difficulty collecting a 24-hour urine sample or when spot-checking the creatinine clearance will assist prompt treatment (as in the case of drug nephrotoxicity). The estimate may be accurate only in patients with chronic renal failure with stable renal function who are not edematous or extremely overweight. The following is the Cockcroft-Gault formula for estimating creatinine clearance:

$$\text{Creatinine Clearance} = \frac{(140 - \text{age}) \times \text{weight (kg)}}{72 \times xP_{Cr}\ (\text{mg/dL})}$$

where P_{Cr} is plasma creatinine; for women, the final result is multiplied by 0.85. Many labs are now routinely reporting the GFR using estimate formulas for creatinine clearance.

When the kidneys are damaged by a disease process, the creatinine clearance decreases, and the serum creatinine concentration rises. The urine creatinine excretion decreases initially until the blood level rises to a point at which the amount of creatinine appearing in the urine is equal to the amount being produced by the body. Because men tend to have a higher proportion of muscle than women, the creatinine and creatinine clearance can be higher in men than women. A healthy person with a serum creatinine concentration of 1 mg/dL and a creatinine excretion of 1 mg/min has a creatinine clearance of 100 mL/min. When the person experiences a 50% loss of renal function, the serum creatinine rises to 2 mg/dL, and the person will continue to excrete 1 mg/min of creatinine in the urine when balance is restored. When the person has rapidly changing renal function and oliguria (eg, acute kidney injury), the creatinine clearance is less reliable. Until renal function stabilizes, serum creatinine levels provide a better indication of the rate and direction of change. In patients with rhabdomyolysis, the serum creatinine is elevated out of proportion to the reduction of GFR as the result of chemical conversion of muscle creatine to creatinine. In this situation, the serum creatinine is less reliable as an indicator of renal function.

BLOOD UREA NITROGEN. The blood urea nitrogen (BUN) level has been used for many years as an indicator of kidney function, but unlike the serum creatinine, the BUN level can be influenced by many factors. At low urine flow rates, more

sodium and water, and consequently more urea, are reabsorbed. Therefore, when the patient is volume depleted, the BUN tends to increase out of proportion to any change in renal function. A normal value for the BUN is considered to be 8 to 20 mg/dL.

Increased urea production can result from increased protein intake (tube feedings and some forms of hyperalimentation), increased tissue breakdown (as with crush injuries), febrile illnesses, steroid or tetracycline administration, and reabsorption of blood from the intestine in a patient with intestinal hemorrhage. The BUN may also be elevated in the dehydrated patient, because the lack of fluid volume causes a concentrated value. The patient in shock and the patient with heart failure may have an elevated BUN secondary to decreased renal perfusion. The opposite is true for patients with decreased protein intake or liver disease (both of which reduce urea production) and for patients with large urine volumes secondary to excessive fluid intake.

However, the BUN can be of significant value when used as a comparison with the serum creatinine concentration. Normally, there is a urea/creatinine ratio of 10:1. Discrepancies in this ratio might suggest a potentially correctable situation, as Box 29-2 shows.

OSMOLALITY. The osmolality of a solution is an expression of the total osmoles per kilogram (Osm/kg) of solvent and is independent of the size, molecular weight, and electrical charge of the molecules. All substances in solution contribute to the osmolality. For example, 1 mol (gram molecular weight) of sodium chloride (NaCl) dissociates incompletely into sodium (Na) and chloride (Cl) ions and produces 1.86 Osm when dissolved in 1 kg of solvent (such as plasma). A mole of nonionic solute (eg, glucose or urea) produces only 1 Osm when dissolved in 1 kg of solvent. The total concentration of particles in a solution equals the osmolality and is normally reported in units of osmoles per kilogram of solvent. In the clinical setting (because of much smaller concentrations), the osmolality is reported in milliosmoles (thousandth of an osmole, abbreviated mOsm) per kilogram of solvent (plasma or serum).

The normal serum osmolality consists primarily of sodium and its accompanying anions, with urea and glucose contributing about 5 mOsm each. Therefore, when the serum sodium, urea, and glucose concentrations are known, the osmolality of plasma can be calculated by the following formula:

$$\text{Osmolality} = 2(\text{Na}) + \frac{\text{Glucose}}{18} + \frac{\text{BUN}}{2.8}$$

BOX 29-2 Factors Affecting the Serum Urea: Creatinine Ratio

Decreased Urea/Creatinine Ratio (<10:1)
- Liver disease
- Protein restriction
- Excessive fluid intake

Increased Urea/Creatinine Ratio (>10:1)
- Volume depletion
- Decreased "effective" blood volume
- Catabolic states
- Excessive protein intake

The normal adult average osmolality is 280 to 290 mOsm/kg and remains quite constant. Because water can move freely between the blood, interstitial fluid, and tissues, any change in the osmolality of one body compartment produces a shift in body fluids. Therefore, the osmolality of the plasma is the same as that of other body compartments except in rapidly changing conditions, when a slight lag may occur.

A decrease in the serum osmolality can occur only when the serum sodium is decreased. An increase in the serum osmolality can occur whenever the serum sodium, urea, or glucose is elevated or when abnormal compounds are present in the blood, such as drugs, poisons, or metabolic waste products, such as lactic acid. Symptoms usually do not occur until the osmolality exceeds 350 mOsm/kg. Coma can occur when the osmolality is 400 mOsm/kg or greater.

The calculated osmolality normally is within 10 mOsm of the measured osmolality. Comparing the calculated and measured osmolality can be useful in determining potential substances present. An elevated osmolar gap provides evidence for the presence of a significant amount of abnormal solutes. Ethanol, methanol, and ethylene glycol are three examples of solutes that, when present in appreciable amounts, cause an elevated osmolar gap. If ingestion of one of these substances is suspected, calculate the osmolar gap.

NONSPECIFIC STUDIES. Changes in hematocrit, hemoglobin, platelets, and uric acid levels may be indications of a disorder.

Hematocrit and Hemoglobin. The normal hemoglobin for men is 13.5 to 17.5 g/dL and is 12 to 16 g/dL for women. The normal hematocrit should be 40% to 52% for adult men and 37% to 48% for adult women. False elevations of hematocrit can be seen with dehydration or after dialysis. Low hematocrits may be a dilutional value due to hypervolemia. The kidney is the primary site for the production of erythropoietin. It stimulates the bone marrow to release mature RBCs. Many patients with CKD produce insufficient amounts of erythropoietin, which can result in chronic anemia.

Platelets. Patients with uremia are particularly susceptible to platelet dysfunction. Gastrointestinal (GI) bleeding may be a presenting symptom. The bleeding time, indicative of platelet function, may be prolonged. One mechanism of platelet dysfunction is that uremic platelets tend to synthesize less thromboxane-A2, the chemical that gives platelets their sticky function.

Uric Acid. Uric acid is a nitrogenous end product of protein and purine metabolism. Humans produce only small quantities of uric acid under normal conditions, and the normal uric acid serum level is between 2 and 8.5 mg/dL. Uric acid is excreted primarily by the kidneys, with some in the stool. The value may be elevated because of excessive production from cell breakdown or inadequate excretion by the kidney.

Diagnostic Studies

Radiological Studies

Radiological studies of the kidneys that may be useful in evaluating renal abnormalities include roentgenography, ultrasonography, and radionuclide studies. Table 29-3 summarizes these studies and their purposes.

Table 29-3 Radiological Study of Kidneys

Diagnostic Test	Definition	Purpose
Roentgenography		
Radiograph of kidney–ureter–bladder	Also known as abdominal x-ray. Standard x-rays capture image.	Detects abnormal calcifications and renal size
Tomography	Standard x-rays capture series of cross-sectional scans made along single axis of bodily structure or tissue. Computer software is used to construct three-dimensional image of that structure.	Determines renal outlines and abnormalities
Intravenous pyelography (IVP)	X-ray of renal structures using contrast material. Images are captured on real-time basis. Contrast material is injected intravenously and then collected in renal system; this turns areas bright white, allowing assessment of anatomy and function of kidneys and lower urinary tract.	Detects anatomical abnormalities of kidneys and ureters
Retrograde pyelography	Similar to IVP, with use of x-ray and contrast material. Contrast material is injected through urinary catheter. This test is typically performed at the same time as cystoscopy.	Assesses renal size, evaluates ureteral obstruction, and localizes and diagnoses tumors as well as obstructions
Antegrade pyelography	Similar to IVP and retrograde pyelography, this x-ray test uses contrast material to visualize structures of urinary tract. Contrast dye, however, is injected into ureter. Thus, structures of upper urinary tract are well visualized.	Distinguishes cysts from hydronephrosis
Renal arteriography and venography	Vessel (artery or vein) is accessed and contrast dye is injected to visualize structures "downstream."	Evaluates possible renal arterial stenosis, renal mass lesions, renal vein thrombosis, and venous extension of renal cell carcinoma
Digital subtraction angiography	X-ray with a computer technique that compares x-ray image of kidney vessels before and after contrast dye is injected. Tissues and blood vessels on first image are digitally subtracted from second image, leaving clear picture of artery, which can then be studied independently from rest of the body.	Visualizes major arterial vessels
Ultrasonography	Imaging technique that is excellent means of visualizing tissues and organs to assess their size, structure, and possible pathology. Images are created by emission and receiving of sound waves.	Delineates renal outlines Measures longitudinal and transverse dimensions of the kidneys Evaluates mass lesions Examines perinephric area Detects and grades hydronephrosis
Radionuclide scintillation imaging (renal scan)	Nuclear medicine test that uses small amounts of radioactive materials (radioisotopes) to measure kidney function	
Static imaging	Gives information about the size, shape, and position of the kidneys; and whether there are scars on the kidney from a previous infection.	Evaluates location, size, and contour of functional renal tissue; may reveal areas of inhomogeneity or filling defects
Dynamic imaging	Gives information about the blood flow to the kidneys and how well each kidney is functioning for the production of urine	Monitors passage of radiopharmaceutical agent through vascular, renal parenchymal, and urinary tract compartments; also indicates whether there are any obstructions in urine output
Magnetic resonance imaging	Uses nonionizing radiofrequency signals (as opposed to computed tomography, which uses ionizing radiation) to acquire its images and is best suited for noncalcified tissue.	Determines anatomical abnormalities

Table 29-4 Indications for Renal Biopsy

Clinical Condition	Biopsy Indicated	Expected Gain
Orthostatic proteinuria	No	—
Isolated hematuria and/or proteinuria	No*	—
Hematuria and/or proteinuria with ↓ GFR	Yes	D,P,T
Nephrotic syndrome	Yes	D,P,T
Systemic disease with renal abnormalities	Yes†	D,P,T
Classic ARF	No	—
ARF with		
1. Azotemia for >3 wk	Yes	D,P
2. Moderate proteinuria	Yes	D,T
3. Anuria	Yes	D,T
4. Eosinophilia or eosinophiluria	Yes	D,T
Posttransplant ↓ in GFR	Yes	D,P,T

*Biopsy may be indicated for insurance, administrative reasons, and so forth.

†Biopsy may or may not be indicated, depending on clinical picture.

GFR, glomerular filtration rate; D, diagnosis; P, prognosis; T, therapy; ARF, acute renal failure.

Renal Biopsy

Renal biopsy is the most invasive diagnostic test and has been shown to have a good sensitivity and specificity for malignancy (97.7% and 100%, respectively).[3] It is used to define the histological counterpart of the clinical picture, provide etiological clues for diagnosis, assess prognosis, and guide therapy. Table 29-4 lists the indications for renal biopsy. Contraindications to biopsy include serious bleeding disorders, excessive obesity, and severe hypertension.

Renal biopsies are usually performed percutaneously with a biopsy needle, but an open renal biopsy under general anesthesia still is performed. Preparation for a renal biopsy includes obtaining informed consent, prebiopsy clotting studies, preoperative blood typing, and sedation (usually diazepam, 5 to 10 mg). It is necessary to establish intravenous (IV) access to prevent or treat complications. After the biopsy, the patient's vital signs are checked frequently for the first 24 hours as the patient is monitored during the emergency from sedation and for signs of bleeding. The patient's urine is examined for blood. The major complication is bleeding, which can occur either retroperitoneally or into the urinary tract. Other complications that can occur are biopsy of other abdominal viscera such as bowel, pancreas, liver, spleen, or vessels, and tears in the diaphragm or pleura.

Renal Angiography

Assessment of the renal vasculature may be accomplished by ultrasonography. When precise measurements are required, evaluation of renal blood flow through angiography may be used. This procedure may be performed in conjunction with cardiac catheterization. Access is obtained by percutaneous technique: an introducer, or sheath, is inserted into the femoral artery, and a small catheter is passed to the bifurcation of the renal arteries. Contrast medium is injected to provide radiological visualization of blood flow. Preparation for a renal angiogram is similar to that for renal biopsy; it includes obtaining informed consent, preprocedure clotting studies, preoperative blood typing, and sedation, as well as establishing IV access to prevent or treat complications. After the angiogram, the patient's vital signs are checked frequently for the first 24 hours as the patient is monitored for emergence from sedation and for bleeding. Pressure is applied locally when the arterial access is removed. Because the artery has been accessed, life-threatening bleeding can ensue. Therefore, the access site is assessed for bleeding with the same frequency as the vital signs are assessed. Diligence in application of pressure to the site and conducting site assessment is imperative. Watch for development of bradycardia, because pressure applied to the groin area may stimulate the vagus nerve.

▲ Assessment of Electrolytes and Acid–Base Balance

The role of the kidney is central in maintaining fluid volume and ionic composition of body fluids. When the kidneys properly regulate the excretion of water and ions, homeostasis is achieved. When the kidneys fail to adapt adequately, imbalances occur. Table 29-5 summarizes electrolyte values and signs and symptoms of imbalance. The critical care nurse needs to monitor closely all of the electrolytes because minor shifts can be lethal.

Sodium Balance

Serum sodium concentration is normally 135 to 145 mEq/L. It is regulated by the kidneys and depends on the sodium concentration in the extracellular fluid (ECF). When the concentration of sodium rises, antidiuretic hormone (ADH) is secreted from the posterior pituitary gland, and the kidneys retain water in response. When the concentration falls, aldosterone promotes sodium retention by the kidneys (see Chapter 42, Fig. 42-9). When the kidneys malfunction, this balance is not maintained. Low serum sodium usually indicates water intake in excess of sodium and is characterized by an increase in body weight. High serum sodium usually indicates water loss in excess of sodium and is reflected in weight loss. Sodium is essential for maintaining the osmolality of ECFs, neuromuscular function, acid–base balance, and various other cellular chemical reactions.

Hyponatremia is important because it can produce a wide range of neurological symptoms, including death. The severity of symptoms depends on the degree of hyponatremia and the rate at which it has developed. Usually, symptoms do not occur until the serum sodium level is below 120 mEq/L.[2] For patients with hyponatremia, the severity of symptoms encountered depends on how rapidly the sodium concentration was lowered, as well as the value. Hyponatremia requires further evaluation. Figure 29-5 illustrates the etiologies and evaluation for hyponatremia.

Table 29-5 Disorders of Electrolyte Balance

Electrolyte Imbalance	Signs and Symptoms	Diagnostic Test Results
Hyponatremia	• Muscle twitching and weakness • Lethargy, confusion, seizures, and coma • Hypotension and tachycardia • Nausea, vomiting, and abdominal cramps • Oliguria or anuria	• Serum sodium <135 mEq/L • Decreased urine specific gravity • Decreased serum osmolality • Urine sodium >100 mEq/24 h • Increased red blood cell count
Hypernatremia	• Agitation, restlessness, fever, and decreased level of consciousness • Muscle irritability and convulsions • Hypertension, tachycardia, pitting edema, and excessive weight gain • Thirst, increased viscosity of saliva, and rough tongue • Dyspnea, respiratory arrest, and death	• Serum sodium >145 mEq/L • Urine sodium <40 mEq/24 h • High serum osmolality
Hypokalemia	• Dizziness, hypotension, dysrhythmias, electrocardiogram (ECG) changes, and cardiac and respiratory arrest • Nausea, vomiting, anorexia, diarrhea, decreased peristalsis, abdominal distention, and paralytic ileus • Muscle weakness, fatigue, and leg cramps	• Serum potassium <3.5 mEq/L • Coexisting low serum calcium and magnesium levels not responsive to treatment for hypokalemia usually suggest hypomagnesemia • Metabolic alkalosis • ECG changes, including flattened T waves, elevated U waves, depressed ST segment
Hyperkalemia	• Tachycardia changing to bradycardia, ECG changes, and cardiac arrest • Nausea, diarrhea, and abdominal cramps • Muscle weakness and flaccid paralysis	• Serum potassium >5 mEq/L • Metabolic acidosis • ECG changes, including tented and elevated T waves, widened QRS complex, prolonged PR interval, flattened or absent P waves, depressed ST segment
Hypochloremia	• Muscle hyperexcitability and tetany • Shallow, depressed breathing • Usually associated with hyponatremia and its characteristic symptoms, such as muscle weakness and twitching	• Serum chloride <96 mEq/L • Serum pH > 7.45, serum CO_2 <32 mEq/L (supportive values)
Hyperchloremia	• Deep, rapid breathing • Weakness • Lethargy, possibly leading to coma	• Serum chloride > 108 mEq/L • Serum pH < 7.35, serum CO_2 <22 mEq/L (supportive values)
Hypocalcemia	• Anxiety, irritability, twitching around the mouth, laryngospasm, seizures, positive Chvostek's and Trousseau's signs • Hypotension and dysrhythmias due to decreased calcium influx	• Serum calcium <8.5 mg/dL • Low platelet count • ECG changes: lengthened QT interval, prolonged ST segment, and dysrhythmias
Hypercalcemia	• Drowsiness, lethargy, headaches, irritability, confusion, depression, apathy, tingling and numbness of fingers, muscle cramps, and convulsions • Weakness and muscle flaccidity • Bone pain and pathological fractures • Heart block • Anorexia, nausea, vomiting, constipation, dehydration, and abdominal cramps • Flank pain	• Serum calcium >10.5 mg/dL • ECG changes: signs of heart block and shortened QT interval • Decreased parathyroid hormone level • Calcium stones in urine
Hypomagnesemia	• Nearly always coexists with hypokalemia and hypocalcemia • Hyperirritability, tetany, leg and foot cramps, positive Chvostek's and Trousseau's signs, confusion, delusions, and seizures • Dysrhythmias, vasodilation, and hypotension	• Serum magnesium <1.8 mEq/L • Coexisting low serum potassium and calcium levels
Hypermagnesemia	• Central nervous system (CNS) depression, lethargy, and drowsiness • Diminished reflexes, muscle weakness to flaccid paralysis • Respiratory depression • Heart block, bradycardia, widened QRS, and prolonged QT interval • Hypotension	• Serum magnesium >2.5 mEq/L • Coexisting elevated potassium and calcium levels

(continued on page 629)

Table 29-5 Disorders of Electrolyte Balance (continued)

Electrolyte Imbalance	Signs and Symptoms	Diagnostic Test Results
Hypophosphatemia	• Muscle weakness, tremor, and paresthesia • Tissue hypoxia • Bone pain, decreased reflexes, and seizures • Weak pulse • Hyperventilation • Dysphagia and anorexia	• Serum phosphate <2.5 mg/dL • Urine phosphate >1.3 g/24 h
Hyperphosphatemia	• Usually asymptomatic unless leading to hypocalcemia, then evidenced by tetany and seizures • Hyperreflexia, flaccid paralysis, and muscular weakness	• Serum phosphate > 4.5 mg/dL • Serum calcium < 8.5 mg/dL • Urine phosphate < 0.9 g/24 h

From Anatomical Chart Company: Atlas of Pathophysiology, 3rd ed. Ambler, PA: Lippincott Williams & Wilkins, 2010, pp 32–33.

Symptoms of hypernatremia generally are the same as those of hyperosmolality and result from central nervous system (CNS) dehydration. Mental confusion, stupor, seizures, coma, and death may occur, in addition to other signs of dehydration, such as fatigue, muscle weakness and cramps, and anorexia. The serum osmolality usually is above 350 mOsm/L before significant symptoms are noted. This corresponds to a serum sodium level of 165 to 170 mEq/L.

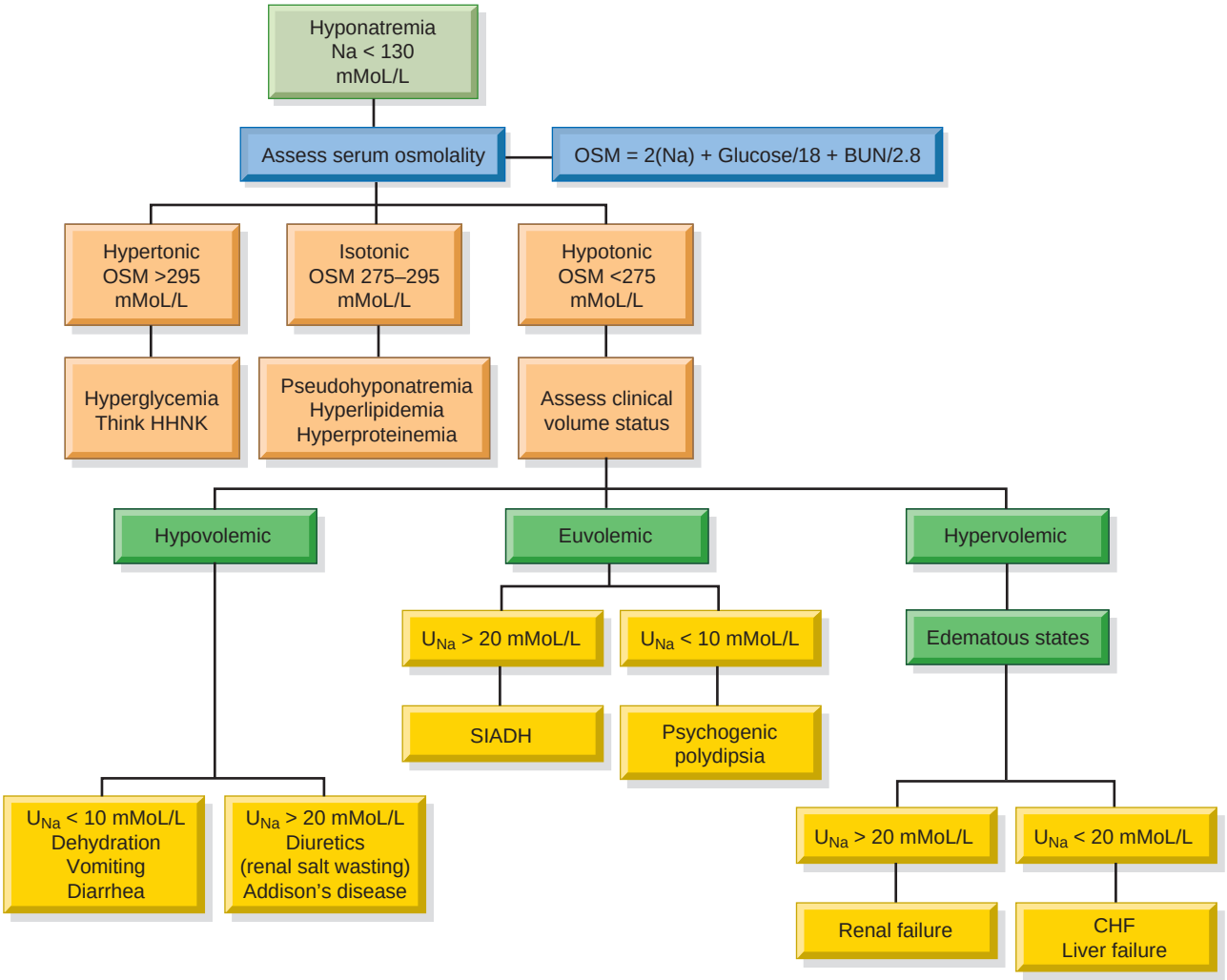


FIGURE 29-5 ▲ Assessing hyponatremia. (BUN, blood urea nitrogen; CHF, congestive heart failure; HHNK, hyperglycemic hyperosmolar nonketotic [coma]; Na, sodium; OSM, osmolality; SIADH, syndrome of inappropriate antidiuretic hormone secretion; UNa, urine sodium.)

Potassium Balance

Potassium is essential for regulating nerve impulse conduction and muscle contraction and is involved in numerous other body functions, including intracellular osmolality and acid–base balance. The normal serum potassium concentration is 3.5 to 5 mEq/L. Potassium balance is maintained by dietary intake and renal excretion. Ninety-eight percent of potassium is located in the skeletal muscle; therefore, the balance of this electrolyte also is strongly tied to the exchanges between the intracellular and extracellular compartments in the body.

Hypokalemia can result from inadequate potassium intake, excessive potassium loss through the kidneys, GI loss, and extracellular-to-intracellular potassium shifts. Also, diuretic therapy can contribute to potassium excretion, further compounding the problem.

Hyperkalemia may be caused by a decrease in the renal excretion of potassium or transcellular shifts of potassium. This is seen most often in acidosis, cell injury or destruction, and hyperglycemia.

Calcium and Phosphate Balance

Calcium and phosphate are regulated reciprocally in the body by vitamin D, parathyroid hormone, and calcitonin. The calcium and phosphate salts are normally deposited in bone. When calcium levels are high, phosphate levels are low. Because in renal failure, the kidneys are unable to eliminate phosphate, patients with renal failure often have high phosphate and low calcium levels.

Calcium's primary function is maintenance of bone and tooth strength. It also plays an important role in myocardial and skeletal contractility. Calcium also maintains cellular permeability and assists in blood coagulation. The normal serum concentration of calcium is 8.5 to 10.5 mg/dL. The total serum calcium is composed of two major fractions: the diffusible or ultrafiltrable (or ionized) calcium and the nondiffusible or protein-bound (primarily to albumin) calcium. Many critically ill patients have low albumin, which will result in low serum calcium. This result does not necessarily mean that the patient's calcium is low. It is necessary to either assess the ionized calcium (if available) or to correct the serum calcium for the albumin level, using the following formula:

$$\text{Corrected calcium} = [0.8 \times (\text{normal albumin} - \text{patient's albumin})] + \text{serum}$$

Phosphate is essential for the formation of adenosine triphosphate. Phosphate also assists in maintaining cell membrane structure, oxygen delivery, and cellular immunity. The normal phosphate level is 3 to 4.5 mg/dL.

Magnesium Balance

The magnesium ion is the second major intracellular ion. The normal serum concentration is 1.4 to 2.1 mEq/L. Magnesium balance is necessary for the functional integrity of the neuromuscular system. The parathyroid glands regulate both magnesium and calcium. Sodium is necessary for magnesium reabsorption. Magnesium can accumulate in the serum, bone, and muscle in renal failure, causing numerous problems.

Acid–Base Balance

A normal acidity or alkalinity (pH 7.35 to 7.45) of the body fluid is essential for life. The body maintains acid–base balance by the buffer system, the respiratory system, and the renal system. The buffer and respiratory systems are able to react quickly to changes in body pH. However, the kidneys take more time to adjust to changes in body pH.

Five major processes are associated with the regulation of acid–base balance by the renal system: hydrogen ion excretion; sodium ion reabsorption; bicarbonate ion generation and reabsorption; phosphate salt and titratable acid excretion; and ammonia synthesis and ammonium excretion. Acid–base imbalances may result when the kidneys are unable to perform those processes adequately. Table 29-6 summarizes acid–base balances.

The Anion Gap

To maintain chemical neutrality, the total concentration of cations and anions in the blood (and other body fluids) must be equivalent in terms of milliequivalents per liter. However, because a number of anions and cations are present in blood but not routinely measured, a "gap" exists between the total concentration of cations and anions and the concentration normally measured in the plasma.

The anion gap is composed primarily of an excess of unmeasured anions, including plasma proteins, inorganic phosphates and sulfates, and organic acids. The unmeasured cations that exist in smaller concentrations are primarily calcium and magnesium.

The anion gap usually is calculated by subtracting the anions (chloride and bicarbonate) from the cations (sodium and potassium) using the following formula:

$$\text{Anion gap} = ([Na^+] + [K]) - ([Cl^-] + [HCO_3])$$

The normal mean is approximately 12 mEq/L (range, 8 to 16 mEq/L). However, departures from this "normal" anion gap may have important diagnostic significance in acid–base disorders, especially metabolic acidoses.

The most common abnormality of the anion gap is an increase that is associated with increased concentrations of lactate, ketone bodies, or inorganic phosphate and sulfate that are found in lactic acidosis, ketoacidosis, and uremia, respectively. Other forms of acidosis associated with ingestion of toxins, such as ethylene glycol, methanol, paraldehyde, and salicylates, also may produce significant increases in the anion gap.

Decreases in the anion gap are less common but equally important. They can occur because of increases in unmeasured cations or because of decreases in unmeasured anions. Table 29-7 lists the causes of altered anion gap.

Table 29-6 Disorders of Acid–Base Balance

Disorder/Causes	Pathophysiology	Signs/Symptoms	Diagnosis
Respiratory Acidosis			
• Airway obstruction or parenchymal lung disease • Mechanical ventilation • Chronic metabolic alkalosis as respiratory compensatory mechanisms try to normalize pH • Chronic bronchitis • Extensive pneumonia • Large pneumothorax • Pulmonary edema • Asthma • Chronic obstructive pulmonary disorder • Drugs • Cardiac arrest • CNS trauma • Neuromuscular diseases • Sleep apnea	When pulmonary ventilation decreases, partial pressure of carbon dioxide in arterial blood ($PaCO_2$) increases and CO_2 level rises. Retained CO_2 combines with water (H_2O) to form carbonic acid (H_2CO_3), which dissociates to release free hydrogen (H^+) and bicarbonate (HCO_3^-) ions. Increased $PaCO_2$ and free H^+ ions stimulate the medulla to increase respiratory drive and expel CO_2. As pH falls, 2,3-diphosphoglycerate accumulates in red blood cells, where it alters hemoglobin (Hgb) to release oxygen. The Hgb picks up H^+ ions and CO_2 and removes them from the serum. As respiratory mechanisms fail, rising $PaCO_2$ stimulates kidneys to retain HCO_3^- and sodium (Na^+) ions and excrete H^+ ions. As the H^+ ion concentration overwhelms compensatory mechanisms, H^+ ions move into cells and potassium (K^+) ions move out. Without enough oxygen, anaerobic metabolism produces lactic acid.	• Restlessness • Confusion • Apprehension • Somnolence • Asterixis • Headaches • Dyspnea and tachypnea • Papilledema • Depressed reflexes • Hypoxemia • Tachycardia • Hypertension/hypotension • Atrial and ventricular dysrhythmias • Coma	• Arterial blood gas (ABG) analysis: $PaCO_2$ > 45 mm Hg; pH <7.35–7.45; and normal HCO_3^- in the acute stage and elevated HCO_3^- in the chronic stage
Respiratory Alkalosis			
• Acute hypoxemia, pneumonia, interstitial lung disease, pulmonary vascular disease, or acute asthma • Anxiety • Hypermetabolic states such as fever and sepsis • Excessive mechanical ventilation • Salicylate toxicity • Metabolic acidosis • Hepatic failure • Pregnancy	As pulmonary ventilation increases, excessive CO_2 is exhaled. Resulting hypocapnia leads to reduction of H_2CO_3, excretion of H^+ and HCO_3^- ions, and rising serum pH. Against rising pH, the hydrogen–potassium buffer system pulls H^+ ions out of cells and into blood in exchange for K^+ ions. H^+ ions entering blood combine with HCO_3^- ions to form H_2CO_3, and pH falls. Hypocapnia causes an increase in heart rate, cerebral vasoconstriction, and decreased cerebral blood flow. After 6 h, kidneys secrete more HCO_3^- and less H^+. Continued low $PaCO_2$ and vasoconstriction increases cerebral and peripheral hypoxia. Severe alkalosis inhibits calcium (Ca^{++}) ionization; increasing nerve/muscle excitability.	• Deep, rapid breathing • Light-headedness or dizziness • Agitation • Circumoral and peripheral paresthesias • Carpopedal spasms, twitching, and muscle weakness	ABG analysis showing $PaCO_2$ < 35 mm Hg; elevated pH in proportion to decrease in $PaCO_2$ in the acute stage but decreasing toward normal in the chronic stage; normal HCO_3^- in the acute stage but less than normal in the chronic stage
Metabolic Acidosis			
• Excessive acid accumulation • Deficient HCO_3^- stores • Decreased acid excretion by the kidneys • Diabetic ketoacidosis • Chronic alcoholism • Malnutrition or a low-carbohydrate, high-fat diet • Anaerobic carbohydrate metabolism • Underexcretion of metabolized acids or inability to conserve base • Diarrhea, intestinal malabsorption, or loss of sodium bicarbonate from the intestines • Salicylate intoxication, exogenous poisoning, or, less frequently, Addison's disease • Inhibited secretion of acid	As H^+ ions begin accumulating in the body, chemical buffers (plasma HCO_3^- and proteins) in cells and extracellular fluid (ECF) bind them. Excess H^+ ions decrease blood pH and stimulate chemoreceptors in the medulla to increase respiration. Consequent fall of partial pressure of $PaCO_2$ frees H^+ ions to bind with HCO_3^- ions. Respiratory compensation occurs but is not sufficient to correct acidosis. Healthy kidneys compensate, excreting excess H^+ ions, buffered by phosphate or ammonia. For each H^+ ion excreted, renal tubules reabsorb and return to blood one Na^+ ion and one HCO_3^- ion. Excess H^+ ions in ECF passively diffuse into cells. To maintain balance of charge across cell membrane, cells release K^+ ions. Excess H^+ ions change the normal balance of K^+, Na^+, and Ca^{++} ions, impairing neural excitability.	• Headache and lethargy progressing to drowsiness, CNS depression, Kussmaul's respirations, hypotension, stupor, and coma and death • Associated gastrointestinal (GI) distress leading to anorexia, nausea, vomiting, diarrhea, and possibly dehydration • Warm, flushed skin • Fruity-smelling breath	• Arterial pH < 7.35; $PaCO_2$ normal or <35 mm Hg as respiratory compensatory mechanisms take hold; HCO_3^- may be <22 mEq/L • Urine pH < 4.5 in the absence of renal disease • Elevated plasma lactic acid in lactic acidosis • Anion gap >14 mEq/L in high–anion gap metabolic acidosis, lactic acidosis, ketoacidosis, aspirin overdose, alcohol poisoning, renal failure, or other disorder characterized by accumulation of organic acids, sulfates, or phosphates • Anion gap 12 mEq/L or less in normal anion gap metabolic acidosis from HCO_3^- loss, GI or renal loss, increased acid load, rapid intravenous (IV) saline administration, or other disorders characterized by HCO_3^- loss

(continued on page 632)

Table 29-6 Disorders of Acid–Base Balance (continued)

Disorder/Causes	Pathophysiology	Signs/Symptoms	Diagnosis
Metabolic Alkalosis			
• Chronic vomiting • Nasogastric tube drainage or lavage without adequate electrolyte replacement • Fistulas • Use of steroids and certain diuretics (furosemide [Lasix], thiazides, and ethacrynic acid [Edecrin]) • Massive blood transfusions • Cushing's disease, primary hyperaldosteronism, and Bartter's syndrome • Excessive intake of bicarbonate of soda, other antacids, or absorbable alkali • Excessive amounts of IV fluids, high serum concentrations of bicarbonate or lactate • Respiratory insufficiency • Low serum chloride • Low serum potassium	Chemical buffers in ECF and intracellular fluid bind HCO_3^- in the body. Excess unbound HCO_3^- raises blood pH, depressing chemoreceptors in the medulla, inhibiting respiration, and raising $PaCO_2$. CO_2 combines with H_2O to form H_2CO_3. Low oxygen limits respiratory compensation. When blood HCO_3^- rises to 28 mEq/L, the amount filtered by renal glomeruli exceeds reabsorptive capacity of the renal tubules. Excess HCO_3^- is excreted in urine, and H^+ ions are retained. To maintain electrochemical balance, Na^+ ions and water are excreted with HCO_3^- ions. When H^+ ion levels in ECF are low, H^+ ions diffuse passively out of cells and extracellular K+ ions move into cells. As intracellular H^+ ion levels fall, calcium ionization decreases, and nerve cells become permeable to Na^+ ions. Na^+ ions moving into cells trigger neural impulses in peripheral nervous system and in CNS.	• Irritability, picking at bedclothes (carphology), twitching, and confusion • Nausea, vomiting, and diarrhea • Cardiovascular abnormalities due to hypokalemia • Respiratory disturbances (such as cyanosis and apnea) and slow, shallow respirations • Possible carpopedal spasm in the hand, due to diminished peripheral blood flow during repeated blood pressure checks	• Arterial blood pH > 7.45; HCO_3^- > 26 mEq/L • Low potassium (<3.5 mEq/L), calcium (<8.9 mg/dL), and chloride (<98 mEq/L)

From Anatomical Chart Company: Atlas of Pathophysiology, 3rd ed. Ambler, PA: Lippincott Williams & Wilkins, 2010, pp 34–35.

▲ Assessment of Fluid Balance

The nurse's role in the assessment of problems of fluid balance includes accurate measurement of intake and output, weight, and vital signs. Although vital signs can provide supporting data, they may not be abnormal until significant volume or water deficits occur. Assessment of fluid imbalance needs to be based on keen observation and recognition of pertinent symptoms.

Weight

Weight is one of the single most important tests for critically ill patients. The admission weight is compared with that obtained in the history. Of note is whether the weight has changed significantly over the past 1 to 2 weeks. Weights should be carefully measured at the same time, with the same scale, and with the same linens and clothing daily. Variations in the procedure should be noted and made known to the

Table 29-7 Causes of an Altered Anion Gap

Increased Anion Gap	Decreased Anion Gap
Increased unmeasured anions • Endogenous metabolic acidosis Lactic acidosis Ketoacidosis Uremic acidosis • Exogenous anion ingestion Ethylene glycol Methanol Paraldehyde Salicylates Penicillin Carbenicillin • Increased plasma proteins Hyperalbuminemia Decreased unmeasured cations • Hypokalemia • Hypocalcemia • Hypomagnesemia	Increased unmeasured cations • Normal cations Hypercalcemia Hyperkalemia Hypermagnesemia • Abnormal cations Increased globulins (eg, myeloma) Lithium Decreased unmeasured anions • Hypoalbuminemia

physician. One liter of fluid equals 1 kg of body weight, equivalent to 2.2 pounds. A kilogram scale provides for greater accuracy because drug, fluid, and diet measurements can be calculated easily using the metric system. An increase in weight does not specify where the weight is gained. For example, a patient may be intravascularly volume depleted yet show an increase in weight because of third spacing of fluid (ie, movement of water to the interstitial space).

Rapid daily gains and losses of weight usually are associated with changes in fluid volume and not nutritional factors. Critically ill patients often experience unmeasured insensible losses, such as ventilation and wound losses. Fever can increase the amount of fluid lost through the skin and lungs by as much as 75 mL/1°F above baseline. Serial weights often are more reliable, and weight changes usually detect imbalances before any symptoms are apparent. In addition to the fluid balance perspective, body weights are also used to calculate drug dosages and, for the patient receiving dialysis, determine the amount of fluid to be removed during therapy.

Intake and Output

An accurate intake and output record provides valuable data for evaluating and treating fluid and electrolyte imbalances. It is important that the nurse teach the patient or visitors to assist in this assessment. Intake and output are measured and recorded as they occur and totaled at the end of every shift. In the presence of excessive losses or deterioration of cardiac, hepatic, renal, or respiratory function, more detailed recording of every source of fluid intake and output is necessary, and calculations may be required every 1 to 4 hours.

In the critically ill patient, intake and output are monitored every 1 to 2 hours. The intake and output values are summed to provide an overall balance at the end of a 24-hour period. A net balance is calculated by subtracting the output from the intake:

$$\text{Fluid balance} = \text{total fluid intake} - \text{total fluid output}$$

Depending on the patient's condition, daily therapeutic goals, and response to interventions, the net balance may be neutral, positive, or negative. The 24-hour balance is compared with the daily weight to assess overall balance. If the net daily balance is positive, but the daily weight reflects a loss over the past 24 hours, insensible losses may be the cause of the discrepancy.

Intake should include all liquids, such as water, juices, or soup, and any foods that are high in water content (eg, oranges, grapefruit, gelatin, and ice cream). It is useful to keep a list of equivalents for fruits, ice cubes and chips, and other sources of fluid. Output should include urinary and intestinal losses and estimates of respiratory and cutaneous losses when the patient's temperature or the ambient temperature is high. Also recorded are other sources of fluid loss that are present, such as ileostomy or other enteric drainage, wound drainage, or thoracic drainage.

In severe electrolyte and fluid imbalances, the time and type of fluid intake and the time and amount of each voiding must be recorded. In the event that renal function decreases, this information may aid immeasurably in the diagnosis and possible prevention of prerenal azotemia or acute kidney injury. Box 29-3 gives risk factors for excessive fluid loss.

Hypovolemia and Hypervolemia

The critical care nurse must be continually on the alert to detect early changes in the patient's volume status. Seldom is the diagnosis made on the basis of one parameter. The first clue may be the patient's general appearance; after observing this, the nurse seeks and notes more specific parameters.

Symptoms vary with the degree of imbalance; some are seen early in imbalance states, and others are not evident until severe imbalances are present. Table 29-8 lists the signs and symptoms of hypovolemia and hypervolemia.

In volume depletion, the patient may complain of orthostatic light-headedness when assuming the sitting or standing position (this also can occur from inactivity and autonomic dysfunction). Development of tachycardia on assuming the upright position and a decrease in blood pressure (orthostatic hypotension), as opposed to the normal rise, are frequent early findings. Later, the pulse may become rapid, weak, and thready. There may be early dryness of the skin,

BOX 29-3 PATIENT SAFETY

Risk Factors for Excessive Fluid Loss

- **Fever:** A patient with a body temperature of 40°C (104°F) and a respiratory rate of 40 breaths/min can lose as much as 2,500 mL of fluid in a 24-hour period from the respiratory tract and from the skin.
- **Environment:** Hot, dry climates can increase evaporative sweat losses to 1,500 mL/h to maintain body evaporative heat loss. This can increase to between 2 and 2.5 L/h for short times in acclimatized people exercising in hot climates.
- **Hyperventilation:** Hyperventilation can increase respiratory water losses as a result of either disease or use of nonhumidified respirators or oxygen delivery systems.
- **Gastrointestinal tract:** Vomiting, nasogastric suction, diarrhea, and enterocutaneous drainage or fistulas can increase gastrointestinal losses.
- **Third spacing:** Formation of pleural or peritoneal effusions and edema from liver, renal, or hepatic disease or from the diffuse capillary leak syndrome can result in a loss of effective intravascular volume. Drainage of peritoneal or pleural fluid, when for of these third spaces still is occurring, can result in further effective intravascular losses because of continued fluid shifts from the vascular compartment to the third space.
- **Burns:** Fluid loss into burned tissues can result in a significant decrease in effective intravascular volume. Because both evaporative and transudative losses through the burned skin can result in very large losses of fluid daily, the burned patient requires special attention to maintain fluid and electrolyte balance. Formulas for determining burn area and fluid resuscitation are discussed in Chapter 53.
- **Renal losses:** Inappropriate solute and fluid loss from the kidneys can occur because of renal salt wasting. This is seen in the diuretic phase of acute tubular necrosis, in some rare patients with true renal salt wasting, and as a result of excessive diuretic administration. It also may occur as a result of solute diuresis from high-protein or high-saline enteral and parenteral alimentation and from administration of osmotic agents, such as mannitol and radiocontrast agents. Finally, fluid can be lost during the generation phase of metabolic alkalosis, in which compensatory urinary bicarbonate excretion obligates renal sodium excretion. This frequently results in volume depletion.

Table 29-8 Signs and Symptoms of Hypovolemia and Hypervolemia

Parameters	Hypovolemia	Hypervolemia
Skin and subcutaneous tissues	Dry, less elastic	Warm, moist, pitting edema over bony prominences; wrinkled skin from pressure of clothing
Face	Sunken eyes (late symptom)	Periorbital edema
Tongue	Dry, coated (early symptom); fissured (late symptom)	Moist
Saliva	Thick, scanty	Excessive, frothy
Thirst	Present	May not be significant
Temperature	May be elevated	May not be significant
Pulse	Rapid, weak, thready	Rapid
Respirations	Rapid, shallow	Rapid dyspnea, moist rales, cough
Blood pressure	Low, orthostatic hypotension; small pulse pressure	Normal to high
Weight	Loss	Gain

with loss of elasticity, sunken eyes, loss of axillary sweating, and a dry, coated tongue. When severe volume depletion occurs, thirst, decreased urine volume, and weight loss may be noted; however, weight loss and orthostatic blood pressure and pulse changes may be the only findings.

Laboratory studies, such as a high urine osmolality and low urinary sodium, may facilitate the diagnosis. Other guidelines, such as elevated hematocrit, decreased central venous pressure, and decreased pulmonary wedge pressure, may corroborate the diagnosis.

In fluid overload, the patient, if alert, may complain of puffiness or stiffness in the hands and feet. Later, periorbital edema or puffiness, followed by pitting edema of the dependent parts (feet and ankles if upright; sacral area and posterior thighs if supine) will occur, followed by dyspnea or ascites, depending on etiology (ie, cardiac decompensation and systemic fluid overload versus hepatic disease). Urine volume and urine sodium may be normal, increased, or decreased, depending on the etiology. In most diseases with fluid retention, except for the syndrome of inappropriate ADH secretion, urine sodium is reduced. The hematocrit is decreased, reflecting hemodilution.

The pulse may be rapid, and auscultation of the heart may reveal a third heart sound (S_3), fourth heart sound (S_4), or murmur secondary to volume overload. Respirations may be increased because of pulmonary congestion, and auscultation of the chest may reveal rales. A chest film may reveal pulmonary vascular congestion, increased alveolar lung markings, cardiac dilation, frank pulmonary congestion, and pleural effusions.

All data should be evaluated in the light of other evidence. Trends usually are more significant than isolated values. For example, when a decrease in urine output is noted, a systematic assessment should be done to determine why this is happening and what nursing interventions are most appropriate. Depending on the stability of the patient, the health care team may use advanced physiological monitoring (eg, pulmonary artery catheter) to guide assessment and management. Table 29-9 lists factors affecting water balance.

Table 29-9 Factors Affecting Water Balance

	Water Excess	Water Deficiency
Intake		
Thirst	Decreased thirst threshold Increased osmolality Potassium depletion Hypercalcemia Fever Dry mucous membranes Poor oral hygiene Unmisted O_2 administration Hypotension Psychiatric disorders	Increased thirst threshold Decreased osmolality Lack of access Psychiatric disorders
Parenteral fluids	Excessive D_5W	Deficient replacement Osmotic loads Parenteral nutrition Hyperglycemia Mannitol Radiographic contrast agents

(continued on page 635)

Table 29-9 Factors Affecting Water Balance (continued)

	Water Excess	Water Deficiency
Output		
Sweating		High ambient temperature High altitude Fever

Renal excretion	Inappropriate ADH release
	Appropriate ADH release
	  Congestive failure
	  Decompensated cirrhosis
	  Volume depletion
	  Adrenal insufficiency
	  Renal salt wasting
	  Hemorrhage
	  Diuretics
	  Burns
	  Hypothyroidism
	Renal disease
	  ARF
	  Chronic renal failure
	  Nephrotic syndrome
	  Acute glomerulonephritis
	Nonsteroidal anti-inflammatory agents
		Excess excretion
		  Central
		  Nephrogenic
		  Potassium depletion
		  Hypercalcemia
		  Lithium administration
		  Demeclocycline (Declomycin)
		  Methoxyflurane (Penthrane)

D_5W, dextrose 5% in water; ADH, antidiuretic hormone; ARF, acute renal failure

Table 29-10 Etiologies of Altered Preload

Hemodynamic Parameter	Increased	Decreased
Preload	Renal failure Volume or blood administration Vasopressors Cardiogenic shock Bradycardia Cardiac tamponade Constrictive pericarditis	Hemorrhage Diuresis Diaphoresis Vomiting Diarrhea Poor intake Third spacing Vasodilators Septic shock Neurogenic shock Anaphylactic shock Tachycardia Loss of atrial kick
Right-sided preload	Right ventricular failure Tricuspid/pulmonic valve disease Ventricular septal defect Right ventricular papillary muscle dysfunction	
Left-sided preload	Left ventricular failure Mitral/aortic valve disease Left ventricular papillary muscle dysfunction	

Hemodynamic Monitoring

Hemodynamic monitoring offers the clinician an improved assessment of the patient's overall status. For a detailed discussion, refer to Chapter 17. Although physical assessment may provide insight into the volume status, changes in physical assessment are reflected later than changes in hemodynamic assessment parameters, such as central venous pressure. Through improved monitoring, interventions are guided by information on a real-time basis. Table 29-10 provides an overview of the causes of altered parameters for the assessment of preload.

Based on data from the history, physical examination, and laboratory and diagnostic tests, nursing diagnoses are developed for the patient with renal problems. Box 29-4 lists possible nursing diagnoses.

BOX 29-4 EXAMPLES OF NURSING DIAGNOSES

For the Patient with Renal, Fluid, or Electrolyte Problems

- Acute Pain related to urinary retention
- Chronic Pain related to urinary retention
- Acute Pain: Dysuria related to infection
- Acute Pain: Dysuria related to urinary obstruction
- Impaired Urinary Elimination related to urinary tract obstruction
- Disturbed Body Image
- Excess Fluid Volume related to impaired kidney function
- Risk for Deficient Fluid Volume related to impaired kidney function
- Activity Intolerance
- Acute Confusion
- Risk for Electrolyte Imbalance
- Risk for Falls
- Risk for Ineffective Renal Perfusion
- Impaired Gas Exchange

▲ Clinical Applicability Challenges

CASE STUDY

Mr. J. is admitted to the intensive care unit following cardiac bypass surgery. He has a history of diabetes, type 2, and is controlled with oral hypoglycemic agents. His glycalated hemoglobin is 6.5%. He had a preoperative ejection fraction of 20%. His bypass time was 45 minutes with a total surgery time of 3 hours. He received 4 units of packed RBCs. Postoperatively, his hemodynamic profile is as follows:

- Heart rate 63 beats/min
- Blood pressure 115/65
- Pulmonary capillary wedge pressure 22 mm Hg
- Central venous pressure 8 mm Hg
- Cardiac index 1.5 L/min/m^2

Urine output has been 30 to 45 mL/h over the past 4 hours. The next morning, his lab values are as follows:

- Na^+ 135 mEq/L
- K^+ 4.2 mEq/L
- Cl^- 90 mEq/L
- BUN 35 mg/dL
- Cr 1.4 mg/dL

1. Describe the risk factors Mr. J. has for developing acute kidney injury. How can each factor contribute to the development of acute kidney injury?
2. What clinical assessment and physiologic data support the diagnosis for acute kidney injury?
3. What are some additional diagnostic tests that may be performed?
4. Outline the monitoring plan for Mr. J. related to his acute kidney injury.

References

1. Hicks D, Li CY: Management of macroscopic haematuria in the emergency department. Emerg Med J 24:385–390, 2007
2. Pépin MN, Bouchard J, Legault L, et al: Diagnostic performance of fractional excretion of urea and fractional excretion of sodium in the evaluations of patients with acute kidney injury with or without diuretic treatment. Am J Kidney Dis 50(4):566–573, 2007
3. Maturen KE, Nghiem HV, Caoili EM, et al: Renal mass core biopsy: Accuracy and impact on clinical management. AJR Am J Roentgenol 188:563–570, 2007

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

30 Patient Management: Renal System

Angela C. Muzzy and Kara Adams Snyder

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Explain the physiological principles involved in renal replacement therapy: hemodialysis, continuous renal replacement therapies, and peritoneal dialysis.
2. Analyze the differences in equipment and procedures used in renal replacement therapy.
3. Explain the types of vascular access used in hemodialysis and continuous renal replacement therapies.
4. Compare and contrast the indications, assessment and management, and complications for each renal replacement therapy.
5. Discuss the psychosocial and teaching needs surrounding renal replacement therapy for patients and their families.
6. Describe the nursing assessments and interventions for patients receiving fluid therapy.
7. Analyze the specific fluid therapies chosen based on physiological alterations.
8. Explain the nursing management for patients with selected electrolyte disorders.

Renal function may be replaced by a process called dialysis, which is a life-maintaining therapy used in acute kidney injury and chronic kidney disease (also known as chronic renal failure). Critical care nurses may encounter patients suffering from the effects of acute kidney injury or patients already on some form of chronic dialysis who subsequently become critically ill. Critical care nurses must be familiar with various dialysis therapies to help care for patients with complex illnesses. This chapter discusses the three most common forms of renal replacement therapy: hemodialysis, continuous renal replacement therapies (CRRTs), and peritoneal dialysis. Common fluid and electrolyte imbalances experienced by critically ill patients also are explored.

▲ Physiology

All forms of dialysis make use of the principles of osmosis and diffusion to remove waste products and excess fluid from the blood. A semipermeable membrane is in the dialysis circuit between the blood and the dialysate. Dissolved substances, such as urea and creatinine, diffuse across the membrane from an area of greater concentration (blood) to an area of lesser concentration (dialysate). Water molecules move across the membrane by osmosis to the solution that contains fewer water molecules. Dialysate is formulated with varying concentrations of dextrose or sodium to produce an osmotic gradient, thereby pulling excess water from the circulatory system. This process of fluid moving across a semipermeable membrane in relation to forces created by osmotic and hydrostatic pressures is called ultrafiltration. These basic principles are the foundation of any dialysis therapy. The manner in which they are accomplished varies depending on the therapy.

▲ Extracorporeal Therapies

Hemodialysis and the CRRTs use an extracorporeal (outside the body) circuit. Therefore, they require access to the patient's circulation and anticoagulation of the circuit.

Access to Circulation

The three most common methods used to access a patient's circulation are through a vascular catheter, an arteriovenous fistula, or a synthetic vascular graft. Patients who suddenly need hemodialysis or CRRT have a venous catheter, whereas

BOX 30-1 NURSING INTERVENTIONS

For the Patient With Dialysis Vascular Access

Dual-Lumen Venous Catheter

- If in upper torso, verify central line catheter placement radiographically before use.
- Do not inject IV fluids or medication into the catheter. Both lumens of the catheter may be filled with concentrated heparin.
- Do not unclamp the catheter unless preparing for dialysis therapy. This can cause blood to fill the lumen and clot.
- Maintain sterile technique in handling vascular access.
- Observe catheter exit site for signs of inflammation or catheter kinking.

Arteriovenous Fistula or Graft

- Do not take blood pressure or draw blood from the access limb.
- Listen for bruit and palpate for thrill every 8 hours and before and after accessing.
- Make sure there is no tight clothing or restraints on the access limb.
- Check access patency more frequently when patients are hypotensive. Hypotension can predispose the blood to clotting.
- In the event of postdialysis bleeding from the needle site, apply just enough pressure to stop the flow of blood and hold the pressure until bleeding stops. Do not occlude the vessel.

patients already receiving chronic hemodialysis typically have either an arteriovenous fistula or a synthetic vascular graft. Box 30-1 lists nursing interventions for the patient with dialysis vascular access.

Venous Catheters

Dual-lumen catheters inserted into large central veins are used for acutely ill patients who need hemodialysis, continuous venovenous hemofiltration (CVVH), or continuous venovenous hemofiltration with dialysis (CVVH/D). These catheters are also used for hemodialysis when there is no other means of access to the circulation. Veins commonly used are the femoral, internal jugular, and subclavian. The site chosen depends on the patient's anatomy and vein accessibility and the physician's experience and preference.

Dual-lumen venous catheters are also used temporarily for patients on acute dialysis who are critically ill or patients on chronic dialysis who are waiting for a more permanent access to mature. Tunneled dual-lumen central venous catheters are often used as a permanent means of access for patients in whom all other means of entry into the circulatory system have been exhausted. The tunneled catheter has an implantable cuff around which tissue grows and acts as a barrier against infection. If possible, the catheter should be placed in the right or left internal jugular vein because catheters placed in the subclavian vein can cause stenosis. The stenosis can cause increased venous pressure and edema that may thwart future efforts to create an arteriovenous fistula or place a graft.

Whenever venous catheters are used, care must be taken to avoid accidental slippage and dislodgment during hemodialysis. For safety, catheters are usually secured with sutures as well as tape to avoid movement. The length of time catheters are left in place depends on catheter function and institutional policy. In general, central venous catheters may be used for up to 3 to 4 weeks. To reduce the risk of catheter-related bloodstream infection, the guidelines from the Centers for Disease Control suggest placing a tunneled catheter if hemodialysis is expected to last longer than 3 weeks.[1] More permanent internal jugular vein catheters often function for many months before problems force their removal. Catheters left in place between dialysis treatments usually are filled with a concentrated heparin-saline solution or 0.9% normal saline solution (dependent upon end cap used and institutional policy) after dialysis to prevent clotting. These catheters should never be used for any purpose other than hemodialysis without first checking with dialysis unit personnel. Cleansing and dressing of the insertion site are the same as with other central lines using strict aseptic technique.

If the catheters are removed at the end of dialysis, pressure is applied to the puncture sites until complete clotting occurs. The site is checked for several hours thereafter so that any recurrent bleeding can be detected. Removal of the more permanent tunneled catheter requires use of local anesthetic at the exit site and careful dissection around the cuff to free it from the attached subcutaneous tissue.

Catheter patency must be maintained. Thrombolytics may be used to dissolve clots in venous catheters. Thrombolytics are enzymes derived from streptococcal bacteria that are capable of activating the fibrinolytic system and dissolving intravascular thrombi. These agents can help preserve vascular access and reduce the need for surgery or catheter reinsertion. However, their use is associated with inherent risks and side effects, including bleeding and an allergic response. The key element in catheter patency is in preventing the formation of thrombosis via proper and routine flushing.

In the early days of dialysis, vascular access was created at every treatment by cannulating an artery to remove blood from the body and a vein to return dialyzed blood to the patient. The lines carrying blood to the dialyzer were called arterial lines, and the lines returning blood to the body were called venous lines. The two lumens of the venous catheter used in dialysis are still designated as arterial and venous. The "arterial" lumen is longer than the venous lumen, so it can pull the venous blood flowing by and allow it to be pumped out of the body and to the dialyzer. Blood is returned upstream from the "arterial" lumen to avoid pulling out the blood that has just been dialyzed and returned to the body. The lumens are distinguished by the presence of colored ends: red on the "arterial" lumen and blue on the venous lumen.

Arteriovenous Fistulas

The arteriovenous fistula technique was developed in 1966 in an effort to provide long-term access for hemodialysis. To create the arteriovenous fistula, a surgeon anastomoses an artery and a vein, creating a fistula or artificial opening between them (Fig. 30-1A). Arterial blood flowing into the venous system results in a marked dilation of the vein, which can then be punctured easily with a 15- or 16-gauge dialysis

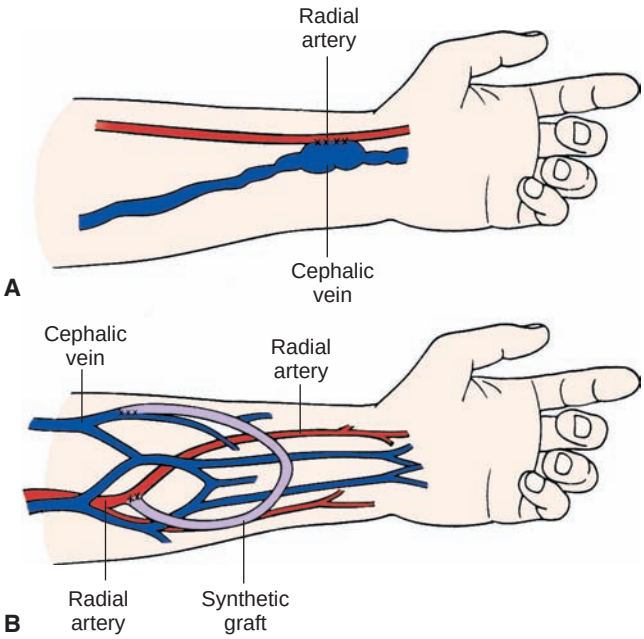


FIGURE 30-1 ▲ Methods of vascular access for hemodialysis. **A:** Arteriovenous fistula. **B:** Synthetic graft.

BOX 30-2 TEACHING GUIDE *Caring for an Arteriovenous Fistula*

- Wash the fistula site with antibacterial soap each day and always before dialysis.
- Refrain from picking the scab that forms after completion of dialysis therapy.
- Check for redness, feeling of excess warmth, or the beginning of a pimple on any area of access.
- Ask the dialysis care team to rotate needles at the time of dialysis treatment.
- Check blood flow several times each day by feeling for a pulse or thrill. If this is not felt, or if there is a change, call your health care provider or dialysis center.
- Refrain from wearing tight clothes or jewelry on the access arm. Also avoid carrying anything heavy or doing anything that will put pressure on the access site.
- Avoid sleeping with your head on the arm where the access site is located.
- Remind care givers and staff not to use a blood pressure cuff on, or draw blood from, the arm where the access site is located.
- Apply only gentle pressure to the access site after the needle is removed. Too much pressure stops flow of blood to the access site.

fistula needle. Two venipunctures are made at the time of dialysis: one for blood outflow and one for blood return.

After the arteriovenous fistula incision has healed, the site is cleansed by normal bathing or showering. To avoid scar formation, excessive bleeding, or hematoma of the arteriovenous fistula, care is taken to avoid traumatic venipuncture, excessive manipulation of the needles, and repeated use of the same site for venipuncture. Adequate pressure must be put on the puncture sites after the needles are removed. In addition, blood pressure measurements and venipunctures should not be performed on the arm with the fistula. For inpatients, a sign posted above the patient's head of bed is advisable regarding these precautions. This aids in ensuring communication regarding the fistula precautions.

Most arteriovenous fistulas are developed and ready to use 1 to 3 months after surgery. After initial healing has occurred, patients are taught to exercise the arm to assist in vessel maturation. They also are encouraged to become familiar with the quality of the "thrill" felt at the site of anastomosis so that they can report any change in its presence or strength. A loud, swishing sound termed a *bruit* plus a palpable thrill indicates a functioning fistula. Box 30-2 presents a patient teaching guide for the care of arteriovenous fistulas.

Although arteriovenous fistulas usually have a long life, complications may occur. These include thrombosis, aneurysm or pseudoaneurysm, or arterial insufficiency causing a "steal syndrome." Steal syndrome occurs when shunting of blood from the artery to the vein produces ischemia of the hand, causing pain or coldness in the hand. Surgical intervention can remedy these problems and restore adequate fistula flow.

Synthetic Grafts

The synthetic graft is made from polytetrafluoroethylene (PTFE), a material manufactured from an expanded, highly porous form of Teflon. The graft is anastomosed between an artery and a vein and is used in the same manner as an arteriovenous fistula (see Fig. 30-1B).

For many patients whose own vessels are not adequate for fistula formation, PTFE grafts are extremely valuable. PTFE segments are also used to patch areas of arteriovenous grafts or fistulas that have stenosed or developed areas of aneurysm. It is best to avoid venipuncture in new PTFE grafts for 2 to 4 weeks while the patient's tissue grows into the graft. When tissue growth progresses satisfactorily, the graft has an endothelium and wall composition similar to the patient's own vessels.

The procedures for preventing complications in grafts are the same as those used for arteriovenous fistulas. However, certain complications are seen more frequently with grafts than with fistulas, including thrombosis, infection, aneurysm formation, and stenosis at the site of anastomosis.

Anticoagulation

Blood in the extracorporeal system, such as the dialyzer and blood lines, clots rapidly unless some method of anticoagulation is used. Heparin is most commonly used because it is simple to administer, it increases clotting time rapidly, it is monitored easily, and its effect may be reversed with protamine. Citrate solutions may also be used for anticoagulation during dialysis. This agent chelates calcium, thereby inactivating the clotting cascade. Evidence suggests that the use of citrate in CRRTs is associated with fewer life-threatening bleeding complications than heparin.[2,3]

Specific anticoagulation procedures vary, but the primary goal of all methods is to prevent clotting in the dialyzer with the least amount of anticoagulation. Two methods of heparinization commonly used are intermittent and continuous infusion. Regardless of the type of anticoagulation used, close monitoring of appropriate laboratory values is necessary to ensure patient safety.

Systemic Anticoagulation

Typically, the circuit is initially primed with a dose of heparin, followed by smaller intermittent doses of anticoagulant or heparin administered at a constant rate by an infusion pump. This results in systemic anticoagulation, in which the clotting times of the patient and the dialyzer essentially are the same.

Definitive guidelines are difficult to provide because methods and dialyzer requirements vary. The normal clotting time of 6 to 10 minutes may be increased to 30 to 60 minutes. The effect of heparin is generally monitored by the activated partial thromboplastin time (aPTT).

The patient's need for heparinization and an appropriate beginning heparin dose should be assessed routinely before dialysis, especially in the critically ill patient who may be actively bleeding or at risk for bleeding. In addition, there is also a need to assess for the development of heparin-induced thrombocytopenia with the administration of all heparin products (refer to Chapter 49 for a discussion of hematologic abnormalities), and there is evidence that the use of argatroban for CRRT may be of benefit.[4] The patient's platelet count, serum calcium level, and results of coagulation studies are valuable in assessing current function of the clotting process. Often, little or no heparin may be used when the patient has serious alterations in one or more factors needed for effective clotting.

Regional Anticoagulation

Regional anticoagulation is another option to maintain blood flow during extracorporeal therapies. Regional anticoagulation is where the patient's clotting time is kept normal while the clotting time of the dialyzer is increased. This is accomplished by infusing the anticoagulant at a constant rate into the dialyzer and simultaneously neutralizing its effects with its antidote before the blood returns to the patient. Typical combinations include heparin/protamine sulfate or trisodium citrate/calcium.

Regional anticoagulation has no associated standard ratios of anticoagulants to antidotes. Frequent monitoring of the clotting times with adjustment of the antidote rate is the best way to achieve effective regional anticoagulation. One patient safety concern is bleeding secondary to overanticoagulation. Causes of overanticoagulation include infusion pump malfunction, errors in setting delivery rates, and infrequent monitoring of clotting times. Because of these hazards, anticoagulation delivery must be monitored carefully and frequently, with meticulous checking of pump rates.

Another way to prevent dialyzer clotting and reduce the risk for bleeding from anticoagulation is to administer the anticoagulant with intermittent boluses and use frequent normal saline flushes. Occasionally, saline flushes are used alone.

When regional citrate anticoagulation is used, citrate is infused into the system before the dialyzer binds calcium, obstructing the normal clotting pathway. The patient's sodium levels may rise because the citrate is administered in the form of sodium citrate.[3] Citrate has a higher pH, and therefore, patients may also develop metabolic alkalosis.

▲ Intermittent Hemodialysis

In hemodialysis, water and excess waste products are removed from the blood as it is pumped by the dialysis machine (Fig. 30-2) through an extracorporeal circuit (Fig. 30-3) into a device called a dialyzer or artificial kidney. The blood is in one compartment, and the dialysate is in another compartment. There, the blood flows through a semipermeable membrane. The semipermeable membrane is a thin, porous sheet made of cellulose or a synthetic material. The pore size of the membrane permits diffusion of low-molecular-weight substances such as urea, creatinine, and uric acid. In addition, water molecules are small and move freely through the membrane, but most plasma proteins, bacteria, and blood cells are too large to pass through the pores of the membrane. The difference in the concentration of the substances in the two compartments is called the concentration gradient.

FIGURE 30-2 ▲ Hemodialysis delivery unit, which includes an automatic blood pressure cuff, heparin infusion pump, and blood pump. This machine displays a continuous readout of ultrafiltration goal, rate, and total fluid removed and monitors dialysate temperature and conductivity. It can vary the sodium concentration of the dialysate. (Courtesy of Fresenius 2008T Fresenius VSA, Inc., Concord, CA.)

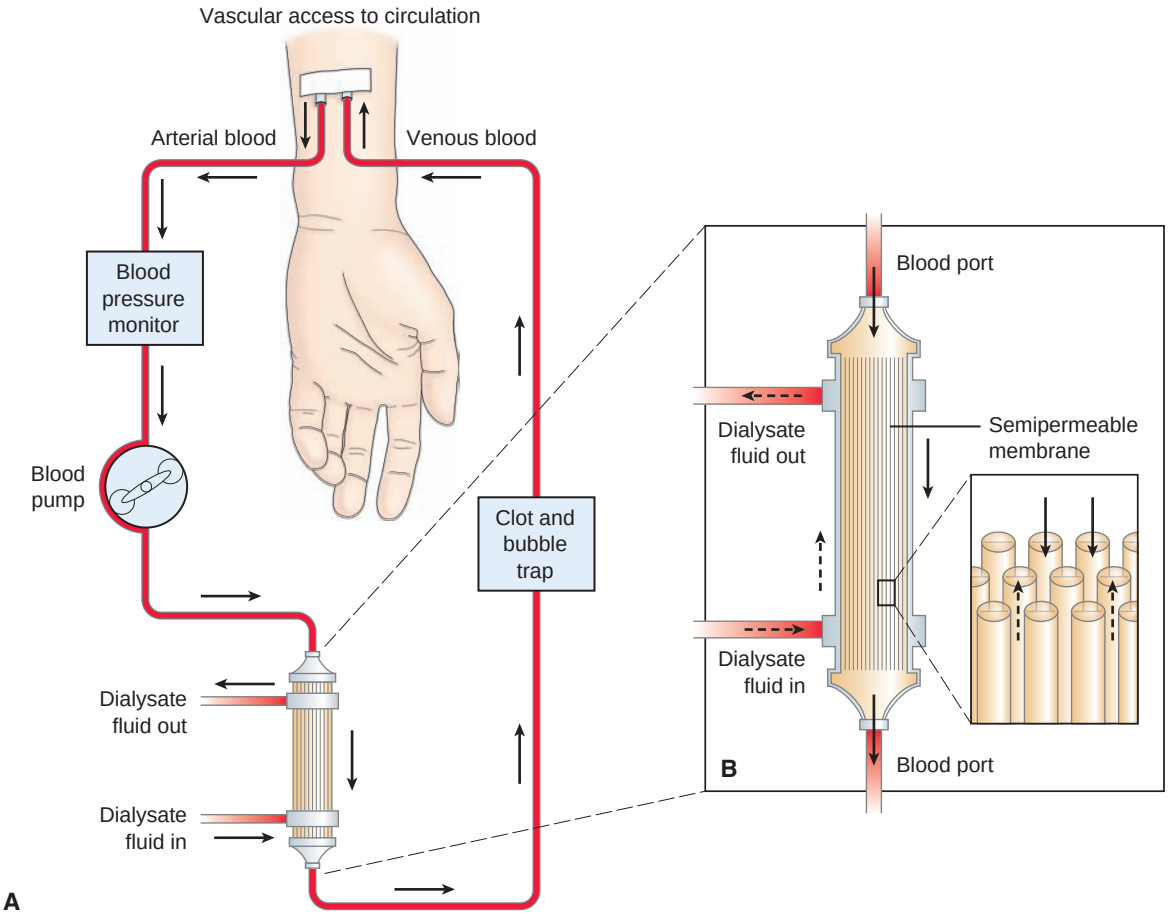


FIGURE 30-3 ▲ Hemodialysis system. **A:** Blood from an artery is pumped into a dialyzer (**B**), where it flows through the cellophane tubes, which act as the semipermeable membrane (**inset**). The dialysate, which has the same chemical composition as the blood except for urea and waste products, flows in around the tubules. The waste products in the blood diffuse through the semipermeable membrane into the dialysate. (From Smeltzer SC, Bare BG, Hinkle JL, Cheever KH: Brunner & Suddarth's Textbook of Medical-Surgical Nursing, 12th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 1334.)

The blood, which contains waste products such as urea and creatinine, flows into the blood compartment of the dialyzer, where it comes into contact with the dialysate, which contains no urea or creatinine. A maximal gradient is established so that these substances move from the blood to the dialysate. These waste products fall to more normal levels as the blood passes through the dialyzer repeatedly at a rate ranging from 200 to 400 mL/min over 2 to 4 hours.

Excess water is removed by a pressure differential created between the blood and fluid compartments. This pressure differential is aided by the action of the dialyzer pump and usually consists of positive pressure in the blood path and negative pressure in the dialysate compartment. This is the process of ultrafiltration.

In summary, hemodialysis:

- Removes by-products of protein metabolism, such as urea, creatinine, and uric acid
- Removes excess water
- Maintains or restores the body's buffer system
- Maintains or restores electrolyte levels in the body.

Indications for Hemodialysis

Hemodialysis is indicated in chronic renal failure and for complications of acute kidney injury. These include uremia, fluid overload, acidosis, hyperkalemia, and drug overdose. Table 30-1 compares hemodialysis, CRRT, and peritoneal dialysis, and Box 30-3 lists indications for dialysis.

Contraindications to Hemodialysis

Hemodialysis may be contraindicated in patients with coagulopathies because the extracorporeal circuit needs to be heparinized. Intermittent hemodialysis may also be difficult to perform in patients who are hypotensive, have extremely low cardiac output, or are sensitive to abrupt changes in volume status. For these critically ill patients in these situations, CRRT may be the optimal choice. In addition, intermittent hemodialysis may not keep up with the metabolic needs of a highly catabolic patient. In this case, CRRT would likely be chosen as the dialysis method. Patients treated chronically for renal failure may be given the choice to undergo hemodialysis or peritoneal dialysis.

Table 30-1 Comparison of Hemodialysis, Continuous Renal Replacement Therapy, and Peritoneal Dialysis

	Hemodialysis	CRRT	Peritoneal Dialysis
Access	Arteriovenous fistula or graft; dual-lumen venous catheter	Dual-lumen venous catheter	Temporary or permanent peritoneal catheter
Anticoagulation requirements	Systemic heparinization or frequent saline flushes	Systemic anticoagulation with heparin or trisodium citrate may be indicated depending on patient's coagulation studies before starting therapy	May only need heparin intraperitoneally Not absorbed systemically
Length of treatment	3–4 h, three or more times per week, depending on patient acuity and need	Continuous throughout day; may last as many days as needed	Continuous (cycled) or intermittent exchanges; time between exchanges = 1–6 h
Advantages	Quick, efficient removal of metabolic wastes and excess fluid Useful for drug overdoses and poisonings	Best choice for patient who is hemodynamically unstable because less blood is outside body than with hemodialysis and blood flow rates are slower; amount of fluid removed can still be achieved but over a much longer period of time Good for hypercatabolic patients who receive large amounts of IV fluids	Continuous removal of wastes and fluid Better hemodynamic stability Fewer dietary restrictions
Disadvantages	May require frequent vascular access procedures Places strain on a compromised cardiovascular system Potential blood loss from bleeding or clotted lines Requires specially skilled staff to perform therapy Risk of bloodstream infection	Requires vascular access procedures; potential blood loss from clotting or equipment leaks; uses an extra piece of equipment Requires specially skilled staff to perform therapy Costly Risk of bloodstream infection	Contraindicated after abdominal surgery or in presence of many scars Waste products may be removed too slowly in a catabolic patient Danger of peritonitis Abdominal discomfort

Assessment and Management

The degree and complexity of problems arising during hemodialysis vary among patients and depend on many factors. Important variables are the patient's diagnosis, stage of illness, age, other medical problems, fluid and electrolyte balance, prior experience with hemodialysis, and emotional state. Because an increasing number of older adults are receiving dialysis, it is also important to consider the normal renal and urinary system changes resulting from the aging process (Table 30-2).

Preprocedure

A predialysis assessment is the first step in managing the patient receiving hemodialysis. It consists of a review of the patient's history and clinical findings, response to previous dialysis treatment, laboratory results (such as electrolytes), consultation with other care givers, and the nurse's direct assessment of the patient.

BOX 30-3 Indications for Dialysis

- Anuria secondary to acute/chronic kidney failure
- Symptomatic pulmonary edema unresponsive to diuretic therapy
- Severe electrolyte disturbances (ie, hyperkalemia and hyperphosphatemia refractory to medical therapy)
- Metabolic acidosis
- Uremic complications involving other organs (ie, pericarditis, encephalopathy)
- Drug/toxin overdoses that are dialyzable (ie, salicylates)
- Evolving use of hemodialysis in the literature: Treatment of sepsis

The nurse evaluates fluid balance before dialysis so that corrective measures may be initiated at the beginning of the procedure. Blood pressure, pulse, weight, intake and output, tissue turgor, and other symptoms assist the nurse in estimating fluid overload or depletion. Monitoring tools, such as pulmonary artery catheters and central venous pressures, also help determine cardiovascular fluid load.

The term *dry weight* or *ideal weight* is used to express the weight at which fluid volume is in a normal range for a patient who is free of the symptoms of fluid imbalance. It provides a guideline for fluid removal or replacement. The figure is not absolute. It requires frequent review and revision, especially in patients receiving dialysis in whom frequent weight changes occur.

After reviewing the data and while consulting with the physician and the bedside nurse (as applicable), the dialysis nurse establishes objectives regarding fluid removal and restoration of electrolyte balance for the dialysis treatment. The objectives vary from one dialysis to the next in the patient whose condition may change rapidly. For example, fluid removal may take precedence over correction of an electrolyte imbalance or vice versa.

Anxiety and apprehension, especially during the first dialysis, may contribute to change in blood pressure, restlessness, and gastrointestinal upset. The presence of a competent and

Table 30-2 Renal and Urinary System Changes Resulting From the Aging Process

Pathophysiologic Changes	Physiologic Effects	Nursing Implications
Decrease in number and function of nephrons	Decreased ability to concentrate urine and conserve water. At risk for dehydration; dry mouth.	Provide routine oral care and offer fluids liberally (as ordered).
Decreased GFR	Decreased secretion of sodium, water, urea, ammonia, and drugs. Increased risk for confusion, dry skin, thirst. Less clearance of renally eliminated drugs leading to toxicity.	Fall precautions as needed. Thorough skin assessments with position changes every 2 h. Review medications/prescriptions with multidisciplinary team to ensure appropriate medications and doses for age.
Decline in kidney efficiency	Predisposes to hypernatremia, fluid overload, drug reactions.	Routine assessment for heart failure symptoms (crackles, edema, S3 heart tones). Daily weights and I&O as ordered.
Reduction in bladder tonicity and capacity	Increased residual urine, nocturnal urination. Risk for incontinence and associated skin breakdown. Increased risk of falls associated with incontinence.	Monitor for incontinence and skin breakdown. Provide skin protection as appropriate. Offer toileting program. Call bell in reach at all times. Teach Kegel exercises.
Decreased regulatory functions such as sensation of thirst, secretion of aldosterone, calcium absorption, response to vasopressin	Increased risk for dehydration. Less ability to conserve sodium and excrete potassium. Altered bone formation. Risk for injury. Increased needs for supplements. Risk for hypotensive episodes.	Provide adequate fluid intake and encourage as appropriate. Monitor for fluid and electrolyte imbalances. Assess gait and balance, place on fall precautions if indicated.

caring nurse during dialysis may increase the patient's sense of security enough to avoid the need for an antianxiety drug that might precipitate changes in vital signs.

A basic explanation of the procedure and its place in the total plan of care for the patient may also allay some of the anxiety experienced by the patient and family. They must understand that dialysis is being used to support normal body function rather than to "cure" the kidney disease process.

Procedure

The nurse begins the procedure by checking the equipment (Box 30-4). After predialysis preparation and a safety check of equipment, the nurse is ready to begin hemodialysis. Access to the circulatory system is gained by one of several options: a dual-lumen catheter, an arteriovenous fistula, or a graft. The dual-lumen catheter is opened under aseptic conditions according to institutional policy. Two large-gauge (15- or 16-gauge) needles are needed to cannulate a graft or fistula.

BOX 30-4 NURSING INTERVENTIONS

For Checking Hemodialysis and Continuous Venovenous Hemofiltration With Dialysis Equipment

- Prime lines and dialyzer or filter to expel all air before starting treatment.
- Test all alarms before connecting the patient to the circuit.
- Respond to all alarms immediately.
- Replace wet pressure transducers if they interfere with transmission of pressure reading.
- Inspect and tighten all connections before initiating treatment.

Figure 30-3 on page 641 illustrates the hemodialysis circuit. After vascular access is established through strict aseptic technique, blood begins to flow, assisted by the blood pump. The part of the disposable circuit before the dialyzer is designated the arterial line, both to distinguish the blood in it as blood that has not yet reached the dialyzer and in reference to needle placement. The arterial needle is placed closest to the arteriovenous anastomosis in a graft or fistula to maximize blood flow. A clamped bag of saline solution is always attached to the circuit just before the blood pump. In episodes of hypotension, blood flow from the patient can be clamped while the bag of saline solution is opened and allowed to infuse rapidly to correct blood pressure. Blood transfusions and plasma expanders also can be attached to the circuit at this point and allowed to drip in, assisted by the blood pump. Heparin infusions may be located either before or after the blood pump, depending on the equipment in use.

The dialyzer is the next important component of the circuit. Blood flows into the blood compartment of the dialyzer, where exchange of fluid and waste products takes place. Blood leaving the dialyzer passes through an air detector that shuts down the blood pump if any air is detected. At this point in the pathway, any medications that can be given during dialysis are infused through a medication port. However, unless otherwise ordered, most medications are withheld until after dialysis.

Blood that has passed through the dialyzer returns to the patient through the venous (postdialyzer) line. After the prescribed treatment time, dialysis is terminated by clamping off blood from the patient, opening the line for saline solution, and rinsing the circuit to return the patient's blood.

A dialysis nurse is in constant attendance during acute hemodialysis. Blood pressure and pulse are recorded at least every half hour when the patient's condition is stable. All

machine pressures and flow rates are checked and recorded on a regular basis. The nurse assesses the patient's responses to fluid and solute removal and the condition and function of the patient's vascular access. Gloves are always worn by the nurse performing hemodialysis because of the risk for exposure to blood. The dialysis nurse and critical care nurse work together to care for the patient because they must coordinate their specific patient care responsibilities.

Postprocedure

The results of a dialysis treatment can be determined by assessing the amount of fluid removed (as assessed by postdialysis weight) and the degree to which electrolyte and acid–base imbalances have been corrected. Blood drawn immediately after dialysis may show falsely low levels of electrolytes, urea nitrogen, and creatinine. The process of equilibration is thought to continue for some time after dialysis because these substances move from inside the cell to the plasma. To ensure accuracy of laboratory data after dialysis, a minimum of 2 to 3 hours should elapse before samples for laboratory tests are taken from the patient.

Complications

Dialysis Dysequilibrium

Uremia must be corrected slowly to prevent dysequilibrium syndrome, which is a set of signs and symptoms ranging from headache, nausea, restlessness, and mild mental impairment to vomiting, confusion, agitation, and seizures. This is thought to occur as the plasma concentration of solutes, such as urea nitrogen, is lowered. Blood urea and nitrogen play a role in calculating the serum osmolarity. Because of the blood–brain barrier, solutes are removed much more slowly from brain cells. Therefore, plasma becomes hypotonic in relation to the brain cells. This results in a shift of water from plasma to the brain cells and causes cerebral edema and symptoms of dysequilibrium syndrome. This syndrome can be avoided by dialyzing patients for short periods, such as 1 to 2 hours on 3 or 4 consecutive days.

Hypovolemia

Fluid overload is treated during dialysis by removing excess water. Because this removal depends on shifting fluid from other body compartments to the vascular space, clinicians must take care to avoid removing fluid so rapidly during dialysis that it leads to volume depletion. Excessive fluid removal may lead to hypotension, and little is gained if intravenous (IV) fluids are given to correct the problem. Therefore, it is better to reduce the volume overload over two or three dialyses, unless pulmonary congestion is life threatening.

Hypotension

Generally, normal saline solution in bolus amounts of 100 to 200 mL is used to correct hypotension. Dialysis machines now aid in preventing hypotension because the amount of ultrafiltration is controlled at the push of a button. It is also possible to vary the sodium concentration of dialysate. A higher sodium level in the dialysate means that less sodium is removed from the blood. A higher serum sodium assists the body as it shifts fluid from the interstitial to the intravascular compartment. Blood volume expanders, such as albumin, are sometimes used in patients with a low serum protein.

The use of antihypertensive drugs in patients who undergo dialysis may precipitate hypotension during dialysis. To avoid this, standard practice in many dialysis units is to omit antihypertensive drugs 4 to 6 hours before dialysis. Restriction of fluids and sodium before and during the dialysis phases is a more desirable method for controlling hypertension. Sedatives and tranquilizers also may cause hypotension and should be avoided, if possible.

Hypertension

Fluid overload, dysequilibrium syndrome, renin response to ultrafiltration, and anxiety are the most frequent causes of hypertension during dialysis. Hypertension during dialysis is usually caused by sodium and water excess. This can be confirmed by comparing the patient's present weight to his or her ideal or dry weight. If fluid overload is the cause of hypertension, ultrafiltration usually brings about a reduction in the blood pressure.

Some patients who may be normotensive before dialysis become hypertensive during dialysis. The blood pressure rise may occur either gradually or abruptly. The cause is not well understood, but it may be the result of renin production in response to ultrafiltration and an increase in renal ischemia. Patients must be carefully monitored because the vasoconstriction caused by the renin response is limited. Once a decrease in blood volume surpasses the ability to maintain blood pressure through vasoconstriction, hypotension can occur precipitously.

Muscle Cramps

Muscle cramps may occur during dialysis as a result of excess fluid removal, which results in diminished intravascular volume and reduced muscle perfusion. During dialysis, cramps may be treated by lowering the rate of ultrafiltration and administering hypertonic solutions, normal saline solution boluses, mannitol, or glucose in an attempt to increase perfusion to the muscles.

Dysrhythmias and Angina

Dysrhythmias and angina may occur in patients with underlying cardiac disease in response to fluid and electrolyte removal. Decreasing the rate of fluid removal may help. Medication may be needed to control the patient's cardiac rhythm.

▲ Continuous Renal Replacement Therapies

In continuous renal replacement therapy (CRRT), blood circulates outside the body through a highly porous filter similar to that used with hemodialysis. The process is similar to hemodialysis in that water, electrolytes, and small to medium-sized molecules are removed by ultrafiltration. CRRT is accompanied by a simultaneous reinfusion of a physiological solution, and it occurs continuously for an extended period. A pump, slightly different from that used in hemodialysis,

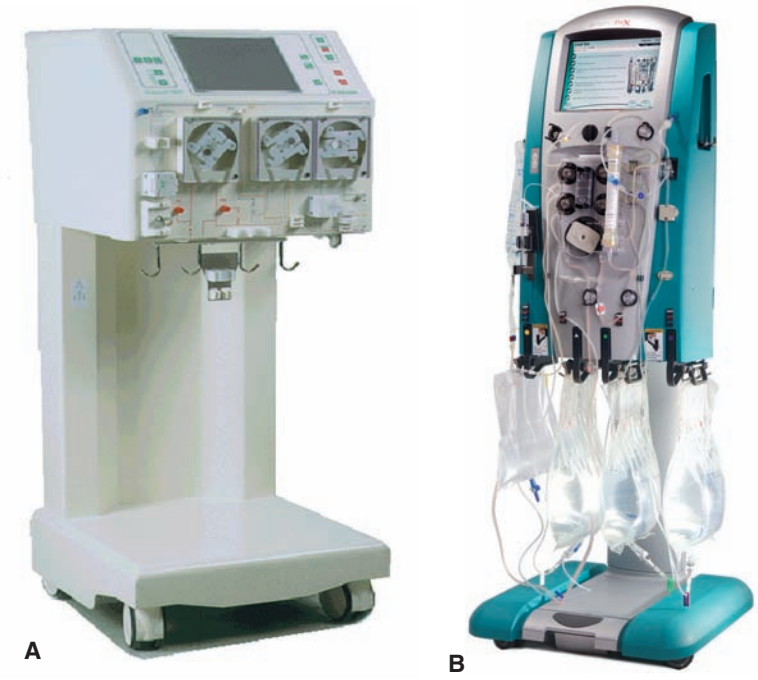


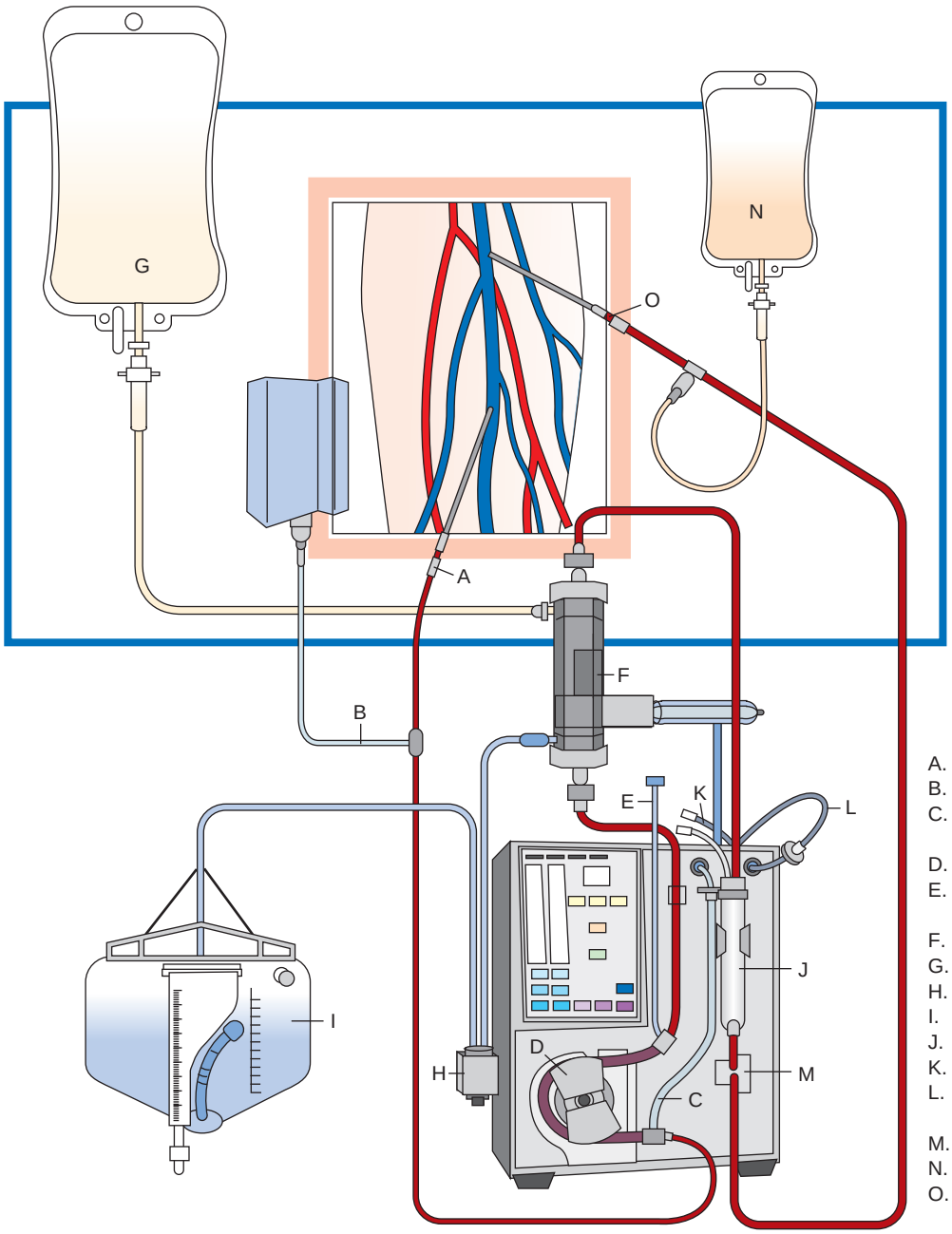
FIGURE 30-4 ▲ Devices for administering continuous renal replacement therapy (CRRT) offer an integrated fluid warmer for the heating of infusion and dialysate fluids, a weighing system to reduce the possibility of error in assessing fluid balance, and a battery backup that allows treatments to continue when the patient is moved.
A: Diapact pump. (Courtesy B-Braun McGraw Corporation.)
B: PRISMAFLEX. (Courtesy of Gambro Renal Products, Inc.)

is used and often incorporates a weighing system so that fluids can be intricately balanced hour to hour (Fig. 30-4).

The most common types of CRRTs include CVVH, CVVH/D, and slow continuous hemofiltration (Table 30-3). This discussion focuses primarily on CVVH and CVVH/D because these therapies are replacing previous arteriovenous procedures. Access to the circulation for CVVH and CVVH/D is generally a large-bore dual-lumen central venous catheter designed for helodialysis. The extracorporeal circuit is similar to the hemodialysis circuit (Fig. 30-5). A pump is added to assist blood flow. The rate of blood flow is typically much slower than in hemodialysis (mimicking the patient's

Table 30-3 Continuous Renal Replacement Therapies

Type of Therapy	Mechanism of Action	Indications
Continuous venovenous hemodialysis (CVVHD)	Blood is driven from the access port through the low-permeability dialysis filter; there is a countercurrent flow of dialysis solution into the dialysate compartment and to the ultrafiltrate bag. There is hydrostatic pressure pushing molecules (electrolytes and toxins) and fluid and osmotic pressure pulling additional molecules (electrolytes and toxins) across the filter. Both actions create the effluent which drains into the ultrafiltrate collection bag. Solute clearance is mainly achieved by diffusion; replacement solution is not needed. Efficiency of clearance is limited to small molecules.	Fluid and solute clearance
Continuous venovenous hemofiltration (CVVH)	Blood is driven from the access port through the high-permeability dialysis filter; replacement fluid is added to the system, typically just prior to the filter This results in a "push-only" phenomena: there is hydrostatic pressure pushing molecules (electrolytes and toxins) and fluid across the filter. The addition of replacement fluid will increase this hydrostatic pressure to help facilitate clearance. The effluent created then drains into the ultrafiltrate collection bag. Fluid removed may or may not be replaced depending upon the needs of the patient. Ultrafiltration is in excess of patient weight loss; replacement solution is needed.	Fluid and solute clearance
Continuous venovenous hemofiltration with dialysis (CVVHD)	Both replacement fluid and dialysate are used. Blood is driven through highly permeable dialyzer, and countercurrent flow of dialysis solution is delivered on the dialysate compartment. Replacement fluid is added typically just prior to connection to the filter. Solute clearance is obtained both by diffusion and convection; replacement solution is needed to obtain fluid balance (sometimes referred to as "push–pull" dialysis).	For combined convection and diffusion clearance



A. Blood exiting the body
B. Heparin infusion
C. Arterial pressure monitor (prefilter pressure)
D. Blood pump
E. Saline infusion line (saline not shown here)
F. Filter
G. Dialysate
H. Blood leak detector
I. Graduated collection device
J. Air and foam detector
K. Syringe line
L. Venous pressure monitor (postfilter pressure)
M. Clamp
N. Replacement fluid
O. Blood returns to body

FIGURE 30-5 ▲ Continuous venovenous hemofiltration with dialysis. (Courtesy of Baxter Health Care Corporation, Renal Division, McGaw Park, IL.)

natural blood flow). The ultrafiltration rate is titrated to reach an hourly goal and is based on the patient's cardiac and pulmonary status.

When CVVH is used, a replacement fluid is ordered and is connected either before or after the filter, depending on patient characteristics and institutional practice. When dialysis is added to the CVVH process, it is called CVVH/D. Adding the dialysate increases the ability to remove wastes. Therefore, it is used when uremia must be aggressively managed, such as with the highly catabolic patient. CVVH and CVVH/D can be performed and managed by the critical care nurse. Typically, didactic training, competency assessment, and validation are performed before the nurse cares for patients receiving CRRT.

Indications for Continuous Renal Replacement Therapy

CRRT is indicated in the following circumstances: in patients with a high risk for hemodynamic instability who do not tolerate the rapid fluid shifts that occur with hemodialysis, in those who require large amounts of hourly IV fluids or parenteral nutrition, and in those who need more than the usual 3- to 4-hour hemodialysis treatment to correct the metabolic imbalances of acute renal failure. CVVH is used when patients primarily need excess fluid removed, whereas CVVH/D is used when patients also need waste products removed because of uremia. For a comparison of CRRT with hemodialysis and peritoneal dialysis, see Table 30-1 on page 642.

Contraindications to Continuous Renal Replacement Therapy

CRRT is contraindicated when patients become hemodynamically stable or no longer require continuous therapy, and intermittent hemodialysis should be used. It may be difficult to achieve access to circulation in some patients with coagulopathies, which may prolong initiation of therapy. Patient and family discussion is imperative before initiation of therapy; patients may not wish to receive CRRT, and it is essential that patient wishes be considered.

Equipment

A typical CVVH/D setup is shown in Figure 30-5. Blood exits the body through the arterial limb of the vascular access. The first infusion line shown is for anticoagulation. Located just before the blood pump is a line that measures pressure in the prefilter portion of the circuit, known as the arterial pressure. The blood pump, which propels blood into the filter, is next. An infusion port just after the blood pump is usually connected to normal saline solution for flushing the circuit or for attaching the replacement fluid. A bag of dialysate is shown flowing through the filter and surrounding the hollow fibers in which the blood travels. As the dialysate exits the filter, it passes through a sensor that detects microscopic amounts of blood, thereby warning of filter rupture. The dialysate and excess fluid removed from the patient are collected in a graduated/weighed collection device for easy measurement. Meanwhile, the blood exits the filter and passes into a drip chamber, where air and foam are trapped instead of entering the patient's circulation. The drip chamber also contains a line to which a syringe can be attached to raise and lower the blood level and another line that measures pressure in the postfilter section of the circuit, known as venous pressure. A clamp is located after the drip chamber and automatically engages if air tries to pass through it. The arterial and venous pressure transducers are protected by a disposable filter. As blood returns to the body, replacement fluid is infused. In some systems, the line for replacement fluid is placed before the blood pump so that it can be infused before the blood reaches the filter. The total amount of blood in the circuit is about 150 to 200 mL.

Assessment and Management

Preprocedure

Baseline hemodynamics, vital signs, laboratory values (ie, electrolytes and coagulation results), and weight are obtained before initiation of therapy. The potential exists for uncontrolled losses of a large amount of fluid. Because of this, an hourly fluid balance goal is set by the physician. Fluid is either removed or replaced each hour in varying amounts to achieve the fluid balance goal (Box 30-5).

Procedure

Before therapy is initiated, the equipment is checked (see Box 30-4, p. 643). The lines and filter are primed to expel air from the circuit. Arterial and venous lines are connected to the corresponding port of the access catheter, and the blood pump is turned on. Blood starts to flow through the tubing. Ultrafiltration begins to produce plasma water (ultrafiltrate) that starts to flow into the collection device. Blood flow rates through the circuit average 30 to 60 mL/min and up to 200 mL/min for optimal clearance. Substances are adequately cleared when ultrafiltration produces 500 to 600 mL/h of ultrafiltrate.

Anticoagulation, if indicated, is administered as therapy begins. Low-dose heparin is the standard anticoagulant used, and aPTT values should be monitored frequently. It may also be used along with saline flushes to prevent circuit clotting, which is the most common mechanism for interruption of CRRT. Saline flushes without low-dose heparin may be used when the patient has a low platelet count. A typical protocol is to flush 50 to 100 mL through the circuit every hour. Another method of anticoagulation is to infuse citrate before the filter. This anticoagulates only the extracorporeal part of the circuit. It chelates calcium, which is then replaced through infusion through the venous return line or peripherally to maintain normal ionized calcium levels.[4] Therefore, the patient needs to be closely monitored to prevent hypercalcemia or hypocalcemia.

Hourly maintenance of the CVVH/D system includes measuring blood and dialysate flows, calculating net ultrafiltration and replacement fluid, titrating anticoagulants, assessing the integrity of the vascular access, and monitoring hemodynamic parameters and blood circuit pressures. In consultation with the interdisciplinary team, the nephrologist sets a goal for hourly fluid balance, and the critical care nurse is responsible to see that it is met. Box 30-6 lists nursing interventions in monitoring fluid and electrolyte balance. By comparing total intake and output, the hourly net fluid balance is calculated. The amount of replacement fluid is determined by the difference between desired and net fluid balance. Fluid balance and replacement should be carefully documented in the patient's intake and output record.

In CVVH, replacement fluid may be infused before or after the filter. Both techniques have advantages and disadvantages. Fluid given before the filter decreases blood viscosity and increases blood flow through the filter. This enhances ultrafiltrate (plasma fluid) production and solute removal and decreases the frequency of clotting. The disadvantage is the increased need for fluid replacement. If replacement fluid is given after the filter, there is less total fluid loss and less need

BOX 30-5 Example Showing Hourly Fluid Goal With Intake and Output in Fluid Replacement in CVVH/D

1. The patient needs to have 100 mL of fluid removed per hour.
2. The patient receives 450 mL/h of IV fluid (eg, a blood transfusion and IV medications).
3. The patient has 100 mL of chest tube drainage and 50 mL of nasogastric drainage in 1 hour.
4. Dialysate is added at the rate of 1,000 mL/h to increase clearance.
5. The total amount of fluid in the collecting bag at the end of 1 hour is 1,500 mL. (Remember, 1,000 mL is 1 liter dialysate, so 500 mL has been filtered from the patient's plasma.)

To calculate the amount of replacement fluid, add the input fluid of 450 mL of IV fluid and 1,000 mL of dialysate. Output is the 500 mL that has been filtered from the patient's plasma, 50 mL of nasogastric drainage, and the 100 mL of chest tube drainage. Total output is 1,650 mL, and the total input is 1,450 mL. The difference is 200 mL. The hourly fluid removal goal of 100 mL is subtracted, indicating 100 mL has been removed in excess of the goal and needs to be replaced this hour.

BOX 30-6 NURSING INTERVENTIONS

For Monitoring Fluid and Electrolyte Balance During CVVH/D

- Monitor and record patient weight daily—preferably with the same scale and at the same time.
- Draw blood for electrolyte, blood urea nitrogen, and creatinine analysis before initiating treatment and then at least twice daily.
- Assess vital signs, central pressure readings (if available), intake, and output before initiating treatment and at least every hour during treatment.
- Collaborate with nephrologist to determine hourly fluid balance.
- Record all intake and output when calculating replacement fluid for the next hour.
- Administer replacement fluid tailored to the patient's electrolytes or obtain custom-mixed dialysate from the pharmacy.
- If hypotension occurs, administer boluses of saline solution as ordered (100 to 200 mL), reduce ultrafiltration and, if necessary, obtain an order for 5% albumin.
- Observe patient for signs of electrolyte imbalances (ie, electrocardiographic changes and muscle weakness, as with hypokalemia).

for replacement fluid. However, there is an increased incidence of filter clotting and decreased filter life. The method chosen depends on the system used and institutional preference.

Electrolytes, urea nitrogen, creatinine, and glucose levels are measured before the procedure is started and then at least every 6 to 12 hours. Electrolyte imbalances can be corrected by altering the composition of the replacement fluid or by custom-mixing the dialysate. Anticoagulation is monitored by checking activated clotting times or prothrombin time (PT) and partial thromboplastin time (PTT). Although frequency is determined by each institution, it is not unusual to check clotting times every 1 or 2 hours to prevent clotting of the filter and blood lines.

No one policy delineates the optimal time to change the circuit. Many institutions put a 24- to 48-hour limit on circuit life, although there are reports of filters lasting an average of 4 days. System performance is monitored by checking the amount of urea nitrogen in the filtrate compared with the amount of urea nitrogen before the filter. A decreasing ratio indicates inadequate performance. A decreasing rate of ultrafiltration and increases in the venous pressure indicate clotting in the filter.

Treatment may be interrupted to transport the patient for a diagnostic test or to fix a mechanical problem with the circuit or vascular access. Treatment may be terminated if the patient shows signs of recovering renal function. When it is determined that continuous therapy can be terminated, the blood is returned to the patient. First, the ultrafiltrate outlet is clamped, and the dialysate is turned off. Then, anticoagulation is turned off, and the blood is returned to the patient through a saline flush. Once the lines are clear, they are disconnected from the vascular access. Then, the vascular access is flushed and locked per unit policy. Documentation includes fluid balance, condition of the access, and the patient's response to treatment. The tubing and filter are disposable. When working with the circuit and ultrafiltrate, the nurse uses Standard Precautions. Box 30-7 lists some nursing diagnoses associated with hemodialysis and CRRT.

BOX 30-7 EXAMPLES OF NURSING DIAGNOSES

For the Patient Undergoing Hemodialysis or Continuous Renal Replacement Therapy

- Excess Fluid Volume related to renal impairment
- Deficient Fluid Volume related to renal replacement fluid removal
- Ineffective Renal Tissue Perfusion as manifested by decreased renal perfusion
- Risk for Infection related to invasive devices, malnutrition

Technical Complications in Continuous Venovenous Hemofiltration With Dialysis

Access Problems

Blood flows used in CVVH/D are much lower than for hemodialysis, making it more likely that a catheter will provide adequate flow. However, poorly functioning access jeopardizes the entire CVVH/D procedure. Depending on the location of the access catheter (especially femoral or internal jugular), the position of the patient can affect blood flow. An obstruction, such as a clot or kink in the arterial lumen of the catheter, results in less blood being delivered to the circuit and manifests as lowered arterial and venous pressures. Clots or kinks in the venous lumen of the catheter raise venous pressures as blood tries to return against an obstruction. The treatment may be temporarily halted while the nurse manually flushes each lumen to determine patency. If blood flow still cannot be established, the physician is notified for further intervention, such as administration of a thrombolytic to restore patency to the dialysis catheter or to replace the catheter.

Clotting

An early sign of filter clotting is a reduced rate of ultrafiltration that cannot be corrected by increasing blood flow. As clotting progresses, venous pressure rises, arterial pressure drops, and the blood lines appear dark. Clotting times are low. A bolus of saline solution may help determine the location and extent of clotting. It may be possible to return some of the patient's blood before changing the circuit, but if clotting is extensive, this should not be attempted. Box 30-8 lists some nursing interventions for maintaining blood flow through a CVVH/D circuit.

Air in the Circuit

If the connections are loose, or a prefilter infusion line runs dry, air disrupts the system by collecting in the drip chamber and setting off the air detector alarm and triggering the clamp on the venous line to close. The nurse assesses the circuit's integrity to detect the source of air. Before resetting the line clamp, the nurse makes sure all bubbles have been tapped out of the drip chamber, all connections are tight, and there is no danger of air getting into the patient's bloodstream.

BOX 30-8 NURSING INTERVENTIONS

For Maintaining Blood Flow Through A Continuous Venovenous Hemofiltration With Dialysis Circuit

- Check clotting times at initiation and at the prescribed intervals throughout treatment.
- Flush system as often as needed with saline solution to assess appearance of filter and circuit.
- Monitor ultrafiltration rates, venous and arterial pressure, and color of blood in circuit.
- If system is clotting, return as much blood as possible to the patient before changing the system.

Blood Leaks

Blood appears in the ultrafiltrate if there is any rupture inside the filter. The blood leak alarm sounds, and the blood pump stops. Testing the ultrafiltrate with a dipstick can verify a microscopic leak. Blood can be safely returned to the patient as long as there is no gross blood in the ultrafiltrate. Then the circuit should be changed. A gross leak is readily identifiable. Blood should not be returned to the patient, and the patient's hematocrit should be checked to determine the need for transfusion.

Physiological Complications in Continuous Venovenous Hemofiltration With Dialysis

Hypotension

If blood pressure and intravascular filling pressures fall below optimal, the nurse can increase the infusion rate of replacement fluid, decrease the amount of fluid removal, give a normal saline bolus, or titrate existing vasoactive IV drips. An infusion of 5% albumin may also help stabilize blood pressure. If this situation persists, the physician is consulted to adjust the net ultrafiltration goal.

Hypothermia

Some patients experience chills and lowered body temperature while their blood is circulating outside the body. If this happens, it may be advisable to use a blood warmer to warm either the dialysis lines, dialysate, or the replacement fluid. Advancements in the technologies used to perform CRRT have improved the precision of fluid balance and reduced the hypothermia that can develop with any extracorporeal therapy. And because hypothermia can cause clotting deficiencies and dysrhythmias, close monitoring of the patient's temperature along with interventions to achieve normothermia is essential.[2]

Psychological Aspects of Renal Replacement Therapies

The psychological impact of short-term renal replacement therapy is different from that of lifelong therapy. Although the patient depends on a machine in both situations, in short-term therapy, there is usually hope that the patient may recover renal function. Therefore, concerns usually focus on the discomfort associated with insertion of the temporary vascular access and the dialysis treatment. Once these situations are handled, the patient and family then must cope with the uncertainty of how long renal failure will last and how long dialysis will be necessary.

Patients who develop chronic renal failure must deal with the fact that renal replacement therapy will be necessary for the rest of their lives. At first, patients usually deny a great deal of what is happening to them. This may continue for some time and prevent some patients from accepting necessary aspects of their treatment regimen. Other patients who feel considerably better after starting dialysis may enter a "honeymoon phase" and appear quite euphoric for a while. Patients should progress through the normal grieving stages and develop healthy coping mechanisms to deal with their long-term treatment.

Hemodialysis Applied to Other Therapies

The technical equipment and knowledge needed to perform hemodialysis are often applied to other therapies that involve an extracorporeal blood process, such as hemoperfusion and therapeutic apheresis. Hemoperfusion is used primarily for treating drug overdose. Blood is pumped from the body and perfused through a column of charcoal or other absorbent materials that bind the drug. This leads to a rapid reduction in serum levels and avoids potential tissue damage caused by an abnormally high drug level. This therapy is particularly useful for drugs that are fat bound or whose molecular structure is too large to be removed by hemodialysis. With this in mind, critical care nurses may need to take the hemodialysis schedules of their patients into consideration when timing administration of medications. For a list of medications that are removed during dialysis, see Box 30-9.

Therapeutic plasma exchange, or apheresis, is another therapy that may be performed using standard hemodialysis equipment in conjunction with a plasma separator cell and replacement fluids. Apheresis is used to treat diseases caused or complicated by circulating immune complexes or their abnormal proteins. During the procedure, the patient's whole blood is separated into its major components, and the offending components are removed.

BOX 30-9 Examples of Medications Commonly Hemodialyzed

Acetaminophen, acyclovir, allopurinol, amoxicillin, ampicillin, aspirin, atenolol
Captopril, cefazolin, cefepime, cefoxitin, ceftazidime, cimetidine, ciprofloxacin
Enalapril, esmolol
Ferrous sulfate, fluconazole
Ganciclovir, gentamicin
Imipenem
Lisinopril, lithium
Mannitol, meropenem, metformin, methotrexate, methylprednisolone, metoprolol, metronidazole, morphine
Nitroprusside
Penicillin, phenobarbital, piperacillin, procainamide
Salsalate, sotalol, streptomycin, sulfamethoxazole
Theophylline, tobramycin

▲ Peritoneal Dialysis

Peritoneal dialysis and hemodialysis accomplish the same objective and operate on the same principle of diffusion. However, in peritoneal dialysis, the peritoneum is the semipermeable membrane, and osmosis is used to remove fluid, rather than the pressure differentials used in hemodialysis. To access the peritoneal cavity, a Tenckhoff (peritoneal) catheter is inserted (Fig. 30-6). Intermittent peritoneal dialysis (IPD) is an effective alternative method of treating acute renal failure when hemodialysis is not available or when access to the bloodstream is not possible. It sometimes is used as an initial treatment for renal failure while the patient is being evaluated for a hemodialysis program. For a comparison of peritoneal dialysis with hemodialysis and CRRT, see Table 30-1 on page 642.

Peritoneal dialysis has the following advantages over hemodialysis:

- The required technical equipment and supplies are less complicated and more readily available.
- There is less training required by personnel.
- The adverse effects associated with the more efficient hemodialysis are minimized. This may be important for patients with severe cardiac disease, who cannot tolerate rapid hemodynamic changes.
- Patients can learn to manage their own peritoneal dialysis at home.

Peritoneal dialysis also has a few disadvantages, which are as follows:

- It requires more time to remove metabolic wastes adequately and to restore electrolyte and fluid balance.
- Repeated treatments may lead to peritonitis.
- Long periods of immobility may result in complications, such as pulmonary congestion and venous stasis.

Because fluid is introduced into the peritoneal cavity, peritoneal dialysis is contraindicated in patients who have existing peritonitis, in those who have undergone recent or extensive abdominal surgery, and in those who have abdominal adhesions. In the event of a cardiac arrest, the patient's abdomen is drained immediately to maximize the efficiency of chest compressions.

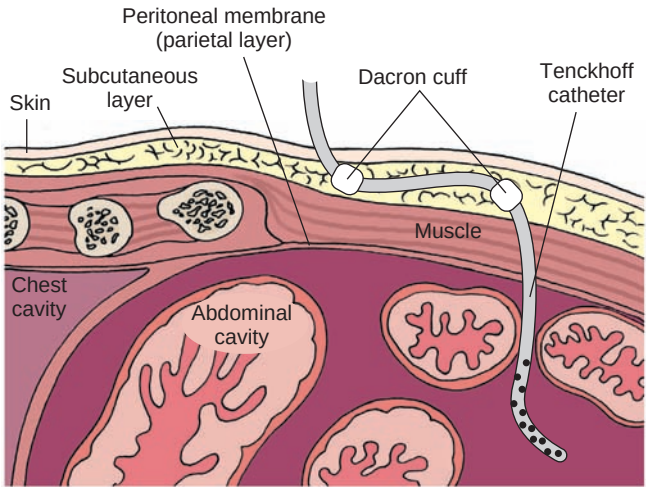


FIGURE 30-6 ▲ A Tenckhoff (peritoneal) catheter is used to access the peritoneal cavity. A Dacron cuff wrapped around the catheter helps to reduce complications related to infection.

Equipment

Solutions

As in hemodialysis, peritoneal dialysis solutions contain "ideal" concentrations of electrolytes but lack urea, creatinine, and other substances that are to be removed. Unlike dialysate used in hemodialysis, solutions must be sterile. Dextrose concentrations of the solutions vary; a 1.5%, 2.5%, or 4.25% dextrose solution can be used. Use of 2.5% or 4.25% solutions usually are reserved for more fluid removal and occasionally for better solute clearance. If peritoneal dialysate does not contain potassium, a small amount of potassium chloride may have to be added to the dialysate to prevent hypokalemia. The patient's serum potassium must be monitored closely to regulate the amount of potassium to be added.

Automated Peritoneal Dialysis Systems

Automated peritoneal dialysis systems have built-in monitors and a system of automatic timing devices that cycle the infusion and removal of peritoneal fluid. For this reason, they are called cyclers, and they may be used in the intensive care setting. They are convenient because they eliminate the need to change solution bags constantly. Most cyclers also have a log that retains cycle-by-cycle information on ultrafiltration. Setting up the cycler requires attaching the appropriate strength of large-volume (5 L) solution bags to the cycler tubing, using aseptic technique. The cycler is programmed to deliver a set amount of dialysate per exchange for a certain length of time. When the time is up, the patient is automatically drained and then refilled. Cyclers are usually used when patients have a permanent peritoneal access device.

Assessment and Management

Preprocedure

Before peritoneal dialysis begins, the nurse must perform the following interventions:

1. If a new catheter is required, prepare the patient for catheter insertion and the dialysis procedure by giving a thorough explanation of the procedure. Depending on hospital policy, a signed consent form may be necessary.
2. Ask the patient to empty his or her bladder just before the procedure.
3. Give a preoperative medication, as ordered, to enhance relaxation during the procedure.
4. Warm the dialyzing fluid to body temperature or slightly warmer, using a device manufactured solely for this purpose. Avoid warming peritoneal dialysate in microwave ovens because of uneven heating of the fluid and inconsistency from one microwave to another.
5. Take and record baseline vital signs, such as temperature, blood pressure, pulse, respirations, and weight. An in-bed scale is ideal for frequent monitoring of the patient's weight.
6. Take the patient's history, identifying abdominal surgery or trauma.
7. Examine the abdomen before inserting the catheter.
8. Follow specific orders, obtained before the procedure, regarding fluid removal, replacement, and drug administration.

Procedure

The following items are needed for the catheter placement procedure:

- Peritoneal dialysis administration set
- Peritoneal dialysis catheter set, which includes the catheter, a connecting tube for connecting the catheter to the administration set, and a metal stylet
- Trocar set of the physician's choice.
- Ancillary drugs: local anesthetic solution (2% lidocaine), aqueous heparin (1,000 units/mL), potassium chloride, broad-spectrum antibiotics

The physician makes a small midline incision just below the umbilicus under sterile conditions. A trocar is inserted through the incision into the peritoneal cavity. The obturator is removed, and the catheter is inserted and secured. The placement of the peritoneal dialysis catheter is ideally performed in a special procedures laboratory or in the operating room.

The dialysis solution flows into the abdominal cavity by gravity as rapidly as possible (5 to 10 minutes; Fig. 30-7). If it flows in too slowly, the catheter may need to be repositioned. When the solution is infused, the tubing is clamped, and the solution remains in the abdominal cavity for 30 to 45 minutes. Next, the solution bottles or bags are placed below the abdominal cavity, and the fluid drains out of the peritoneal cavity by gravity. If the system is patent and the catheter well placed, the fluid drains in a steady, forceful stream. Drainage should take no more than 20 minutes.

This cycle is repeated continuously for the prescribed time, which varies from 12 to 36 hours, depending on the purpose of the treatment, the patient's condition, and the proper functioning of the system. Dialysis effluent is considered a contaminated fluid, and gloves are worn while handling it.

Postprocedure

After the procedure, the nurse must perform the following interventions:

1. Maintain accurate records of intake and output and weights obtained from the same scale for the assessment of volume depletion or overload.
2. Monitor blood pressure and pulse rate. Orthostatic blood pressure changes and increased pulse rate are valuable clues that help the nurse evaluate the patient's volume status.
3. Detect signs and symptoms of peritonitis early. Low-grade fever, abdominal pain, and cloudy peritoneal fluid all are possible signs of infection.
4. Maintain sterility of the peritoneal system. Masks and sterile gloves must be worn while the abdominal dressing is being changed and when the catheter is being accessed or discontinued from the exchange. Solution bags or bottles are changed in as controlled a physical environment

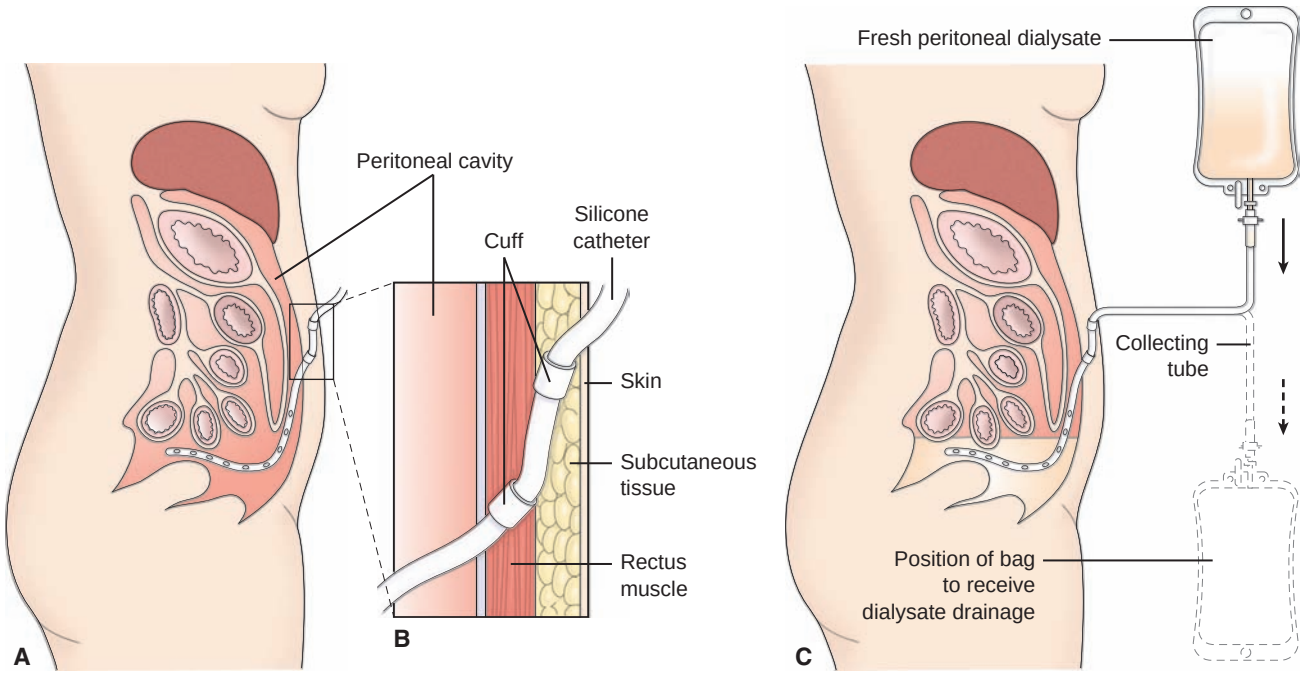


FIGURE 30-7 ▲ Continuous ambulatory peritoneal dialysis. **A:** The peritoneal catheter is implanted through the abdominal wall. **B:** Dacron cuffs and a subcutaneous tunnel provide protection against bacterial infection. **C:** Dialysate flows by gravity into the peritoneal catheter and then into the peritoneal cavity. After a prescribed period of time, the fluid is drained by gravity and discarded. New solution is then infused into the peritoneal cavity until the next drainage period. Dialysis thus continues on a 24-hour-a-day basis during which the patient is free to move around and engage in his or her usual activities. (From Smeltzer SC, Bare BG, Hinkle JL, et al: Brunner & Suddarth's Textbook of Medical–Surgical Nursing, 12th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 1341.)

as possible to avoid contamination (eg, avoiding areas of high traffic and high air flow).
5. Detect and correct technical difficulties early before they result in physiological problems. Slow outflow of the peritoneal fluid may indicate early problems with the patency of the peritoneal catheter.
6. Prevent complications of bed rest and provide an environment that helps the patient in accepting bed rest for prolonged periods.
7. Prevent constipation. Difficult or infrequent defecation decreases the clearance of waste products and causes the patient more discomfort and distention.

Technical Complications

Incomplete Recovery of Fluid

The fluid that is removed should equal or exceed the amount infused. Commercially prepared dialysate contains about 1,000 to 2,000 mL of fluid. If, after several exchanges, the volume drained is less (by 500 mL or more) than the amount infused, an evaluation must be made. Signs of fluid retention include abdominal distention or complaints of fullness. The most accurate indication of the amount of unrecovered fluid is weight.

If the fluid drains slowly, the catheter tip may be buried in the omentum or clogged with fibrin. Turning the patient from side to side, elevating the head of the bed, and gently massaging the abdomen may facilitate drainage.

If fibrin or blood exists in the outflow drainage, heparin needs to be added to the dialysate. The specific dose, which is ordered by the physician, is 500 to 1,000 units/L.

Leakage Around the Catheter

Superficial leakage after surgery may be controlled with extra sutures and a decrease in the amount of dialysate instilled into the peritoneum. Increases in intra-abdominal pressure may also cause dialysate leaks. Therefore, continued vomiting, coughing, and jarring movements should be avoided during the initial postoperative period. The abdominal dressing must be checked frequently to detect leakage. Dialysate leaks can be distinguished from other clear fluids by checking with a dextrose test strip. Dialysate tests positive because of its dextrose content. A leaking catheter must be corrected because it acts as a pathway for bacteria to enter the peritoneum.

BOX 30–10 NURSING INTERVENTIONS

For Preventing Infection During Peritoneal Dialysis

- Maintain aseptic technique throughout dialysis procedure.
- Use sealed plastic dialysate bags.
- Change dialysis tubing regularly per protocol.
- Swab or soak tubing connections and injection ports with bactericidal solution before adding medications or breaking closed system.
- Assess patient continuously for signs and symptoms of peritonitis (pain, cloudy effluent, fever).
- Change exit site dressing daily using aseptic technique until healing occurs. Assess daily for increase in inflammation or drainage.
- If infection is suspected, obtain appropriate culture and begin antibiotic according to protocol or physician's order.

Blood-Tinged Peritoneal Fluid

Blood-tinged peritoneal fluid is expected in the initial outflow but should clear after a few exchanges. Gross bleeding at any time is an indication of a more serious problem and must be investigated immediately.

Physiological Complications

Peritonitis

Peritonitis is a serious, but manageable, complication of peritoneal dialysis. Signs of peritonitis include low-grade fever, abdominal pain when fluid is being inserted, and cloudy peritoneal drainage fluid. Early detection and treatment reduce the patient's discomfort and prevent more serious complications.

Treatment begins as soon as a sample of peritoneal fluid is obtained for culture and sensitivity. The patient is started on a broad-spectrum antibiotic, which is usually added to the dialysate solution, although it also can be given intravenously. Depending on the severity of the infection, the patient's condition should improve dramatically after 8 hours of antibiotic therapy.

Catheter Infection

During the daily dressing change, the nurse examines the exit site closely for signs of infection, such as tenderness, redness, and drainage around the catheter. In the absence of peritonitis, a catheter infection usually is treated with an oral, broad-spectrum antibiotic. Box 30-10 lists nursing interventions for preventing infections during peritoneal dialysis.

Hypotension

Hypotension may occur if excessive fluid is removed. Vital signs are monitored frequently, especially if a hypertonic solution is used. Lying and sitting blood pressure readings are especially useful for evaluating fluid status. Progressive drops in blood pressure and weight are signs of fluid deficit.

Hypertension and Fluid Overload

If all the dialysate solution is not removed in each cycle, hypertension and fluid overload may occur. If there is hypertension and a weight increase, the nurse assesses catheter patency and notes the exact amount of fluid in the dialysate bag. Some manufacturers add 50 mL to a 1,000-mL bag. Over a period of hours, this can make a considerable difference.

The nurse also observes the patient for signs of respiratory distress and pulmonary congestion. In the absence of other symptoms of fluid overload, hypertension may be the result of anxiety and apprehension. Nonpharmacological measures to reduce anxiety are preferable to administering sedatives and tranquilizers.

High Blood Urea Nitrogen and Creatinine Levels

Blood urea nitrogen and creatinine levels are closely monitored because they help evaluate the effectiveness of the dialysis. When levels remain high, it indicates inadequate clearance of these waste products.

Hypokalemia

The serum potassium is monitored closely because hypokalemia is a common complication of peritoneal dialysis. When the serum potassium level is low, potassium chloride is added to the dialysate.

Hyperglycemia

Supplemental insulin can be added to the dialysate to control hyperglycemia. Blood glucose levels should be monitored closely in patients with diabetes mellitus and hepatic disease.

Pain

Patients may experience mild abdominal discomfort at any time during the procedure. It is probably related to the constant distention or chemical irritation of the peritoneum. If a mild analgesic does not provide relief, injecting 5 mL of 2% lidocaine directly into the catheter may help. The patient may be more comfortable if nourishment is given in small amounts, when the fluid is draining out rather than when the abdominal cavity is distended.

Severe pain may indicate more serious problems of infection or paralytic ileus. Infection is not likely in the first 24 hours. Aseptic technique and prophylactic antibiotics minimize the risk for infection. Periodic cultures of the outflowing fluid help in the early detection of pathogenic organisms.

Immobility

Immobility may lead to hypostatic pneumonia, especially in the debilitated or older patient. Deep breathing, turning, and coughing should be encouraged during the procedure. Leg exercises and the use of elastic stockings may prevent the development of venous thrombi and emboli.

Discomfort

Peritoneal dialysis results in slower clearance of waste products than hemodialysis; therefore, it is rarely associated with the dysequilibrium seen with hemodialysis. However, boredom is a frequent problem because the treatment is longer. Nursing measures are directed toward making the patient as comfortable as possible. Diversions such as reading, watching television, and visitors should be encouraged. Educating the patient about peritoneal dialysis and involving the patient in the care may reduce some of the anxiety and discomfort.

Peritoneal Dialysis as a Chronic Treatment

Intermittent peritoneal dialysis (IPD) has been used for chronic therapy for some time, but it requires the patient to remain stationary for up to 10 to 14 hours, three times per week. Because of this inconvenience to the patient and increased staff time needed if this therapy is performed in-center, IPD seldom is used and is not available in many dialysis centers.

Peritoneal dialysis has gained popularity as a chronic form of dialysis therapy, especially since continuous ambulatory peritoneal dialysis (CAPD) has become available. CAPD is easily taught to patients and does not limit ambulation between dialysate fluid exchanges. It uses the dialysis fluid that is continuously present in the peritoneal cavity 24 hours a day, 7 days a week. Dialysis fluid is drained by the patient and replaced with fresh solution three to five times per day. The number of solution exchanges needed per day depends on the patient's individual needs. Although the patient is required to perform dialysis techniques every day, CAPD is attractive to many patients with end-stage renal disease (ESRD) because they can accomplish it easily and independently. CAPD may also be preferred in patients who benefit from a slow, continuous removal of sodium and water, such as in those with refractory congestive heart failure.

Continuous cyclic peritoneal dialysis (CCPD) is another variation of chronic peritoneal dialysis therapy. Patients who choose this form of therapy perform IPD at night during sleep using a cycling machine and in the morning instill dialysis fluid, which remains in the abdomen during the entire day. This is most convenient for patients who require the help of working family members to perform their exchanges.

As with acute peritoneal dialysis, peritonitis is the greatest potential problem associated with chronic forms of dialysis. Peritoneal catheters are permanent and inserted in the operating room. Such catheters have one or two Dacron cuffs that the surgeon sutures to the abdominal wall, subcutaneous tissue, or both to anchor the catheter and provide a permanent seal against invading bacteria. Patients are taught how to recognize any potential problem associated with the catheter or treatment and to seek help from the CAPD team when needed.

Patients who perform IPD, CAPD, or CCPD at home usually visit the dialysis unit every 4 to 8 weeks. At this time, a nursing assessment is performed, techniques are reviewed, and required blood studies are obtained. All health team members, including the physician, nurse, dietitian, and social worker, work together with the patient and family to ensure successful adaptation to the chosen mode of treatment. Box 30-10 presents a discharge planning guide for the patient undergoing chronic peritoneal dialysis.

▲ Pharmacological Management of Renal Dysfunction

When the kidneys fail, treatment such as dialysis may be used to achieve fluid and electrolyte balance. Pharmacological treatment may be initiated to enhance an already functional kidney, attempt to recover renal function, or optimize fluid balance.

Diuretics

Diuretics are drugs that promote fluid removal through increased urine production. There are three major classes of diuretics: loop, thiazide, and potassium sparing. Table 30-4 presents information about the various diuretics. In addition, acetazolamide (a carbonic anhydrase inhibitor) and mannitol (an osmotic diuretic) may be used to promote fluid removal. It may be necessary to use combination therapy to achieve the desired therapeutic end point. Drugs from different classes are chosen to maximize urine production in combination therapy.

Table 30-4 Diuretic Drugs

Drug	Mechanism	Complication(s)	Management and Prevention
Loop diuretics Examples: furosemide (Lasix), ethacrynic acid (Edecrin), bumetanide (Bumex)	Powerful diuretics that act primarily in the thick segment of the medullary and cortical ascending limbs of loop of Henle Cause loss of sodium, chloride, and potassium Increase calcium excretion	Volume depletion Hypokalemia Hyponatremia Hypocalcemia Hypomagnesemia	Monitor daily weights, intake and output, and signs and symptoms of volume depletion. Potassium supplements or extradietary potassium may be necessary when loop diuretics are used routinely. Monitor calcium levels and administer supplemental calcium as indicated. Usually no intervention is required because body stores of magnesium are quite high.
Thiazides Examples: chlorothiazide (Diuril), hydrochlorothiazide	Act by inhibiting sodium reabsorption in distal tubule and, to a lesser extent, inner medullary collecting duct Cause loss of sodium, chloride, and potassium Decrease urinary calcium excretion	Hypercalcemia Hypokalemia Hypomagnesemia	Monitor calcium levels; loop diuretic may be indicated if calcium levels are persistently elevated. Potassium supplements or extradietary potassium may be necessary when these agents are used routinely. Usually no intervention is required because body stores of magnesium are quite high.
Potassium-sparing diuretics Examples: spironolactone (Aldactone), triamterene (Dyrenium), amiloride (Midamor)	Conserve potassium Spironolactone inhibits action of aldosterone, thereby reducing sodium reabsorption while increasing potassium reabsorption Triamterene acts on distal renal tubule to depress exchange of sodium Effects of amiloride are apparently due to inhibition of sodium entry into cell from luminal fluid	Hyperkalemia	Potassium supplements are contraindicated, as are salt substitutes containing potassium. Often combined with thiazides for effective diuresis; hypokalemic tendency of thiazides may offset hyperkalemic tendency of triamterene and spironolactone.
Carbonic anhydrase inhibitors Example: acetazolamide (Diamox)	Decrease proximal tubular sodium reabsorption Not very effective when administered alone Facilitate excretion of bicarbonate	Metabolic acidosis	Monitor pH and bicarbonate.
Osmotic diuretics Example: mannitol	Nonreabsorbable polysaccharide that pulls water into vascular space, thereby increasing glomerular flow	Hyperosmolarity	Monitor serum osmolarity. Withhold mannitol therapy if serum osmolarity is >300–305 mMol/L.

Diuretics may be administered orally or IV. The effect is more immediate with IV therapy. The patient is monitored for breath sounds, hemodynamic value changes, weight, and peripheral edema to determine his or her response to therapy. Careful laboratory assessment of the blood urea nitrogen and creatinine level is required to monitor for development or worsening of acute renal failure. Ideally, the patient's pulmonary status and fluid balance improve while the glomerular filtration rate (GFR) remains normal.

Diuretics have both desirable and undesirable effects (see Table 30-4). Overdiuresis is the most common side effect. The nurse must monitor for fluid volume depletion, especially when diuretic regimens are altered or initiated. Signs of volume depletion are discussed in Chapter 29. Other side effects include hyponatremia, hypokalemia, hyperkalemia, hypocalcemia, hypercalcemia, hypomagnesemia, and acid–base disturbances. A reduction in volume from vomiting, third-spacing of fluid, diuretic therapy, or other conditions may have the same consequences. Box 30-11 lists nursing diagnoses for patients taking diuretics.

Hypokalemia is another common side effect of diuretics, particularly the loop and thiazide diuretics. In general, hypokalemia is a benign condition that can be managed effectively with potassium supplementation. If left untreated, patients may experience harmful, sometimes life-threatening, cardiac dysrhythmias.

BOX 30-11 EXAMPLES OF NURSING DIAGNOSES

For the Patient Undergoing Diuretic Therapy

- Impaired Gas Exchange related to increased pulmonary hydrostatic pressure secondary to congestive heart failure
- Risk for Deficient Fluid Volume related to effect of diuretic therapy
- Risk for Electrolyte Imbalance
- Risk for Acute Confusion related to electrolyte imbalance/fluid shifts

Vasoactive Drugs

Sometimes, the cause of decreased effective circulatory volume is reduced cardiac contractility. In such a case, an inotropic agent (eg, dobutamine or milrinone) may be added to the plan of care to improve the forward flow of the heart, thereby improving the effective circulatory volume and stopping the cascade of counterproductive compensatory mechanisms. A failing heart, such as in congestive heart failure, can cause reduced blood flow to the kidney and potentiate acute renal failure. The same compensatory mechanisms used in volume depletion operate in an attempt to restore renal function. Namely, the renin–angiotensin–aldosterone system is activated to increase sodium and water retention and achieve renal and peripheral vasoconstriction. Other common inotropic agents are described in detail in Chapter 18.

Dopamine is a vasoactive drug at higher doses but can stimulate dopaminergic receptors in the kidneys when infused at lower doses (1 to 3 mcg/kg/min). Stimulation of dopamine receptors increases renal blood flow and promotes natriuresis. Although this practice may sometimes still be used to prevent or treat acute renal failure in some settings, several studies have found that there is no improvement in clinical outcome and a lack of sufficient clinical evidence to support its routine use.[5]

▲ Disorders of Fluid Volume

Critically ill patients often have imbalances in fluid homeostasis related to their primary underlying disease. Fluid imbalance occurs when there is an excess or deficit of fluid and may be either absolute or relative. Medications, such as diuretics, put patients at increased risk for fluid imbalance. Infection increases metabolic demand and insensible loss, and fluid volume deficits may develop. Regardless of patient diagnosis, assessment of fluid balance (see Chapter 29) and careful management are mainstays of patient care in the critical care setting.

Fluid Volume Deficit

When fluid loss exceeds intake, a fluid volume deficit exists. A fluid volume deficit is a physiological situation in which fluids are lost in an isotonic fashion (both fluid and electrolytes are lost together). Dehydration is the loss of water alone, resulting in a hyperosmolar state. Although the critically ill patient typically can have both a fluid volume deficit and dehydration states simultaneously, this discussion is limited strictly to disorders of fluid volume deficit.

Several patient populations are particularly vulnerable to the development of fluid volume deficits. Young children at prespeech developmental levels cannot communicate thirst; therefore, during times when fluid requirements increase, they do not increase their fluid intake of their own accord. Debilitated patients, for example, patients affected by stroke, may have difficulty communicating their needs or have swallowing disturbances and cannot manage their own intake of fluid. Elderly patients are at particular risk for a fluid volume deficit because of the multisystem changes associated with aging. For a review of the changes associated with aging and nursing implications for fluid volume assessment and management, see Table 30-2 on page 643.

Causes

GASTROINTESTINAL LOSS. Physiologically, the body produces approximately 5 L of gastrointestinal fluid. In the gastrointestinal tract, fluids help act as a carrier of important enzymes and buffers to aid in digestion. In the distal small intestine and large intestine, fluid is reabsorbed, leaving only approximately 150 mL lost through the stool daily.

Excess loss from any site from which fluids are ordinarily lost may cause a fluid imbalance. Conditions such as vomiting and diarrhea may cause an increase beyond the typical 150 mL and result in a fluid volume deficit. In addition, surgically placed drainage tubes and nasogastric tubes used for suction may cause such a deficit.

INFECTION. Infection causes fluid deficits in several ways as follows:

1. Infection can increase metabolic demand, increasing insensible water loss. When patients are not critically ill, they often mitigate this imbalance by increasing fluid intake. When they have widespread infections or a self-care deficit, which may occur in elderly people, fluid intake may not be sufficient to restore fluid balance.
2. Mediators are released as part of the immune response. These mediators cause a loosening of the capillary tight junctions, resulting in the third-spacing of fluids.
3. Carbon dioxide production increases due to increased metabolism. To maintain pH balance, tachypnea may develop. Although only a very small amount of fluid is lost daily through the respiratory tract, water loss may become clinically significant when the respiratory rate is greater than 35 breaths/min.

RENAL LOSS. The kidneys filter approximately 180 L/d. However, urine output is only 1% to 2% of total blood volume filtered. Reabsorption of fluid is influenced by a complex regulatory system that includes the actions of aldosterone, angiotensin, and antidiuretic hormone (ADH). A defect in any one of the regulatory functions can cause a disruption in renal fluid balance.

Several endocrine disorders may disrupt the renal regulatory system. Adrenal insufficiency, the absence of glucocorticoids and aldosterone, can reduce the absorption of sodium, thereby promoting water loss. Diabetes insipidus is a profound reduction in ADH, which reduces the amount of fluid reabsorbed at the distal convoluted tubule. Water loss predominates in diabetes insipidus, and therefore, volume imbalance is related to dehydration (see Chapter 44).

Serum osmolarity is predicted by sodium, glucose, and blood urea nitrogen. Normally, glucose does not influence the overall osmolarity. However, in profound hyperglycemia, the influence of glucose increases greatly. Serum osmolarity increases and is sensed by the osmoreceptors, thereby pulling fluids into the vascular space and initiating an osmotic diuresis. Two conditions that pathologically increase glucose are diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar nonketotic coma. Both of these disorders are discussed in more detail in Chapter 44.

Diuretic therapy is intended to treat fluid volume excess. However, overadministration of diuretics may result in a fluid volume deficit. It is important to recognize the immediate onset that diuretics can have when administered intravenously, initiated for the first time, or adjusted in dosage (see Table 30-4 for more information).

THIRD-SPACING OF FLUID. Third-spacing of fluid is the movement of fluid from the vascular space to the interstitial space. To create a movement of fluid between body compartments, there is an alteration in capillary permeability because of inflammation, ischemia, or injury. Causes of third-spacing of fluids are numerous and include infection; systemic inflammatory response syndrome, such as in pancreatitis; hypoalbuminemia, such as in liver failure; burns; intestinal obstruction; and surgery. The amount of fluid lost depends on the degree of the pathophysiological alteration. Regardless of cause, the fluid lost is not functioning to maintain vascular volume, and therefore, a fluid volume deficit exists. When fluid leaks out of the vascular space, daily weights can increase, paradoxically, despite intravascular volume depletion.

Management: Fluid Volume Replacement

To correct a fluid volume deficit, it is necessary to treat the underlying cause and replace the lost fluid. The main purposes of fluid administration include replacement of lost fluid, maintenance of fluid balance, and replacement of lost electrolytes. Several types of fluids, which have different physiological effects, are available. Administration of fluids may occur using the gastrointestinal tract or an IV or intraosseous route. When chronic replacement is required, such as in patients with long-term tube feeding, the gastrointestinal approach is used. Enteral access is required when patients cannot take fluids by mouth. When rapid restoration of fluid balance is required, the IV route is preferred. Occasionally, both routes are used.

MAINTENANCE FLUIDS. Under normal conditions, the average healthy adult requires about 2.5 L/d. This volume replaces fluids lost through the feces, the respiratory tract, sweating, and the urine. Patients who are unable to consume their usual intake of fluid are often prescribed IV maintenance fluids of 2 to 3 L/d. When determining the rate of administration of maintenance fluid, factors such as medical history (renal failure), age (young or old), confounding water excesses (heart failure), and ongoing assessment parameters (edema formation) must be considered.

REPLACEMENT FLUIDS. Critically ill patients are often unable to consume the additional fluid required to replace the lost fluid. In this case, IV administration beyond baseline maintenance fluids is required for homeostasis. This is achieved either by administering a bolus of fluid or by increasing the total daily fluid intake. When fluid loss occurs acutely, the loss must be replaced immediately to maintain tissue perfusion. The type of fluid given depends on the type of fluid lost. When whole blood is lost, such as in trauma or surgery, blood may be administered. When intravascular volume is depleted, such as in diarrhea, isotonic solutions may be administered. The rate of administration depends on the patient's medical history and amount of volume lost.

Crystalloids Crystalloid solutions are prepared with a specified balance of water and electrolytes. Box 30-12 provides a description of commonly used crystalloid solutions, and although here they are described separately, they are most commonly used in combination. Fluids are classified as hypotonic (osmolarity <250 mEq/L), isotonic (osmolarity approximately 310 mEq/L), or hypertonic (osmolarity > 376 mEq/L).

Dextrose solutions are given to provide free water and some calories to prevent protein catabolism. The 5% solution contains 50 g of dextrose for every liter of fluid and provides approximately 170 cal/L. When pure dextrose solutions, such as 5% dextrose in water (D_5W), are administered, the dextrose is metabolized, resulting in the administration of free water. When given intravenously, free water decreases the plasma osmolarity, thereby promoting the movement of water evenly into all body compartments. Free water does not stay in the vascular space; therefore, pure dextrose solutions should not be used when intravascular replacement of fluids is required.

Saline solutions are commonly used and are available in different strengths, such as 0.9% and 0.45%. Normal saline, or 0.9% saline, is an isotonic solution. Approximately one fourth of the fluid administered remains in the vascular space, and the remaining fluid moves into the extracellular space 1 hour after administration. During critical illness, the amount that exits into the extracellular space can increase as a result of increased capillary permeability.

Half-strength (0.45%) saline solution, in comparison, is a hypotonic solution. Additional free water is administered

BOX 30-12 Common Crystalloid Solutions

5% dextrose in water (D_5W): no electrolytes, 50 g dextrose
- Supplies about 170 cal/L and free water to aid in renal excretion of solutes
- Should not be used in excessive volumes in patients with increased ADH activity or to replace fluids in hypovolemic patients

0.9% NaCl (isotonic saline): Na^+ 154 mEq/L, Cl^- 154 mEq/L
- Isotonic fluid commonly used to expand the extracellular fluid in presence of hypovolemia
- Because of relatively high chloride content, it can be used to treat mild metabolic alkalosis

0.45% NaCl (½ strength saline): Na^+ 77 mEq/L, Cl^- 77 mEq/L
- A hypotonic solution that provides sodium, chloride, and free water (sodium and chloride provided in fluid allow kidneys to select and retain needed amounts)
- Free water desirable as aid to kidneys in elimination of solutes

0.33% NaCl (1/3 strength saline): Na^+ 56 mEq/L, Cl^- 56 mEq/L
- A hypotonic solution that provides sodium, chloride, and free water
- Often used to treat hypernatremia (because this solution contains a small amount of sodium, it dilutes the plasma sodium while not allowing the level to drop too rapidly)

3% Saline Solution
- Grossly hypertonic solution used only to treat severe hyponatremia
- This solution used only in settings where the patient can be closely monitored

Lactated Ringer's solution: Na^+ 130 mEq/L, K^+ 4 mEq/L, Ca^{2+} 3 mEq/L, Cl^- 109 mEq/L, lactate (metabolized to bicarbonate) 28 mEq/L
- Approximately isotonic solution that contains multiple electrolytes in about same concentrations as found in plasma (note that this solution is lacking magnesium and phosphate)
- Used in treating hypovolemia, burns, and fluid lost as bile or diarrhea
- Useful in treating mild metabolic acidosis

with this solution, making it an ideal maintenance fluid. Occasionally, half-strength saline solution is administered to replace fluids lost when there is concurrent hypernatremia.

Saline solutions, such as 3% saline, are hypertonic and may be given to treat symptomatic hyponatremia. The hypertonicity pulls fluid from the extravascular space to the vascular space. Hypertonic solutions should be administered only when patients may be closely monitored because fluid volume excess can develop rapidly. Some studies have shown that hypertonic saline solutions, such as 3% or 7.5% saline, may be beneficial during resuscitation.[6,7]

Colloids. Colloids are high-molecular-weight substances and therefore do not cross the capillary membrane under normal conditions. Table 30-5 describes commonly prepared colloid solutions.

Albumin is the most abundant circulating protein in the body and accounts for 80% of the colloidal oncotic pressure. For therapeutic uses, albumin is prepared from donor plasma. With albumin, there is no risk of bloodborne diseases, such as hepatitis or human immunodeficiency virus infection. Albumin is available in two concentrations, 5% and 25%, and both preparations contain some sodium. The 5% solution is similar in osmolarity to plasma. In contrast, the 25% solution is hypertonic, thereby pulling extravascular water into the vascular space. Both preparations of albumin can cause the intravascular volume to expand beyond the volume of albumin infused because of the increased oncotic pressure generated. Care must be taken when administering albumin to patients at high risk for volume overload. Use of albumin should also be limited in patients with profound capillary leak syndrome (eg, in sepsis, acute respiratory distress syndrome, and pancreatitis[7]). Although albumin is a protein, it is inefficient and expensive when used for malnutrition.

The starches dextran and hetastarch, which differ from each other only slightly, have an oncotic pressure similar to albumin. Both substances are used to expand plasma volume by exerting an oncotic pressure and thereby pulling water from the extravascular space to the vascular space. Hetastarch is metabolized by both the kidneys and liver. The diuresis that may occur with hetastarch is an osmotic diuresis and does not reflect an increase in effective renal circulatory volume. Both dextran and hetastarch may cause coagulopathies; however, dextran has a more profound effect on coagulation.

Fluid Volume Excess

Fluid volume excess occurs when there is retention of sodium, resulting in the reabsorption of water. Electrolytes typically remain unchanged when there is an increase in total body water and electrolytes increase in parallel. Many critically ill patients may have mixed disturbances with manifestations of the confounding compensatory mechanisms. Causes of fluid volume excess include overadministration of fluids, edematous disorders (eg, congestive heart failure, kidney, or liver failure), excessive sodium intake, and medications (eg, steroids, desmopressin acetate).

When the kidneys are functioning normally and regulating fluid balance, the body typically rids itself of excess fluid, and fluid overload is not manifested clinically. When the kidneys sense a decrease in effective circulatory volume, the compensatory mechanisms prevent the excretion of excess water, such as in congestive heart failure.

Management of fluid volume excess is directed toward correcting the underlying disorder. If this is not feasible, efforts are geared to preventing pulmonary compromise by attempting to rid the body of the excess sodium and water. In cases of volume overload, there is an increase in pulmonary hydrostatic pressure, which promotes movement of water into the alveoli, thereby impeding gas exchange. Sodium restriction reduces the amount of water reabsorption but does contribute to acute correction of

Table 30-5 Common Colloid Solutions

Solution	Contents	Indications	Comments
Albumin	Available in two concentrations: 5%: oncotically similar to plasma 25%: hypertonic Both 5% and 25% solutions contain about 130–160 mEq/L of sodium	Used as volume expander in treatment of shock May be useful in treating burns and third-spacing shifts	Cost is ~25–30 times more than for crystalloid solutions. Increased interstitial oncotic pressure in disease states in which there is increased capillary leaking (eg, burns, sepsis) may occur; this may result in increased vascular space loss of fluid. Use caution with rapid administration; watch for volume overload.
Hetastarch	Synthetic colloid made from starch (6%) and added to sodium chloride solution	May be used to expand plasma volume when volume is lost from hemorrhage, trauma, burns, and sepsis	Plasma volume expansion effects decrease over 24–36 h Starch is eliminated by kidneys and liver; therefore, use caution in patients with liver and kidney impairment. Mild, transient coagulopathies may occur. Transient rise in serum amylase may occur.
Dextran	Glucose polysaccharide substance, available as low-molecular-weight dextran (dextran 40) or high-molecular-weight dextran (dextran 70) No electrolyte content	May be used to expand plasma volume when volume is lost from hemorrhage, trauma, burns, and sepsis	Has been associated with greater risk for allergic reaction than albumin or hetastarch. Interference with blood cross-matching may occur. May cause coagulopathy; has more profound effect on coagulation than hetastarch.

volume overload. Diuretics are the mainstay of treatment for acute resolution of fluid volume excess (see Table 30-4).

▲ Management of Electrolyte Imbalances

Electrolyte disorders commonly occur in critically ill patients, typically in combination with other conditions. Management of the underlying problem ensures long-term restoration of balance. However, acute management of electrolyte disorders is often required to maintain cellular integrity.

Sodium

Sodium is the major extracellular cation. It is a major predictor of serum osmolarity and controls movement of water. Disorders of sodium are typically associated with water disorders (Table 30-6).

Table 30-6 Management of Electrolyte Disorders

Electrolyte	Selected Medical Conditions Associated With Disturbance	Collaborative Interventions
Sodium		
Hyponatremia	Congestive heart failure Liver failure Kidney failure Hyperlipidemia Hypoproteinemia SIADH GI loss Adrenal insufficiency Thiazide diuretics Drugs: NSAIDs, tricyclic antidepressants, SSRIs, chlorpropamide, omeprazole Tumors associated with ectopic excessive ADH production: oat cell carcinoma, leukemia, lymphoma Pulmonary disorders: pneumonia, acute asthma AIDS	Review medication profile and patient history. Monitor for sites of fluid losses or gains. Monitor fluid balances and for signs and symptoms of electrolyte disturbance. Attempt to manage underlying cause. Correction of electrolyte may require sodium replacement (3% saline solution) or water restriction, depending on etiology of disorder.
Hypernatremia	Profound dehydration usually in patients not able to ask for water (eg, debilitated elderly or children), in those with impaired thirst regulation (eg, elderly), or in those with heatstroke Hypertonic tube feedings without water supplementation Increased insensible water loss (eg, excessive sweating, second- and third-degree burns, hyperventilation) Excessive administration of sodium-containing fluids (3% saline, sodium bicarbonate) Diabetes insipidus	Assess in patients at particular risk for hypernatremia, including debilitated or elderly patients, acutely or critically ill children, and patients receiving tube feedings. Monitor laboratory values closely in patients with insensible fluid losses and in those receiving parenteral administration of sodium-containing fluids. For comprehensive review of management of diabetes insipidus, see Chapter 44. Administer therapeutic medications, including vasopressin, DDAVP. Administer hypotonic fluids (1/2 saline to free water, D_5W).
Potassium		
Hypokalemia	GI loss: diarrhea, laxatives, gastric suction Renal loss: potassium-losing diuretics, hyperaldosteronism, osmotic diuresis, steroids, some antibiotics Intracellular shifts: alkalosis, excessive secretion or administration of insulin, hyperalimentation Poor intake: anorexia nervosa, alcoholism, debilitation	Monitor laboratory values closely in patients at particular risk for hypokalemia. Pay particular attention to potassium level in patients receiving digoxin. Administer potassium either orally or IV (see Box 30-13, p. 661). Monitor magnesium levels in patients who are refractory to potassium replacement.
Hyperkalemia	Pseudohyperkalemia: prolonged tight application of tourniquet; fist clenching and unclenching immediately before or during blood draws; hemolysis of blood sample Decreased potassium excretion: oliguric renal failure, potassium-sparing diuretics, hypoaldosteronism High potassium intake: improper use of oral potassium supplements; rapid IV potassium administration Extracellular shifts: acidosis, crush injuries, tumor cell lysis after chemotherapy	Ensure that minimal negative pressure is used to obtain all laboratory samples, particularly when drawn through small-gauge needles. Restrict potassium-sparing diuretics. Promote excretion: sodium polystyrene sulfonate orally or rectally, dialysis, potassium-losing diuretics (eg, furosemide) Emergency management measures: calcium IV, sodium bicarbonate, IV insulin with glucose, β_2-adrenergic agonists

(continued on page 659)

Table 30-6 Management of Electrolyte Disorders (continued)

Electrolyte	Selected Medical Conditions Associated With Disturbance	Collaborative Interventions
Calcium		
Hypocalcemia	Surgical hypoparathyroidism Primary hypoparathyroidism Malabsorption (alcoholism) Acute pancreatitis Excessive administration of citrated blood Alkalotic states Drugs (loop diuretics, mithramycin, calcitonin) Hyperphosphatemia Sepsis Hypomagnesemia Medullary carcinoma of thyroid Hypoalbuminemia	Monitor for signs and symptoms associated with low calcium, especially for seizures, and stridor. Administer calcium IV for acute replacement (see Box 30-13, p. 661). Ensure adequate dietary intake for patients at particular risk.
Hypercalcemia	Hyperparathyroidism Malignant neoplastic disease Drugs (thiazide diuretics, lithium, theophylline) Prolonged immobilization Dehydration	Administer bisphosphonates, such as etidronate or mithramycin, especially when disorder is related to malignancy. Administer diuretics, such as loop diuretics, to promote renal excretion. Provide fluid replacement with 0.9% saline solution.
Magnesium		
Hypomagnesemia	Inadequate intake: starvation, total parenteral nutrition without adequate Mg^{2+} supplementation, chronic alcoholism Increased GI loss: diarrhea, laxatives, fistulas, nasogastric tube suction, vomiting Increased renal loss: drugs (loop and thiazide diuretics, mannitol, amphotericin B), diuresis (uncontrolled diabetes mellitus, hypoaldosteronism) Changes in magnesium distribution: pancreatitis, burns, insulin, blood products	Monitor for hypokalemia in patients with low magnesium because kidneys are not able to conserve potassium when magnesium level is low. Administer magnesium IV for acute replacement (see Box 30-13, p. 661). Administer PO preparations for long-term replacement.
Hypermagnesemia	Renal failure Excessive intake of magnesium-containing compounds (eg, antacids, mineral supplements, laxatives)	Avoid administration of magnesium-containing compounds to patients in renal failure. In extreme cases, dialysis may be indicated.
Phosphorus		
Hypophosphatemia	Refeeding syndrome Alcoholism Phosphate-binding antacids Respiratory alkalosis Administration of exogenous insulin IV Burns	Ensure nutritional intake. Monitor phosphorus for the first few days after initiation of enteral or parenteral nutrition. Administer by oral supplementation (Neutra-Phos capsules) or IV (see Box 30-13, p. 661).
Hyperphosphatemia	Renal failure Chemotherapy Excessive administration of phosphate compounds	Prevention is mainstay of therapy; avoid administration of phosphorus to patients in renal failure. Administer calcium acetate. Administer IV fluids to promote renal excretion. In severe cases, administration of high levels of glucose with insulin may help shift phosphorus intracellularly.

ADH, antidiuretic hormone; AIDS, acquired immunodeficiency syndrome; DDAVP, desmopressin acetate; GI, gastrointestinal; NSAID, nonsteroidal anti-inflammatory drug; SIADH, syndrome of inappropriate antidiuretic hormone; SSRI, selective serotonin reuptake inhibitor.

Hyponatremia may be associated with volume excess, such as in edematous disorders (eg, heart, kidney, or liver failure), or with volume deficit, such as when volume loss is exceeded by sodium loss (eg, in gastrointestinal fluid, diuretic overuse, or adrenal insufficiency). Low sodium with euvolemia is manifested as the syndrome of inappropriate antidiuretic hormone secretion (SIADH; see Chapter 44). Pseudohyponatremia may occur in association with hyperlipidemia and hypoproteinemia; the total-body sodium remains unchanged, but the actual sodium measurement is altered.

Management of hyponatremia is aimed at correcting the underlying cause (see Table 30-6). When the hyponatremia is associated with hypervolemia, diuretics may be beneficial. When the disorder is associated with euvolemia, such as in SIADH, water restriction may be useful. In conditions in which there is both sodium loss and water loss, administration of hypertonic saline solution, typically started in symptomatic patients, at slow rates may help improve this clinically significant hyponatremia.

Hypernatremia may occur as an isolated condition when there is a loss of free water, which raises the sodium level. Increased insensible loss of fluid, such as occurs in sweating, hyperventilation, or fever, is the most common cause of this type of hypernatremia. The fluid volume deficit associated with the hypernatremia depends almost entirely on the degree of insensible loss. Endocrine disorders, such as hyperaldosteronism or Cushing's disease, can result in hypernatremia and are associated with total-body water excess. Administration of hypertonic fluids, such as sodium bicarbonate, 3% saline solution, or albumin, may also cause hypernatremia.

Management of hypernatremia is primarily aimed at restoring fluid balance (see Table 30-6). Correcting the underlying cause of the increased sodium is also important.

Potassium

Potassium is the major intracellular ion. Potassium plays a key role in neuromuscular functioning, and high or low levels may result in alterations in the cardiac rhythm. Because of the narrow range of extracellular potassium balance, renal function is essential to regulation of potassium. In critically ill patients, disorders of potassium are common and have numerous causes (see Table 30-6).

Hypokalemia is most commonly caused by an absolute deficiency in potassium. Losses of potassium occur through the kidneys, gastrointestinal tract, sweat, and intracellular shifting. Although relative deficiencies may occur, such as in metabolic alkalosis, they are rare compared with the absolute deficits. Management of hypokalemia involves replacing depleted potassium to restore potassium balance. It may be necessary to check the magnesium level in patients who do not respond to potassium replacement. Box 30-13 presents nursing considerations in potassium replacement.

Hyperkalemia is caused by reduced renal excretion, excessive administration of potassium replacements, transcellular shifts, and measurement error. Patients with chronic renal failure or acute kidney injury are at particular risk. Dialysis is typically used to manage hyperkalemia in patients with ESRD. Noncompliance with dialysis can certainly cause hyperkalemia and is a frequent reason for hospital admission. Potassium replacement therapy, although performed frequently in critical care settings, must be performed carefully; particular attention should be paid to cardiac signs and laboratory reassessments. Acidosis of any cause can potentiate hyperkalemia; therefore, patients who are acidotic must be carefully monitored for potassium shifts. In this situation, the primary goal of management is resolution of the acidosis. Clinically significant hyperkalemia must be resolved using the measures described in Table 30-6. Temporizing measures for hyperkalemia center on stabilization of cell membrane (calcium) and shifting potassium from the extracellular to the intracellular spaces (bicarbonate, insulin). These measures resolve the potassium imbalance temporarily and give clinicians time to address the underlying problem. If it is anticipated that correction of this problem will take some time, the excretion of potassium is facilitated by administering sodium polystyrene, dialysis, and diuretics. Drawing laboratory samples can cause hemolysis of cells, which causes liberation of the abundant intracellular potassium. Evaluating trends and assessing the overall clinical picture prevent unnecessary treatment and therefore prevent hypokalemia.

Calcium

Almost all of the calcium in the body is contained in the bone, and the remaining 1% is either bound to albumin (50% plasma calcium) or in an ionized form. The primary function of calcium is promotion of the neuromuscular impulse. Several clotting factors also depend on calcium.

Hypocalcemia has numerous causes (see Table 30-6). Most hypocalcemia is a relative deficiency; causes include intracellular shifting, decreased circulating protein, and binding with fatty acids (pancreatitis). The relative hypocalcemia that occurs with a massive transfusion of blood is common in the critical care setting. The blood is mixed with citrate to prevent coagulation; when the blood is infused, the citrate binds to calcium, causing a relative calcium deficiency. Citrate used for anticoagulation in CRRT also results in hypocalcemia. Other causes of hypocalcemia include increased renal excretion (loop diuretics) or decreased absorption (malabsorption syndromes).

Calcium is transported in its ionized form, provides some of the structural components in bone, and is also bound to albumin. A low albumin level can therefore be one cause of a low calcium level. The calcium level should be corrected for the low albumin before considering calcium replacement. Replacement of calcium is required to prevent complications of bleeding and decreased impulse transmission. For a review of nursing considerations in calcium replacement, see Box 30-13.

Hypercalcemia, which is less common in the critical care setting, is most often caused by malignancy. Treatment is supportive and involves administration of diuretics and IV fluids, sometimes simultaneously.

Magnesium

About two thirds of the magnesium in the body is in the skeletal system, and the remaining one third is in the intracellular space. About 1% circulates in the extracellular space. Magnesium is a catalyst for hundreds of enzymatic reactions and plays a role in neurotransmission and cardiac contraction. Magnesium is primarily excreted by the kidneys.

Hypomagnesemia is caused by loss of magnesium through the gastrointestinal tract or (less commonly) the kidneys. Alcoholism is a significant cause. The etiological mechanism

BOX 30-13 NURSING INTERVENTIONS

For Intravenous Electrolyte Replacement

Potassium

Dilution

- Do not administer undiluted potassium directly IV.
- Keep all vials of undiluted potassium away from patient care area.
- Dilution of potassium depends on the amount of fluid the patient can tolerate. Highly concentrated potassium solutions can cause irritation, pain, and sclerosing of vein.
- Typical concentrations of potassium are 10 to 40 mEq/100 mL. Premixed bags are available.

Peripheral IV Administration

- In collaboration with prescribing provider, consider the addition of small volume of lidocaine to minimize pain.
- Administer in central vein if available.
- For mild to moderate hypokalemia, rates of 10 to 20 mEq/h are recommended.
- Rates exceeding 40 mEq/h are not recommended.
- Use infusion pump to administer replacement.

Monitoring

- Monitor urinary output, blood urea nitrogen, and creatinine in patients receiving potassium replacement. Patients with impaired renal function or oliguric renal failure may experience transient hyperkalemia. Consider smaller replacement dosages and periodic reevaluation.
- When rate of administration exceeds 10 mEq/h, monitoring of cardiac rhythm is recommended.
- Assess magnesium level because correction of potassium may be refractory to potassium replacement with concurrent hypomagnesemia.

Calcium

Dilution

- Calcium can be delivered as calcium gluconate (4.5 mEq of elemental Ca^{2+}) or calcium chloride (13.5 mEq of elemental Ca^{2+}).
- Calcium can be irritating to veins. If peripheral administration is required, calcium gluconate is recommended because damage can occur to surrounding soft tissues.

Administration

- Administer by slow IV push through central vein or administer by mixing with compatible IV fluids.
- Administer slowly (over 1 to 2 hours) for patients receiving digoxin.

Magnesium

Administration

- Administer with caution to patients with renal failure because magnesium is primarily excreted by the kidneys.
- During emergencies, such as torsades de pointes, magnesium may be injected directly.
- In mild to moderate hypomagnesemia, a rate of infusion of 1 to 2 g over 1 hour is advisable.

Monitoring

- Monitor for hypotension or flushing during administration.
- Monitor deep tendon reflexes periodically during administration.

Phosphorus

- Phosphorus IV replacement is available as sodium or potassium phosphate. Note that phosphorus is dosed in millimoles, whereas sodium and potassium are dosed in milliequivalents.
- Administer sodium phosphate for patients with renal failure.
- Do not administer with calcium.
- Administer over several hours, typically 15 to 30 mmol phosphorus over 4 to 6 hours.

is not completely understood, but it is thought that decreased dietary intake due to malnutrition, decreased absorption, and increased gastrointestinal losses (due to periodic emesis) all play a role. Several drugs may also cause hypomagnesemia, including loop diuretics, aminoglycosides, amphotericin B, *cis*-platinum, cyclosporine, and citrate. For a review of the causes of hypomagnesemia, see Table 30-6.

Magnesium is available in a variety of preparations, including 50%, 20%, or 10% solutions. It is important to pay particular attention to how the replacement preparation is ordered; the replacement solution should be "dosed" in grams instead of milliliters. For a review of nursing considerations in magnesium replacement, see Box 30-13.

Phosphorus

Phosphorus is the major intracellular anion. The source of adenosine triphosphate (ATP), phosphorus is implicated in many life-sustaining processes, such as muscle contraction, neuromuscular impulse conduction, and the regulation of several intracellular and extracellular electrolyte balances.

Hypophosphatemia may be caused by several metabolic disorders, including refeeding syndrome and alcoholism, intracellular shifting due to respiratory alkalosis, binding by medications, such as phosphate-binding magnesium-containing antacids, and excessive excretion of phosphate, such as in DKA (see Table 30-6). Refeeding syndrome occurs when the patient is fed, either enterally or parenterally, after some time of starvation. During starvation, protein catabolism occurs, depleting all of the intracellular phosphorus. When a large glucose load is administered, as occurs with refeeding, it is thought that the insulin response shifts the phosphorus intracellularly.

Management of hypophosphatemia may be problematic, particularly for patients on a mechanical ventilator. Contraction of all muscles, including the diaphragm, depends on ATP. Replacement of phosphorus is indicated in critically ill patients to achieve adequate pulmonary function. Once the critical illness abates, the hypophosphatemia typically resolves as well. However, replacement with either sodium or potassium phosphate is indicated in the meantime. For a review of nursing considerations in phosphorus replacement, see Box 30-13.

Hyperphosphatemia is commonly associated with renal failure due to reduced elimination of phosphorus. Because of the inverse relationship with calcium, the high phosphorus may also be associated with hypocalcemia. Administration of phosphate binders and calcium supplementation are indicated.

▲ Clinical Applicability Challenges

CASE STUDY

Mrs. B. is a 68-year-old female with a history of diabetes and hypertension. Two years ago, she was diagnosed with an abdominal aortic aneurysm (AAA). Four days ago, she was admitted to the cardiovascular intensive care unit (CVICU) postoperatively for repair of her AAA. Immediate postoperative data are as follows: temperature, 97.3°F (36.3°C); pulse rate, 120 beats/min; blood pressure, 87/50 mm Hg; respiratory rate, 18 breaths/min; and oxygen saturation by pulse oximetry (SpO_2), 98% (on 50% fraction of inspired oxygen [FiO_2]). Laboratory test on admission included Na^+, 130 mEq/L; K^+, 3.5 mEq/L; Cl^-, 100 mEq/L; CO_2, 18 mmol/L; blood urea nitrogen, 28 mg/dL; creatinine, 1.4 mg/dL; and glucose, 162 mg/dL. This morning, the data included temperature, 101.5°F (38.5°C); pulse rate, 80 beats/min; blood pressure, 127/85 mm Hg; respiratory rate, 16 breaths/min; oxygen saturation by pulse oximetry (SpO_2), 96% (on 2 L/min by nasal cannula); Na^+, 132 mEq/L; K^+, 4.5 mEq/L; Cl^-, 105 mEq/L; CO_2, 17 mmol/L; blood urea nitrogen, 45 mg/dL; creatinine, 2.1 mg/dL; and glucose, 122 mg/dL.

1. Explain the pathophysiological basis of Mrs. B.'s blood urea nitrogen and creatinine values before and after surgery.
2. What are Mrs. B.'s risk factors for developing renal insufficiency?
3. What additional diagnostic tests might you anticipate to be ordered to evaluate Mrs. B.'s renal function?
4. What are some of the nursing care priorities for Mrs. B.? What treatments could you anticipate for her?

References

1. CDC. Recommendations and Reports: Guidelines for the Prevention of Intravascular Catheter-Related Infections, August 9, 2002
2. Dirkes S, Hodge K: Continuous renal replacement therapy in the adult intensive care unit: History and current trends. Crit Care Nurse 27(2): 61–81, 2007
3. Morgera S, Schnieder M, Slowinski T, et al: A safe citrate anticoagulation protocol with variable treatment efficacy and excellent control of the acid-base status. Crit Care Med 37(6):2018–2024, 2009
4. Link A, Girndt M, Selejan S, et al: Argatroban for anticoagulation in continuous renal replacement therapy. Crit Care Med 37(1):105–110, 2009
5. Holmes CL, Walley KR: Bad medicine: Low-dose dopamine in the ICU. Chest 123(4):1266–1275, 2003
6. DuBose JJ, Kobayashi L, Lozornio A, et al: Clincal experience using 5% hypertonic saline as a safe alternative fluid for use in trauma. J Trauma 68(5):1172–1177, 2010.
7. Singh A, Carlin BW, Shade D, et al: The use of hypertonic saline for fluid resuscitation in sepsis: A review. Crit Care Nurs Q 32(1):10–13, 2009.

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

31 Renal Failure

Dorene M. Holcombe and Nancy Kern Feeley

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Explain the causes of acute kidney injury (AKI).
2. Describe urine production during the nonoliguric, oliguric, and diuretic phases of acute tubular necrosis.
3. Differentiate between the three types of AKI based on history and physical examination, laboratory values, and diagnostic tests.
4. Discuss the major causes and the clinical stages of chronic kidney disease (CKD).
5. Explain factors that can contribute to the progression of CKD.
6. Discuss the clinical manifestations and management of renal failure.

Previously, acute kidney injury (AKI) was known as acute renal failure (ARF). More than 35 different definitions for ARF were contained in the medical literature. This lack of uniformity resulted in varying reported incidences and conflicting reports regarding morbidity and mortality. This had an adverse effect on research studies.[1] Consequently, in 2004, a group of expert intensivists and nephrologists formed the Acute Dialysis Quality Initiative (ADQI) to develop a consensus definition for ARF/AKI. The resultant consensus definition (formulated by ADQI) is known as the Risk-Injury-Failure-Loss-and End-stage (RIFLE) classification.

▲ Acute Kidney Injury

Acute kidney injury (AKI) occurs in up to 15% to 18% of hospitalized patients and in as many as 66% of the patients treated in intensive care units (ICUs).[1–3] Furthermore, due to increased recognition, the incidence of AKI has dramatically increased over the last decade.[4] Regardless of the underlying etiology, AKI is associated with increased in-hospital morbidity, mortality, and costs along with increased long-term mortality and morbidity.[1,2,4] Patients with AKI who are treated with renal replacement therapy (RRT) have a mortality rate between 50% and 60%; this rate has not changed in the past several decades despite advances in technology and dialysis.[1,3,5] However, the demographics of patients have changed, with patients in general being older and having an increased number of comorbid conditions.[1,6] On an optimistic note, more than 80% of survivors of severe AKI (those requiring RRT) are independent of dialysis by discharge.[1]

AKI is a common clinical syndrome in which there is a sudden onset of reduced renal function that can result in derangements in fluid and electrolyte balance, acid–base homeostasis, calcium and phosphate metabolism, blood pressure (BP) regulation, and erythropoiesis. The hallmark of AKI is a decreased glomerular filtration rate (GFR), reflected by an accumulation of blood urea nitrogen (BUN) and serum creatinine—a condition termed azotemia. Serum creatinine is the better marker because increases in serum creatinine are relatively unaffected by metabolic factors. As defined by the RIFLE classification[7,8] (Table 31-1), there are three increasing grades of severity of AKI (risk, injury, and failure) based on a relative increase in serum creatinine or a period of decreased urine output. Also, two outcome criteria (loss and end-stage kidney disease) are defined by duration of loss of kidney function, 4 weeks and 3 months, respectively.[7]

In 2007, the RIFLE criteria were modified by the Acute Kidney Injury Network (AKIN), which included the ADQI group as well as other representatives from nephrology and intensive care societies. The AKIN-proposed diagnostic criteria for AKI is an abrupt (within 48 hours) increase in the serum creatinine of 0.3 mg/dL or more from baseline, a percentage increase in the serum creatinine concentration of 50% or more, or a urine output of less than 0.5 mL/kg/h for more than 6 hours.[9] Since its creation, the RIFLE criteria has been widely adopted in research and has been validated in clinical settings for predicting patient outcomes.[7,10] In the future, it is likely that functional markers of renal failure (urine output and serum creatinine) will be replaced or augmented by biological injury markers, analogous to how troponin is now used to help diagnose an acute myocardial infarction. Hopefully, such markers of kidney cellular injury

Table 31-1 Acute Kidney Injury Staging Criteria

Stage	Creatinine Criteria	Urine Output Criteria
RIFLE Criteria		
Risk	Creatinine increase of 1.5–2 times baseline value	<0.5 mL/kg/h × 6 h
Injury	Creatinine increase of 2–3 times baseline value	<0.5 mL/kg/h × 12 h
Failure	Creatinine increase of 3 or more times baseline value or a creatinine value >4 mg/dL with an acute increase of 0.5 mg/dL or more	<0.3 mL/kg/h × 24 h or anuria × 12 h
Loss	Persistent ARF for >4 wk	
End-stage kidney disease	Persistent ARF for >3 mo	
AKIN Criteria*		
1	Creatinine increase of 1/5–2 times baseline value or increases in creatinine of 0.3 or more mg/dL	<0.5 mL/kg/h × 6 h
2	Creatinine increase of 2–3 times baseline value	<0.5 mL/kg/h × 12 h
3	Creatinine increase of 3 or more times baseline value or a creatinine value >4 mg/dL with an acute increase of 0.5 mg/dL or more	

*Reduction in renal function must occur within 48 h.

will not only define AKI but will also offer the potential to diagnose the disorder before functional decline.

Urine output patterns in AKI can manifest as oliguria (<400 mL/d), nonoliguria (>400 mL/d), or anuria (<100 mL/d). Categorization of AKI as oliguric or nonoliguric is diagnostically significant because the oliguric form is associated with higher morbidity and mortality rates. This may be mediated in part by the more pronounced fluid retention in oliguric versus nonoliguric patients.[11] Anuria is rare and is most often seen in two conditions: shock and complete bilateral urinary tract obstruction. Any sudden and complete cessation of urinary flow in a patient with a Foley catheter should alert the nurse to inspect, flush, or change the urinary catheter.

Causes of Acute Kidney Injury

Many pathophysiological pathways may lead to the syndrome of AKI. To aid in establishing a diagnostic and management plan, AKI is organized into three general categories according to precipitating factors and the symptoms manifested (Box 31-1).

Prerenal Acute Kidney Injury

Prerenal AKI is characterized by any physiological event that results in renal hypoperfusion. Most commonly, precipitating events include hypovolemia and cardiovascular failure; however, any other event that leads to an acute decrease in "effective renal perfusion" can fall into this category (see Box 31-1). For example, in sepsis, a systemic inflammatory response triggers a cascade of events that results in a vasodilated hypotensive state despite no net loss in body fluids.

Intrarenal Acute Kidney Injury

The intrarenal category of AKI is characterized by actual damage to the renal parenchyma and has many possible associated causes. One way to categorize these causes is by anatomical compartment: glomerular, vascular, interstitial, and tubular. The glomerular etiologies, which result in acute glomerulonephritis, include immune complex–mediated causes (eg, as seen with poststreptococcal glomerulonephritis) and diseases that cause vasculitis, such as Wegener's granulomatosis and antiglomerular basement membrane disease. Interstitial causes include acute allergic interstitial nephritis, usually caused by pharmacological agents, and infectious causes such as pyelonephritis. Vascular etiologies include malignant hypertension as well as microangiopathic processes, such as atheroembolic disease or hemolytic–uremic syndrome (HUS) and thrombotic thrombocytopenic purpura (TTP). Finally, the tubules of the kidney can be primarily affected because of obstruction or acute tubular necrosis (ATN). Obstructive causes include multiple myeloma and acute urate nephropathy.

A common cause of intrarenal hospital-acquired AKI is ATN. ATN results from either a prolonged prerenal condition (ischemic ATN) or the effects of toxins on the tubules (toxic ATN). Examples of potential toxins to the tubules include pharmacological agents, such as aminoglycosides, amphotericin B, and chemotherapeutic agents; heavy

BOX 31-1 Precipitating Causes of Acute Kidney Injury

Prerenal
- Decreased intravascular volume
  - Dehydration
  - Hemorrhage
  - Hypovolemic shock
  - Hypovolemia (gastrointestinal losses, diuretics, diabetes insipidus)
  - Third spacing (burns, peritonitis)
- Cardiovascular failure
  - Heart failure
  - Myocardial infarction
  - Cardiogenic shock
  - Valvular heart disease
  - Renal artery stenosis or thrombosis
- Drugs
  - ACE inhibitors
  - NSAIDs—inhibit prostaglandin-mediated afferent arteriolar vasodilation
  - Calcineurin inhibitors (eg, tacrolimus, cyclosporine)—cause preglomerular vasoconstriction
- Decreased "effective renal perfusion"
  - Sepsis
  - Cirrhosis
  - Neurogenic shock

Intrarenal
- Acute glomerulonephritis
  - Immune complex–mediated (postinfectious, lupus nephritis, cryoglobulinemia, immunoglobulin A [IgA] nephropathy)
  - With vasculitis (Wegener's granulomatosis, antiglomerular basement membrane disease, polyarteritis nodosa)
- Vascular disease
  - Malignant hypertension
  - Microangiopathic hemolytic–uremic syndrome (HUS)
  - Thrombotic thrombocytopenic purpura (TTP)
  - Scleroderma
  - Eclampsia
  - Atheroembolic disease
  - Acute cortical necrosis
- Acute interstitial disease
  - Allergic interstitial nephritis
  - Acute pyelonephritis
- Tubular obstruction
  - Multiple myeloma
  - Acute urate nephropathy
  - Ethylene glycol or methanol toxicity
- Acute tubular necrosis (ATN)
  - Ischemia
  - Nephrotoxins (contrast dye, drugs, heme pigments)
  - Kidney transplant rejection

Postrenal
- Ureteral obstruction
  - Intrinsic (stones, transitional cell carcinoma of the ureter, blood clots, stricture)
  - Extrinsic (ovarian cancer; lymphoma; metastatic cancer of the prostate, cervix, or colon; retroperitoneal fibrosis)
- Bladder problems
  - Tumors
  - Blood clots
  - Neurogenic bladder (spinal cord injury, diabetes mellitus, ischemia, drugs)
  - Stones
- Urethral obstruction
  - Prostate cancer or benign prostatic hypertrophy
  - Stones
  - Stricture
  - Blood clots
  - Obstructed indwelling catheter

metals; organic solvents; heme pigments (eg, myoglobin and hemoglobin); and radiocontrast media (Box 31-2).

Postrenal Acute Kidney Injury

Postrenal AKI accounts for approximately 10% of hospital cases.[10] Any obstruction in the flow of urine from the collecting ducts in the kidney to the external urethral orifice can result in postrenal AKI. Postrenal obstruction can result from ureteral blockage (as with bilateral renal stones), urethral blockage (as from stricture and benign prostatic hypertrophy), or an extrinsic source, such as a retroperitoneal tumor or fibrosis. Another source of postrenal AKI is a dysfunctional bladder (eg, as might be caused by ganglionic blocking agents that interrupt autonomic supply to the urinary system). Elderly men and the young are populations particularly susceptible to postrenal AKI. Children are at risk secondary to congenital anomalies, and elderly men are at risk because of the high prevalence of benign or malignant prostatic hypertrophy.

Pathophysiology of Acute Kidney Injury

Prerenal Acute Kidney Injury

The pathophysiology of prerenal AKI is centered on the kidneys' response to inadequate perfusion. A decrease in renal perfusion results in the release of the enzyme renin from juxtaglomerular cells in the walls of the afferent arterioles. This activates the renin–angiotensin–aldosterone cascade, the end result being the production of angiotensin II and the release of aldosterone from the adrenal cortex. Angiotensin II causes profound systemic vasoconstriction, and aldosterone induces sodium and water retention. These effects help the body preserve circulatory volume to maintain adequate blood flow to essential organs such as the heart and brain. In the kidneys, angiotensin II also helps maintain the GFR by increasing efferent arteriolar resistance and by stimulating intrarenal vasodilator prostaglandins (which dilate the afferent arteriole), increasing hydrostatic pressure in the glomeruli.[12] In this way, the kidneys can preserve the GFR over a wide range of mean arterial pressures. However, when renal perfusion is severely compromised, the capacity for autoregulation is overwhelmed, and the GFR decreases.

Even with moderate hypovolemia or congestive heart failure, certain drugs, such as angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), and nonsteroidal anti-inflammatory drugs (NSAIDs), can overwhelm the kidney's ability to autoregulate. These drugs disrupt some of the autoregulatory mechanisms, such as prostaglandin-mediated afferent arterial vasodilation, in the case of NSAIDs, and increased efferent arteriolar resistance,

BOX 31-2 Common Causes of Acute Tubular Necrosis

Ischemic
Hemorrhagic hypotension
Severe volume depletion
Surgical aortic cross-clamping
Cardiac surgery
Defective cardiac output
Septic shock
Pancreatitis
Immunosuppression (cyclosporine, tacrolimus)
NSAIDs

Nephrotoxic
Drugs, including antimicrobials (aminoglycosides, amphotericin), cyclosporine, anesthetics, chemotherapeutic agents
Heavy metals (mercury, lead, cisplatinum, uranium, cadmium, bismuth, arsenic)
Radiological contrast agents
Heme/pigments (myoglobin, hemoglobin)
Organic solvents (carbon tetrachloride)
Fungicides and pesticides
Plant and animal substances (mushrooms, snake venom)

in the case of ACE inhibitors and ARBs. Predisposing factors for NSAID- and ACE inhibitor–induced prerenal failure are hypovolemia, baseline renal insufficiency, liver disease, heart failure, and diseases of the renal arteries.[12,13]

In prerenal AKI, once autoregulatory capacity is overwhelmed and the GFR decreases, changes in urinary composition and volume occur in a predictable pattern. When the GFR decreases, the amount of tubular fluid is reduced, and the fluid travels through the tubule more slowly. This results in increased sodium and water reabsorption. Because of the reduced renal circulation, the solutes reabsorbed from the tubular fluid are removed more slowly than normal from the interstitium of the renal medulla. This results in increased medullary tonicity, further augmenting water reabsorption from the distal tubular fluid. As a result of these events, the urinary volume is reduced to less than 400 mL/d (<17 mL/h), the urine specific gravity is increased, and the urine sodium concentration is low (usually <5 mEq/L; Fig. 31-1). Because of these characteristic changes associated with renal underperfusion, measurement of urinary volume, urinary sodium, and specific gravity is a simple method of determining the effect of management on renal perfusion.

An increase in systemic BP does not necessarily imply improvement in renal perfusion. This may be especially evident when drugs, such as norepinephrine, are used to correct the hypotension associated with states of volume depletion. These drugs may be associated with further reduction in renal blood flow as a consequence of constriction of the renal arteries. This is manifested by a further fall in urinary volume and rise in specific gravity.

In turn, if the hypoperfusion state is more appropriately and specifically treated by replacement of volume, improvement of cardiac output, correction of dysrhythmias, or a combination of these approaches, the improved renal perfusion is manifested as an increased urinary volume and urine sodium concentration and as a decreased specific gravity of

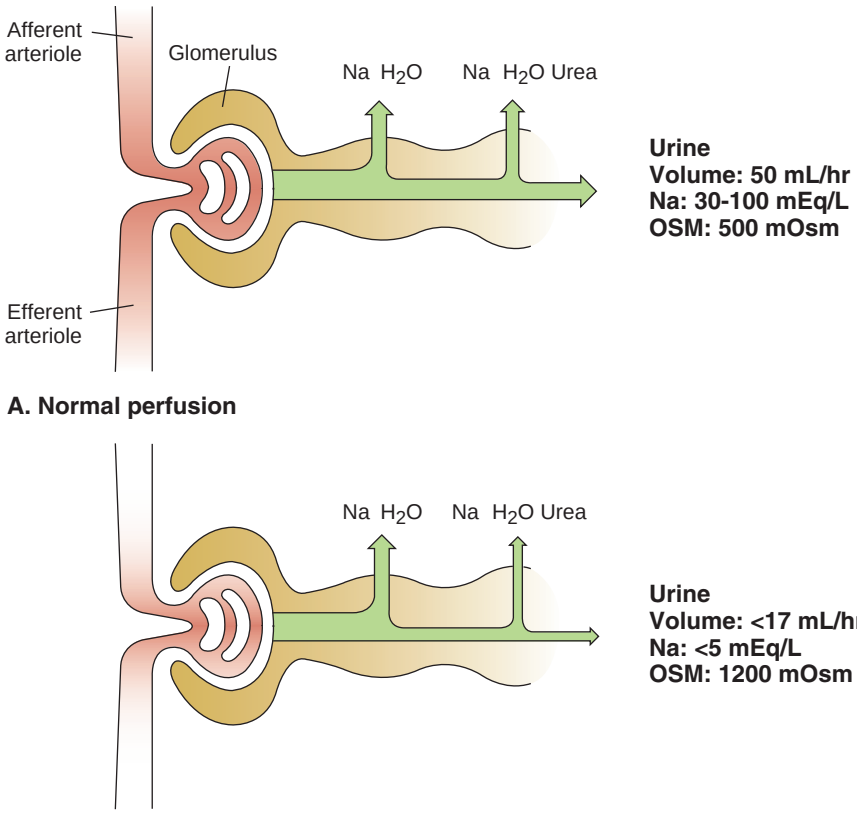


FIGURE 31-1 ▲ Normal perfusion (**A**) of the kidney compared with underperfusion (**B**), as seen in prerenal AKI. Underperfusion of the kidney results in decreased renal blood flow and glomerular filtration, an increase in the fraction of filtrate reabsorbed in the proximal tubule, and low urine flow with low sodium (Na) content and increased concentration. H_2O, water; OSM, osmolarity.

the urine. This ability to reverse prerenal AKI is the key to its diagnosis.

Intrarenal Acute Kidney Injury

Just as there are many causes of intrarenal AKI, there are also many pathophysiological pathways that lead to it (Fig. 31-2). Because ATN is the most common hospital-acquired form of intrarenal AKI, this discussion focuses on the pathophysiology of ATN, which is complex, but intense and ongoing research has increased understanding of the factors contributing to this condition. Ischemia and nephrotoxicity are two major underlying causes of ATN (Fig. 31-3).

Ischemic Acute Tubular Necrosis

Ischemic ATN results from prolonged hypoperfusion. Thus, prerenal AKI and ischemic ATN are actually a continuum, a fact that underscores the importance of prompt recognition and treatment of the prerenal state. When renal hypoperfusion persists for a sufficient time (the exact duration of which is unpredictable and varies with clinical circumstances), renal tubular epithelial cells become hypoxic and sustain damage to the point that restoration of renal perfusion no longer effects an improvement in glomerular filtration. Ischemia results in decreased adenosine triphosphate production in renal cell mitochondria, which robs the cells of a needed energy supply. Part of this energy is used to keep the proper concentration of electrolytes in the cell through electrolyte exchange channels. Some of the cellular electrolyte disturbances from ischemia are decreased intracellular potassium, magnesium, and phosphate and increased intracellular sodium, chloride, and calcium. Increased intracellular calcium specifically has been shown to predispose the cells to injury and dysfunction.[12,14]

During reperfusion, cellular insults also occur from the formation of oxygen free radicals. Eventually, these cellular insults cause the tubular cells to swell and become necrotic. The necrotic cells then slough off and may obstruct the tubular lumen. These sloughed cells also allow back leak of tubular fluid because of altered function of their basement membrane, which contributes to the decreased GFR seen in this disorder.

A final contributor to the pathophysiology of ischemic ATN is profound renal vasoconstriction, which reduces renal blood flow by as much as 50%.[12] These hemodynamic disturbances further compromise renal oxygen delivery and add to the ischemic damage. Vasoconstrictors involved include norepinephrine from sympathetic nervous system activation, angiotensin II, thromboxane A_2, adenosine, leukotrienes C4 and D4, prostaglandin H_2, and endothelin, a powerful vasoconstrictor released by damaged vascular endothelial cells of the kidney.[12]

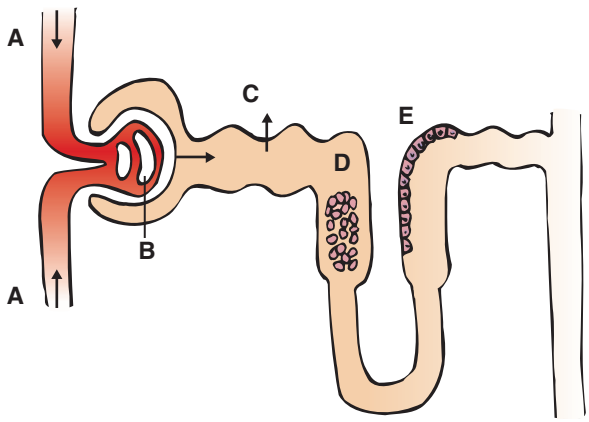


FIGURE 31-2 ▲ Potential mechanisms of intrarenal AKI include decreased filtration pressure because of constriction in the renal arterioles (**A**), decreased glomerular capillary permeability (**B**), increased permeability of the proximal tubules with back leak of filtrate (**C**), obstruction of urine flow by necrotic tubular cells (**D**), and increased sodium delivery to the macula densa (**E**), which causes an increase in renin–angiotensin production and vasoconstriction at the glomerular level.

Toxic Acute Tubular Necrosis

The pathophysiology of toxic ATN begins with a concentration of a nephrotoxin in the renal tubular cells, which causes necrosis. These necrotic cells then slough off into the tubular lumen, possibly causing obstruction and impairing glomerular filtration in a manner similar to that of ischemic ATN. However, there are significant differences between toxic ATN and ischemic ATN. In toxic ATN, the basement membrane of the renal cells usually remains intact, and the injured necrotic areas are more localized. In addition, nonoliguria occurs more often with toxic ATN, and the healing process is often more rapid.

Although the potential nephrotoxins in toxic ATN are many (see Box 31-2), aminoglycoside antibiotics and radiocontrast dye deserve special mention because of the frequency with which they are seen as causes of toxic ATN in hospitalized patients. Nephrotoxicity occurs in 10% to 20% of patients treated with aminoglycosides for 10 days or more.[13,14] The onset of AKI secondary to aminoglycosides is usually delayed, often beginning 5 to 10 days after the onset of therapy. The toxicity of these agents is dose dependent, and because these agents are primarily eliminated by the kidneys, dosage must be adjusted in patients with preexisting renal impairment. To ensure that the correct therapeutic range is being achieved, blood is drawn frequently for peak and trough level analysis. Offending agents (listed in decreasing order of the severity with which they produce dose-dependent proximal tubule damage) are neomycin, gentamicin, amikacin, tobramycin, and streptomycin. Several studies have suggested that a single daily dose of an aminoglycoside may result in less nephrotoxicity than giving the same total amount of medication in three daily doses.[13,15] Besides dosage, increased risk factors for aminoglycoside toxicity are volume depletion, advanced age, concurrent use of other nephrotoxic agents, and hepatic dysfunction.[13,16]

Contrast-induced nephropathy (CIN), the sudden decline of renal function following intravascular injection of contrast media, accounts for a significant number of hospital-acquired cases of AKI. It usually begins within 24 to 48 hours of intravenous (IV) radiocontrast administration and peaks within 5 to 7 days. Typically, CIN is nonoliguric, transient, and reversible; however, in high-risk patients, dialysis may be required on an intermittent or permanent basis.[17,18] Patients at greatest risk for CIN are those with diabetes, especially those with diabetes and underlying renal impairment. In

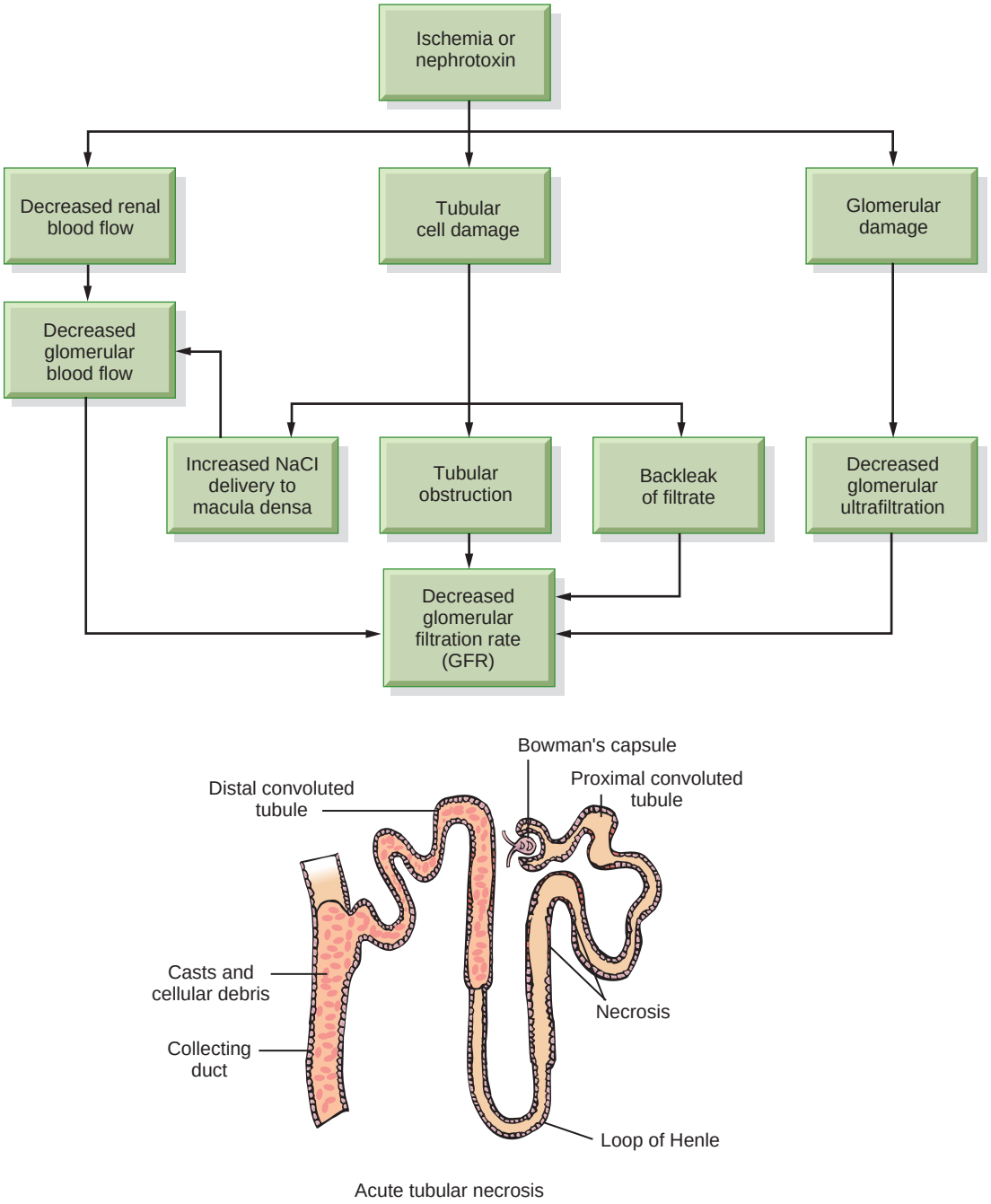


FIGURE 31-3 ▲ Ischemic ATN results from prolonged hypoperfusion. A sequence of pathophysiological processes results in the sloughing off of necrotic cells that block the tubular lumen. Toxic ATN occurs when a nephrotoxin becomes concentrated in the renal tubular cells and causes necrosis. The necrotic cells slough off and obstruct the tubular lumen, similar to ischemic ATN. In toxic ATN, the basement membrane of the renal cells usually remains intact, and the necrotic areas are more localized.

these patients, the incidence of CIN may be as high as 50%.[17] Other patients at risk are elderly patients; those with intravascular volume depletion, congestive heart failure, or anemia; and those who receive a large contrast load.[17,18]

The only proven way to reduce the risk for CIN is by aggressive volume expansion with isotonic crystalloids (normal saline solution) before and after contrast agent administration.[17] Because CIN is believed to involve the production of oxygen free radicals, it is postulated that alkalinization of the urine with sodium bicarbonate may confer greater protection than IV fluids alone. However, two recent meta-analyses of multiple randomized trials have demonstrated no clear evidence of benefit for hydration with sodium bicarbonate versus hydration with saline solution to prevent CIN.[19,20]

Other interventions to reduce the incidence of CIN include using the minimal necessary dose of contrast media, using low or iso-osmolar nonionic contrast media instead of ionic hyperosmolar agents, stopping the intake of nephrotoxic drugs 24 hours before contrast media

injection, and avoiding short intervals between contrast procedures.[17,18] *N*-Acetylcysteine (NAC), an antioxidant and a potent vasodilator, is part of the protocol in many hospitals to prevent CIN based on clinical trials demonstrating its renoprotective effects in patients receiving IV contrast media. However, NAC has been the subject of many meta-analyses, and, overall, there has been insufficient evidence to support the universal use of NAC to prevent CIN.[17,18] Studies have shown that other pharmacological interventions, such as calcium channel blockers, dopamine, mannitol, and atrial natriuretic peptide, do not consistently reduce the incidence of CIN and may even be harmful.

Of course, avoiding any use of iodinated contrast media in high-risk patients is the best prevention, and alternative studies, such as ultrasonography, computerized tomography (CT) scanning without contrast, and contrast-enhanced magnetic resonance imaging (MRI), should be considered. The MRI contrast agents in use are mostly chelates of gadolinium and have been found to be less nephrotoxic than iodinated radiocontrast, particularly when used in small doses.[21] This has led to the use of gadolinium-based contrast agents (GBCAs) as alternatives to iodinated contrast agents for digital subtraction angiography or interventional procedures, especially in patients with iodinated contrast allergies. However, one important caveat regarding the use of GBCA in patients with acute or severe chronic kidney disease (CKD; GFR < 30 mL/min) is the rare but serious risk for developing nephrogenic systemic fibrosis (NSF). NSF, a fibrosing disorder seen only in patients with kidney disease, is characterized by thickening and hardening of the skin overlying the extremities and trunk. Occasionally, fibrosis of deeper structures (such as muscles, the lungs, and the heart) occurs as well. Because the condition can be devastating to a patient (resulting in significant loss of mobility and even death), gadolinium agents should be avoided in patients with a GFR less than 30 mL/min.[21–23] If it is determined that a study using a GBCA must be performed, the Food and Drug Administration recommends that the lowest possible dose of GBCA needed to conduct the study be used and that there be allotted a sufficient period of time for elimination of the agent from the body prior to readministration. In addition, for patients receiving hemodialysis, prompt hemodialysis following GBCA administration is recommended to enhance elimination of the GBCA.[23]

Postrenal Acute Kidney Injury

Obstruction can occur at any point in the urinary tract. When urine cannot get around the obstruction, resulting congestion causes retrograde pressure through the collecting system and nephrons. This slows the rate of tubular fluid flow and lowers the GFR. As a result, the reabsorption of sodium, water, and urea is increased, leading to a lowered urine sodium concentration and increased urine osmolality and BUN. Serum creatinine levels also increase. With prolonged pressure from urinary obstruction, the entire collecting system dilates, compressing and damaging nephrons. This results in dysfunction of the concentrating and diluting mechanism, and the urine osmolality and urine sodium concentration become similar to that of plasma. This circumstance can be avoided by prompt removal of the obstruction.

Because a single well-functioning kidney is adequate to maintain homeostasis, the development of AKI from obstruction requires blockage of both kidneys (ie, urethral or bladder neck obstruction or bilateral ureteral obstruction) or unilateral ureteral obstruction in patients with a single kidney. After relief of the obstruction, there is often a profound diuresis that may be as great as 5 to 8 L/d.[14] If electrolytes and water are not replenished as needed, this diuresis can lead to hemodynamic compromise, dysrhythmias, and ATN.

Clinical Course of Acute Tubular Necrosis

The course of ATN can be divided into four clinical phases: onset phase, oliguric or nonoliguric phase, diuretic phase, and recovery phase.

Onset Phase

The onset (initiating) phase begins with an initial insult and lasts until cell injury occurs. During this phase, injury is evolving, and the health care team members need to attempt to prevent disease progression. The onset phase lasts from hours to days, depending on the cause, and is heralded by an increase in serum creatinine. The major goal during this phase is to determine the cause of the ATN and initiate treatment to prevent irreversible tubular damage.

Oliguric or Nonoliguric Phase

The second phase of ATN is characterized as either oliguric or nonoliguric. Patients who present with oliguric ATN are less likely to recover renal function and have a higher associated mortality rate.[14] This presentation of ATN is most commonly the result of ischemic insult. Oliguric ATN is marked by fluid overload, azotemia, electrolyte abnormalities (ie, hyperkalemia, hyperphosphatemia, and hypocalcemia), metabolic acidosis, and symptoms of uremia. The main goal during this period is to support renal function and keep the patient alive until renal injury heals. Complications during this period are from hyperkalemia, hypoxemia, gastrointestinal bleeding, and infection. Oliguric ATN lasts approximately 7 to 14 days, although it may persist for weeks depending on the extent of renal injury.[14]

The nonoliguric presentation of ATN is most commonly associated with toxic injury (eg, from aminoglycoside antibiotics). It is characterized by a renal concentrating defect rather than the impaired urinary flow seen in oliguric ATN. Urine volume in nonoliguric ATN ranges from normal to as much as 2 L/h.[14] Because of the higher volume of urine output, fluid complications are minimized. However, because potassium excretion parallels that seen in oliguria, hyperkalemia remains a major risk. The duration of nonoliguric ATN is typically short, lasting an average of 5 to 8 days.

Diuretic Phase

The diuretic phase lasts 1 to 2 weeks and is characterized by a gradual increase in urine output as renal function starts to resume. The degree of diuresis, which can exceed

10 L/d, is primarily determined by the state of hydration at the time the patient enters this stage. Thus, patients who are receiving hemodialysis or who are nonoliguric tend to diurese less. The diuresis is thought to result from the osmotic pull of retained substances (ie, urea and sodium), which act as osmotic agents. Although the urine output may be normal or elevated, renal concentrating ability is still impaired. This puts patients at risk for fluid volume deficits and electrolyte abnormalities, such as hyponatremia and hypokalemia. Primary goals during this stage are maintenance of hydration, prevention of electrolyte depletion, and continued support of renal function.

Recovery Phase

The recovery phase of ATN lasts from several months to a year. It is the time it takes for renal function to return to normal or near-normal levels. If significant renal cell damage has occurred, especially to the basement membrane (which cannot regenerate), residual renal impairment may result. Major goals of the health care team in this phase revolve around patient education. To foster and maintain the return of renal function, it is critical that patients and family members understand what precipitated the AKI episode as well as what follow-up care and preventive measures are necessary to prevent a recurrence.

Diagnosis of Acute Kidney Injury

Diagnosis of AKI begins with a determination of whether the AKI is prerenal, intrarenal, or postrenal. The assessment tools used to make this determination include the history and physical examination, laboratory tests, and diagnostic studies. Special considerations for assessing renal function in older patients are given in Box 31-3.[24]

History and Physical Examination

Essential to any assessment is the health history and physical examination. By taking a detailed history, clues to the categorization and exact cause of the AKI can be obtained. Important indications in the history that suggest prerenal AKI include any event or condition that may have contributed to decreased renal perfusion (eg, acute myocardial infarction, cardiovascular surgery, cardiac arrest, high fever, any shock state, and the use of certain drugs, such as NSAIDs). Also, a history of atherosclerotic disease may be a clue to renal artery stenosis, another precipitant of prerenal AKI. Clues to an intrarenal cause provided by the history include any prolonged prerenal event or condition as well as exposure to nephrotoxins, especially aminoglycoside antibiotics and radiocontrast media. It is also important to collect information regarding systemic diseases such as lupus or vasculitis, recent streptococcal infections, and causes for heme pigment toxicity, such as rhabdomyolysis (eg, a history of trauma or a patient found unconscious for an unknown amount of time). In addition, a history of cardiac catheterization, anticoagulation, and thrombolytic therapy increases the possibility of atheroembolic intrarenal diseases. Findings that may point to postrenal AKI include any history of abdominal tumors or calculi, and especially a history of benign prostatic hypertrophy in elderly men. A family history of urolithiasis or benign prostatic hypertrophy may be contributory.

The physical examination, particularly fluid status, is critical to the diagnosis of AKI. In prerenal AKI, a state of decreased renal perfusion related to dehydration or hypovolemia is heralded by poor skin turgor, dry mucous membranes, weight loss, and reduced jugular venous distention. In contrast, when decreased perfusion is related to vasodilation, third spacing, cardiovascular disease (eg, heart failure), liver disease, or a combination of these factors, findings of increased extracellular fluid may be manifested. These findings include edema, ascites, and weight gain. For critical care patients, hemodynamic monitoring values help determine intravascular fluid status as well as cardiac functioning. Surveillance values include central venous pressure (CVP), pulmonary artery occlusion pressure (PAOP), and cardiac output (or cardiac index). By correlating physical examination findings with the history, hemodynamic values, and

BOX 31-3 CONSIDERATIONS FOR THE OLDER PATIENT

Physiological Changes Affecting the Renal System

As the body ages, physiological systemic and kidney-specific changes occur that are important to take into consideration when addressing the kidney.

- Vascular changes: At 30 years of age, arteriosclerosis starts to develop, including in the renal arteries; this can result in significant damage.
- Musculoskeletal changes: In elderly people, there is a decreased muscle mass and body weight. These changes must be kept in mind when assessing renal function because of the possibility of a consequent decreased baseline serum creatinine value. A minimum rise in serum creatinine value in elderly patients, which may be within normal limits for a young adult, may actually signify major renal impairment.
- Kidney-specific changes: With aging, there is a decrease in the total number of functioning glomeruli, a decrease in renal blood flow, and a decrease in GFR of about 0.75 mL/min/1.73 m^2 per year after 30 years of age.[24]

In view of these systemic and kidney-specific changes, an accurate assessment of GFR using a 24-hour urine study or an isotopic study is essential. The Cockroft-Gault formula or the Modification of Diet in Renal Disease (MDRD) formulas below, which take into account gender and age, can also be used. It is important to realize that these formulas are not extensively validated in patients older than 70 years. After true GFR is realized, therapy (eg, drug dosages) can be guided more safely.

Cockcroft-Gault Formula for Creatinine Clearance (mL/min)

Men = (140 – age) × weight in kg/72 × serum creatinine

Women = 0.85 × creatinine clearance for men

MDRD Formula for GFR (adults; mL/min)

175 × Serum creatinine concentration$^{-1.154}$

× Age$^{-0.203}$

× 0.742 (if female)

× 1.210 (if black)

Websites are available to aid in the calculation of the GFR through these formulas. These include http://www.kidney.org and http://www.nephron.com.

laboratory tests, potential prerenal etiologies can be narrowed down.

Although no specific physical examination finding prompts consideration of intrarenal AKI, many examination findings are helpful clues to potential causes of intrarenal AKI. For example, signs of a streptococcal throat infection, lupus (eg, a butterfly mask rash), or embolic phenomena (eg, discolored toes and livedo reticularis, a semipermanent bluish mottling of the skin in the extremities) may all suggest an intrarenal cause. Again, correlation with the history and laboratory studies helps narrow the list of potential causes. Findings on physical examination that may suggest a postrenal cause include a distended bladder, an abdominal mass, an enlarged or nodular prostate gland, and, most obviously, a kinked or obstructed Foley catheter.

Laboratory Studies

Laboratory assessment, critical to the diagnosis and categorization of AKI, includes both serum and urinary values. For a basic comparison of laboratory values in prerenal AKI, postrenal AKI, and ATN, see Table 31-2. In addition to helping differentiate between prerenal, intrarenal, and postrenal AKI, blood and urine tests are also helpful for diagnosing the underlying etiology of the AKI (Box 31-4).

URINARY VALUES. Obtaining a urine specimen for diagnostic evaluations is invaluable in establishing the diagnosis and determining the type of AKI. The urine specimen should be obtained before a diagnostic challenge dose of diuretics is administered because these agents may alter the urine's chemical composition. The urine sodium concentration, osmolality, and specific gravity are especially helpful in distinguishing between prerenal AKI and ATN because these values reflect the concentrating ability of the kidney. In prerenal failure, the hypoperfused kidney actively reabsorbs sodium and water in an attempt to increase circulatory volume. Consequently, the urine sodium level and the fractional excretion of sodium (FE_{Na}) are low (<20 mEq/L and <1%, respectively), whereas the urine osmolality and concentration of nonreabsorbable solutes are high. In contrast, in ATN in which parenchymal damage affects the kidney, the tubular cells can no longer effectively reabsorb sodium or concentrate the urine. As a result, the urine sodium concentration is often greater than 40 mEq/L, the FE_{Na} is greater than 1%, and the urine osmolality is close to that of plasma (isosthenuria). Unfortunately, there is a limit to the usefulness of these indices because of overlap in these values for prerenal AKI and ATN (ie, urine sodium concentration values in the 20- to 40-mEq/L range). Values at the extremes thus are most useful.

Although controversial, one test that may be helpful in distinguishing prerenal AKI from ATN in patients who have already been given diuretics is the fractional excretion of urea nitrogen (FE_{un}). Urea, like sodium, is reabsorbed in a prerenal hypoperfused kidney, but unlike sodium, its reabsorption is primarily dependent on passive forces and is not inhibited by loop and thiazide diuretic administration.[25] In prerenal AKI, the fractional excretion of urea nitrogen is less than 35%, whereas in ATN and normally, it is more than 50%.

The sediment in a urinalysis is also very helpful in diagnosing and distinguishing the types of AKI. In prerenal AKI, the urinary sediment is normal with only a few hyaline casts, whereas in ATN, coarse, muddy-brown granular casts and tubular epithelial cells are typically found. In postrenal AKI, the sediment is often normal but can be helpful in diagnosing kidney stones.

BLOOD UREA NITROGEN AND CREATININE LEVELS. Serum tests for blood urea nitrogen (BUN) and creatinine are essential not only for diagnosing AKI but also for helping to distinguish between prerenal AKI and ATN or postrenal AKI. In prerenal AKI, the BUN-to-creatinine ratio is increased from the normal ratio of 10:1 to more than 20:1. This finding is caused by a state of dehydration and by the fact that as the tubules become more permeable to sodium and water in prerenal AKI, urea is also passively reabsorbed. In ATN and postrenal AKI, when the concentrating ability of the kidneys is impaired, both the BUN and creatinine increase proportionally, maintaining the normal 10:1 ratio.

Diagnostic Studies

One important diagnostic test in the evaluation of AKI is renal ultrasonography. This test is especially useful in ruling out an obstruction and has the advantage of being

Table 31-2 Acute Kidney Injury: Comparison of Laboratory Findings in Prerenal Failure, Postrenal Failure, and Acute Tubular Necrosis

Value	Prerenal	Postrenal	Acute Tubular Necrosis
Urine volume	Oliguria	May alternate between anuria and polyuria	Anuria, oliguria, or nonoliguria
Urine osmolality	Increased (>500 mOsm/kg H_2O)	Varies, increased, or equal to serum	250–300 mOsm/kg H_2O
Urine specific gravity	Increased (>1.020)	Varies	Approximately 1.010
Urine sodium	<20 mEq/L	Varies	>40 mEq/L
Urine sediment	Normal, few casts	Normal, may be crystals	Granular casts, tubular epithelial cells
FE_{Na}	<1%	>1%	>1% (often >3%)
BUN:Cr	>20:1	10:1 to 15:1	10:1 to 15:1

FE_{Na}, fractional excretion of sodium; BUN:Cr, blood urea nitrogen/creatinine ratio.

BOX 31-4 Diagnostic Clues in Acute Kidney Injury

Urine
- Urate crystals: tumor lysis, especially lymphoma (urate nephropathy)
- Oxalate crystals: ethylene glycol nephrotoxicity, methoxyflurane nephrotoxicity
- Eosinophils: allergic interstitial nephritis, especially methicillin
- Positive peroxidase test without red blood cells: hemoglobinuria or myoglobinuria
- Pigmented casts: hemoglobinuria or myoglobinuria
- Massive proteinuria: acute interstitial nephritis, thiazide diuretics, hemorrhagic fevers (eg, Korean, Scandinavian)
- Abnormal urine protein electrophoresis: multiple myeloma
- Anuria: renal cortical necrosis, bilateral obstruction, renal vascular catastrophe

Plasma
- Marked hyperkalemia: rhabdomyolysis, tissue necrosis, hemolysis
- Marked hypocalcemia: rhabdomyolysis
- Hypercalcemia: hypercalcemic nephropathy
- Hyperuricemia: tumor lysis, rhabdomyolysis, toxin ingestion
- Marked acidosis: ethylene glycol, methyl alcohol
- Elevated creatine kinase or myoglobin levels: rhabdomyolysis
- Low complement levels: systemic lupus erythematosus (SLE), postinfectious glomerulonephritis, subacute bacterial endocarditis
- Abnormal serum protein electrophoresis: multiple myeloma
- Positive antibody/glomerular basement membrane ratio: Goodpasture's syndrome
- Positive antineutrophilic cytoplasmic antibody: small vessel vasculitis (Wegener's granulomatosis or polyarteritis nodosa)
- Positive antinuclear antibody or antibody to double-stranded DNA: SLE
- Positive antibodies to streptolysin O: poststreptococcal glomerulonephritis
- Elevated lactate dehydrogenase level, elevated serum bilirubin level, or decreased haptoglobin level: HUS or TTP

noninvasive. With a high-grade obstruction, dilation of the urinary collecting system is detectable on ultrasonography within 1 to 2 days of the onset of the obstruction. Ultrasonography may also reveal proximal renal calculi as a cause of postrenal obstruction. In addition, it can be used to estimate renal size, which is helpful in distinguishing between AKI and advanced CKD. Often in advanced CKD, the kidneys are small (<9 cm) and echogenic.

Other studies may be useful in diagnosing AKI. These include CT and MRI to evaluate for masses, vascular disorders, and filling defects in the collecting system as well as renal angiography to evaluate for renal artery stenosis. It is notable that the iodinated contrast media used in some studies are allergenic and nephrotoxic and that GBCAs can cause NSF in patients with severe kidney disease (GFR < 30 mL/min). If available, alternative technology, such as the use of carbon dioxide gas in digital subtraction angiography, should be considered for patients allergic to iodinated agents or with advanced kidney failure.[26] For any diagnostic test, the benefits of the study must be weighed against potential risks. Finally, renal biopsy may be helpful in patients thought to have intrarenal AKI that is not ATN, especially if significant proteinuria or unexplained hematuria is revealed on urinalysis. In addition to having diagnostic value, the results of a biopsy may help determine prognosis and therapy.

▲ Chronic Kidney Disease

Chronic kidney disease (CKD) is a slow, progressive, irreversible deterioration in renal function that results in the kidney's inability to eliminate waste products and maintain fluid and electrolyte balance. Ultimately, it leads to end-stage renal disease (ESRD) and the need for RRT or renal transplantation to sustain life.

Currently, there are more than 520,000 dialysis and renal transplant recipients in the United States, which is an 18% increase in prevalence since the year 2000. In 2007 alone, more than 110,000 patients were newly diagnosed with ESRD. Among ESRD patients, incidence rates are 24% higher in men than in women and are higher with increasing age. The incidence rates in the African American population are 3.7 times greater than in the white population. Hispanics and Native Americans also have higher incidence rates than whites, but the difference in rates is not as dramatic.[27] These differences in incidence rates are important to remember when considering patient risk factors and populations to which increased health education regarding prevention should be targeted.

Although the exact reason for the increase in ESRD is unclear, it is postulated that changes in the demographics of the population, differences in disease burden among racial groups, underrecognition and undertreatment of earlier stages of CKD, and underrecognition of the risk factors for CKD may partially explain this increase.[28] Increasing evidence shows that early detection and treatment of CKD may prevent or at least delay progression to ESRD.[29–31] Consequently, it is important that opportunities to prevent and treat CKD are not lost secondary to underdiagnosis, undertreatment, or both.

Definition and Classification

In an effort to address the growing public health problem of CKD, the Kidney Disease Outcome Quality Initiative (K/DOQI) of the National Kidney Foundation published clinical practice guidelines for CKD in 2002. The goals of the working group that developed these guidelines were as follows: to define CKD and classify its stages, to evaluate laboratory measurements for clinical assessment of kidney disease, to associate the level of kidney function with the complications of CKD, and to stratify risk for the loss of kidney function and the development of cardiovascular disease.[28]

The K/DOQI defines CKD as either kidney damage with or without decreased GFR for 3 or more months *or* a GFR of less than 60 mL/min/1.73 m^2 for greater than 3 months (Box 31-5). Markers of damage include abnormal findings in the blood or urine tests or imaging studies. Examples are proteinuria, abnormalities in the urine sediment, increased serum creatinine, and multiple renal cysts detected on ultrasound in a patient with a family history of polycystic kidney disease (see Spotlight on Genetics 31-1).

BOX 31-5 Definition of Chronic Kidney Disease

1. Kidney damage for greater than or equal to 3 months as defined by structural or functional abnormalities of the kidney, with or without decreased GFR, manifested by *either:*
   a. Pathological abnormalities; *or*
   b. Markers of kidney damage, including abnormalities in the composition of the blood and urine, or abnormalities in imaging tests.
2. GFR less than 60 mL/min/1.73 m^2, with or without kidney damage

From National Kidney Foundation: K/DOQI clinical practice guidelines for chronic kidney disease: Evaluation, classification, and stratification. Am J Kidney Dis 39(2 Suppl 1): S1–S266, 2002.

A GFR (considered to be the best overall measure of kidney function) of less than 60 mL/min/1.73 m^2 was chosen for two reasons: (1) it represents a loss of half or more of the adult level of normal kidney function, and (2) below this level, the prevalence of complications from CKD increases.

Because predictable complications and management issues are based on the level of kidney dysfunction, regardless of the specific underlying etiology of CKD, the K/DOQI working group also developed a classification system for CKD based on the measured GFR (Table 31-3).[32] This classification system provides a common language for practitioners and patients to improve communication, enhance education, and promote research. Most importantly, it also provides a framework for evaluation and development of a treatment plan for patients with various stages of CKD, as outlined below.[14]

- Stage 1 is characterized by the lack of a clear filtration deficit and is defined as normal or increased kidney function (GFR ≥ 90 mL/min/1.73 m^2) in association with evidence of kidney damage. This damage most often is represented by persistent albuminuria defined as two consecutive spot urine albumin-to-creatinine ratios (mg/g) of greater than 17 mg/g in men and greater than 25 mg/g in women.
- Stage 2 is a mild reduction in kidney function (GFR, 60 to 89 mL/min/1.73 m^2) that occurs in association with kidney damage.
- Stages 3 and 4 are defined as moderately decreased kidney function (GFR, 30 to 59 mL/min/1.73 m^2) and severely decreased kidney function (GFR, 15 to 29 mL/min/1.73 m^2), respectively. These degrees of reduced GFR are classified as CKD regardless of any additional evidence of kidney damage.
- Stage 5 CKD is defined as a GFR of less than 15 mL/min/1.73 m^2 or the need for dialysis therapy. The term ESRD, widely used in regulatory and administrative circles, continues to be used by K/DOQI to represent those patients receiving or eligible for RRT by dialysis or transplantation.

It is important to note (see Table 31-3) that stage 5, which basically correlates to patients who are classified as having ESRD, represents only the tip of the iceberg of the total number of people who have CKD. According to the Third National Health and Nutrition Examination Survey (NHANES III), conducted between 1988 and 1994, which examined the health and nutritional status of 15,625 participants age 20 years or older, the prevalence of adults in the United States with stage 1 to 4 CKD was 19.2 million (11%), and 8.3 million (4.7%) had stages 3 to 5 CKD, with a GFR of less than 60 mL/min/m^2.[33] This is when most of the complications of CKD begin or have already occurred. The most recent NHANES data collected from 1999 to 2004 reveal increasing prevalence estimates for all stages of CKD, with an overall prevalence of 16.9%.[32] Compounding these grave statistics is the fact that CKD is associated with an increased risk of mortality, particularly from cardiovascular disease.

SPOTLIGHT ON GENETICS 31-1

POLYCYSTIC KIDNEY DISEASE

- Polycystic kidney disease is one of the most common disorders caused by mutations in a single gene. It affects about 500,000 people in the United States. The autosomal dominant form of the disease is much more common than the autosomal recessive form. Autosomal dominant polycystic kidney disease affects 1 in 500 to 1,000 people, while the autosomal recessive type occurs in an estimated 1 in 20,000 to 40,000 people. Clusters of fluid-filled sacs, called cysts, develop in the kidneys and interfere with their ability to filter waste products from the blood.
- Mutations in the PKD1, PKD2, and PKHD 1 genes cause polycystic kidney disease.
- Mutations in either the PKD1 or PKD2 gene can cause autosomal dominant polycystic kidney disease. These genes provide instructions for making proteins whose functions are not fully understood. Researchers believe that they are involved in transmitting chemical signals from outside the cell to the cell's nucleus. The two proteins work together to promote normal kidney development, organization, and function. Mutations in the PKD1 or PKD2 gene lead to the formation of thousands of cysts, which disrupt the normal functions of the kidneys and other organs.
- Genetic tests for Autosomal dominant and Autosomal recessive type of polycystic kidney disease are available

Genetic Home Reference: http://ghr.nlm.nih.gov. Accessed July 14, 2011.

Patch C, Charlton J, Roderick PJ, et al: Use of antihypertensive medications and mortality of patients with autosomal dominant polycystic kidney disease: A population-based study. Am J Kidney Dis 57(6):856–862, 2011

Causes

The causes of CKD are numerous (Box 31-6). By far, the two most common causes are diabetes mellitus and hypertension, which account for more than 54% and 33% of incident cases of ESRD, respectively.[27] Other causes include glomerulonephritis (both primary and secondary to systemic diseases), interstitial nephritis, congenital malformations, genetic disorders, neoplasms, hepatorenal syndrome, obstructive uropathy, and microangiopathic etiologies, such as scleroderma and atheroembolic disease.

Pathophysiology

Although many diseases can cause CKD, there appear to be common pathophysiologic pathways for disease progression.

Table 31-3 Stages and Prevalence of Chronic Kidney Disease in the U.S. Adult Population

Stage	Description	GFR (mL/min/1.73 m²)	Prevalence in U.S. Adult Population
1	Kidney damage with normal or increased GFR	90 or more	5.7
2	Kidney damage with mild or decreased GFR	60–89	5.4
3	Moderately decreased GFR	30–59	5.4
4	Severely decreased GFR	15–29	0.3
5	Kidney failure	<15 or dialysis	0.1

GFR, glomerular filtration rate.

Modified from Jacobs C, Opolinsky D. The Little Handbook of Dialysis. Boston, MA: Jones & Bartlett, 2010.

The outstanding common morphologic features seen in CKD include fibrosis, loss of native renal cells, and infiltration by monocytes and macrophages. The mediators of the process are many and include abnormal glomerular hemodynamics, hypoxia, proteinuria, and vasoactive substances, such as angiotensin II.[34,35]

In discussing glomerular hemodynamics, it is important to understand intact nephron theory. Because each of the more than 1 million nephrons in each kidney is an independent functioning unit, as renal disease progresses, nephrons can lose function at different times. When an individual nephron becomes diseased, nephrons in close proximity increase their individual filtration rates by increasing the rate of blood flow and hydrostatic pressure in their glomerular capillaries. This hyperfiltration response in the nondiseased nephrons enables the kidneys to maintain excretory and homeostatic functions, even when up to 70% of the nephrons are damaged. However, eventually, the intact nephrons reach a point of maximal filtration, and any additional loss of glomerular mass is accompanied by an incremental loss in GFR and subsequent accumulation of filterable toxins.

Although hyperfiltration is an adaptive measure to nephron loss, over time it actually can accelerate the loss of nephrons because the hyperfiltration causes endothelial injury, stimulation of profibrotic cytokines, infiltration by monocytes and macrophages, and detachment of glomerular epithelial cells. In addition, hypertrophy of the nondiseased nephrons caused by hyperfiltration leads to increased wall stress and even more injury.[14] This is why many interventions to slow down the progression of renal failure involve measures that reduce glomerular hydrostatic pressure. One such example is the use of ACE inhibitors and ARBs, which prevent angiotensin II–mediated efferent arteriolar vasoconstriction and subsequent nephron hyperfiltration.

Other possible mediators of CKD progression are hypoxia and angiotensin II. In CKD, the loss of peritubular capillaries by various causes results in reduced capillary perfusion of the tubules. The resultant hypoxia favors the release of proinflammatory and profibrotic cytokines, leading to fibrosis and cell injury. Angiotensin II stimulates growth factors and cytokines that contribute to fibrosis aside from its hemodynamic effects on the glomerulus.[11,34,35]

Proteinuria, the result of glomerular hypertension and abnormal glomerular permeability, also contributes to CKD progression. Abnormally filtered protein is reabsorbed by proximal tubular cells through endocytosis and accumulates in the cells, causing the production of cytokines. These proinflammatory factors ultimately cause fibrosis and scarring of the tubulointerstitium.[34,35] Proteinuria is a very strong predictor of CKD progression, consistent with its role in the pathophysiology of CKD.

BOX 31-6 Causes of Chronic Kidney Disease

- Diabetes mellitus
- Hypertension
- Glomerulonephritis
  - Primary (immunoglobulin A nephropathy, postinfectious glomerulonephritis)
  - Secondary (HIV nephropathy, lupus, cryoglobulinemia, Wegener's granulomatosis, Goodpasture's syndrome, polyarteritis nodosa, amyloidosis)
- Interstitial nephritis (allergic interstitial nephritis, pyelonephritis)
- Microangiopathic vascular disease (atheroembolic disease, scleroderma)
- Congenital disease
- Genetic disease (polycystic kidney disease, medullary cystic kidney disease)
- Obstructive uropathy
- Neoplasms or tumors
- Transplant rejection
- Hepatorenal syndrome

Diabetic Nephropathy

Because of the extremely high prevalence of diabetes and hypertension as causes of CKD, an understanding of the renal pathophysiology specific to these entities and knowledge of interventions designed to slow down or even prevent progression to stage 5 CKD is imperative. Diabetic nephropathy is a major complication of diabetes, with an incidence of approximately 20% to 40%.[36]

In diabetes, the microvasculature in the organ systems of the body, including the kidneys, is damaged. In the kidneys, primarily the afferent and efferent arterioles and the glomerular capillaries are affected. Glomerular changes include thickening of the basement membrane, mesangial expansion from overproduction and underdegradation of extracellular matrix proteins, and diffuse glomerulosclerosis. Late in diabetic nephropathy, tubular atrophy and interstitial fibrosis also occur. The exact physiological

mechanism for these structural alterations is unclear, but hyperglycemia is a major contributor. In the classic Diabetes Control and Complications Trial (DCCT)—a prospective, randomized, multicenter trial performed to assess the effectiveness of tight blood glucose control on the complications of type 1 diabetes—researchers found that strict blood glucose control delayed and possibly even prevented the progression of diabetic nephropathy.[37] More recently, the follow-up study to the DCCT, called the Epidemiology of Diabetes Interventions and Complications (EDIC) study, revealed that the benefits of tight control persist for a number of years.[38] In addition, the classic United Kingdom Prospective Diabetes Study (UKPDS) reached conclusions in people with type II diabetes that were similar to those of the DCCT.[39]

At the onset of diabetic nephropathy, patients may have an increased GFR (as high as 140 mL/min) because of hyperfiltration, slightly enlarged kidneys, and microalbuminuria (30 to 300 mg/d of albumin in the urine). Over the course of approximately 10 to 15 years, hypertension and protein leakage increase. Eventually, protein leakage is massive, with consequent hypoalbuminemia and edema as well as mild azotemia. At this point, kidney damage is extensive, often requiring dialysis therapy within a few years.

Hypertensive Nephrosclerosis

The effect of systemic hypertension on the kidneys results in a condition known as nephrosclerosis. Hypertensive nephrosclerosis involves the development of sclerotic lesions in the renal arterioles and glomerular capillaries that cause them to become thickened, narrowed, and eventually necrotic. Hypertensive nephrosclerosis can be benign or malignant. In benign nephrosclerosis, associated with chronic mild or moderate hypertension, renal impairment occurs over many years. Malignant nephrosclerosis, associated with malignant hypertension, can lead to permanent renal failure rapidly if BP is not immediately reduced. Often, symptoms such as blurred vision and a severe headache accompany this crisis situation.

Because hypertensive nephrosclerosis is directly caused by hypertension, its incidence is greater in populations with a higher incidence of primary hypertension (eg, elderly people, African Americans). Among African Americans, the risk for hypertension-induced ESRD is nearly two times that of Caucasians.[40] The signs of hypertensive nephrosclerosis vary depending on the severity of the renal damage and the acuteness of the hypertension. Some signs that may be present include proteinuria, azotemia, and hematuria with red blood cell casts. Unfortunately, like those with hypertension, patients often remain asymptomatic until extensive damage has occurred. To prevent or delay the progression of hypertensive nephropathy, BP control is essential, and often multiple different antihypertensive medications are required. This is an area in which patient education can have a great impact in decreasing the incidence of ESRD. Educating patients about the complications of uncontrolled hypertension is particularly important and may foster the patient's active involvement in controlling his or her BP.

Preventing the Progression of Chronic Kidney Disease

An important characteristic of chronic kidney disease (CKD) is continuous progression. Slowing the rate of progression after CKD is diagnosed is a focus of extensive and ongoing research. Regardless of the primary cause of CKD, specific identifiable secondary insults to the kidney can rapidly accelerate the loss of nephrons. Such secondary insults include an alteration in renal perfusion, as observed in congestive heart failure or intravascular volume depletion; the administration of nephrotoxic agents; urinary obstruction; and urinary infections. Consequently, monitoring for and avoiding these insults or aggressively treating them, if they occur, is paramount.

It is also important to educate patients and their families about the dangers of these insults. Patients and families should be instructed, for instance, about the signs and symptoms and the need for prompt treatment of urinary infections as well as common nephrotoxic drugs to avoid. Common over-the-counter and prescription analgesics, such as NSAIDs, can cause rapid deterioration in renal function and should be avoided in patients with CKD.

Strict control of blood glucose levels is critical to preventing and retarding the progression of renal failure in people with diabetes. The targets for key parameters of glucose control set by the American Diabetes Association for people with diabetes are a glycosylated hemoglobin of less than 7.0%, a preprandial plasma glucose of 70 to 130 mg/dL, and a peak postprandial plasma glucose of less than 180 mg/dL.[36]

BP control is also essential for preventing the progression of renal failure from almost any primary etiology, not just hypertension or diabetes. According to the K/DOQI Clinical Practice Guidelines on Blood Pressure Management and Use of Antihypertensive Agents in Chronic Kidney Disease (2004), the target of therapy is a BP of less than 130/80 mm Hg.[41] Control of hypertension entails lifestyle changes (eg, exercise, salt restriction, smoking cessation, and avoidance of excessive alcohol) as well as pharmacological therapy if necessary. Regarding pharmacological therapy, ACE inhibitors and ARBs have been shown to offer a selective advantage in slowing the progression of diabetic and other proteinuric syndromes. Both drugs have been proved to lower BP, reduce proteinuria, and slow the progression of kidney disease,[29–31] presumably because of their ability to decrease intraglomerular pressure by blocking the effect of angiotensin II on the afferent and efferent arterioles.

A protein-restricted diet as a means to slow the progression of renal failure is controversial, but the evidence appears to support the view that moderate protein restriction of 0.8 g/kg/d in patients may help.[30,31] However, caution with protein restriction, especially in critically ill patients who are in a catabolic state, is important to prevent malnutrition. Malnutrition itself is a major determinant of morbidity and mortality in patients with renal failure.[14,32] Ways to avoid malnutrition include providing protein with high biological value, ensuring that adequate caloric requirements are met, and closely monitoring nutritional assessment parameters (ie, body weight, serum albumin and prealbumin levels, and

total protein levels). Because of the complexity of nutritional requirements in critically ill patients, collaboration with a dietitian is essential.

Finally, controlling serum lipid levels may slow the progression of renal failure. Hyperlipidemia is commonly seen in patients with CKD, particularly those with diabetes and nephrotic syndrome, and is hypothesized to contribute to renal failure.[42] In addition, numerous secondary analyses of data from lipid trials suggest that high lipid levels are associated with a faster rate of progression and that statins slow this rate.[30] The mechanisms by which dyslipidemias may contribute to renal failure are unclear, but the formation of oxygen free radicals, the expression of growth factors and cytokines, the proliferation of mesangial cells, and the inhibition of nitric oxide have been postulated. Besides their lipid-lowering effect, statins have pleiotrophic effects that may directly oppose these mechanisms.[43] Because the use of lipid-lowering statins have favorable side effect profiles in patients with CKD, their use may be prudent, especially because lowering lipid levels decreases the risk for cardiovascular events (the primary cause of mortality in patients with ESRD).

▲ Management of Renal Failure

Although some distinct differences exist between the ways AKI and CKD are managed, many of the clinical manifestations and complications encountered are the same. Thus, the general management of renal failure is addressed here, noting any differences between AKI and CKD as necessary. In either type of renal failure, management begins with treating the primary insult. Common nursing diagnoses for patients with AKI are given in Box 31-7. An overview of the management of patients with AKI is provided in the accompanying Collaborative Care Guide (Box 31-8).

BOX 31–7 *Examples of Nursing Diagnoses*

For the Patient with Acute Kidney Injury

- Excess Fluid Volume related to decreased kidney function
- Decreased Cardiac Output related to fluid volume excess, disturbances in renin–angiotensin system
- Imbalanced Nutrition: Less Than Body Requirements related to anorexia, nausea and vomiting, dietary restrictions, and altered oral mucous membranes
- Risk for Skin Integrity related to poor nutritional status, immobility, and edema
- Anxiety related to unexpected serious illness and uncertain prognosis, unfamiliar environment, and current symptoms
- Activity Intolerance related to shortness of breath, fatigue, anemia, uremia, and dialysis procedure
- Disturbed Sleep Pattern related to fragmented sleep in hospital environment
- Risk for Infection related to decreased functioning of immune system
- Deficient Knowledge related to pathophysiology and etiology of acute episode, dietary restrictions, medications, complications, prognosis, and follow-up care

Managing Fluid Balance Alterations

Clinical management of fluid balance is of primary importance in patients with renal failure and is the area in which differences in the management of AKI and CKD are perhaps most dramatic.

Fluid Balance Changes in Acute Kidney Injury

In prerenal AKI and the early stages of ischemic ATN, the cause of the renal failure is inadequate renal perfusion, often from intravascular volume deficits. After using laboratory, physical assessment, and hemodynamic clues to make a rapid diagnosis of intravascular volume depletion, therapy involves prompt administration of replacement fluids, such as blood and crystalloids. The replacement solutions used should reflect the type of losses (eg, for a patient with a hemorrhagic condition, blood would be the replacement fluid of choice). Often in AKI, even if signs and symptoms of intravascular volume deficits are not present, large boluses of IV fluids are given. Reversal of AKI after such a bolus is therapeutic as well as diagnostic of prerenal AKI.

Fluid administration in AKI is also indicated in the diuretic phase of ATN, when extensive diuresis may occur, and for the prevention or alleviation of tubular obstruction seen in obstructive causes of AKI, including ATN and many postrenal etiologies. However, in any oliguric state, caution must be taken to prevent fluid overload. In a sustained oliguric state, such as the oliguric stage of ATN, fluid is restricted to the previous day's urine output amount plus 500 to 800 mL to account for insensible losses.

Diuretics are often used in AKI to increase urinary flow and thereby help alleviate conditions of fluid overload or to prevent tubular obstruction. Furosemide, a loop diuretic, and mannitol, an osmotic diuretic, are often used with hydration to prevent tubular obstruction in certain obstructive causes of AKI, such as acute urate nephropathy, and in heme pigment nephropathy, such as rhabdomyolysis. In states of fluid overload, such as pulmonary edema and heart failure, diuretics are also useful. Often in these situations, furosemide is administered every 6 hours, with the initial dose ranging between 20 and 100 mg depending on whether the patient has taken furosemide regularly. If within an hour the response is inadequate, the dose may then be doubled. This process may be repeated until adequate urine output is achieved. Sometimes even a continuous furosemide drip is required. In addition, a thiazide diuretic, such as chlorothiazide, may be administered with furosemide because of the synergistic action of these diuretics in promoting urinary excretion.

With the use of diuretics, caution must be taken to avoid complications of dehydration, electrolyte imbalances, and side effects. Tinnitus and hearing impairment (reversible and irreversible) have been reported after IV furosemide administration. Ototoxicity is associated with rapid injection, excessively high doses, or concomitant therapy with other ototoxic drugs. The manufacturer recommends controlled IV infusion (not to exceed 4 mg/min) for high-dose parenteral furosemide therapy.

The use of diuretics to convert oliguria to nonoliguria, unlike the aforementioned uses of diuretics, has not been substantiated in medical research and may even be harmful. Additionally, research has not shown that the use of loop diuretics in AKI

BOX 31-8 *COLLABORATIVE CARE GUIDE for the Patient With Acute Kidney Injury*

Outcomes	Interventions
Coordination of Care	
All appropriate team members and disciplines will be involved in the plan of care.	• Develop the plan of care with the patient, family, primary physician, nephrologist, pulmonologist, cardiologist, registered nurse, advanced practice nurse, social worker, respiratory therapist, physical therapist, occupational therapist, dietitian, chaplain, and dialysis staff.
Oxygenation/Ventilation	

Patient will have adequate gas exchange as evidenced by:
• ABGs within normal limits
• Functional oxygen saturation (SpO_2) >92%
• Clear breath sounds
• Normal respiratory rate and depth
• Normal chest x-ray
	• Monitor ABGs and continuous pulse oximetry.
	• Monitor acid–base status.
	• Monitor for signs and symptoms of pulmonary distress from fluid overload.
	• Provide routine pulmonary toilet, including the following:
	  • Airway suctioning
	  • Chest percussion
	  • Incentive spirometer
	  • Frequent turning
	• Mobilize out of bed to chair.
	• Support patient with oxygen therapy, mechanical ventilation, or both as indicated. Involve respiratory therapist.

Circulation/Perfusion	
Patient's BP, heart rate, and hemodynamic parameters will be within normal limits. Patient will have adequate tissue perfusion as evidenced by: • Adequate hemoglobin levels • Euvolemic status • Optimal urine output depending on phase of AKI • Appropriate level of consciousness	• Monitor vital signs every 1–2 h. • Monitor PAOP, right atrial pressure, cardiac output, systemic vascular resistance, and peripheral vascular resistance every 4 h or as ordered if pulmonary artery catheter is in place. • Assess vital signs continuously or every 15 min during dialysis. • Monitor hemoglobin and hematocrit levels daily. • Assess evidence of tissue perfusion (pain, pulses, color, temperature, and signs of decreased organ perfusion such as an altered level of consciousness, ileus, and decreasing urine output). • Administer intravascular crystalloids or blood products as indicated.
Fluids/Electrolytes	
Patient will be euvolemic. Patient will achieve normal electrolyte balance. Patient will achieve optimal renal function.	• Monitor fluid status, including input and output (fluid restriction), daily weight, urine output trends, vital signs, CVP, and PAOP. • Monitor for signs and symptoms of hypervolemia (hypertension, pulmonary edema, peripheral edema, jugular venous distention, and increased CVP). • Monitor serum electrolytes daily. • Monitor renal parameters, including urine output, BUN, serum creatinine, acid–base status, urine electrolytes, urine osmolality, and urine specific gravity. • Administer fluids and diuretics to maintain intravascular volume and renal function, per order. • Replace electrolytes as ordered. • Treat patient with, and monitor response to, dialysis therapies if indicated. • Monitor and maintain dialysis access for chosen intermittent or continuous dialysis method: *Continuous Veno–Veno Dialysis* • Monitor and regulate ultrafiltration rate hourly based on patient's response and fluid status. • Provide fluid replacements as ordered. • Assess and troubleshoot hemofilter and blood tubing hourly. • Protect vascular access from dislodgment. • Change filter and tubing per protocol. • Monitor vascular access for infection.

(*continued on page 678*)

BOX 31–8 *COLLABORATIVE CARE GUIDE for the Patient With Acute Kidney Injury (continued)*

Outcomes	Interventions
Fluids/Electrolytes	
	Peritoneal Dialysis • Slowly infuse warmed dialysate. • Drain after appropriate dwell time. • Assess drainage for volume and appearance. • Send cultures daily or as ordered. • Assess access site for infection. *Intermittent Hemodialysis* • Assess shunt for thrill and buzzing sound (bruit) every 12 h. • Avoid constrictions (ie, BPs), phlebotomy, and IV fluid administration in arm with shunt. • Assess for infection. • Monitor perfusion of related extremity.
Mobility	
Patient will remain free of complications related to bed rest and immobility.	• Initiate deep venous thrombosis prophylaxis. • Reposition frequently. • Mobilize to chair when possible. • Consult physical therapist. • Conduct range-of-motion and strengthening exercises.
Protection/Safety	
Patient will be protected from possible harm.	• Assess need for wrist restraints if patient is intubated, has a decreased level of consciousness, is unable to follow commands, or is acutely agitated, or for affected extremity during hemodialysis. Explain need for restraints to patient and family members. If restrained, assess response to restraints and check every 1–2 h for skin integrity and impairment in tissue perfusion. Follow hospital protocol for use of restraints. • Use siderails on bed and safety belts on chairs as appropriate. • Follow seizure precautions.
Skin Integrity	
Patient will have intact skin.	• Assess skin integrity and all bony prominences every 4 h. • Turn every 2 h. • Consider a pressure relief/reduction mattress. Use Braden Scale to assess risk for skin breakdown. • Use superfatted or lanolin-based soap for bathing and apply emollients for pruritus. • Treat pressure ulcers according to hospital protocol. Involve enterostomal nurse in care.
Nutrition	
Patient will be adequately nourished as evidenced by: • Stable weight not <10% below, or >20% above, ideal body weight • An albumin level of 3.5 to 4.0 g/dL • A total protein level of 6 to 8 g/dL • A total lymphocyte count of 1,000 to 3,000 × 10^6/L	• Consult dietitian to direct and coordinate nutritional support. • Observe sodium, potassium, protein, and fluid restriction as indicated. • Provide small, frequent feedings. • Provide parenteral or enteral feeding as ordered. • Monitor albumin, prealbumin, total protein, hematocrit, hemoglobin, and white blood cell counts, and monitor daily weights to assess effectiveness of nutritional therapy.
Comfort/Pain Control	
Patient will be as comfortable and as pain free as possible as evidenced by: • No complaints of discomfort • No objective indicators of discomfort	• Monitor for signs and symptoms of respiratory distress related to fluid overload and support oxygenation as needed. Keep head of bed elevated and teach breathing techniques to minimize oxygen distress, such as pursed-lip breathing. • Plan fluid restrictions over 24 h, allowing for periodic sips of water and ice chips to minimize thirst.

(*continued on page 679*)

BOX 31–8 COLLABORATIVE CARE GUIDE for the Patient With Acute Kidney Injury (continued)

Outcomes	Interventions
Comfort/Pain Control	
	• Provide frequent mouth and skin care. • Assess quantity and quality of discomfort. • Provide a quiet environment and frequent reassurance. • Observe for complications that may cause discomfort, such as infection of vascular access device, peritonitis or inadequate draining during peritoneal dialysis, and gastrointestinal disturbances (nausea, vomiting, diarrhea, constipation). • Administer analgesics, antiemetics, antidiarrheals, laxatives (non–magnesium and non–phosphate containing), stool softeners, antihistamines, sedatives, or anxiolytics as needed and monitor response.
Psychosocial	
Patient will demonstrate a decrease in anxiety as evidenced by: • Vital signs within normal limits • Level of consciousness within normal limits • Subjective reports of decreased anxiety levels • Objective assessment of decreased anxiety level	• Assess vital signs. • Explore patient and family concerns. • If the patient is intubated, develop interventions for effective communication. • Arrange for flexible visitation to meet needs of the patient and family. • Provide for adequate rest and sleep. • Provide frequent information and updates on condition and treatment, and explain equipment. Answer all questions. • Consult social services and clergy as appropriate. • Administer sedatives and antidepressants as appropriate and monitor response.
Teaching/Discharge Planning	
Patient and family members will understand procedures and tests needed for treatment during the acute phase and maintenance of a patient with chronic disease. Patient and family members understand the severity of the illness, ask appropriate questions, and anticipate potential complications. In preparation for discharge to home, the patient and family members will demonstrate an understanding of RRT, fluid and dietary restrictions, and the medication regimen.	• Prepare the patient and his or her family members for procedures, such as insertion of dialysis access, dialysis therapy, or laboratory studies. • Explain the causes and effects of renal failure and the potential for complications, such as hypertension and fluid overload. • Encourage family members to ask questions related to the pathophysiology of renal failure, dialysis, and dietary or fluid restrictions. • Make appropriate referrals and consults early during hospitalization. • Initiate family education regarding home care of the patient on dialysis, what to expect, maintenance of renal function, and when to seek medical attention.

reduces mortality, shortens the duration of renal failure, or helps avoid or reduce the requirements for RRT.[44,45] Hence, based on the literature, it is reasonable to use diuretics for a short time for volume control but not for therapy for established oliguric AKI.

Dopamine is another agent that has been traditionally used in AKI because of its ability to theoretically cause renal vasodilation at "renal doses" (1 to 3 mcg/kg/min), thereby increasing renal perfusion. However, the efficacy of this agent to affect the course of AKI has not been substantiated despite many clinical trials, and some studies have even shown deleterious effects.[45,46] Thus, based on current evidence, there is no current role for dopamine in preventing AKI.

If fluid complications arise and cannot be controlled by fluid restrictions and pharmacological agents, dialysis (discussed in detail in Chapter 30) or isolated ultrafiltration may be necessary. This is often the case in oliguric patients who are receiving large amounts of IV fluids hourly in the form of medications and nutritional supplements. People in whom AKI develops secondary to hypoperfusion or tubular injury may have a delayed recovery time, necessitating maintenance dialysis until the tissue repairs itself and normal function returns. For these patients, discharge planning should take into consideration the need for outpatient dialysis therapy (which may last for several weeks to months), the need to modify the person's diet and consumption of fluids, and the psychosocial implications of these measures for the patient and family members.

Fluid Balance Changes in Chronic Kidney Disease

In CKD, fluid and salt restriction is a mainstay of therapy to prevent fluid overload. Sodium is restricted to less than 2,400 mg/d, and fluid intake is limited to 500 mL plus the patient's previous day's 24-hour urine output. Diuretics are also used to manage volume overload. Patients usually are able to respond to diuretics until they reach stage 5 CKD, at which point extensive renal damage prevents an adequate

response. By the time CKD progresses to stage 5, oliguria is typically manifested, and signs and symptoms of fluid overload, such as edema, hypertension, pulmonary edema, heart failure, and jugular vein distention, occur unless dialysis therapy is instituted. In these patients, an ongoing assessment of fluid status, including obtaining accurate intake and output measurements with daily weights and monitoring for fluid complications, is imperative.

Managing Acid–Base Alterations

AKI and CKD typically result in metabolic acidosis because of the nephrons' inability to secrete and excrete hydrogen ions and reabsorb bicarbonate ions as renal failure progresses. In critically ill patients, this acid–base disturbance may be intensified because of concurrent conditions, such as lactic acidosis or diabetic ketoacidosis, and because such patients are in a high-catabolic state, which increases the release of intracellular acids into the circulation. Clinical manifestations of metabolic acidosis include headaches, nausea and vomiting, deep and rapid respirations (Kussmaul respirations), altered mental status, hyperkalemia, and tachycardia. In severe metabolic acidosis, bradycardia and hypotension may manifest because of myocardial depression and vasodilation. There is also a dramatic depression of the patient's level of consciousness, often resulting in stupor or coma.

In CKD, metabolic acidosis begins to manifest as the patient reaches stage 3 and the GFR falls below 60 mL/min/1.73 m^2. Although the metabolic acidosis associated with CKD is usually mild (CO_2, 16 to 22 mEq/L), it is associated with many adverse consequences, including fatigue, protein catabolism, and bone demineralization. The bones become demineralized because bone phosphate and carbonate are used as buffers against excess hydrogen ion.

Laboratory assessments of acid–base status using arterial blood gas (ABG) values and venous carbon dioxide content guide therapy. Patients with a plasma bicarbonate level less than 22 mEq/L warrant treatment. Therapy involves the administration of alkaline medications (eg, Bicitra, sodium bicarbonate tablets), dialysis, or both. When using citrate-containing medications, such as Bicitra, it is important that these medications not be given with aluminum-containing phosphate binders. Using these agents together would put the patient at risk for aluminum toxicity because citrate significantly increases aluminum absorption from the gastrointestinal tract.

The use of IV sodium bicarbonate is reserved for severe acidosis (evidenced by a blood pH < 7.2 or a plasma bicarbonate level <12 to 14 mEq/L) because of potential complications of extracellular volume excess, metabolic alkalosis, and hypokalemia. Intractable acidosis is an indication for dialysis, which removes excess hydrogen ions and adds a buffer to the body. In hemodialysis, the buffer is bicarbonate, and in peritoneal dialysis, it is lactate, which is metabolized to bicarbonate. When correcting metabolic acidosis, caution is advised. Rapid correction may result in a suppressed respiratory drive and hypoventilation. Rapid correction can also lead to acute hypocalcemia and tetany because the amount of ionized calcium decreases in an alkalotic state owing to increased binding of calcium with albumin and inorganic substances such as phosphate. Throughout any kind of acid–base therapy, it is necessary to monitor serum bicarbonate, pH, and calcium and potassium levels closely.

Managing Cardiovascular Alterations

Alterations in the cardiovascular system can cause or accelerate AKI and CKD. In addition, cardiovascular complications can arise as a result of renal failure itself. Common cardiovascular complications in AKI and CKD include hypertension and hyperkalemia. Pericarditis, another cardiovascular complication of renal disease, is primarily seen with CKD.

Hypertension

Hypertension as a complication of renal failure results from excess retention of water and sodium, overactivation of the sympathetic nervous system, and stimulation of the renin–angiotensin–aldosterone system. Because controlling BP is essential to prevent end-organ damage and reduce the risk for life-threatening cardiovascular events, adequate treatment is essential. Management may include fluid and sodium restrictions, diuretic administration, antihypertensive therapy, and dialysis to remove excess fluid. Extensive patient teaching regarding nonpharmacological and pharmacological treatment and the potential complications of uncontrolled hypertension is an integral part of management.

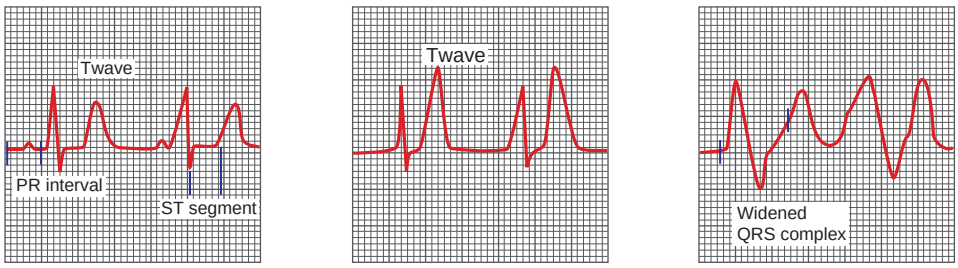
Hyperkalemia

Hyperkalemia is a life-threatening condition seen in patients with AKI and CKD. As the GFR decreases, the ability of the kidneys to excrete excess potassium diminishes. In critically ill patients, this renal impairment is frequently compounded by states of increased catabolism, acidosis, cellular injury, administration of potassium-based medications, and blood transfusions, all of which can raise serum potassium levels. If not recognized and treated, hyperkalemia leads to fatal dysrhythmias.

Assessment of hyperkalemia involves close monitoring of serum potassium levels as well as monitoring the effects of potassium on the electrical conduction system of the heart. Characteristically, electrocardiogram (ECG) changes occur as potassium levels rise (Fig. 31-4). The first ECG changes that occur, usually when serum potassium is in the range of 6 to 7 mEq/L, are the appearance of tall, tented T waves and a prolonged PR interval. Next, there is a loss of the P wave and a slight widening of the QRS complex. At this point, the serum potassium is usually in the range of 8 to 9 mEq/L. From here, the QRS complex continues to widen until a sine wave (wavy line) pattern develops. This ominous sign is closely followed by ventricular fibrillation or standstill.

When evaluating hyperkalemia, note that patients with long-standing elevations in serum potassium are more refractory to its effects on the heart than patients in whom hyperkalemia develops suddenly. Thus, potassium and ECG changes must be evaluated together to determine the acuteness of the situation. Other effects of hyperkalemia that are monitored include paresthesias, hyporeflexia, and muscle weakness (which typically begins in the lower extremities and ascends to the trunk and upper extremities).

Mild hyperkalemia (a serum potassium level <6 mEq/L without ECG changes) may be treated with dietary



A. Peaked T waves, prolonged PR interval, depressed ST segment

B. Lost P wave

C. Widened QRS complex

FIGURE 31-4 ▲ Typical ECG findings indicative of various degrees of hyperkalemia. **A:** When the serum potassium (K^+) level is about 6 to 7 mEq/L, the T waves become peaked, the PR interval is prolonged, and the ST segment is depressed. **B:** At about 8 to 9 mEq/L, the P wave is lost. **C:** At about 10 to 11 mEq/L, the QRS complex widens.

potassium restriction, diuretics, and potassium-binding resins (eg, sodium polystyrene sulfate). Sodium polystyrene is given orally or as an enema. The oral dose of 15 to 30 g in 60 to 120 mL of a 20% sorbitol solution (to prevent constipation) may be repeated every 4 to 6 hours as needed. The rectal dose of 50 g in 50 mL of 70% sorbitol and 150 mL tap water should be retained in the colon for at least 30 to 60 minutes. This drug must be used with caution in critically ill patients with decreased colonic motility, such as postsurgical patients and patients taking large amounts of opiates, because of its association with colonic necrosis in this population. To reduce this risk, a cleansing enema may be given after rectal administration.[14] Sodium polystyrene should never be used in a patient with a gastrointestinal obstruction, and bowel sounds should always be assessed before its administration.

Treatment of life-threatening hyperkalemia entails taking steps to antagonize the effects of potassium on the heart, promote intracellular shifting of potassium, and remove potassium from the body. Antagonizing the effects of potassium on the heart is achieved with IV calcium gluconate or chloride and is the first priority for patients with substantial ECG changes. Intracellular shifting of potassium is done next to bridge the gap until potassium removal from the body can be executed. Means to shift potassium into the cell include IV insulin and dextrose administration and IV bicarbonate administration. β_2-Adrenergic therapy can also effect transcellular potassium shifting but is less commonly used because of the requirement for 10 to 20 times the dose used for reactive airway disease. Removal of potassium from the body entails, as previously mentioned, diuretic administration and the use of potassium exchange resins. If these measures do not control hyperkalemia, dialysis must be initiated. Obviously, in a patient who has stage 5 CKD, and who is likely already receiving dialysis therapy, dialysis is initiated immediately along with other emergent therapy in life-threatening hyperkalemia.

Pericarditis

Pericarditis resulting from uremia (uremic pericarditis) is a complication that can be seen primarily in stage 5 CKD. This type of pericarditis is characterized by an inflammation of the pericardial membrane, which causes the pericardial capillaries to become permeable to fluid, red blood cells, fibrinogen, and albumin. In most cases, the inflammation is aseptic, although it may also result from bacterial or viral infections. The consequent serous or serosanguineous fluid in the pericardial cavity (pericardial effusion) can increase the intrapericardial pressure and compromise ventricular contractility, stroke volume, and cardiac output. Pericardial tamponade, which results when the accumulation of pericardial fluid is so large that adequate cardiac output cannot be maintained, is a life-threatening emergency. The exact etiology of uremic pericarditis is unknown, but it is associated with prolonged inadequate dialysis therapy, uremic toxins, infectious agents, treatment with the antihypertensive agent minoxidil, and heparin administration.

Chest pain, fever, and a pericardial friction rub are the classic triad of findings associated with pericarditis. The chest pain is characteristically sharp and steady and is relieved by sitting forward and intensified by breathing deeply. The pericardial friction rub (a harsh, leathery sound heard over the precordium) may precede the pain, may persist after the pain has subsided, and may disappear when the volume of effusion increases.[14] In addition to these findings, there are typical ECG changes in pericarditis. The most notable are new-onset atrial dysrhythmias and widespread ST elevations with an upward concavity (versus the upward convexity typical in an acute myocardial infarction). In a large pericardial effusion, signs and symptoms are more dramatic and include dyspnea, tachycardia, mental confusion, weakness, increased jugular vein distention, peripheral edema, and a paradoxical pulse greater than 10 mm Hg during inspiration. Tamponade results in distended neck veins, tachypnea, a narrowed pulse pressure, an increased PAOP, muffled heart sounds, diminished peripheral pulses, and a decreased level of consciousness.

Therapy for uremic pericarditis includes aggressive dialysis therapy, usually daily, until symptoms disappear. Also, because anticoagulation during dialysis may precipitate or enhance bleeding into the pericardial space, low-dose, regional, or no heparin may be prescribed. Systemic steroids and NSAIDs, such as indomethacin, may also be used but have variable results. Cardiac tamponade is an emergency that requires urgent pericardiocentesis to relieve the pressure on the heart. For the patient in whom recurrent pericarditis develops or the pericardium becomes constrictive, surgical creation of a pericardial window or pericardiectomy may be necessary.

Cardiovascular Disease in Chronic Kidney Disease

CKD is associated with high cardiovascular morbidity and mortality. In fact, patients with CKD are much more likely to suffer from cardiac disease resulting in cardiovascular death than to eventually require RRT.[47] The predominant cardiac disorders in

CKD are left ventricular hypertrophy (found in 74% of patients on dialysis), coronary artery disease, dysrhythmias, cardiomyopathy, congestive heart failure, and valvular dysfunction.

Because most of these cardiovascular disorders develop over a period of at least a few years, they usually present early in CKD and continue to progress as renal function declines. This association between CKD and cardiovascular disease may occur for the following reasons: (1) cardiovascular disease causes renal dysfunction (ie, heart failure), (2) CKD causes an increased risk for cardiovascular disease, or (3) other factors (eg, hypertension, diabetes mellitus, anemia, or hyperlipidemia) cause or accelerate both renal dysfunction and cardiovascular disease. In any case, monitoring for cardiovascular disease, reducing modifiable risk factors, and treating specific cardiovascular conditions when present are essential to decrease mortality in patients with CKD.

Diagnostic tests useful in assessing for cardiovascular disease in these high-risk patients include routine ECGs, echocardiography, and cardiac stress testing. Pharmacological rather than exercise stress testing is the stress test of choice because patients with CKD are often unable to attain the level of exercise needed to make exercise stress tests useful. More invasive tests for symptomatic patients include a thallium scan and coronary angiography.

Modifiable risk factors that can contribute to cardiovascular disease and that should be addressed as part of managing patients with CKD include hypertension, hyperlipidemia, hypervolemia, anemia, smoking, hyperglycemia, calcium and phosphate imbalances, vitamin D deficiency, hyperhomocysteinemia, and metabolic acidosis.[47,48] Regarding BP control, the Joint National Committee for the Prevention, Detection, Evaluation and Treatment of High Blood Pressure (JNC7) and the K/DOQI Clinical Practice Guidelines recommend strict BP control with a goal of less than 130/80 mm Hg in all patients with CKD.[49,50] Similarly, the K/DOQI Clinical Practice Guidelines recommend that all patients with CKD be included in the highest risk group, justifying strict lipid control with a target low-density lipoprotein cholesterol level of less than 100 mg/dL.[51] As with the general population, disease-specific treatment (eg, antiplatelet therapy and β-blocker administration for coronary artery disease) must be instituted as appropriate.

Managing Pulmonary Alterations

A frequent complication in patients with oliguric AKI or stage 5 CKD is the development of pulmonary edema. This complication results from fluid overload, heart failure, or both. Clinical manifestations include dyspnea; crackles on auscultation; the production of pink, frothy sputum; tachypnea; tachycardia; decreased arterial oxygen saturation (SaO_2); and evidence of fluid overload on chest radiograph. Management involves fluid and sodium restriction, treating underlying cardiac disease, and possibly diuretic medications if the patient's kidneys can respond to them. Frequently, pulmonary edema becomes life threatening, necessitating intubation, emergent dialysis, or both to improve arterial oxygenation and restore fluid balance.

Other pulmonary complications in renal failure include pleural effusions, pleuritic inflammation and pain, uremic pneumonitis, and pulmonary infections. Pleuritic inflammation and uremic pneumonitis occur more frequently with stage 5 CKD and are due to the effect of uremic toxins on the lungs and inadequate dialysis. Pulmonary infections, on the other hand, are common in both AKI and CKD, especially in critically ill patients. Factors associated with renal failure that contribute to pulmonary infections include decreased pulmonary macrophage activity, a generalized immunocompromised state, tenacious sputum, and a depressed cough reflex. Collaborative management includes culturing sputum, administering broad-spectrum antibiotics until organism-specific sensitivities are available, and teaching and encouraging pulmonary hygiene measures (ie, coughing and deep breathing).

Managing Gastrointestinal Alterations

A potentially life-threatening gastrointestinal complication in both AKI and CKD is gastrointestinal bleeding. Proposed etiologies for gastrointestinal bleeding as it relates to renal failure include platelet and blood-clotting abnormalities; anticoagulation with dialysis, access patency, or both; ingestion of irritating drugs (eg, NSAIDs, aspirin); and increased ammonia production in the gastrointestinal tract from urea breakdown. Ammonia is known to be irritating to mucosal surfaces. Physiological stress, especially in critically ill patients, is another proposed contributor. Assessment parameters include examining all vomit and stool for gross and occult blood; monitoring iron, hemoglobin, hematocrit, and red blood cell indices; and paying close attention to signs of intravascular volume depletion. If gastrointestinal bleeding is suspected, radiographic and endoscopic examinations are often required to diagnose and treat specific lesions. Management depends on the specific lesion but often includes volume restoration with crystalloids and blood products as well as administration of histamine-2 receptor (H_2) blockers, proton pump inhibitors (PPIs), or both.

Other gastrointestinal complications associated with renal failure occur primarily in CKD and include anorexia, nausea, vomiting, diarrhea, constipation, gastroesophageal reflux disease (GERD), and oral cavity alterations, such as stomatitis, a metallic taste in the mouth, and uremic fetor (the smell of urine and ammonia on the breath). Oral alterations and symptoms of anorexia, nausea, and vomiting are partially attributable to high levels of uremic toxins, which affect the intestinal mucosa and stimulate vomiting centers in the brain. The reason GERD is common is unclear but may be due to alterations in the hormones that affect lower esophageal sphincter tone and a higher occurrence of hiatal hernias in patients with CKD.[14] Collaborative management involves initiating (or providing) adequate dialysis, providing prophylactic antacids and H_2 blockers or PPIs, and administering antiemetics. Good oral hygiene is also essential.

The complication of constipation is seen frequently in patients with renal failure owing to decreased bulk and fluid in the diet and the administration of oral iron supplements and calcium-based phosphate binders. Diarrhea may also occur as a result of intestinal irritation from uremia. Collaborative management includes increasing dietary bulk; administering bulk-forming laxatives, stool softeners, or both; administering antidiarrheal agents; or a combination of these therapies. For patients with stage 5 CKD, magnesium-containing medications, including cathartics, such as magnesium citrate, should be avoided because of the risk for hypermagnesemia

in these patients. In addition, Fleet enemas, which contain large amounts of phosphate that could be absorbed systemically, should not be used.

Managing Neuromuscular Alterations

Neuromuscular alterations include sleep disturbances, cognitive process disturbances, lethargy, muscle irritability, and peripheral neuropathies, including restless leg syndrome and burning feet syndrome. Restless leg syndrome is characterized by a discomfort in the legs, especially at night, which is sometimes relieved by continuous movement of the extremities. Burning feet syndrome consists of paresthesias and numbness in the soles of the feet and lower parts of the legs. These neuromuscular complications are associated primarily with stage 4 and 5 CKD and are thought to be the result of electrolyte imbalances, metabolic acidosis, and the effect of uremic toxins on motor and sensory nerves. Cognitive process disturbances, such as difficulty concentrating and impaired short-term memory, are linked to elevations of BUN in the cerebral vasculature, which can result in cerebral edema. Extensive cerebral edema can result in seizures, projectile vomiting, and even coma or death.

Frequent assessments for cognitive disturbances, seizure activity, and other neuromuscular alterations are important. In addition to thorough neuromuscular examinations, nerve conduction studies and diagnostic tests, including electroencephalograms and head CT scans, may be used. Collaborative management involves implementing emergency treatment, as in the case of sustained seizure activity; maintaining electrolyte balance; correcting metabolic acidosis; using regular dialysis; and providing extensive patient teaching. Specific points that need to be included during patient teaching are the importance of preventing injury to the extremities by heat or trauma when paresthesias are present and that alterations in neuromuscular function often improve with regular dialysis or transplantation. However, if components of the patient's neuropathies are due to other comorbid conditions, such as diabetes, the problem may respond only minimally to dialysis or renal transplantation.

Cognitive alterations encountered are important to remember during any patient teaching. Because of difficulties in concentrating and impairments in short-term memory, teaching should be provided in short, frequent sessions with reinforcement of material and should include the family as much as possible. These points are especially true for critically ill patients who are, by definition, in a crisis situation.

Managing Hematological Alterations

Hematological system alterations are major complications in AKI and CKD. These alterations include an increased bleeding tendency, an impaired immune system, and anemia.

Increased Bleeding Tendency

The increased bleeding tendency in renal failure is attributable to impaired platelet aggregation and adhesion and an altered platelet response to clotting factor VII (von Willebrand's factor). These alterations are thought to be due to uremia, but their exact pathophysiological mechanisms are unknown. Assessment involves the monitoring of platelet counts and bleeding times, coagulation studies, and assessing for bleeding, especially gastrointestinal bleeding. Collaborative management includes administering blood products as needed, protecting the patient from injury, and avoiding medications that alter platelet function, such as NSAIDs and aspirin. Often heparin (for dialysis) and aspirin (for myocardial infarction prevention) are indicated in patients with renal failure. In such cases, the effects of these medications on platelets must be closely monitored. One potential and serious complication of heparin is heparin-induced thrombocytopenia; the development of this complication mandates discontinuation of the drug.

Impairments in the Immune System

Patients with renal failure are in an immunocompromised state, which sets the stage for infections (a major cause of mortality in AKI and CKD). The impairments in the immune system are thought to be due to malnutrition and the effects of uremia on white blood cells. These effects include, among others, depressed T-cell- and antibody-mediated immunity, impaired phagocytosis and decreased chemotaxis and adherence of white blood cells.[14]

Assessing the patient for infection and monitoring laboratory indicators of infection must be done continuously. Regarding temperature as a gauge of infection, the baseline body temperature in uremic patients is decreased, and thus any increase in temperature above baseline is significant. Collaborative management includes frequent hand washing, removing invasive catheters as soon as possible (or avoiding their use altogether), and culturing blood and other body fluids that may be infected to identify specific organisms and determine appropriate antimicrobial therapy.

Anemia

Anemia associated with renal failure is attributable to three main mechanisms: erythropoietin deficiency, decreased red blood cell survival time, and blood loss from an increased bleeding tendency. Of these three mechanisms, erythropoietin deficiency has the most dramatic effect.

More than 90% of the hormone erythropoietin is produced in the kidneys. It is a glycoprotein that stimulates red blood cell production in response to hypoxia and is essential to maintaining normal red blood cell counts. As kidney disease progresses and nephrons are damaged, this hormone is inadequately synthesized and a hypoproliferative anemia, resulting in normocytic normochromic red blood cells, results. Before the production of erythropoietin by human recombinant techniques, this hormone deficiency caused most patients with CKD to be in a severely anemic state, requiring frequent blood transfusions.

Decreased red blood cell survival time in renal failure occurs in the form of a mild hemolysis. The exact mechanism for this hemolysis is unclear, but it may be related to dialysis therapy or the effect of uremia on red blood cells. The average survival of red blood cells in uremia is only 70 days, which contrasts with the normal 120-day life span of a red blood cell in the general population.

In addition to the three aforementioned mechanisms of anemia, other factors can contribute to anemia in patients with renal failure, particularly those who are critically ill. Examples are malnutrition, frequent laboratory blood sampling, dialyzer

malfunction and sequestration of blood in the dialyzer, and infectious states. Treating anemia in patients with renal failure is extremely important for many different reasons, including increasing the oxygen-carrying capacity of the blood, increasing intravascular volume, and preventing the negative consequences of anemia on the cardiovascular system. Concerning the cardiovascular system, anemia exacerbates myocardial, cerebral, and peripheral ischemia and increases the risk for development (or acceleration) of left ventricular hypertrophy. Correcting anemia has also been shown to have a positive impact on quality-of-life issues in patients with renal failure, including increases in appetite, energy, and work capacity. In addition, CKD anemia is associated with increased hospitalization rates and increased mortality in patients with CKD.[14]

A thorough evaluation of anemia involves diagnostic studies and a history and physical examination. Diagnostic metabolic parameters that should be obtained and monitored include hemoglobin, hematocrit, red blood cell indices, and reticulocyte counts. In addition, the stool or vomit should be tested for occult blood. Iron studies also need to be obtained because iron deficiency itself can cause anemia and because adequate iron stores are needed for erythropoietin to be effective. Specific iron indices that should be obtained include total serum iron, total iron-binding capacity, and serum ferritin levels. Finally, nutritional parameters and levels of folic acid, pyridoxine, and vitamin B_{12}, all of which affect red blood cell production, need to be monitored.

A thorough history and physical examination involves questioning patients about potential sites of bleeding (eg, by asking about stool color), assessing for signs and symptoms of anemia (eg, angina, tachycardia, skin and mucous membrane pallor, appetite suppression, weight loss, decreased energy levels, fatigue), assessing for sources of blood loss, assessing for inflammation or infection, and assessing for other diseases that can cause anemia (eg, lupus, sickle cell anemia).

Collaborative management of anemia includes minimizing blood loss, administering oral or IV iron supplements, providing vitamin supplementation, aggressively treating infections, ensuring adequate nutrition, and administering erythropoietin stimulating agents (ESAs), such as human erythropoietin or darbepoetin, blood products, or both. Goals for iron therapy in the CKD population are a transferrin saturation greater than 20% and serum ferritin levels greater than 100 ng/mL (>200 ng/mL for dialysis patients). These goals and guidelines to achieve them are detailed in the K/DOQI guidelines on anemia.[52] Goals for ESA therapy are less concrete due to potential cardiovascular risks when targeting normal hemoglobin levels. These cardiovascular risks prompted the Federal Drug Administration (FDA) to issue a warning in all package inserts of ESAs. Hemoglobin goals should be individualized, based on patient symptoms and co-morbidities, using the lowest ESA dose to reduce the need for red blood cell transfusions.

Certain points regarding ESA therapy and the management of anemia deserve special mention. One is that the full effect of these medications takes weeks to achieve, and hence in patients with profound anemia, blood administration is indicated. In addition, ESA administration may result in an elevation of BP. In some cases, modification of antihypertensive therapy may be needed. When there is an inadequate response to ESAs despite increased dosages, reasons for erythropoietin resistance need to be explored. These include occult infections, inflammatory states, human immunodeficiency virus infection, hyperparathyroidism, aluminum toxicity, malnutrition, iron deficiency, and bone marrow malignancy.

Important clinical features regarding iron preparations should also be considered by the nurse. One is that oral iron is poorly absorbed if taken with phosphate binders, antacids, H_2 blockers, or PPIs, all of which are commonly prescribed to patients with renal failure. On the other hand, IV iron has much better bioavailability but carries the risk for an allergic, sometimes life-threatening, reaction.

Extensive patient teaching about anemia is crucial. At minimum, teaching should include information about medication therapy; timing of iron supplements; potential causes, signs, and symptoms of worsening anemia; and energy conservation techniques. Instruction about measures to decrease bleeding, such as use of a soft toothbrush and avoidance of NSAIDs, is also helpful.

Managing Alterations in Drug Elimination

Because many pharmacological agents, their metabolites, or both are excreted by the kidneys, extreme caution must be used when administering medication to patients with renal failure. Depending on the patient's GFR, adjustments may need to be made in drug dosage, the interval between drug dosages, or both. Important to consider, especially in AKI, is that the GRF is often unstable, and thus the GFR must be monitored frequently to determine dosages accurately. As in patients without renal failure, monitoring serum levels of certain medications to be sure they are within the therapeutic range is essential. For patients receiving dialysis, the health care team must be cognizant of which drugs are removed during dialysis therapy to ensure appropriate timing of drug administration. For a listing of frequently encountered antimicrobial agents in critical care that are affected by renal failure, hemodialysis, or both, see Table 31-4.

Managing Skeletal Alterations

In renal failure, disturbances in calcium and phosphate balance occur and set the stage for secondary hyperparathyroidism and high-turnover renal osteodystrophy (renal bone disease). As the GFR declines, glomerular filtration of phosphate also decreases, and serum phosphate levels begin to rise. This results in decreased serum ionized calcium levels because of binding of the calcium with the phosphate. Calcium levels also decrease because of the failing kidneys' inability to convert vitamin D to its active form (1,25-dihydroxycholecalciferol, or vitamin D_3), which is needed for adequate intestinal absorption of calcium. In response to decreased ionized calcium levels, elevations in serum phosphorus levels, and reduced vitamin D_3 synthesis, the parathyroid glands secrete parathyroid hormone (PTH). Over time, the continuous PTH stimulation leads to hyperplasia and proliferation of the parathyroid cells, resulting in secondary hyperparathyroidism. PTH causes the reabsorption of calcium and phosphate salts from bones, thus increasing the serum calcium level at the expense of bone density and mass. PTH also causes calcium reabsorption and phosphate excretion in the kidneys; however, as renal failure progresses, this effect of PTH is not realized. Eventually, as calcium and phosphate continue to be reabsorbed from bones, both levels rise in the serum concomitantly. This results in an elevation in the normal calcium–phosphate product (serum calcium

Table 31-4 Impact of Renal Failure and Hemodialysis on Commonly Used Antimicrobials in Critical Care

Drug	Drug Renally Excreted (%)	Adjustment for Renal Failure (GFR [mL/min/1.73 m²]) 10–50	<10	Effect of Hemodialysis
Aminoglycosides*				
Amikacin†	95	100% of normal dose every 24–48 h	100% of normal dose q48–72 h	Dialyzed
Gentamicin†	95	100% of normal dose every 24–48 h	100% of normal dose q48–72 h	Dialyzed
Tobramycin†	95	100% of normal dose every 24–48 h	100% of normal dose every 48–72 h	Dialyzed
Cephalosporins				
Cefazolin†	75–95	100% of normal dose every 12 h	100% of normal dose every 24–48 h	Dialyzed
Cefepime†	85	100% of normal dose every 16–24 h	100% of normal dose every 24–48 h	Dialyzed
Cefotaxime (active metabolite in ESRD)	60	100% of normal dose every 8–12 h	100% of normal dose every 24 h	Dialyzed
Cefotetan	75	50% of normal dose	25% of normal dose	Dialyzed
Ceftazidime	60–85	100% of normal dose every 24–48 h	100% of normal dose every 48 h	Dialyzed
Ceftriaxone	30–65	Normal dose	Normal dose	Dialyzed
Penicillins				
Amoxicillin	50–70	100% of normal dose every 8–12 h	100% of normal dose every 24 h	Dialyzed
Ampicillin	30–90	100% of normal dose every 6–12 h	100% of normal dose every 12–24 h	Dialyzed
Mezlocillin	65	100% of normal dose every 6–8 h	100% of normal dose every 8 h	Not dialyzed
Nafcillin	35	Normal dose	Normal dose	Not dialyzed
Penicillin G	60–85	75% of normal dose	20%–50% of normal dose	Dialyzed
Piperacillin	75–90	100% of normal dose every 6–8 h	100% of normal dose every 8 h	Dialyzed
Ticarcillin†	85	1–2 g every 8 h	1–2 g every 12 h	Dialyzed
Quinolones				
Ciprofloxacin	50–70	50%–75% of normal dose	50% of normal dose	Slightly dialyzed
Levofloxacin	67–87	250 mg every 24–48 h (500 mg initial dose)	250 mg every 48 h (500 mg initial dose)	No data
Tetracyclines				
Doxycycline	35–45	Normal dose	Normal dose	Not dialyzed
Tetracycline†	48–60	100% of normal dose every 12–24 h	100% of normal dose every 24 h	Not dialyzed
Miscellaneous Antibacterials				
Azithromycin	6–12	Normal dose	Normal dose	Not dialyzed
Aztreonam	75	50%–75% of normal dose	25% of normal dose	Moderately dialyzed

(continued on page 686)

Table 31-4 **Impact of Renal Failure and Hemodialysis on Commonly Used Antimicrobials in Critical Care (continued)**

Drug	Drug Renally Excreted (%)	Adjustment for Renal Failure (GFR [mL/min/1.73 m²]) 10–50	<10	Effect of Hemodialysis
Miscellaneous Antibacterials				
Clarithromycin	15–25	75% of normal dose	50%–75% of normal dose	No data; give after dialysis
Erythromycin	15	Normal dose	50%–75% of normal dose	Not dialyzed
Imipenem	20–70	50% of normal dose	25% of normal dose	Dialyzed
Metronidazole	20	Normal dose	50% of normal dose	Dialyzed
Sulfamethoxazole	70	100% of normal dose every 18 h	100% of normal dose every 24 h	Dialyzed
Trimethoprim	40–70	100% of normal dose every 18 h	100% of normal dose every 24 h	Dialyzed
Vancomycin†	90–100	1.0 g every 24–96 h	1.0 g every 4–7d	Not dialyzed
Linezolid‡	30	Normal dose	Normal dose	Not dialyzed
Antifungals				
Amphotericin B	5–10	Normal dose	100% of normal dose every 24–36 h	Not dialyzed
Fluconazole	70	Normal dose	Normal dose	Dialyzed
Ketoconazole	13	Normal dose	Normal dose	Not dialyzed
Antiviral				
Acyclovir	40–75	100% of normal dose every 12–24 h	50% of normal dose every 24 h	Dialyzed
Ganciclovir	90–100	100% of normal dose every 24–48 h	100% of normal dose every 48–96 h	Dialyzed
Antitubercular				
Amantadine†	90	100% of normal dose every 48–72 h	100% of normal dose every 7d	Not dialyzed
Ethambutol	75–90	100% of normal dose every 24–36 h	100% of normal dose every 48 h	Dialyzed
Isoniazid	5–30	Normal dose	Normal dose	Dialyzed
Rifampin	15–30	50%–100% of normal dose	50%–100% of normal dose	Not dialyzed

*Aminoglycosides are nephrotoxic and ototoxic and have a narrow therapeutic window. Serum levels must be monitored frequently for efficacy and toxicity.

†These drugs have adjustment in dose and/or frequency when a patient's GFR is >50 mL/min as well.

‡Metabolites may accumulate in renal failure; significance unknown.

Modified from Aronoff G, Berns J, Brier M, et al: Drug Prescribing in Renal Failure, 5th ed. Philadelphia, PA: American College of Physicians, 2007.

multiplied by serum phosphate) of less than 40 mg/dL. When the product exceeds 55 mg/dL, calcium phosphate crystals can form and precipitate in various parts of the body (a condition known as metastatic calcifications), including the brain, eyes, gums, valves of the heart, myocardium, lungs, joints, blood vessels, and skin. Other insults to bones that can occur in renal disease include bone demineralization in response to metabolic acidosis and low-turnover renal osteodystrophy from aluminum deposits in the bone or overuse of vitamin D_3 therapy. The events related to high-turnover renal osteodystrophy in renal failure are summarized in Figure 31-5.

Complications resulting from renal bone disease include bone pain, fractures, pseudogout from deposits of calcium oxalate in synovial fluid, periarthritis from calcifications of the joints, proximal muscle weakness, spontaneous tendon rupture, and pruritus. Metastatic calcifications can result in calcified blood vessels and valves, skin lesions, red-eye syndrome from crystal deposition in the conjunctiva, and, most seriously, ischemic ulcers. Laboratory data, including levels of calcium, phosphate, aluminum, alkaline phosphatase, and intact PTH, help make the diagnosis. Radiographic findings also may be helpful, particularly in high-turnover bone

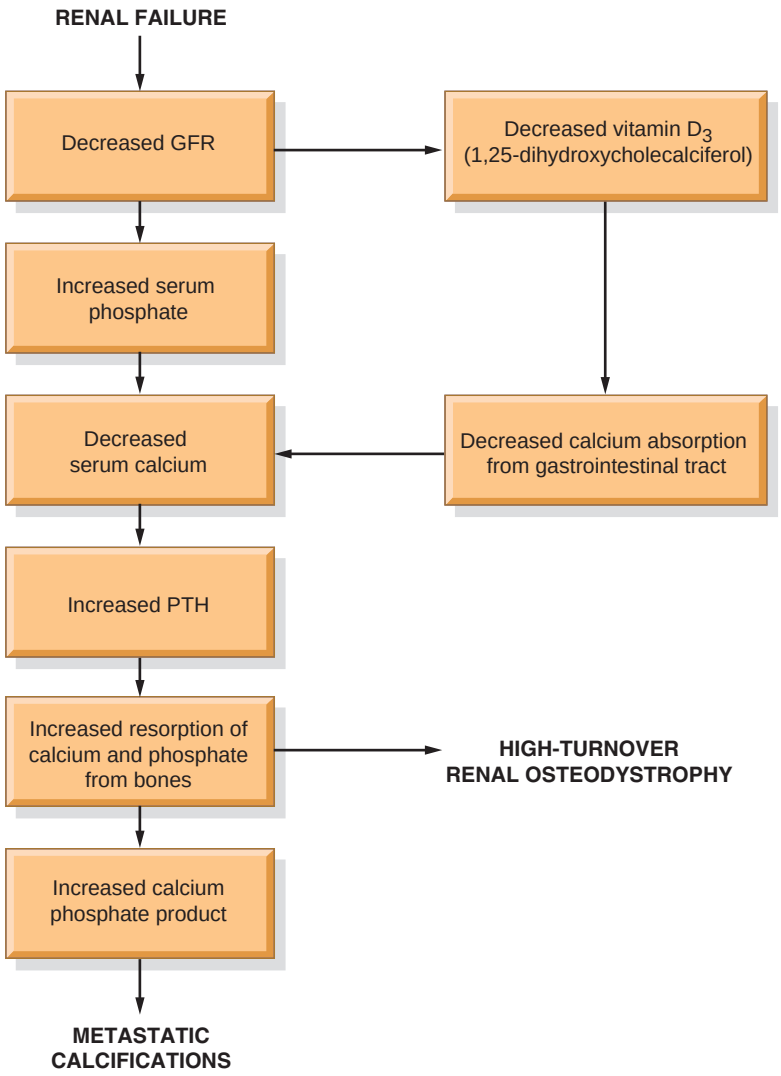


FIGURE 31-5 ▲ Effects of renal failure on the skeletal system.

disease; images may reveal subperiosteal bone thinning, most easily seen in the hands and clavicles. A bone biopsy, considered the gold standard for obtaining a definitive diagnosis of renal bone disease, is not routinely performed secondary to patient discomfort and controversy surrounding the indications for this invasive test.

Management involves phosphate regulation, maintenance of normal calcium levels, treatment of vitamin D deficiency, suppression of PTH, prevention of aluminum toxicity, and control of metabolic acidosis. Measures to control phosphate levels include dietary restrictions and phosphate-binding medications. Commonly used phosphate binders are calcium acetate (Phos-lo), calcium carbonate (Tums), sevelamer hydrochloride (Renagel), and lanthanum carbonate (Fosrenol). Sevelamer and lanthanum carbonate are calcium-free phosphate binders and are preferred over calcium-based binders in patients with high calcium levels because they lessen the risk for hypercalcemia and further elevations in the calcium–phosphate product. Aluminum hydroxide binders, once a mainstay of therapy, are now infrequently used because of the effects of aluminum toxicity on the bones as well as the nervous system. Aluminum toxicity causes erythropoietin resistance as well.

According to the K/DOQI Clinical Practice Guidelines, calcium levels should be maintained in the normal range, preferably toward the lower end of normal (8.4 to 9.5 mg/dL).[53] This is accomplished with diet and calcium supplements. If calcium levels exceed 10.2 mg/dL, therapies that may be contributing to hypercalcemia (eg, administering calcium or vitamin D supplements) should be adjusted to reduce the risk for extraskeletal calcifications.

Vitamin D supplements are administered to suppress PTH secretion. Besides causing a decrease in PTH indirectly through the elevation of serum calcium, active vitamin D also directly inhibits PTH secretion by binding to vitamin D receptors on the parathyroid gland. Active vitamin D may be given orally (calcitriol) or intravenously (Calcijex). In either case, caution must be exercised with the administration of these agents to avoid hypercalcemia and hyperphosphatemia as well as to avoid oversuppression of the parathyroid gland. Two synthetic analogues of active vitamin D that can also be used are paricalcitol (Zemplar) and doxercalciferol (Hectorol). These drugs have the advantage of causing less dramatic increases in serum calcium and phosphate levels while still causing PTH suppression.

The most recent therapeutic agents developed to help suppress PTH and the development of secondary hyperparathyroidism are calcimimetics, which work by increasing the sensitivity of the calcium-sensing receptor in the parathyroid gland to extracellular calcium. In the United States, the Food and Drug Administration approved the calcimimetic cinacalcet hydrochloride (Sensipar) for patients with ESRD. Thus far, it has been shown to be both safe and effective, with the most common side effects being nausea and vomiting and hypocalcemia.[54] Rarely, for patients who are refractory to available treatments for secondary hyperparathyroidism, including vitamin D therapy and calcimimetics, a parathyroidectomy may be necessary.

Patient teaching concerning bone disease and its management is complex and needs to be continually reinforced. Particular areas that should be included are the purpose and timing of medications (eg, phosphate binders must be given with meals to be effective), dietary modifications, and the complications of untreated bone disease.

Managing Integumentary Alterations

Alterations in the integumentary system in renal failure include xerosis (dryness), pruritus, pallor, ecchymosis and purpura, and a pale bronze skin discoloration. Contributing factors to these alterations are anemia, decreased activity of sweat and sebaceous glands, retained skin pigments, platelet dysfunction and capillary fragility, deposition of calcium phosphate crystals into the skin, hyperparathyroidism, hyperphosphatemia, and possibly increased mast cell activity and histamine secretion. In patients with an arteriovenous fistula or graft, pseudo-Kaposi's sarcoma may develop in the area of the fistula, graft, or hands due to overflow of blood or insufficient blood drainage from the area. Patients with stage 5 CKD may also experience bullous lesions and bullous dermatosis, especially male patients who have been on dialysis for long periods.[14] Uremic frost, a white, powdery substance composed of urates on the skin, is due to crystallization of urea. It is usually seen only in severely uremic patients for whom needed dialytic therapy is being withheld. These skin alterations, particularly pruritus and xerosis, may lead to localized infection from excoriation. In addition, substantial patient discomfort and psychological disturbances from skin disfigurement may occur.

Collaborative management for skin alterations includes phosphate regulation, active vitamin D administration, correction of anemia, antihistamine medications, and meticulous skin care and turning to prevent skin breakdown. Dialysis therapy helps as well by removing metabolic waste products. However, because of potential allergies to the dialysis system components, dialysis therapy can also aggravate some conditions, such as pruritus. Patient education should include information on factors contributing to skin alterations, the importance of keeping the skin clean and well moisturized, and ways to avoid excoriation (such as keeping the fingernails trimmed).

Managing Alterations in Dietary Intake

The goals of nutritional therapy in renal failure are to minimize uremic symptoms; reduce the incidence of fluid, electrolyte, and acid–base imbalances; minimize symptoms of anemia; decrease the patient's vulnerability to infections; and limit catabolism. Dietary restrictions related to managing comorbid conditions and reducing cardiovascular risk also need to be considered. Because of the complexity of achieving a nutritional therapy plan that meets these goals, a collaborative health care team approach, including the ongoing participation of a dietitian, is essential. This is particularly the case in critical care, where patients usually are in a catabolic state and are at risk for substantial malnutrition.

Renal diet prescriptions include restrictions in fluid, sodium, potassium, and phosphate intake and may include supplementations of iron, vitamins, and calcium. Calorically, critically ill patients with renal disease need a high-calorie diet with a total of 35 to 45 kcal/kg/d, most of which should come from a combination of carbohydrates and lipids. In addition, adequate protein intake must be administered to prevent catabolism, and at least 50% of protein intake should be of high biological value to ensure that the minimal intake requirements of essential amino acids are met. Protein restriction to decrease symptoms of uremia and slow the progression of renal failure is controversial (refer to the section on preventing the progression of CKD) but may be beneficial. However, protein restriction should never compromise meeting anabolic goals, which would expose the patient to the risk for malnutrition. For patients with CKD, the K/DOQI guidelines recommend a moderate protein restriction of 0.6 to 0.8 g/kg/d in patients not yet receiving dialysis and from 1.2 to 1.3 g/kg/d in patients on dialysis.[55] In critically ill patients, parenteral nutrition may need to be instituted because of impaired bowel function or severe malnutrition. In oliguric patients, the high hourly volume requirements needed for parenteral nutrition often must be offset by dialysis or isolated ultrafiltration.

To determine the effectiveness of nutritional therapy, continual laboratory monitoring of serum protein, cholesterol, albumin and prealbumin, electrolytes, hemoglobin, hematocrit, and urea and creatinine levels is essential. Patient weight, volume status, and energy levels are additional monitoring parameters. Nutritional education, including information on dietary restrictions, the use and timing of phosphate binders, vitamin and mineral supplements, and measures of nutritional status should be provided.

Managing Alterations in Psychosocial Functioning

Patients in AKI and CKD often experience feelings of fear, anxiety, and powerlessness. In addition, patients frequently have an alteration in self-concept as well as body image disturbances because of both physical and functional changes that occur in renal failure. Patients and their families may have difficulty coping owing to stress, limited resources or support, inadequate or ineffective coping mechanisms, interruptions in usual family roles, or a combination of these factors. It is important that the health care team attend to these and other psychosocial complications of renal failure to treat the patient and family holistically. Specific interventions include thorough patient and family teaching, active involvement of the patient and family members in managing the condition, ensuring adequate rest and sleep to the patient, exploring the patient's and family's feelings and concerns, providing support, and obtaining the active involvement of social services and clergy as appropriate.

▲ Clinical Applicability Challenges

CASE STUDY

A 56-year-old white female was admitted to the ICU following a pelvic exenteration at 10 AM. She presented 3 months prior to admission with early satiety and abdominal bloating. Evaluation revealed endometrial cancer and after failed radiation therapy, the patient elected for surgery. Her operative course was prolonged, and there was an estimated blood loss of 4.5 L.

Postoperatively, in the ICU, she remained intubated with an initial BP of 136/86, pulse rate of 120, and respiration rate of 26, and she was afebrile. Initial fluid administration included 2 units of packed red blood cells and lactated Ringer's solution at 125 mL/min.

In the first 6 hours after surgery, her urine output was 20 mL/h. BP remained in the 130/80s and the patient remained tachycardic. IV fluids were increased to 150 mL/h, and an additional 6 hours passed with no increase in urine output. The attending physician ordered Lasix (furosemide) 40 mg IV, which did not improve urinary output.

Midnight laboratory results revealed the following values: sodium 138, potassium 5.3, chloride 105, CO_2 28, BUN 46, and creatinine 2.7. Of note, the patient's preoperative BUN and creatinine values were 23 and 1.1, respectively. An ABG test result showed a pH of 7.47/42/98 (28) while the patient was receiving oxygen therapy (40% of O_2).

Evaluation of her AKI included a renal ultrasound finding of two normal-sized kidneys of normal echotexture without evidence of hydronephrosis.

Urinalysis prior to diuretic administration showed urinary sodium of 42 mg/dL, fractional excretion of sodium of 2.6, and fractional excretion of urea of 66. Analysis of urine obtained from a Foley catheter showed too-numerous-to-count red blood cells, 1+ protein, and muddy brown casts.

1. What is the most likely etiology for this patient's AKI?
2. What special concerns would you have about medication administration in this patient?
3. What electrolyte and acid–base abnormalities may be expected in this patient on subsequent laboratory tests?

References

1. Hoste E, Schurgers M: Epidemiology of acute kidney injury: How big is the problem? Crit Care Med 36(4):S146–S151, 2008
2. Lafrance J-P, Miller D: Acute kidney injury associates with increased long-term mortality. J Am Soc Nephrol 21(2):345–352, 2010
3. Lewington A, Sayed A: Acute kidney injury: how do we define it? Ann Clin Biochem 47(1):4–7, 2010
4. Coca S, Yusuf B, Shlipak M, et al: Long-term risk of mortality and other adverse outcomes after acute kidney injury: a systemic review and meta-analysis. Am J Kidney Dis 53(6):961–973, 2009
5. Goldberg R, Dennen P: Long-term outcomes of acute kidney injury. Adv Chronic Kidney Dis 15(3):297–307, 2008
6. Morgera S, Schneider M, Neumayer H: Long-term outcomes after acute kidney injury. Crit Care Med 36(4):S193–S197, 2008
7. Srisawat N, Hoste E, Kellum J: Modern classification of acute kidney injury. Blood Purif 29(3):300–307, 2010
8. Bellomo R, Ronco C, Kellum J, et al: Acute renal failure: Definition, outcome measures, animal models, fluid therapy and information technology needs. The Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group. Crit Care 8(4): R204–R212, 2004
9. Mehta R, Kellum J, Shah S, et al: Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. Crit Care 11:R31, 2007
10. Lattanzio M, Nelson P, Kopyt D: Acute kidney injury: New concepts in definition, diagnosis, pathophysiology, and treatment. J Am Osteopath Assoc 109 (1):13–19, 2009
11. Payen D, de Pont AC, Sakr Y, et al: A positive fluid balance is associated with a worse outcome in patients with acute renal failure. Crit Care 12(3):R74, 2008
12. Lameire N: The pathophysiology of acute renal failure. Crit Care Clin 21(2):197–210, 2005
13. Pannu N, Nadim M: An overview of drug-induced acute kidney injury. Crit Care Med 36(4):s216–s223, 2008
14. Counts C (ed): Core Curriculum for Nephrology Nursing, 5th ed. Pitman, NJ: AJ Jannetti, 2008.
15. Prescott W, Nagel J: Extended-interval once-daily dosing of aminoglycosides in adult and pediatric patients with cystic fibrosis. Pharmacotherapy 30(1):95–108, 2010
16. Taber S, Pasko D: The epidemiology of drug-induced disorders: the kidney. Expert Opin Drug Saf 7(6):679–690, 2008
17. McCullough P: Contrast-induced acute kidney injury. J Am Coll Cardiol 51(15):1419–1428, 2008
18. Caixeta A, Mehran R: Evidence-based management of patients undergoing PCI: Contrast-induced acute kidney injury. Catheter Cardiovas Interv 75:(Supp 1):S15–S20, 2010
19. Brar S, Hiremath S, Dangas G, et al: Sodium bicarbonate for the prevention of contrast-induced acute kidney injury: A systemic review and meta-analysis. Clin J Am Soc Nephrol 4(10):1584–1592, 2009
20. Hoste E, De Waele J, Gevaert S, et al: Sodium bicarbonate for prevention of contrast-induced acute kidney injury: A systemic review and meta-analysis. Nephrol Dial Transplant 25(3):747–758, 2010
21. Perazella M: Current status of gadolinium toxicity in patients with kidney disease. Clin J Am Soc Nephrol 4(2):461–469, 2009
22. Mayr M, Burkhalter F, Bongartz G: Nephrogenic systemic fibrosis: Clinical spectrum of disease. J Magn Reson Imaging 30:1289–1297, 2009
23. Leiner T, Kucharczyk W: NSF prevention in clinical practice: Summary of recommendations and guidelines in the United States, Canada, and Europe. J Magn Reson Imaging 30:1357–1363, 2009
24. Abdel K, Palevsky P: Acute kidney injury in the elderly. Clin Geriatr Med 25(3):331–358, 2009

25. Diskin C, Stokes T, Dansby L, et al: The comparative benefits of the fractional excretion of urea and sodium in various azotemic oliguric states. Nephron Clin Pract 114(2):C145–C150, 2010
26. Hawkins I, Cho K, Caridi J: Carbon dioxide in angiography to reduce the risk of contrast-induced nephropathy. Radiol Clin North Am 47(5):813–825, 2009
27. U.S. Renal Data System: USRDS 2009 Annual Data Report: Atlas of End-Stage Renal Disease in the United States, National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases, Bethesda, MD, 2009
28. National Kidney Foundation: K/DOQI clinical practice guidelines for chronic kidney disease: Evaluation, classification, and stratification. Am J Kidney Dis 39(2 Suppl 1):S1–S266, 2002
29. Ripley E: Complementary effects of angiotensin-converting enzyme inhibitors and angiotensin receptor blockers in slowing the progression of chronic kidney disease. Am Heart J 157(6 Suppl):S7–S16, 2009
30. Nguyen T, Toto R: Slowing chronic kidney disease progression: results of prospective clinical trials in adults. Pediatr Nephrol 23(9):1409–1422, 2008
31. James M, Hemmelgarn B, Tonelli M: Early recognition and prevention of chronic kidney disease. Lancet 375(9722):1296–1309, 2010
32. Jacobs C, Opolinsky D: The Little Handbook of Dialysis. Boston, MA: Jones & Bartlett, 2010
33. Coresh J, Astor B, Greene T, et al: Prevalence of chronic kidney disease and decreased kidney function in the adult US population: Third National Health and Nutrition Examination Survey. Am J Kidney Dis 41(1):1–12, 2003
34. Coresh J, Byrd-Holt D, Astor B, et al: Chronic kidney disease awareness, prevalence, and trends among U.S. adults, 1999–2000. J Am Soc Nephrol 16(1):180–188, 2005
35. Macconi D: Targeting the renin-angiotensin system for remission/regression of chronic kidney disease. Histol Histopathol 25:655–668, 2010
36. American Diabetes Association Standards of medical care in diabetes—2010: Diabetes Care 33(Suppl 1):s11–s61, 2010
37. Diabetes Control and Complications Trial Research Group: The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. N Engl J Med 329(14):977–986, 1993
38. Sustained effect of intensive treatment of type I diabetes mellitus on development and progression of diabetic nephropathy: The Epidemiology of Diabetes Interventions and Complications (EDIC) study. JAMA 290(16):2159–2167, 2003
39. UK Prospective Diabetes Study (UKPDS) Group: Intensive blood-glucose control with sulphonylureas of insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). Lancet 352(9131):837–853, 1998
40. Appel L, Wright J, Greene T, et al: Long-term effects of rennin-angiotensin system—Blocking therapy and a low blood pressure goal on progression of hypertensive chronic kidney disease in African American. Arch Intern Med 168(8):832–839, 2008
41. National Kidney Foundation: K/DOQI clinical practice guidelines on blood pressure management and use of antihypertensive agents in chronic kidney disease. Am J Kidney Dis 43(Suppl 1):S1–S268, 2004
42. Grone E, Grone H: Does hyperlipidemia injure the kidney? Nat Clin Pract Nephrol 4(8):424–425, 2008
43. Fried L: Effects of HMG-CoA reductase inhibitors (statins) on progression of kidney disease. Kidney Int 74(5):571–576, 2008
44. Karajala V, Mansour W, Kellum J: Diuretics in acute kidney injury. Minerva Anestesiol 75(5):251–257, 2009
45. Venkataraman R: Can we prevent acute kidney injury? Crit Care Med 36(4 Suppl):S166–S171, 2008
46. Bellomo R, Wan L, May C: Vasoactive drugs and acute kidney injury. Crit Care Med 36(4 Suppl):S179–S186, 2008
47. Brosnahan G, Fraer M: Chronic kidney disease: Whom to screen and how to treat, part 1: Definition, epidemiology, and laboratory testing. South Med J 103(2):140–146, 2010
48. Rodriquez-Iturbe B, Correa-Rotter R: Cardiovascular risk factors and prevention of cardiovascular disease in patients with chronic renal disease. Expert Opin Pharmacother 11(Suppl 1):S1–S12, 2010
49. National Kidney Foundation: K/DOQI clinical practice guidelines on blood pressure management and use of antihypertensive agents in chronic kidney disease. Am J Kidney Dis 43(Suppl 1):S1–S268, 2004
50. Chobanian A, Bakris G, Black H, et al: National Heart, Lung and Blood Institute Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure; National High Blood Pressure Education Program Coordinating Committee. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: The JNC 7 report. JAMA 289(19):2560–2572, 2003
51. National Kidney Foundation: K/DOQI clinical practice guidelines on managing dyslipidemias in chronic kidney disease. Am J Kidney Dis 41(Suppl 3):S1–S77, 2003
52. National Kidney Foundation: K/DOQI clinical practice guidelines and clinical practice recommendations for anemia in chronic kidney disease in adults. Am J Kidney Dis 47(5 Suppl 3):S16–S85, 2006
53. National Kidney Foundation: K/DOQI clinical practice guidelines for bone metabolism and disease in chronic kidney disease. Am J Kidney Dis 42(Suppl 3):S1–S210, 2003
54. Drueke T: Cinacalcet treatment in dialysis patients with secondary hyperparathyroidism: Effects and open issues. Ther Apher Dial 12(1):S2–S12, 2008
55. National Kidney Foundation: K/DOQI clinical practice guidelines for nutrition in chronic renal failure. Am J Kidney Dis 35(6 Suppl 2): S1–S140, 2000

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

PART 8

NERVOUS SYSTEM

32

Anatomy and Physiology of the Nervous System

Mary Ciechanowski, Donna Mower-Wade, and Sandra W. McLeskey

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Describe the cellular units of the nervous system.
2. Explain the characteristics of neurons.
3. Describe the components of the central nervous system.
5. List areas of the cerebrum and their corresponding function.
6. Explain the functions of the thalamus.
7. Define the reticular activating system.
8. Briefly define the sensory system and the motor system.
9. Explain the baroreceptor reflex, and list three spinal cord reflexes.
10. Explain the anatomy and physiology of pain.
11. Explain the concept of homeostasis.
12. Describe the acute stress response.
13. Discuss why the stress response could be helpful or harmful, depending on the situation.

The brain is a central organ that coordinates activity of most, if not all, body systems through its influence on the endocrine and immune systems as well as its more generally appreciated influence on skeletal muscle and autonomic function. Its influence is modulated by sensory perceptions that convey a picture of the external and internal environments and also by internal circuits having to do with emotional state and levels of arousal. Therefore, the brain can be thought of as the integrative organ that drives our responses to environmental influence. Moreover, separation of the brain and spinal cord from the periphery is more anatomical than conceptual, and modern nurses must always keep in mind that the brain has a profound influence on almost everything that happens in the periphery, and vice versa.

Traditionally, the nervous system is discussed with reference to both anatomical and functional divisions. Anatomical components are the central nervous system (CNS), comprising the brain and spinal cord, and the peripheral nervous system (PNS), comprising the cranial and spinal nerves. The nervous system is functionally separated into the sensory, integrative, and motor (somatic and autonomic) divisions. Content in this chapter is ordered according to both divisions. However, cell anatomy and physiology are discussed first.

▲ Cells of the Nervous System

The cellular units are the neuron—the basic functional unit—and its attendant cells, the neuroglia.

Neuroglia

Neuroglia constitute the supportive tissue associated with the neurons. In the CNS, there are four types of neuroglia: microglia, astrocytes, ependymal cells, and oligodendroglia. The microglia are phagocytic cells of the nervous system similar to macrophages in the periphery. The astrocytes are supportive cells of the nervous system and make up the blood–brain barrier. The ependymal cells line the ventricles and aid in the production and circulation of the cerebrospinal fluid (CSF). The oligodendroglia are mostly found in the white matter and produce the myelin that covers nerve fibers in the CNS. In the PNS, the counterpart of the myelin-producing oligodendroglial cell is the Schwann cell.

Under most circumstances, neurons lose their ability to undergo mitosis early in the life of the individual. However, neuroglia retain mitotic abilities throughout a person's life span. Because of this, malignant or benign proliferative lesions originating in the CNS involve neuroglia rather than neurons. However, as the neuroglial tumor enlarges, it adversely affects adjacent neurons—early by exerting pressure and later by promoting an inflammatory reaction along with the pressure.

Neurons

The basic functional unit of the nervous system is the neuron (or nerve cell), and all information and activity, whether sensory, motor, or both, is accomplished by neurons. The neuron consists of a nerve cell body or soma that contains nuclear and cytoplasmic material and processes, either axons or dendrites, that arise from the soma (Fig. 32-1). Axons normally carry nerve impulses away from the cell body, whereas dendrites conduct impulses toward the cell body. Axons and dendrites may be merely microscopic knobs or areas on the cell body surface, or they may be cylindrical processes that can extend to more than 1 m (3.25 ft) long. A specialized structure at the end of the axon is called the axon terminal. This is a bulbous ending (sometimes called a bouton) that forms a synapse with another neuron. The axon terminal contains vesicles of neurotransmitter that is released

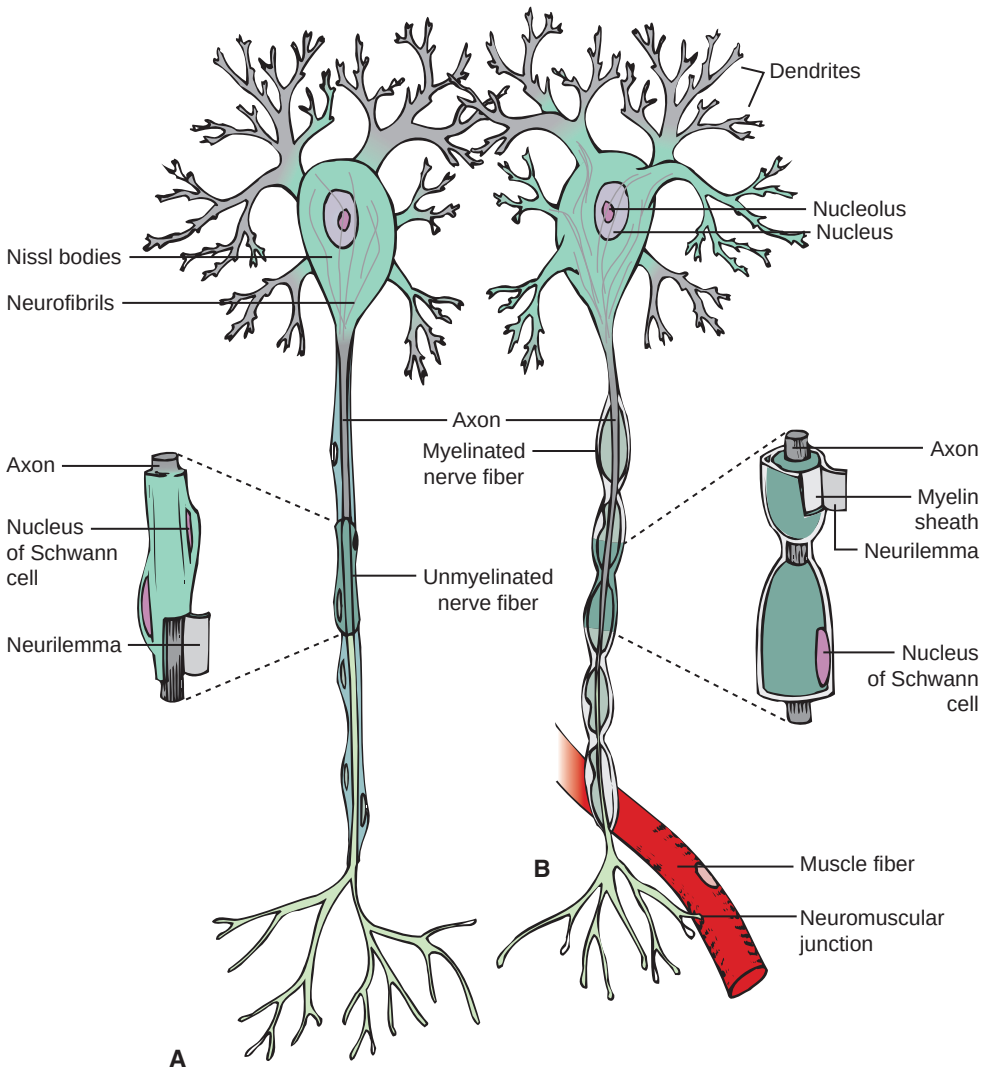


FIGURE 32-1 ▲ Typical efferent neurons. **A**: Unmyelinated fiber. **B**: Myelinated fiber.

into the synapse, diffuses across to the postsynaptic neuron, and binds to specific receptors on the membrane of the postsynaptic cell. Binding of a particular neurotransmitter to its specific receptor on the postsynaptic neuron either depolarizes or hyperpolarizes the postsynaptic neuron in the area of the synapse. Axons and dendrites are referred to collectively as nerve fibers. A bundle of nerve fibers together with their coverings is called a tract in the CNS and a nerve in the periphery.

Some nerve fibers are covered with a white lipid–protein sheath termed the myelin sheath. This covering is what differentiates white matter from gray matter in the CNS. The myelin sheath is formed in the CNS by the oligodendrocytes. Other fibers remain unmyelinated. All nerve fibers in the PNS are covered by a neurilemma. This is a sheath formed by the Schwann cells, which wrap themselves around the fiber. Some Schwann cells around particular fibers secrete myelin; others do not (see Fig. 32-1). The neurilemma of a myelinated fiber comes in contact with the axon at periodic intervals. These periodic constrictions of the neurilemmal sheath are termed the nodes of Ranvier. The nodes of Ranvier produce faster nerve impulse conduction by allowing the impulse to jump from one node to the next (saltatory conduction).

Neurons are very diverse, with many specialized anatomical features that are important to their function. Some neurons are extremely large or may give rise to extremely long nerve fibers. Transmission velocities in long, myelinated fibers may be as high as 100 m/s, whereas unmyelinated neurons with very short, unmyelinated processes demonstrate velocities of 1 m/s. Some neurons connect to many different neurons, perhaps thousands of other neurons, in a "network," whereas others have relatively few connections to other cells of the nervous system.

It is estimated that the human CNS has 12 billion neurons. Three fourths of these neurons are located in the cerebral cortex, where conscious thought and feeling reside, along with integrative processing and smoothing of planned motor movements. This processing includes not only the determination of appropriate and effective responses but also the storage of memory and the development of associative motor and thought patterns.

▲ Characteristics of Neurons

Resting Membrane Potential

As is true of all cells, the neuronal cell membrane contains sodium–potassium pumps that keep the inside of the neuron more negatively charged than the outside interstitial fluid. The cytoplasm of all cells contains anions (negatively charged ions) that are too large to leave the cell. Many ions, including sodium, potassium, and chloride, are small enough to diffuse through tiny pores in the cell membrane. If it were not for the sodium–potassium pump, the concentrations of these ions would be equal on the inside of the cell compared with the outside. However, the sodium–potassium pump in the cell membrane pumps sodium ions out of the cell almost as fast as they enter. For every two sodium ions that are pumped out, one potassium ion is pumped into the cell. Because of this, there is a net positive charge leaving the cell, and the large anions cannot be counterbalanced. Thus, under resting conditions when no impulse is being conducted, the inside of the neuron is negative with respect to the outside. This internal relative negativity is the resting membrane potential of the neuron and typically measures about −85 mV.

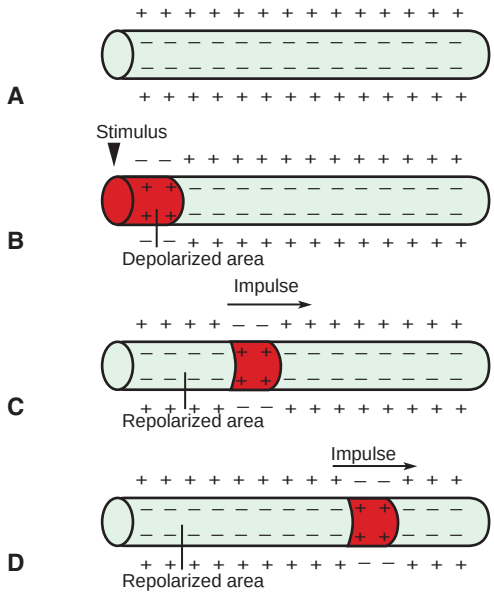


FIGURE 32-2 ▲ Propagation of impulses. **A**: Resting membrane. **B**: Action potential, first stage: stimulation of fiber results in depolarization. **C**: Action potential, second stage: repolarization occurs as the resting potential is restored. **D**: Propagation of impulses continues in direction of *arrow*.

In addition, as a result of activity of the sodium–potassium pump, sodium ion concentration inside the cell is much lower than outside, and potassium ion concentration is much higher inside the cell than outside. These concentration gradients are important for depolarization produced by synaptic transmission and also for conduction of the action potential down the axon (Fig. 32-2).

Synaptic Transmission

Submicroscopic spaces between the axon (or axons) of one neuron and the dendrite (or dendrites) or soma of another are called synapses. Axons or dendrites may branch, enabling the axon of one neuron to synapse with dendrites or somas of several neurons. A synapse consists of a presynaptic axon terminal, a postsynaptic neuron, and the small (150 to 1,000 Å) space between elements called a synaptic cleft (Fig. 32-3A). When an action potential is conducted down the presynaptic axon and depolarizes the axon terminal, vesicles of neurotransmitter fuse with the plasma membrane, releasing neurotransmitter into the synaptic cleft. The molecules of neurotransmitter diffuse across the cleft and bind to specific receptors on the postsynaptic cell membrane. The binding of neurotransmitter to its receptor causes a change in the membrane potential of the postsynaptic cell, either a depolarization or a hyperpolarization.

In very short time (millionths of a second), the neurotransmitter detaches from the receptor site. It may then reattach or be inactivated. Inactivation occurs in two basic ways, depending on the neurotransmitter. For example, the

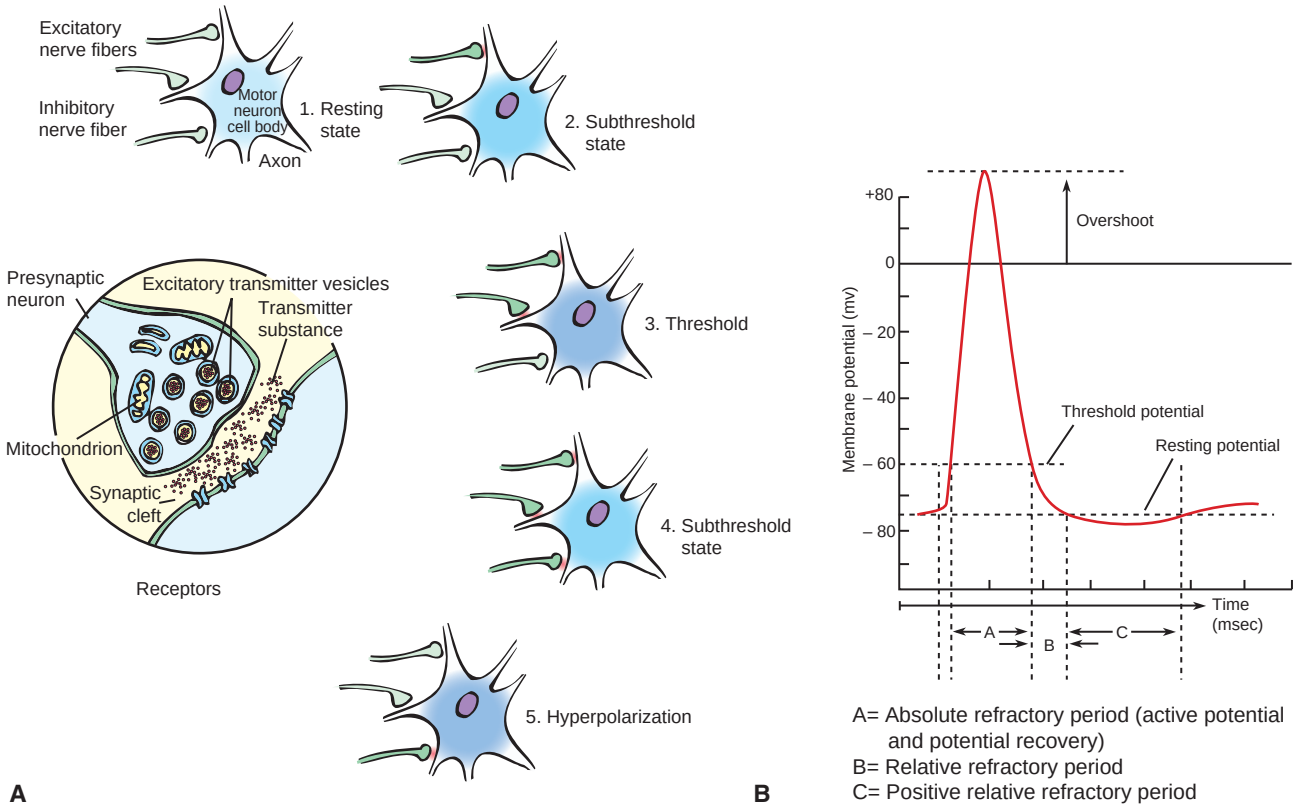


FIGURE 32-3 ▲ Conduction at synapses. **A**: A neuron may be excited or inhibited by transmitter substances liberated by presynaptic nerve fiber endings. Two excitatory fibers and one inhibitory fiber are shown. (1) During the resting state, no impulses are received. (2) During the subthreshold state, impulses from only one excitatory fiber cannot cause the postsynaptic neuron to mount an action potential. (3) The threshold is reached by the addition of impulses from a second excitatory fiber. This enables the postsynaptic neuron to mount an action potential. (4) The subthreshold state is restored by impulses from an inhibitory fiber. (5) When the inhibitory fiber alone is carrying impulses, the postsynaptic neuron is in a state of hyperpolarization and is unable to fire. **B**: The time course of a neural action potential.

catecholamine neurotransmitters and serotonin are taken back into the axon terminal by specific reuptake pumps and are repackaged in vesicles to be reused. In contrast, acetylcholine is destroyed by an enzyme, acetylcholinesterase, present in the synaptic cleft. In either case, the neurotransmitter is available to bind to its receptors on the postsynaptic membrane only for a very short time. Rapid, repetitive, discrete stimulation of neurons is necessary for activity of a neural pathway to continue. In this way, neural pathways can be stimulated over prolonged periods by repeated depolarizations of presynaptic neurons, or activity of a specific pathway can be turned on or off almost instantaneously.

Synaptic transmission is a one-way street—from the axon across the synaptic cleft to the dendrite or soma of the next neuron. It cannot proceed in the opposite direction. Moreover, decreased destruction or decreased reuptake of a transmitter can increase the effect of this transmitter on the postsynaptic membrane. Similarly, increased destruction or increased reuptake of a transmitter reduces its postsynaptic effects. Several classes of pharmacological agents take advantage of these facts. For instance, acetylcholinesterase inhibitors, such as neostigmine, are used to increase the amount of acetylcholine remaining in the synapse of the neuromuscular junction to counteract the effects of paralytic drugs given during anesthesia. Serotonin or norepinephrine reuptake pump inhibitors increase the amount of serotonin or norepinephrine in the synapse and have therapeutic effects in depressed patients.

Each neuron synthesizes and stores only one major neurotransmitter in its axon terminal. Major neurotransmitters include serotonin, acetylcholine, gamma-aminobutyric acid (GABA), glycine, glutamate, and the catecholamines, dopamine, norepinephrine, and epinephrine. Examples of neuropeptide neurotransmitters are the endogenous opioids (endorphins and enkephalins) and substance P, all of which appear to be involved in pain sensation. The endorphins and enkephalins, often described as the body's own morphine, contribute to a decrease in pain sensation. Substance P excites sensory neurons that respond to painful stimuli, so it is thought to be involved in transmission of pain information from the periphery to the CNS.

Each major neurotransmitter has multiple receptors. For instance, epinephrine can bind to α_1, α_2, β_1, and β_2 receptors, and acetylcholine can bind to neuronal nicotinic, skeletal muscle nicotinic, or muscarinic receptors, which are further subdivided into m_1, m_2, and m_3. The postsynaptic membrane of each synapse contains only one receptor type for the particular neurotransmitter synthesized by the presynaptic neuron. The receptor subtype dictates the effects of a neurotransmitter in a particular synapse on the postsynaptic cell

(hyperpolarization or depolarization). Therefore, the same neurotransmitter might be either hyperpolarizing or depolarizing to a given postsynaptic neuron, depending on the receptor subtype present on the postsynaptic cell. However, all GABA receptor types are hyperpolarizing, and GABA is the most important inhibitory neurotransmitter in the nervous system. Likewise, glutamate and glycine are always depolarizing (excitatory) neurotransmitters.

Neuronal Thresholds and the Action Potential

A depolarizing impulse that reaches a neuron's dendrites or soma through action of a neurotransmitter binding to its receptors causes the membrane to depolarize locally through action of the receptor. The local depolarization causes voltage-sensitive sodium channels locally in the membrane to open, and sodium ions are transmitted down their concentration gradient from outside the neuron to inside, causing further local depolarization. If enough sodium channels open locally, the resulting depolarization is large enough to open sodium channels in adjacent areas, depolarizing a larger area of the membrane. Conversely, release of an inhibitory neurotransmitter, such as GABA, at a synapse may cause hyperpolarization of the postsynaptic neuron through receptor action.

As mentioned, for a given nerve cell, typically there are many other neurons that synapse with its soma or dendrites. Some of these synapsing neurons release an excitatory neurotransmitter that interacts with the nerve cell's receptors to depolarize the postsynaptic neuron. Other synapsing neurons release inhibitory neurotransmitters that interact with their receptors to hyperpolarize the postsynaptic neuron. The nerve cell body algebraically sums the positive depolarizing (excitatory) and negative hyperpolarizing (inhibitory) influences. If the depolarizing influences outweigh the hyperpolarizing influences, the membrane potential of the nerve cell body may reach a value called threshold. At that point, an action potential is generated at the point where the axon leaves the soma. The action potential is propagated down the length of the axon by the process of sodium channel openings in the area of the advancing action potential, followed by complete depolarization of that area. After this process, sodium channels close, and the membrane in that area can repolarize through activity of the sodium–potassium pump and through opening of voltage-sensitive potassium channels. As potassium accumulates within the cell, either by entry through the voltage-sensitive potassium channels or the sodium–potassium pump, the membrane potential is reestablished.

The action potential is normally propagated down the entire length of the axon to the axon terminal. At that point, depolarization of the axon terminal causes neurotransmitter to be released, which diffuses across the synapse and binds to specific receptors, causing a depolarizing or hyperpolarizing change in the postsynaptic neuron.

Neuronal activity can be influenced by hormones. For example, thyroxine lowers thresholds of certain neurons, and one sign of hyperthyroidism is the presence of exaggerated spinal reflexes, such as the knee jerk and ankle jerk.

Figure 32-3B depicts the time course of a neuronal action potential as monitored by electrodes inserted into an axon. Compared with cardiac action potentials, neuronal action potentials are quite short, with durations of approximately 5 to 15 ms. Like the cardiac action potential, there are absolute and relative refractory periods during which the neuron cannot easily be reexcited. However, these refractory periods are very short because repeated impulse conduction is necessary to maintain tonic activity of particular neural pathways. For instance, the motor pathways that supply muscles of posture must be tonically active to maintain a steady contraction in these muscles that keep us erect. Other pathways that have tonic activity include the autonomic motor pathways (sympathetic and parasympathetic), as discussed later in this chapter.

The electrical activity of the action potential can be monitored in certain clinical situations. For example, the electroencephalogram depicts multiple action potentials from surface neurons of the brain. Nerve conduction studies can be done on peripheral nerves to diagnose areas of compression or entrapment, which slow action potentials.

Remodeling of Connections in the Nervous System

Although neurons in adults, and all but the youngest children, do not divide to form more neurons, and in fact neurons die throughout our life span, our CNS is in a constant state of remodeling (often referred to as *plasticity*), with formation of new connections between neurons (synapses) and regression of previous connections. This is an area of continuing research, but evidence suggests that sensory information that is communicated to the brain as a result of our experiences is responsible for the remodeling. These alterations in brain circuitry may explain phenomena, such as emotional maturation and motor learning. They may also explain the development of mental illness at certain times in a person's life or the intense cravings that appear with substance abuse disorders.

▲ Nerve Regeneration

If a nerve fiber is severed, the portion distal to the cut dies, and the part still attached to the cell body regenerates. In peripheral neurons, the neurilemma itself provides a channel that can be followed by a regenerating fiber so that it may become reattached to its original anatomical connection (Fig. 32-4). Regeneration also occurs in the absence of a neurilemma, as in the case of CNS neurons. Because there is no channel to ensure correct anatomical reconnection, most such regenerations do not produce recovered function. The regrowing stump may wind aimlessly among other structures or curl into a useless tangle. However, a bigger hindrance to functional regeneration in the CNS has been discovered—an overgrowth of neuroglial cells that occurs in response to injury. This produces a neuroglial thicket that acts as a barrier to the reconnection of severed neuronal networks.

Note that nerve processes, axons, or dendrites that are cut by an injury may regenerate, but if the soma is damaged by injury or the nerve cell is killed by lack of oxygen or a neurotoxin, there will be no regeneration. Moreover, under ordinary circumstances, a neuron that dies cannot be replaced because neurons do not normally undergo mitosis in people older than age 2.

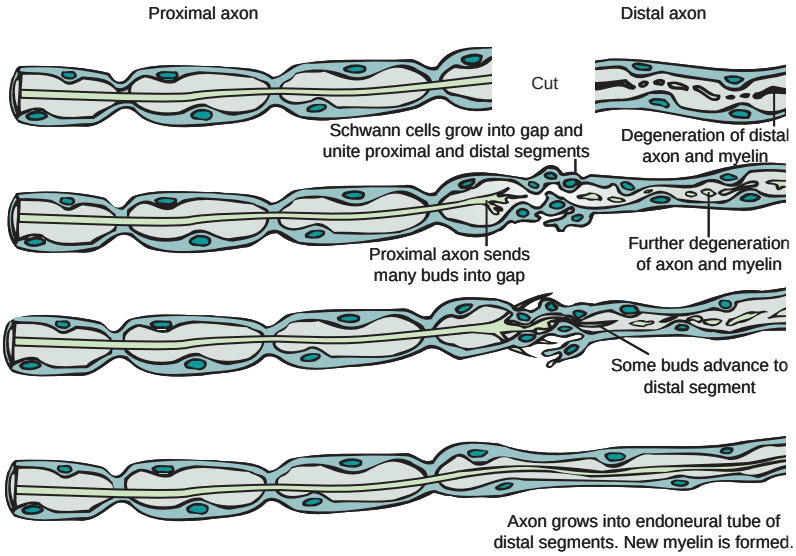


FIGURE 32-4 ▲ Diagram of changes that occur in a nerve fiber that has been cut and then regenerates.

▲ Central Nervous System

The CNS comprises the brain and spinal cord. It receives sensory input through sensory neurons whose dendrites run within spinal and cranial nerves, and it sends out motor impulses through axons of motor neurons that run in these same nerves. The CNS also contains large numbers of neurons that are entirely contained within it. These neurons are termed internuncial neurons, or interneurons, and exist inside the brain or the spinal cord or connect one with the other.

Skull

The skull (cranium) is one of the hardest bones of the body and, along with the facial bones, functions to protect the brain from traumatic injury (Fig. 32-5). The facial bones help protect the brain from injury by absorbing some of the traumatic forces. The skull, which surrounds the soft structures of the brain, is composed of eight bones fused together at suture lines. The bones that compose the main part of the skull are the frontal, parietal, temporal, and occipital bones, and these bones are joined together during early childhood (see Fig. 32-5).

Meninges

The CNS, including the spinal cord, is covered by three layers of tissue collectively called the meninges (Fig. 32-6). The pia mater is the layer that lies next to the CNS. Next is the arachnoid layer, which contains a substantial vascular supply. The last layer is the dura mater, the thickest layer of all, lying next to the bones surrounding the CNS. Between the pia and arachnoid layers lies the subarachnoid space. CSF

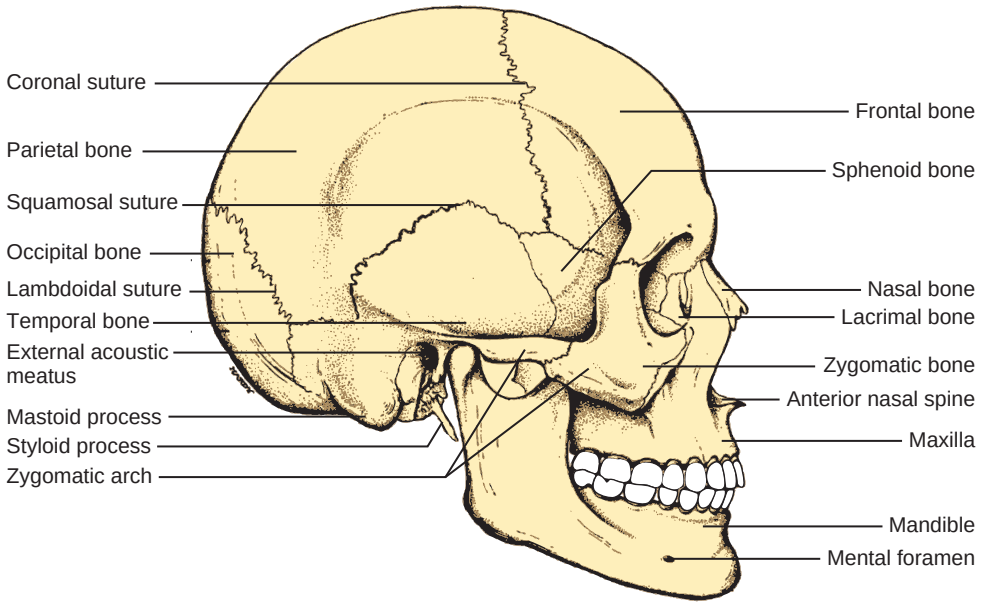


FIGURE 32-5 ▲ Lateral view of the skull. (From Hickey JV: The Clinical Practice of Neurological and Neurosurgical Nursing, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 47.)

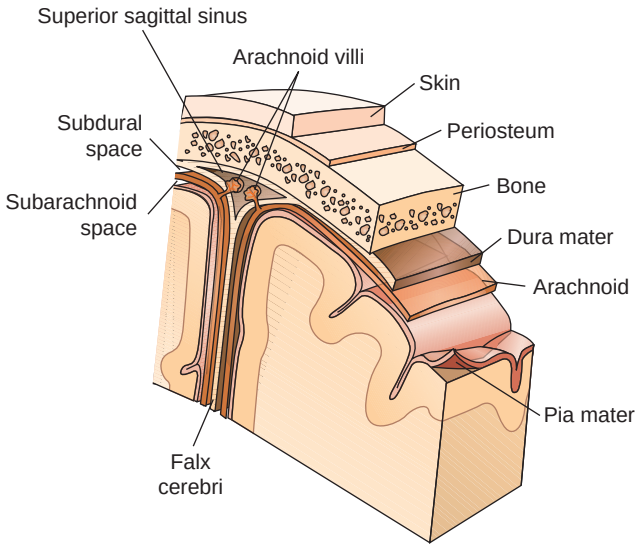


FIGURE 32-6 ▲ The cranial meninges. The arachnoid villi, shown within the superior sagittal sinus, are one site of passage of cerebrospinal fluid (CSF) into the blood. (From Porth CM: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Wolters Kluwer Health | Lippincott Williams & Wilkins, 2009, p 1211.)

circulates through the subarachnoid space. In addition, the subarachnoid space contains the cerebral vasculature. When a cerebral vascular abnormality rupture occurs, it bleeds into the subarachnoid space, causing a subarachnoid hemorrhage. The space between the arachnoid and dura mater is named the subdural space. The space between the dura mater and skull bone is known as the epidural space. The epidural space that surrounds the spinal cord is used for epidural pain management.

The CNS is richly supplied with blood vessels that bring oxygen and nutrients to the cells. However, many substances cannot easily be exchanged between the blood and the brain because the endothelial cells of the vessels and the astrocytes of the CNS form extremely tight junctions collectively referred to as the blood–brain barrier. In particular, polar molecules and large molecules, such as proteins, do not cross the blood–brain barrier, but lipid-soluble molecules cross with ease. Many drugs penetrate the brain poorly because they do not have sufficient lipid solubility to cross the blood–brain barrier.

The space between the arachnoid layer and the pia mater, termed the subarachnoid space, contains CSF, which is another means of supplying nutrients, but not oxygen, to the CNS. CSF also serves a protective function by cushioning the brain and spinal cord.

Cerebrospinal Fluid

CSF, a clear, colorless fluid, flows in the ventricles of the brain and the subarachnoid space of the brain and spinal cord. Functioning as a fluid shock absorber, CSF keeps the delicate CNS tissues from being mechanically injured by surrounding bony structures. CSF is actually a plasma filtrate that is exuded by the capillaries in the roofs of each of the four ventricles of the brain. As such, it is similar to plasma without the large plasma proteins, which stay behind in the bloodstream. Red blood cells, which contain the hemoglobin that is responsible for most oxygen transport in blood, are not present in CSF. Therefore, the CSF is a poor source of oxygen, although it contains glucose, amino acids, and other nutrients that might be needed by the cells of the CNS.

Most of the CSF is made in the lateral ventricles, which are located in each cerebral hemisphere. The choroid plexus produces approximately 500 mL of CSF each day, or 25 mL/h. The CSF moves from there through the ducts into the third ventricle of the diencephalon (Fig. 32-7). From there, it travels through the aqueduct of Sylvius of the midbrain and enters the fourth ventricle in the medulla oblongata. Most of it then passes through holes (foramina) in the roof of this ventricle and enters the subarachnoid space. A small amount diffuses down into the spinal canal. In the subarachnoid space, the CSF is reabsorbed into the bloodstream at certain structures called the arachnoid villi.

The formation and reabsorption of CSF is governed by the same hydrostatic and colloid osmotic forces that regulate the movements of fluid and small molecules between the plasma and interstitial fluid compartments. Briefly reviewed, the action of these forces is as follows. Two opposing push–pull forces influence the movement of water and small molecules through the semipermeable capillary membranes. One force is composed of plasma oncotic pressure and CSF hydrostatic pressure. It favors movement of water and small molecules from the CSF compartment into the plasma. The movement of water and small molecules in the opposite direction is influenced by the force of plasma hydrostatic pressure and CSF oncotic pressure. These two opposing forces are exerted simultaneously and continually. In the lateral ventricles, the flow of CSF out of the ventricles reduces CSF hydrostatic pressure. This tips the influence in favor of the movement

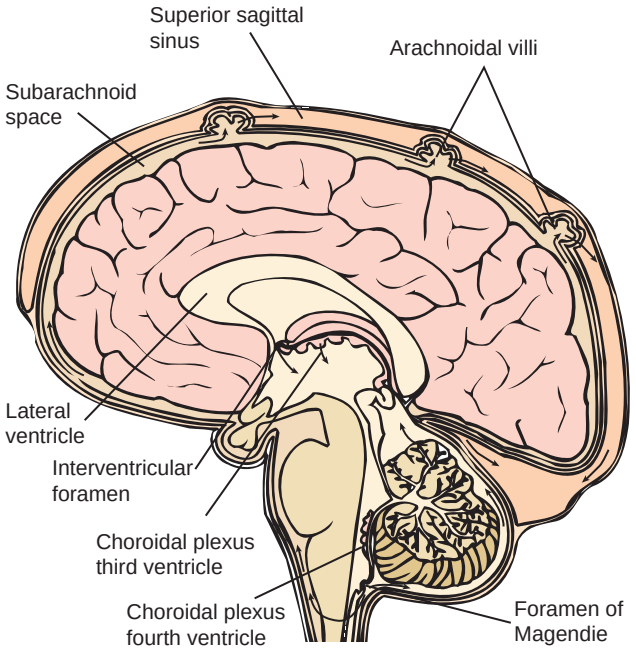


FIGURE 32-7 ▲ Diagram of the flow of CSF from the time of its formation from blood in the choroid plexuses until its return to the blood in the superior sagittal sinus. (Adapted from Hickey JV: The Clinical Practice of Neurological and Neurosurgical Nursing, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 52.)

of water and small molecules from plasma to the ventricles. The low plasma hydrostatic pressure of blood in the venous sinuses next to the arachnoid villi tips the scales in favor of the movement of water and solute from the CSF compartment back into the bloodstream. These forces are modulated by death of cells lining the CSF compartment, which releases proteins into the CSF. This elevates CSF oncotic pressure and retards reabsorption (while also hastening CSF formation if the damage is in ventricle walls). Increased CSF proteins from this or other causes can provoke or exacerbate a condition of excess CSF called hydrocephalus. If at any time the arachnoid villi become blocked or the flow of CSF is otherwise disrupted, hydrocephalus occurs.

Because the CSF is formed in the ventricles and must travel to the arachnoid to be reabsorbed, any impediment to its flow impairs its absorption. The aqueduct of Sylvius or the foramina in the roof of the fourth ventricle may become clogged by adhesions from an infection (meningitis), clots from a subarachnoid hemorrhage, a tumor, or a congenital abnormality. This produces an obstructive hydrocephalus with increased pressure in the CSF. Communicating hydrocephalus, caused by infection or subarachnoid hemorrhage, occurs when CSF cannot be reabsorbed by the arachnoid villi.

Cerebral Vasculature

Because the brain requires a continuous supply of oxygen and glucose to survive, it receives about 20% of the cardiac output at a rate of approximately 750 mL/min. Two major sets of vessels supply the brain with blood: the two internal carotid arteries and the two vertebral arteries. The left common carotid artery arises from the aortic arch, and the right common carotid artery is formed from the bifurcation of the short brachiocephalic trunk, which arises from the aorta. Each carotid artery bifurcates to form the internal and external carotid arteries. The internal carotid arteries supply most of the cerebrum and upper portion of the diencephalon, and the external carotid artery supplies the face and scalp. The vertebral arteries arise from the subclavian arteries. After entering the foramen magnum, the vertebral arteries join to form the basilar artery, which sends branches to the cerebellum, brainstem, and posterior diencephalon. The basilar artery joins the circle of Willis by bifurcating to form the two posterior cerebral arteries.

The circle of Willis, located in the subarachnoid space, is the area in which the branches of the basilar and internal carotid arteries unite (Fig. 32-8). This area is composed of the two anterior cerebral arteries, the anterior communicating artery, the two posterior cerebral arteries, and the two posterior communicating arteries. These arteries send branches to supply the various lobes of the cerebral cortex. This circular network permits blood to circulate from one hemisphere to the other and from the anterior to the posterior areas of the brain. The anterior circulation consists of the middle and anterior cerebral arteries and one anterior communicating cerebral artery. The posterior circulation consists of posterior and posterior communicating cerebral arteries and one basilar artery. This system allows for collateral circulation if one vessel is occluded.

It is not unusual for a vessel in the circle of Willis to be atrophic or even absent. This accounts for different clinical presentations among patients with the same lesion. For example, a person with an occluded carotid artery and a fully patent circle of Willis may be totally asymptomatic, but a patient in whom the circle of Willis is incomplete may demonstrate a massive cerebral infarction.

Brain

The basic anatomy of the brain is illustrated in Figure 32-9. The parts of the brain, in descending order, are the cerebral

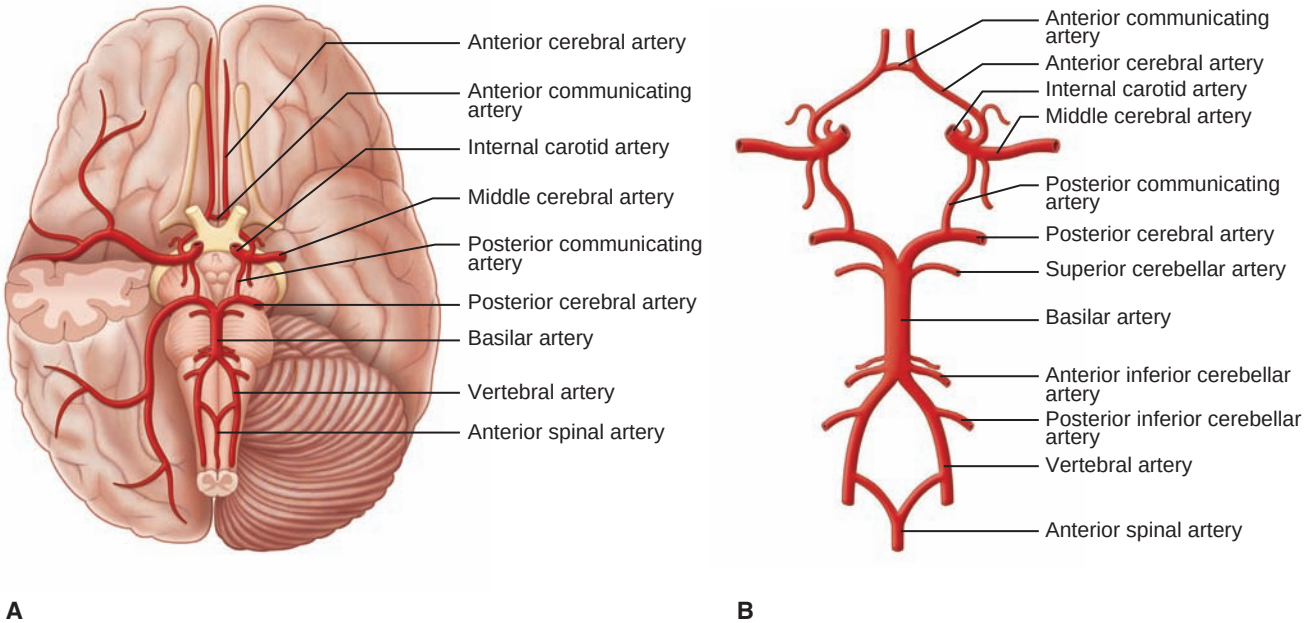


FIGURE 32-8 ▲ Circle of Willis (arterial blood supply to the brain). **A**: The circle of Willis seen from below the brain. **B**: Schematic of the circle of Willis.

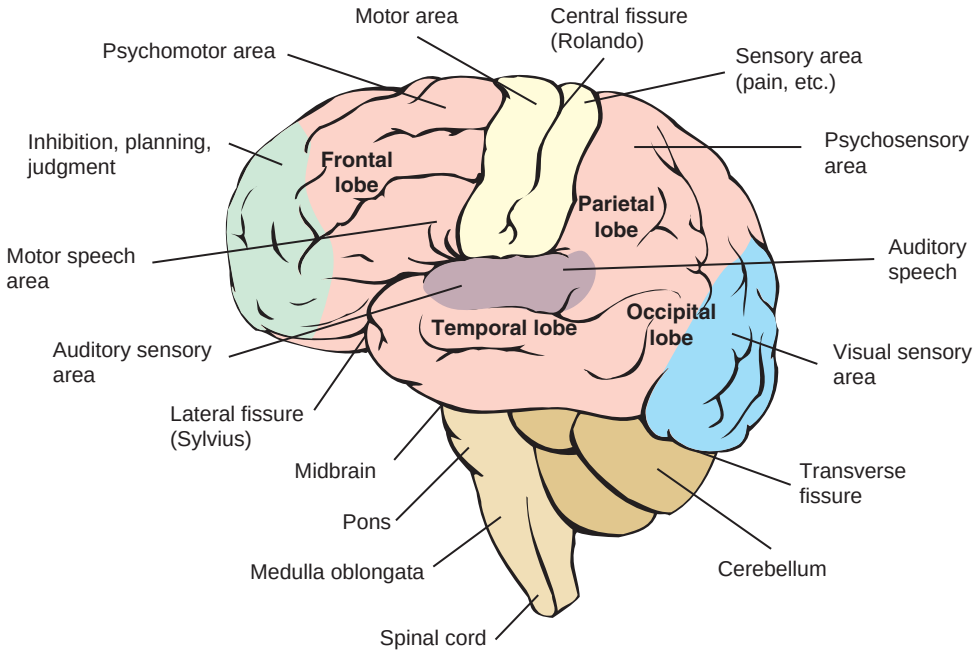


FIGURE 32-9 ▲ The human brain, showing the lobes and fissures of the cerebrum. Major functional areas are also indicated. The cortex is comprised of the frontal lobe, the parietal lobe, the temporal lobe, and the occipital lobe.

hemispheres (the cerebrum), diencephalon, midbrain, pons varolii (usually called the pons), medulla oblongata (usually called the medulla), and cerebellum.[4] The general appearance of the brain can be viewed as a stem extending upward from the spinal cord with an inferior small flowering overgrowth (cerebellum) covering the lower part of the stem and a large superior flowering overgrowth (cerebrum) covering most of the upper portion of the stem. The medulla, pons, and midbrain compose the brainstem.

Cerebrum

Each of the two cerebral hemispheres (left and right) has a layer of cortex covering the surface. This cortical layer consists of several different types of neurons with accompanying neuroglia arranged in six distinctive layers according to cell type and function. Some of these neurons project myelinated axons deeper into the cortex with ultimate destinations lower in the CNS or in the opposite cortex. Areas of myelinated axons appear as white matter. Gray matter is made up of nerve cell bodies that are vascular in nature and have a gray appearance. Deep within each hemisphere is a lateral ventricle containing CSF, along with several collections of nerve cell bodies, termed the basal ganglia. The left and right hemispheres are connected and communicate with each other by a transverse band of white matter termed the corpus callosum, formed by myelinated axons traveling between each side of the cortex. For the most part, each hemisphere serves the contralateral side of the body (fibers cross over in the CNS). However, one notable exception is Broca's speech area. This area of the cortex subserves all motor speech functions and is located in a posterolateral area of the left frontal lobe for all right-handed and most left-handed people. Damage to this area in an adult produces motor dysphasia, which includes dysarthria (difficulty with spoken words) and dysgraphia (difficulty with written words).

Each hemisphere has four lobes that are named for the skull bones that cover them: frontal, parietal, temporal, and occipital. Primary functions of each lobe are as follows. The frontal lobes perform high-level cognition, memory, and voluntary motor movement. The parietal lobes deal mostly with sensation. The temporal lobes are primarily responsible for a variety of sensory functions such as learning, memory, emotion, and visual stimuli. The occipital lobes primarily function to interpret.

Many areas of the cerebrum operate together to produce coordinated human function. The process of communication provides a good example of this coordination. Verbal communication depends on the ability to interpret speech and translate thought into speech. Ideas usually are communicated between people by either the spoken or the written word. With the spoken word, the input of sensory information occurs through the primary auditory cortex. In auditory association areas, the sounds are interpreted as words and the words as sentences. These sentences are then interpreted by a common integrative area of the cortex as thoughts.

The common integrative area also develops thoughts to be communicated. Letters seen by the eyes are associated with words and sentences in the visual association areas and then integrated into thought in the common integrative area. Operating in conjunction with facial regions of the somesthetic sensory area, the common integrative area initiates a series of impulses, each representing a syllable or word, and transmits them to the secondary motor area controlling the larynx and mouth. The speech center, in addition to controlling motor activity of the larynx and mouth, sends impulses to the respiratory center of the secondary motor cortex to provide appropriate breath patterns for the speech process.

Cortex

As mentioned, the cortex is the most superficial layer of the cerebrum. It is responsible for all higher mental functions, such as judgment, language, memory, creativity, and abstract thinking. It also functions in the perception, localization, and interpretation of all sensations and governs all voluntary

motor activities (see Fig. 32-9). Various areas of the cortex have been identified as having different motor and sensory functions, but some of these areas are being implicated in other functions as well. For example, the occipital area, which usually takes sensory impulses from the eyes and integrates them into visual images, is now known to function in some learning processes of blind people.

Basal Ganglia

The basal ganglia function in cooperation with other lower brain parts in providing circuitry for basic and subconscious bodily movements. They provide the necessary background muscle tone for discrete voluntary movements, smoothness and coordination in functions of muscle antagonists, and the basic automatic subconscious rhythmic movements involved in walking and balance. Lesions of the basal ganglia produce various clinical abnormalities, such as chorea, hemiballismus, and Parkinson's disease.

Diencephalon

The diencephalon, a major division of the cerebrum, lies below the cerebral hemispheres. The diencephalon is a paired structure on each side of the third ventricle, directly above the brainstem. The most important areas of the diencephalon are the thalamus and the hypothalamus, described in the following sections. The subthalamus is the ventral portion of the thalamus, and the epithalamus is an area that contains the pineal gland, thought to play an important role in diurnal rhythms (Fig. 32-10).

Thalamus

The thalamus (see Fig. 32-10) functions as a sensory and motor relay center. Its neurons receive sensory impulses from synapsing neurons that originate at lower levels in the spinal cord or brainstem and relay sensory input, including sight, sound, and touch, to the sensory cortex. The thalamus also functions in the gross awareness of certain sensations, most notably pain. Discrete localization and the finer perceptual details of sensations are cortical functions, but awareness occurs at the thalamic and even midbrain areas. Finally, the thalamus is involved in the reticular activating system (RAS), the neural system that promotes wakefulness and consciousness and possibly some aspects of attention.

Hypothalamus

The hypothalamus is the seat of neuroendocrine interaction. It has a part in controlling visceral, autonomic, endocrine, and emotional function. It is connected to the reticular formation of the brainstem as well as the diencephalon, the cortex, and the pituitary gland. This area of the brain also contains some of the centers for coordinated parasympathetic and sympathetic stimulation, as well as those for temperature regulation, appetite regulation, regulation of water balance by antidiuretic hormone (ADH), and regulation of certain rhythmic psychobiological activities (eg, sleep).

Brainstem

A major subdivision of the brain, the brainstem consists of the midbrain, pons, and medulla and contains respiratory and autonomic control centers, as well as many tracts of myelinated motor axons that are passing through on their way down to the spinal cord or sensory axons passing through on their way up to the thalamus. In addition, areas of the brainstem are important in coordinating activity of the cerebellum with the rest of the brain. Also, 10 of the 12 cranial nerves originate from this area (Fig. 32-11).

Midbrain

The midbrain lies between the diencephalon and the pons. It contains the aqueduct of Sylvius, many ascending and descending nerve fiber tracts (white matter), and centers for auditory and visually stimulated nerve impulses. The Edinger-Westphal nucleus in the midbrain contains the autonomic reflex centers for pupillary accommodations

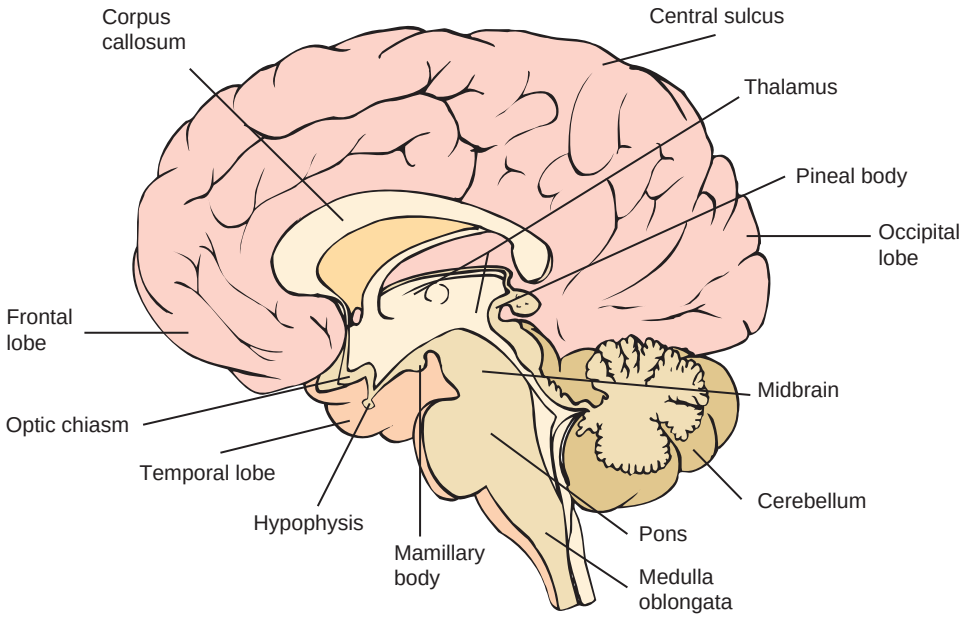


FIGURE 32-10 ▲ Lateral view of the human brain, showing the parts of the brainstem, the cerebellum, and other major landmarks. (From Cohen H: Neuroscience for Rehabilitation, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999.)

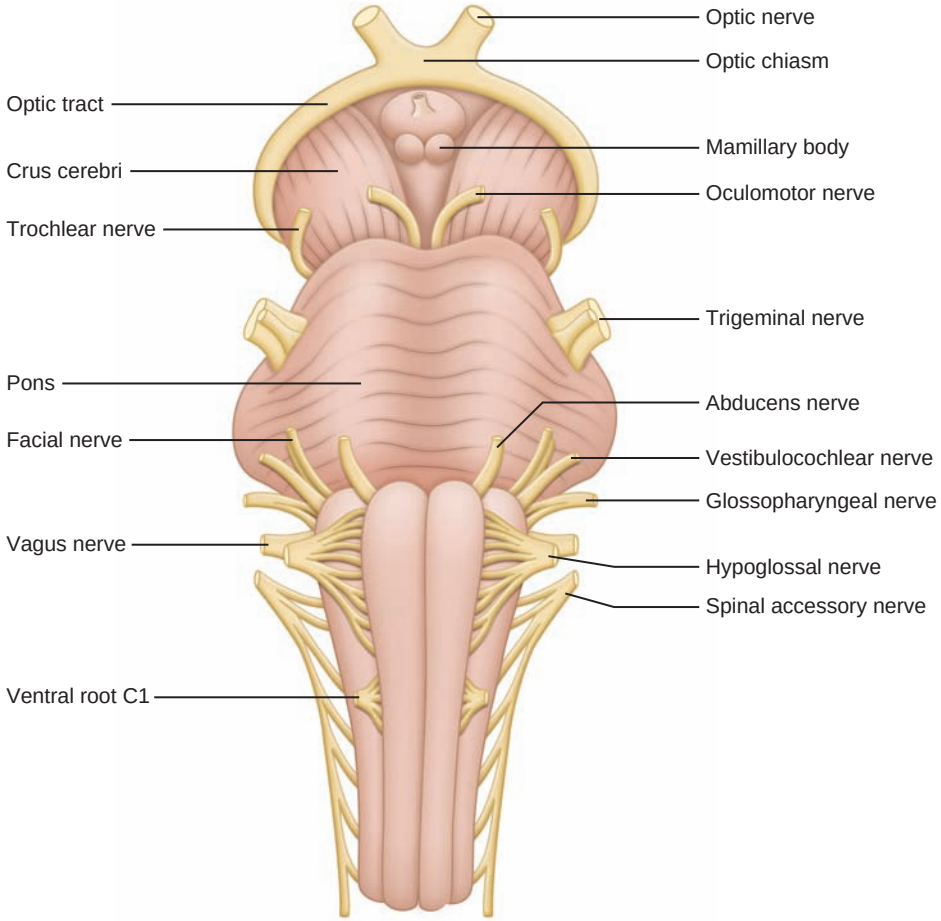


FIGURE 32-11 ▲ Anterior surface of the brainstem, showing the emergence and entrance of most of the cranial nerves.

to light. It receives sensory fibers from the retina through cranial nerve II and sends motor impulses by way of sympathetic and parasympathetic fibers (cranial nerve III) to the smooth muscles of the iris. Impaired pupillary accommodation means that at least one of these inputs or outputs is damaged or that the midbrain is suffering insult (often from tentorial herniation or stroke). Cranial nerve IV also originates in the midbrain.

Pons

The pons lies between the midbrain and the medulla and has cell bodies of fibers contained in cranial nerves V, VI, VII, and VIII. It contains respiratory centers and fiber tracts connecting higher and lower centers, including the cerebellum.

Medulla Oblongata

The medulla lies between the pons and the spinal cord. It contains centers that regulate vital functions, such as breathing, cardiac rate, and vasomotor tone, as well as centers for swallowing, vomiting, gagging, coughing, and sneezing reflex behaviors. It also contains the fourth ventricle. Cranial nerves IX, X, XI, and XII originate in the medulla. Impairment of any of the vital functions or reflexes involving these cranial nerves suggests medullary damage.

Functionally Integrated Brainstem Systems

Four networks of neurons in the brainstem should be mentioned. They are the integrated systems responsible for posture and equilibrium, consciousness, emotional reactions, and sleep.[7]

BULBORETICULAR FORMATION. The bulboreticular formation is a network of neurons in the brainstem that helps maintain balance and erect posture. This area receives sensory information from a variety of sources, including the peripheral sensory receptors that are relayed from the spinal cord, the cerebellum, the inner ear vestibular apparatus, the motor cortex, and the basal ganglia. Therefore, the bulboreticular formation is an integrative network for sensory information and motor information that has to do with body posture and balance. Output from the bulboreticular formation travels down descending fibers to internuncial neurons in the spinal cord, which synapse with motor neurons. This output alters the tonus of muscles maintaining balance and erect posture and positions of major body parts (trunk, appendages) necessary for the performance of discrete actions (eg, writing at a table, walking).

RETICULAR ACTIVATING SYSTEM. The RAS is an ascending nerve fiber system originating in the midbrain and thalamus. The RAS is stimulated by sensory impulses from various sources. These include input from the optic and acoustic cranial nerves, somesthetic impulses from the dorsal

column and spinothalamic pathways, and fibers from the cortex. Therefore, the RAS is an integrative system that receives sensory information concerning light, sound, and touch that may indicate a need for alertness. Excitatory output of the RAS extends to a variety of higher centers, including the cortex. In this way, the RAS can stimulate these centers to maintain alertness. The stimulation of the cortex by the RAS seems to be the major physiological basis for consciousness, alertness, and attention to various environmental stimuli. Decreased activity of the RAS produces decreased alertness or levels of consciousness, including stupor and coma. Inactivation of the RAS can result from anything that interrupts the entry of a critical amount of sensory input or from any damage that prevents the RAS fibers from sending impulses to the cortex.

LIMBIC SYSTEM. The hypothalamus, the cingulate gyrus of the cortex, the amygdala and hippocampus in the temporal lobes, and the septum and interconnecting nerve fiber tracts among these areas compose a functional unit of the brain called the limbic system. This system provides a neural substrate for emotions (eg, terror, intense pleasure, eroticism). This region of the brain is involved in emotional experience and in the control of emotion-related behavior. Also, it is here that neural pathways provide a connection between higher brain functioning and endocrine or autonomic activities.

SLEEP CENTERS. The release of stored serotonin from axon terminals in the diencephalon, medulla, thalamus, and a small forebrain area, collectively called DMTF, results in inactivation of the RAS and activation of the DMTF. DMTF activity results in the four stages of sleep. During sleep stages III and IV, parasympathetic activity (with decreased heart rate, respiratory rate, and so forth) predominates, and sleepwalking, sleep talking, and nocturnal enuresis occur.

Rhythmic discharges (about four to eight times per night, from 10 to 20 minutes per episode) from the pontine nuclei during sleep result in rapid eye movement sleep, during which approximately 80% of all dreaming occurs and sympathetic nervous system activity predominates. Based on circadian rhythmicity and decreasing cerebral serotonin levels, the RAS is reactivated in the morning, after 6 to 8 hours of sleep. See Chapter 2, Box 2-2, on page 19 for a review of the stages and characteristics of sleep.

Cerebellum

The cerebellum (see Fig. 32-10) is located just superior and posterior to the medulla. It receives "samples" of all ascending somesthetic sensory input and all descending motor impulses. Use of these connections enables the cerebellum to match intended motor stimuli (before they reach the muscles) with actual sensory data. This ensures an optimal match for voluntary motor "intention" with actual motor action, with time to alter the motor message in case of error. It sends its own messages to the basal ganglia and cortex and to parts of the brainstem.

The cerebellum functions to produce smooth, steady, harmonious, and coordinated skeletal muscle actions; maintain equilibrium; and control posture without any jerky or uncompensated movements or swaying. The cerebellum is also involved in motor learning and is responsible for reflexive activities that occur when motor learning is complete, such as correcting one's balance when riding a bike. Cerebellar disease can produce typical symptoms, the most prominent of which are disturbances of gait, equilibrium ataxia (overstability or understability of the walk), inability to perform rapid repetitive movements, and characteristic intention tremors.

Spinal Cord

The spinal cord lies within the neural canal of the vertebral column for protection from traumatic injury. The bony structures of the spine, along with those of the skull, are one mechanism for protection of the delicate structures of the CNS (see Fig. 32-5, p. 696 and Fig. 32-12). The vertebral column of the spine is composed of 33 vertebrae: 7 cervical, 12 thoracic, 5 lumbar, 5 sacral (fused into one), and 4 coccygeal (fused into one). Each vertebra is composed of two essential parts, an anterior body

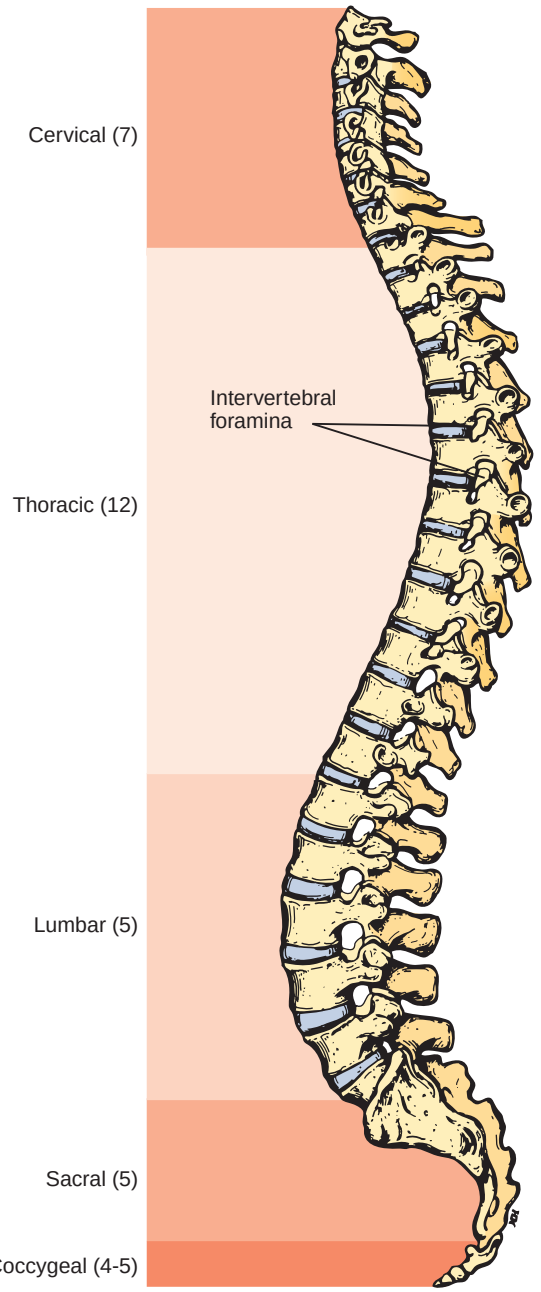


FIGURE 32-12 ▲ Lateral view of the adult vertebral column. (From Hickey JV: The Clinical Practice of Neurological and Neurosurgical Nursing, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 48.)

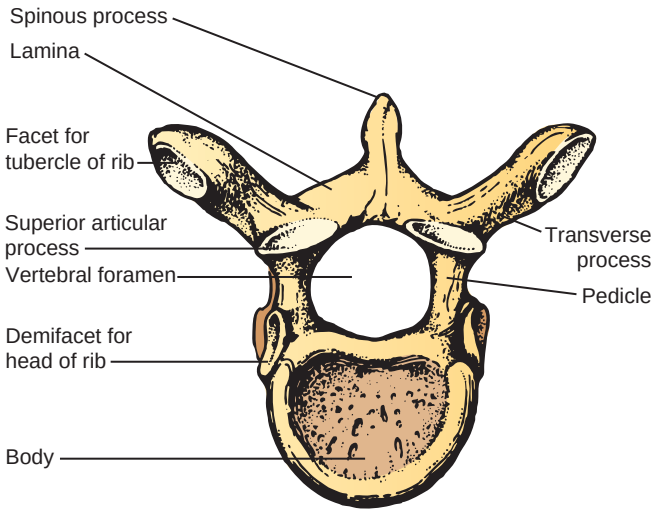


FIGURE 32-13 ▲ The sixth thoracic vertebra with anatomical markings. The vertebral foramen is the site of the spinal cord, and the spinous and transverse processes serve as places for attachments for muscles. The articular processes form synovial joints between the vertebrae. The vertebral body is opposed to superior and inferior intervertebral disks. (From Hickey JV: The Clinical Practice of Neurological and Neurosurgical Nursing, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 416.)

and a posterior arch, which form a protective ring (vertebral foramen) around the spinal cord. The arch has two pedicles and two laminae to support seven processes (four articular, two transverse, and one spinous), on which muscles and ligaments can attach (Fig. 32-13). The first cervical vertebra, the atlas, supports the weight of the head by articulating with the occiput of the skull. The second cervical vertebra, the axis, has a perpendicular projection called the odontoid process that the atlas sits on; this allows for lateral rotation of the head (Fig. 32-14).

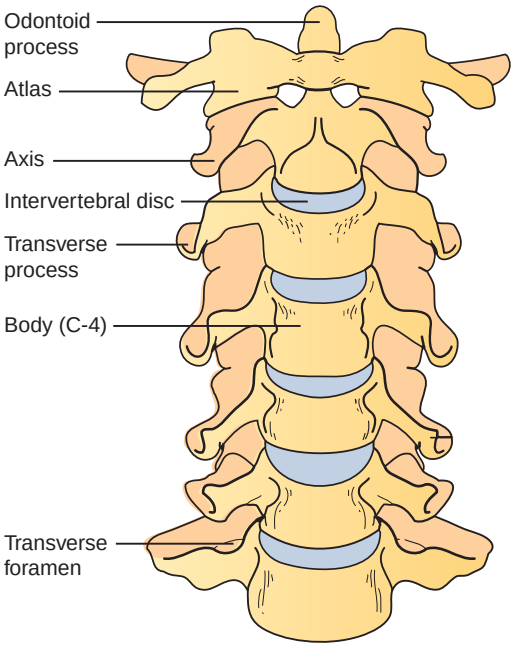


FIGURE 32-14 ▲ The cervical spine. Note odontoid process of C2 and the atlas, C1, positioned on top of C2. (Adapted from Hickey JV: The Clinical Practice of Neurological and Neurosurgical Nursing, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 50.)

In addition to the bony structures of the spinal column, ligaments and the intervertebral disks also protect the spinal cord by providing support and stability to the vertebral column. The anterior longitudinal ligament and the posterior longitudinal ligament hold the disks and vertebral bodies in position. The intervertebral disks are fibrocartilaginous, disk-shaped structures located between the vertebral bodies from the second cervical vertebra to the sacrum. These disks, which act as shock absorbers between the vertebrae, have a central core known as the nucleus pulposus surrounded by a fibrous capsule known as the anulus fibrosus (Fig. 32-15).

The spinal cord extends down and fills the neural canal to the level of about the second lumbar vertebra in an adult. Over the entire length of the vertebral column, a pair of spinal nerves exits between adjacent vertebrae at its respective level (eg, C5, T11, L1, S1). However, because the cord is shorter than the spinal column, the level of the cord that gives rise to a particular pair of spinal nerves is above the vertebra of that level. For instance, the two spinal nerves that leave the spinal column between the L4 and L5 vertebrae have to travel down the neural canal from the cord level L4, which is actually up at about vertebral level T12. Below the point at which the cord terminates, the neural canal is filled with descending spinal nerves collectively

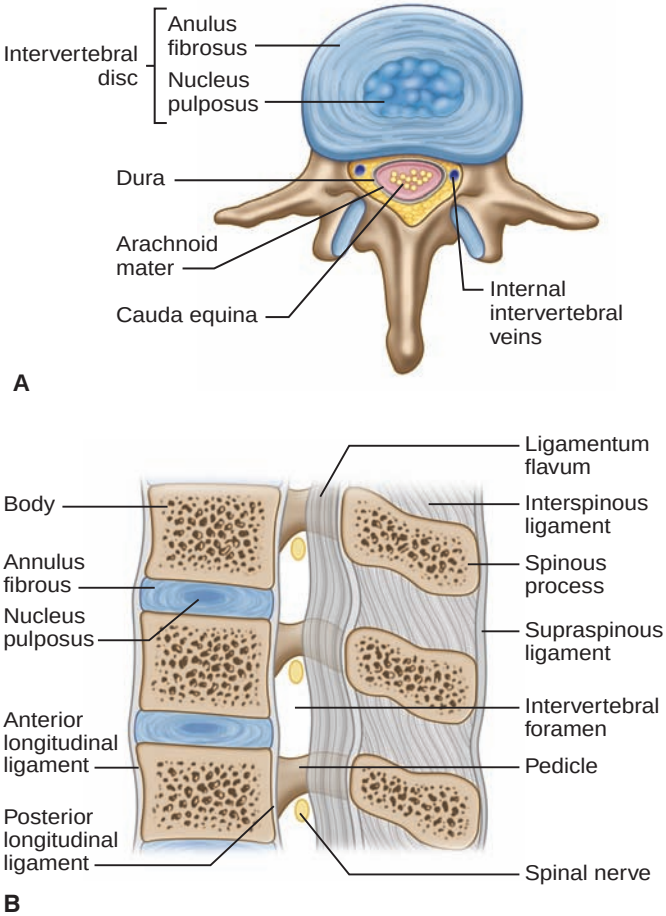


FIGURE 32-15 ▲ **A**: Third lumbar vertebra seen from above, showing the intervertebral disk. **B**: Sagittal section through three lumbar vertebrae, showing the ligaments and the intervertebral disks. (Adapted from Hickey JV: The Clinical Practice of Neurological and Neurosurgical Nursing, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 49.)

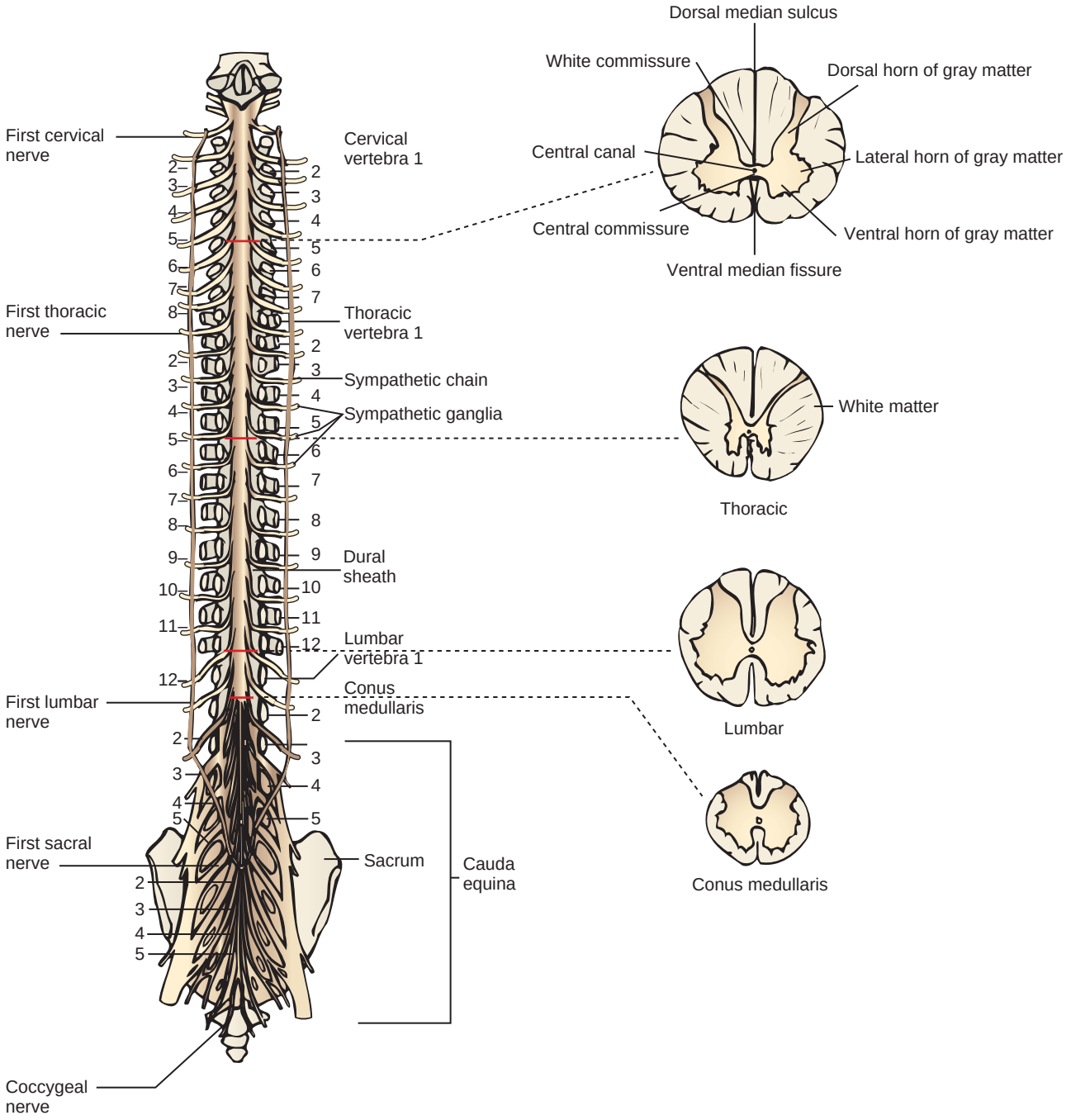


FIGURE 32-16 ▲ The spinal cord within the vertebral canal. The spinal canal and meninges have been opened. The spinal nerves and vertebrae are numbered on the left. Cross (transverse) sections with regional variations in gray matter and increasing proportions of white matter as the cord ascends appear on the right. (Adapted from Hickey JV: The Clinical Practice of Neurological and Neurosurgical Nursing, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 69.)

known as the cauda equina (horse's tail), which exit the neural canal at the vertebra that corresponds to the cord level from which they arose (Fig. 32-16). Because neurons occupy less space in the canal at lower lumbar levels, it is here that spinal taps may be performed safely. This anatomical fact also explains why injuries to lumbar and lower thoracic vertebrae can produce impairment at disproportionately lower body levels.

Within the cord lie ascending sensory fibers and descending motor fibers, many of which are myelinated and appear as white matter. Interneurons, and the nerve cell bodies and dendrites of the second-order somatic (voluntary) and first-order autonomic motor neurons, are not myelinated and appear as gray matter. The central area of the cord contains nerve cell bodies and internuncial neurons (ie, nerve cells contained entirely within the cord) and also appears as gray matter. The gray matter has left and right dorsal and ventral projections (Fig. 32-17). Nerve cell bodies of motor neurons supplying skeletal muscles lie in the ventral horns. Nerve cell bodies of the sympathetic preganglionic neurons lie in left and

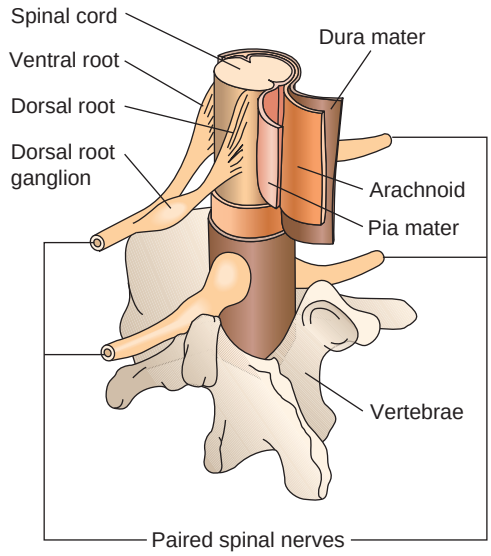


FIGURE 32-17 ▲ Spinal cord and meninges. (From Porth CM: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Wolters Kluwer Health: Lippincott Williams & Wilkins, 2009, p 1211.)

right lateral projections or horns of gray matter referred to as the intermediolateral cell column in the thoracic and upper lumbar cord.

It is important to realize that the spinal cord is really an extension of the brain and contains many integrative and processive functions. For instance, the substantia gelatinosa contains nerve terminals of descending neurons, and also interneurons, which function to moderate ascending pain impulses (see later section on Pain, pp. 716–718).

▲ Peripheral Nervous System

The PNS consists of 12 pairs of cranial nerves and 31 pairs of spinal nerves and includes all neural structures lying outside the pia mater of the spinal cord and brainstem. The parts of the PNS inside the neural canal and attached to the ventral and dorsal surfaces of the cord are called the spinal nerve roots. Those attached to the ventrolateral surface of the brainstem are the cranial nerve roots.

Functionally, the PNS is separated into sensory and motor divisions. The sensory division includes sensory neurons that innervate the skin, muscles, joints, and viscera and provide sensory information about the environment outside and inside the body to the CNS. The motor division includes motor neurons that innervate skeletal muscles and the autonomic nervous system (ANS) that innervates smooth and cardiac muscle and glands. The ANS is responsible for regulating the ongoing functions of many organ systems, such as blood pressure, heart rate, and gastrointestinal activity.

Cranial Nerves

The 12 pairs of cranial nerves supply motor and sensory fibers mostly to the structures of the head, neck, and upper back, although cranial nerve X, the vagus nerve, supplies the viscera to about the level of the waist (Table 32-1). Most cranial nerves originate in the brainstem (see Fig. 32-11, p. 701), except cranial nerves I and II, which originate in the diencephalon. Cranial nerves are classified as either sensory, motor, or mixed (carrying both sensory and motor signals). They bring input from the special senses (vision, hearing, smell) and somatic sensory input from the face and head into the brain. They also send motor commands out to the muscles and glands of the head and neck to control facial expression, eye movements, movements of the structures in the mouth and throat, movements of the head and neck, and autonomic functions of the eyes, salivary glands, and viscera in the chest and upper abdomen. Most cranial nerves contain fibers of more than one functional type; thus, most cranial nerves are associated with more than one nucleus in the brainstem (see Table 32-1).

Spinal Nerves

Spinal nerves are attached to the spinal cord in pairs; there are 8 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 1 coccygeal pair of spinal nerves (see Fig. 32-16). In the cervical spine, the spinal nerves exit above the vertebrae. At C7, an extra spinal nerve exists below C7, giving rise to the C8 spinal nerve. All of the rest of the spinal nerves (ie, the thoracic, lumbar, sacral, and coccygeal spinal nerves) exit below the vertebrae. Spinal nerves contain both sensory and motor fibers. Each spinal nerve attaches to the cord by a dorsal and a ventral root. The dorsal root houses the nerve cell bodies of sensory neurons. Motor axons, whose nerve cell bodies lie in the gray matter of the ventral horn of the cord, traverse the ventral root. Thus, damage to the dorsal root may impair sensory function without impairing motor function, and vice versa. However, a spinal nerve injury distal to the roots could damage both sensory and motor functioning. A dermatome is the area of skin innervated by sensory fibers from a particular spinal nerve emanating from a particular segment of the spinal cord (see Chapter 37, Fig. 37-3, p. 829).

Sensory Division

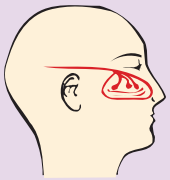
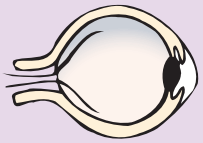
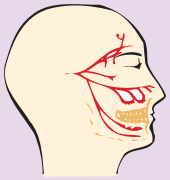
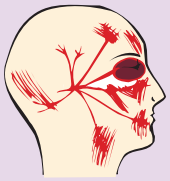
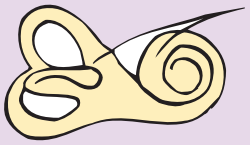
The sensory division of the nervous system is composed of sensory receptors, sensory neurons whose axons form sensory pathways, and perceptive areas of the brain.

Sensations and Sensory Receptors

Sensations often are divided into the special senses (eg, vision, hearing, and smell) and those termed somesthetic sensations (eg, pain, touch, and stretch). In this section, only somesthetic sensations are discussed. Such sensations provide information about, for example, body position and conditions of the external and internal environment. These are called proprioceptive, exteroceptive, and visceral sensations, respectively.

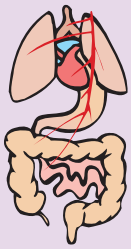
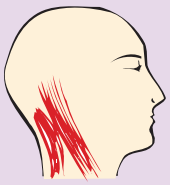
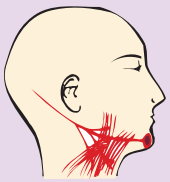
Proprioceptive sensations describe the physical position state of the body, such as muscle tension, joint flexion or extension, tendon tension, and deep pressure in dependent parts, such as the feet while one is standing or the buttocks while one is sitting. Exteroceptive sensations monitor conditions on the body surface, such as temperature and pain.

Table 32-1 The Cranial Nerves

Cranial Nerve	Tract(s)	Function	Location of Origin
			
I. Olfactory	Sensory	Sense of smell	Diencephalon
			
II. Optic	Sensory	Vision	Diencephalon
			
III. Oculomotor	Parasympathetic Motor	Pupillary constriction Elevation of upper eyelid and four of six extraocular movements	Midbrain
IV. Trochlear	Motor	Downward, inward movement of the eye (superior oblique)	Midbrain
			
V. Trigeminal	Motor Sensory	Muscles of mastication and opening jaw Tactile sensation to the cornea, nasal and oral mucosa, and facial skin	Pons
VI. Abducens	Motor	Lateral deviation of eye (lateral rectus)	Pons
			
VII. Facial	Parasympathetic Motor Sensory	Secretory for salivation and tears Movement of the forehead, eyelids, cheeks, lips, ears, nose, and neck to produce facial expression and close eyes Tactile sensation to parts of the external ear, auditory canal, and external tympanic membrane Taste sensation to the anterior two thirds of the tongue	Pons
			
VIII. Vestibulocochlear (also known as acoustic or cochlear)	Sensory	*Vestibular branch:* Equilibrium *Cochlear branch:* Hearing	Pons

(continued on page 707)

Table 32-1 The Cranial Nerves (continued)

Cranial Nerve	Tract(s)	Function	Location of Origin
IX. Glossopharyngeal	Parasympathetic	Salivation	Medulla
	Motor	Voluntary muscles for swallowing and phonation	
	Sensory	Sensation to pharynx, soft palate, and posterior one third of tongue Stimulation elicits gag reflex	
			
X. Vagus	Parasympathetic	Autonomic activity of viscera of thorax and abdomen	Medulla
	Motor	Involuntary activity of visceral muscles of the heart, lungs, and digestive tract Innervation of striated muscles of the soft palate, pharynx, and larynx for voluntary swallowing	
	Sensory	Sensation to the auditory canal, pharynx, larynx, and viscera of the thorax and abdomen	
XI. Spinal accessory	Motor	Sternocleidomastoid and trapezius muscle movements	Medulla
			
XII. Hypoglossal	Motor	Tongue movements	Medulla

Artwork from Evans MJ: Neurologic Neurosurgical Nursing, 2nd ed. Springhouse, PA: Springhouse, 1995, pp 7–8.

Visceral sensations are similar to exteroceptive sensations, except that they originate from within the body and monitor pain, pressure, and fullness from internal organs.

The sensory receptors for somesthetic sensations are basically dendrites, which can have the form of free nerve endings or specialized receptors. Free nerve endings are nothing more than small, filamentous branches of dendrites. They detect crude sensations of touch, pain, heat, and cold. The precision is crude because different neurons have overlapping distributions of their dendrites. These nerve endings are the most widely distributed and most numerous of sensory receptors and perform the general discriminatory functions. The more specialized sensory receptors discriminate between very slight differences in degrees of touch, pain, heat, and cold. Indeed, the special exteroceptive end organs for detecting light touch, warmth, and cold differ structurally from one another and are specific in their function. The physiological basis for this specific function has not been determined but is presumed to be based on some specific physical effect on the receptor itself.

Sensation from the internal organs may come from specialized sensory receptors, such as baroreceptors and chemoreceptors that reside in arterial walls, or stretch receptors in sphincters. In contrast, visceral pain is the result of stimulation of unmyelinated, raw sensory nerve endings, usually by stretch, as might happen during swelling or distention, or by pressure on the organ as might happen from compression by a tumor. For both specialized and unspecialized visceral receptors, the sensory fibers run within the autonomic nerves (sympathetic or parasympathetic) back to CNS centers that are closely associated with autonomic motor responses. For this reason, these sensory fibers are sometimes referred to as autonomic afferent fibers. This is a somewhat misleading name because the ANS is purely a motor system (see later), and the name refers only to the anatomical location of sensory fibers within the nerves that also carry autonomic motor fibers.

Stimulation of a sensory receptor initiates an electrical charge (generator potential) that depolarizes the sensory dendrite, causing a series of nerve impulses to travel along the sensory dendrite to the cell body. As mentioned, the sensory neuron cell body is contained in the dorsal root ganglion just outside the spinal cord. The sensory neuron sends axons into the spinal cord (or brain in the case of cranial nerves), where it synapses with projection neurons in either brain or spinal cord that carry impulses to the appropriate centers in the brain, including the thalamus, where the sensation finally may be perceived consciously. The projection neuron may synapse in the thalamus with another neuron, which relays the sensory impulse to the sensory cortex.

When the sensation first stimulates the sensory receptor in the periphery, there is a burst of impulses; if the stimulus persists, the frequency of impulses transmitted begins to decrease. All sensory receptors show this phenomenon of adaptation to varying degrees and at different rates. Adaptations to light touch and pressure occur in a few seconds, whereas pain and proprioceptive sensations adapt very little, if at all, and at a very slow rate. This adaptation results in our being unaware of the touch of our clothing to our skin or the pressure on our buttocks while we are seated. Determination of the intensity of the sensation is made on a relative rather than an absolute basis.

Although there are structurally different receptors for detecting each type of sensation, the area of the brain to which the information is transmitted determines the modality, or type of sensation, a person feels. The thalamus and sensory cortex operate together to attribute various sensory qualities and intensities to nerve impulse information they receive.

Sensory Pathways

As mentioned, sensory neurons that enter the cord synapse with projection neurons that carry the sensory information up the spinal cord. There are a number of pathways by which sensory information is transmitted up the cord by axons of the projection neurons. Depending on the type of somesthetic receptor involved, fibers of sensory neurons may, on entering the cord, do one of three things.

First, they may send axons up the cord to the medulla on the same side of the body as the sensory receptor. This tract of myelinated axons (white matter) is called the dorsal column. In the medulla, the sensory neurons synapse with projection neurons that cross over to the opposite side of the brain and travel to the thalamus. This tract is called the medial lemniscus (Fig. 32-18). It is used for conducting impulses originating from stimulation of joint, muscle, and tendon proprioceptors; vibration-sensitive receptors; and receptors in the skin involved in precise localization of touch.

Second, the sensory neurons may synapse immediately on entering the cord with projection neurons that immediately cross over to the opposite side of the cord. Fibers from these projection neurons then travel up the white matter of the cord to the thalamus. This is called the spinothalamic pathway (see Fig. 32-18). It conducts impulses concerned with pain, temperature, poorly localized touch, and sex organ sensations. Both the dorsal column–medial lemniscus pathway and the spinothalamic pathway involve crossing of the sensory information from each side of the body to the opposite side of the CNS. Therefore, sensations on each side of the body are perceived by the thalamus and sensory cortex on the opposite side. In the thalamus, neurons of both the dorsal column pathway and the spinothalamic pathway synapse with other neurons that transmit impulses to the appropriate area of the sensory cortex. Because of their final destination in the cortex, impulses from either pathway give rise to consciously perceived sensations. The sensory homunculus (Fig. 32-19) is a pictorial representation of the number of neurons in the sensory cortex that are devoted to receiving sensations from various areas of the body. It should be noted that very sensitive areas, such as the lips, have a large representation in the sensory cortex, whereas less sensitive areas, such as the trunk, have a smaller representation.

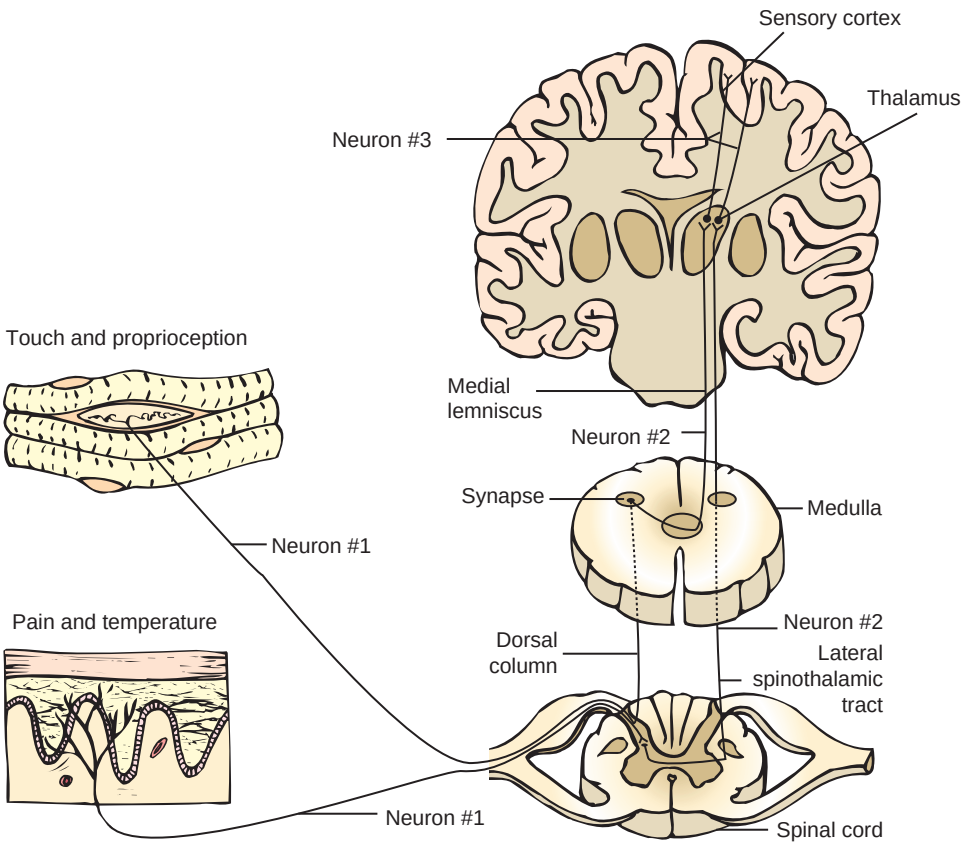


FIGURE 32-18 ▲ Pathways of ascending tracts. Sensory neurons enter the cord at the dorsal horn. Axons of sensory neurons for touch and proprioception ascend in the dorsal columns to the medulla, where they synapse with second-order projection neurons that cross (decussate) to the opposite side before ascending to the thalamus in the tract called the medial lemniscus. First-order neurons for pain and temperature enter the dorsal gray matter of the cord, where they synapse with second-order projection neurons that cross to the opposite side and ascend in the lateral spinothalamic tract to the thalamus. Third-order neurons connect both pathways from thalamus to the sensory cortex.

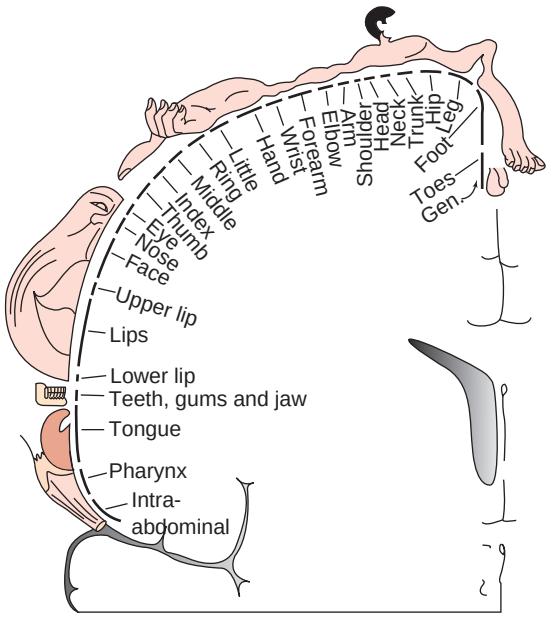


FIGURE 32-19 ▲ The sensory homunculus, a representation of the relative extent of the somatosensory cortex devoted to various body regions, as determined by stimulation studies on the human somatosensory cortex during surgery. (From Penfield E, Rasmussen T: The Cerebral Cortex of Man. New York, NY: Macmillan, 1955. Copyright © by MacMillan Publishing Co., Inc., renewed 1978 by Theodore Rasmussen.)

Third, certain sensory neurons may synapse with a projection neuron belonging to the spinocerebellar pathway. Spinocerebellar neurons do not cross over. They carry impulses only as far as the cerebellum (and possibly lower brainstem). This pathway carries impulses originating from stimulation of joint, muscle, and tendon proprioceptors. Because this pathway ends at the cerebellum, it transmits sensory information that is not perceived consciously. Instead, these data are used in reflex postural adjustments.

Motor Division and the Neuromuscular Junction

The motor division comprises the areas of the brain, descending fiber tracts, and motor neurons involved in producing or altering movement or adjusting tonus of skeletal, cardiac, and smooth muscles and in regulating the secretions of the various exocrine and certain endocrine gland cells. Muscle and glandular tissues are referred to as the effector organs of this system.

The motor division can be divided on the basis of motor neurons and effector organs into somatic and autonomic subdivisions (Fig. 32-20). The former involves skeletal muscles and the motor neurons innervating them. The latter is composed of smooth muscle, cardiac muscle, and gland cells plus the sympathetic and parasympathetic fibers innervating them.

Somatic Motor Division

Figure 32-21 depicts the major descending fiber tracts from motor areas of the cortex. The most prominent of these tracts is the corticospinal tract, often called the pyramidal tract because it originates from pyramid-shaped nerve cell bodies in the cortex. The corticospinal tract is heavily myelinated and appears as white matter in the brain and spinal cord. The fibers cross to the opposite side in an area of the medulla referred to as the decussation (crossing over) of the pyramids. Several other motor tracts originate in the cortex or in the cerebellum. These tracts may cross over in the brain or in cord centers. Motor fibers from the brain ultimately stimulate somatic motor neurons, the nerve cell bodies of which lie in the anterior (ventral) horn of the gray matter in the

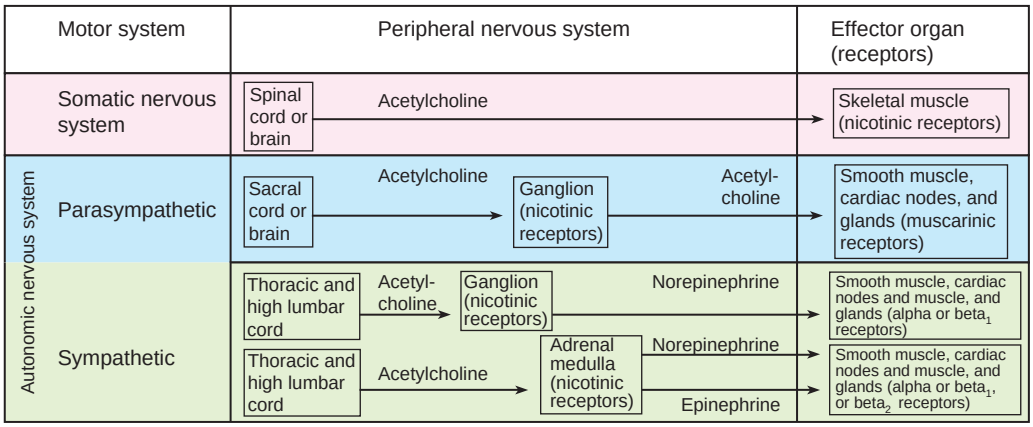


FIGURE 32-20 ▲ A comparison between the divisions of the motor systems. The somatic nervous system (*pink*) sends cholinergic motor axons from the spinal cord or brain to the skeletal muscles. Acetylcholine released from these axon terminals binds to nicotinic receptors on skeletal muscles to cause contraction. The autonomic nervous system (ANS) is composed of parasympathetic (*blue*) and sympathetic (*green*) divisions. For both divisions, preganglionic cholinergic neurons originate in the brain or spinal cord and send their axons to ganglia in the periphery, where they synapse with postganglionic neurons having ganglionic nicotinic receptors. Postganglionic neurons of the parasympathetic division are cholinergic and synapse with muscarinic receptors on end organs. Postganglionic neurons of the sympathetic division are noradrenergic and synapse with α or β_1 receptors on end organs. The adrenal medulla is innervated by preganglionic sympathetic neurons. Acetylcholine released by these neurons binds to ganglionic nicotinic receptors on cells of the adrenal medulla, causing them to release norepinephrine and epinephrine into the bloodstream.

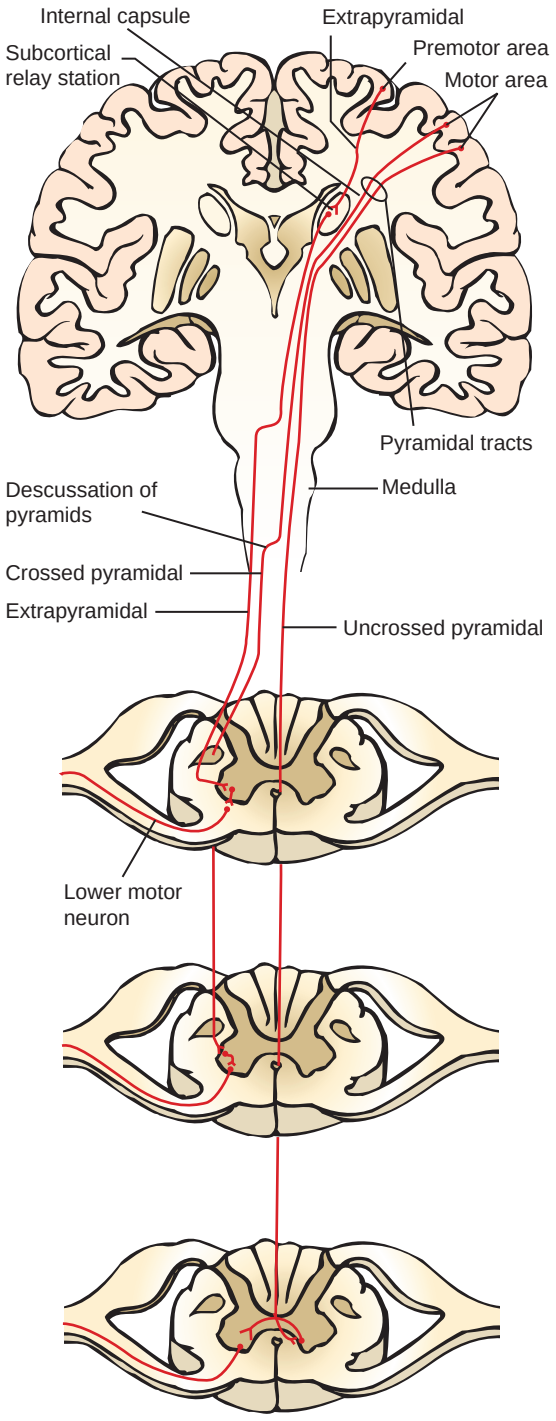


FIGURE 32-21 ▲ Diagram of motor pathways between the cerebral cortex, one of the subcortical relay centers, and lower motor neurons in the spinal cord. Decussation (crossing) of fibers dictates that each side of the brain controls skeletal muscles on the opposite side of the body.

cord. The axons of these motor neurons travel within spinal nerves and terminate at the neuromuscular junction, the synapse between the somatic motor neuron axon and the muscle cell. When a motor neuron depolarizes, acetylcholine is released into the synapse at the neuromuscular junction. It binds to nicotinic receptors on the skeletal muscle membrane, causing depolarization of the muscle cell, which stimulates contraction.

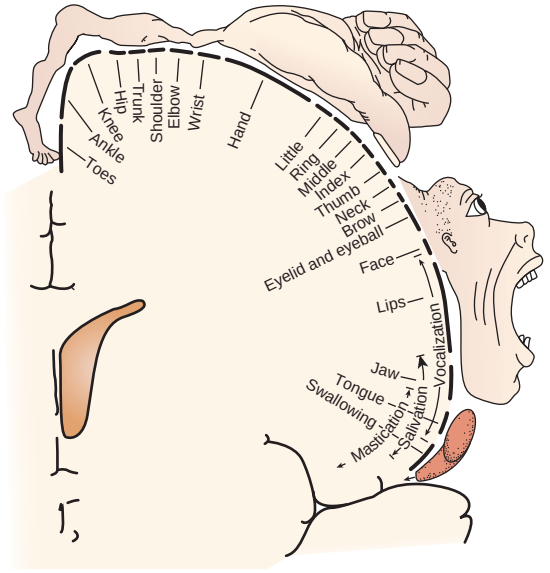


FIGURE 32-22 ▲ The motor homunculus, a representation of the relative extent of the primary motor cortex devoted to various body regions. (From Penfield E, Rasmusen T: The Cerebral Cortex in Man: A Clinical Study of Localization of Function. New York, NY: MacMillan, 1968.)

Figure 32-21 also shows several extrapyramidal (not part of the pyramidal [corticospinal] tracts) tracts arising from the brainstem centers (eg, bulboreticular formation, midbrain). Some of these cross over; others do not. Fibers in these tracts descend the cord and ultimately stimulate either somatic motor neurons, which stimulate skeletal muscle contraction, or other motor neurons (gamma efferent) that alter the tensions of stretch receptor organelles (muscle spindles) in the skeletal muscles. Alteration of spindle tension provokes a spinal reflex arc that efficiently alters skeletal muscle tonus. These extrapyramidal pathways conduct impulses that produce the automatic coordinated alterations in skeletal muscle tonus and movement that are necessary for gross motor movements (eg, walking) and for appropriate posture for conduction of finer movements (eg, sitting at a desk with arm flexed in preparation for writing).

Not shown in Figure 32-21 are descending fiber tracts that stimulate motor neurons responsible for the movement of skeletal muscles of the head (eg, tongue, face, jaw). The general pattern and myoneural transmitter are the same, except the somatic motor neuron nerve cell bodies lie in particular areas of the brain and exit through cranial nerves. These fibers must also cross over from the opposite side before synapsing with the motor neurons.

The motor homunculus (Fig. 32-22) is a pictorial representation of the number of neurons in the motor cortex devoted to each area of the body. It should be noted that areas of the body capable of fine motor movement, such as the fingers and the thumb, have abundant representation in the motor homunculus, whereas those areas not involved with fine motor movement, such as the thigh or trunk, have less abundant representation.

Neuromuscular Junction

As mentioned, motor neurons whose cell bodies are in the ventral horn of the spinal cord at each level send axons out

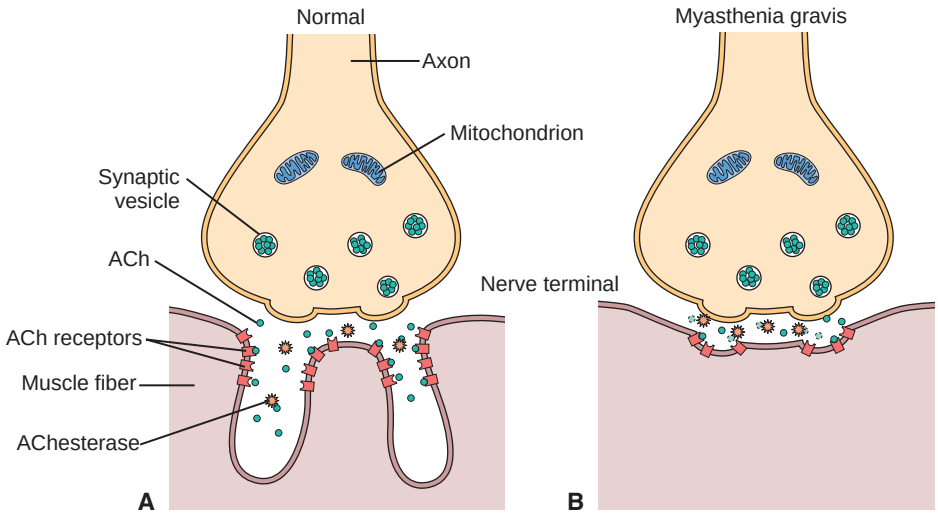


FIGURE 32-23 ▲ The neuromuscular junction. **A**: Acetylcholine (ACh), released from the motor neurons in the myoneural junction, crosses the synaptic space to reach receptors that are concentrated in folds of the endplate of the muscle fiber. Once released, ACh is rapidly broken down by the enzyme acetylcholinesterase (AChesterase). **B**: Decrease in Ach receptors in myasthenia gravis. (From Porth CM: Essentials of Pathophysiology, 3rd ed. Philadelphia, PA: Wolters Kluwer Health | Lippincott Williams & Wilkins, 2011, p 901.)

of the ventral root into each spinal nerve and innervate specific skeletal muscles. Similarly, cranial nerves that innervate skeletal muscles of the head, neck, and shoulders contain axons of motor neurons that originate in nuclei of the brainstem and pons. The axon terminals of these motor neurons end in specialized synapses with skeletal muscle cells, termed the neuromuscular junction.

Motor neurons may innervate several muscle fibers. The combination of axon terminals and the corresponding muscle fibers innervated by one motor neuron is referred to as a motor unit. The axon terminals reach into invaginations on the skeletal muscle fiber, called motor end plates, and form the highly structured synapses that compose the neuromuscular junctions (Fig. 32-23). The cell membrane of the skeletal muscle contains abundant nicotinic skeletal muscle receptors concentrated in the area of the motor end plate. These receptors bind acetylcholine, which is released from the motor neuron in response to an arriving action potential. These nicotinic receptors are slightly different from those located at ganglionic synapses of the ANS, making it possible to design drugs that bind only to skeletal muscle nicotinic receptors, not ganglionic ones. These drugs, such as tubocurarine, are used for neuromuscular blockade during anesthesia or in the intensive care unit.

Also located within the neuromuscular junction, similar to other cholinergic synapses, are many molecules of acetylcholinesterase, an enzyme that degrades acetylcholine into acetate and choline with great rapidity. This enzyme is responsible for terminating the activity of acetylcholine in the synapse. Following the degradation of acetylcholine, choline is quickly taken back up into the motor nerve terminal and used to resynthesize acetylcholine. Acetate can be used for fuel by body cells by conversion to acetyl coenzyme A. Acetylcholinesterase inhibitors are drugs that can be used to prolong the activity of acetylcholine. This may be helpful, as in the reversal of neuromuscular blockade with drugs such as neostigmine, or harmful, as in "nerve gases," such as soman, and organophosphate insecticides, such as malathion.

Myasthenia gravis is a disease of the neuromuscular junction in which antibodies develop to nicotinic skeletal muscle receptors (see Chapter 35). Immune attack of these receptors decreases the number available for activation by acetylcholine, rendering the muscle contraction weaker than normal. Lambert-Eaton syndrome, a similar condition prevalent in cancer patients, is due to decreased release of acetylcholine into the neuromuscular junction.

Autonomic Motor Division

The autonomic division contains both sympathetic and parasympathetic motor fibers. They are responsible for contraction and relaxation of smooth muscle, rate and strength of contraction of cardiac tissue, secretion by exocrine glands, and secretion by the adrenal medulla. They also influence the secretion by the islets of Langerhans in the pancreas.

The sympathetic and parasympathetic sections differ on the basis of the anatomical distribution of nerve fibers, the secretion of two different neurotransmitters by the postganglionic fibers of the two divisions, and the antagonistic effects of the two divisions on some of the organs they innervate. Figure 32-24 shows the anatomy of the sympathetic and parasympathetic nervous systems. The CNS center immediately responsible for sympathetic outflow resides in the thoracic cord. In contrast, 80% of parasympathetic activity originates in the brain and travels through cranial nerve X (the vagus nerve), and approximately 20% originates in the sacral cord and travels through pelvic nerves.

Both the sympathetic and parasympathetic motor pathways are composed of a chain of two neurons carrying nerve impulses from the CNS to the effector organ. The first neuron in the chain is the preganglionic neuron; the second is the postganglionic neuron. (A ganglion is a group of cell bodies.) Nerve cell bodies of preganglionic sympathetic neurons lie in the lateral horns of the gray matter of the thoracic and high lumbar segments of the cord (the intermediolateral cell columns); their axons exit the cord in the spinal nerve roots. The nerve cell bodies of preganglionic parasympathetic neurons lie either in certain areas of the brain and send their axons down cranial nerve X or in the lateral horns of gray matter in the sacral cord and send their axons down the pelvic nerves.

As mentioned, axons of preganglionic sympathetic neurons exit the cord and enter the ventral roots of spinal nerves. They then leave the spinal nerve to enter a nearby sympathetic ganglion by a connecting pathway termed a

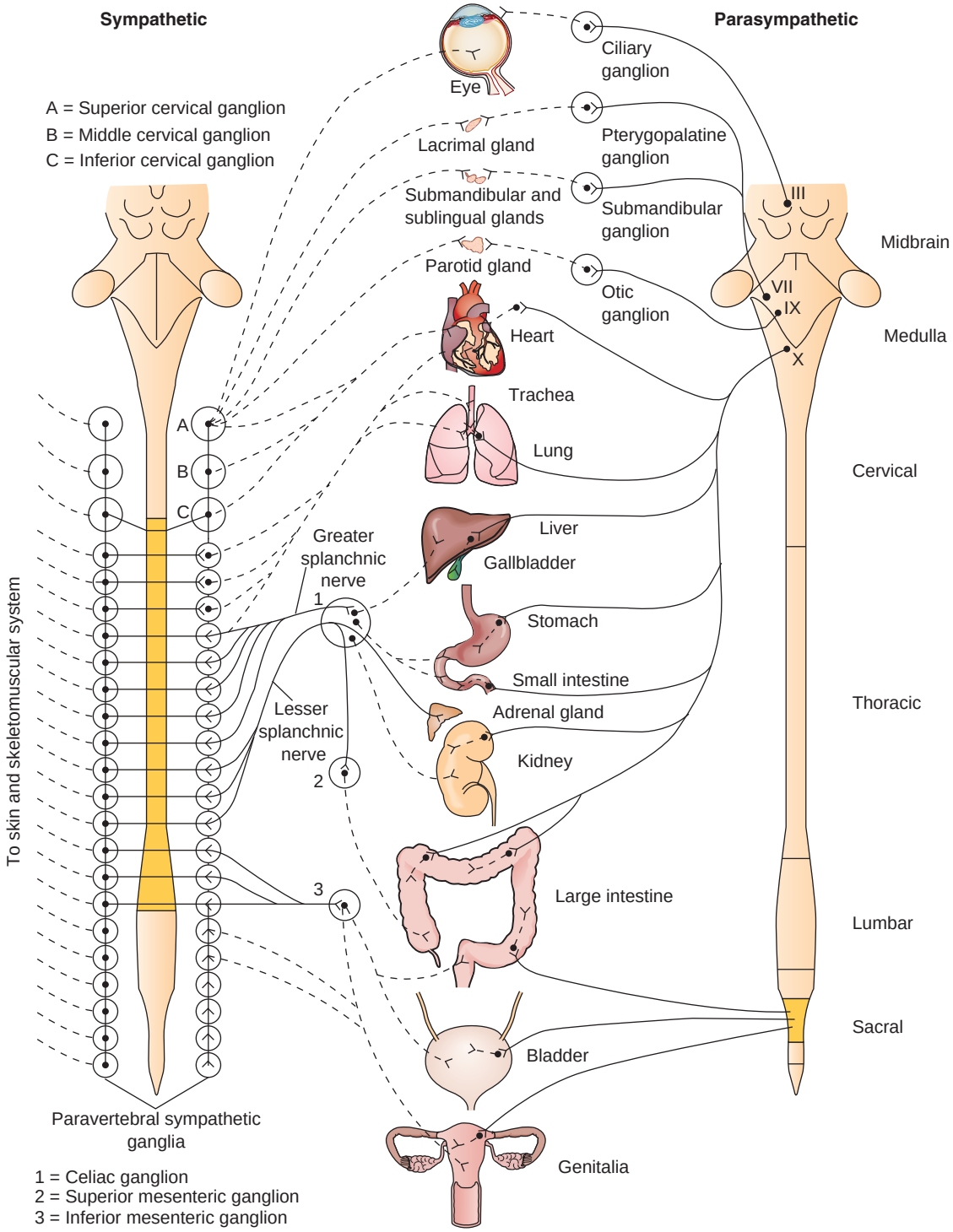


FIGURE 32-24 ▲ The ANS and the organs it affects. The **left side** illustrates the actions of the sympathetic nervous system. The **right side** illustrates the parasympathetic nervous system. (From Porth CM: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009.)

ramus. In a sympathetic ganglion, the preganglionic neuron synapses with a postganglionic one. The postganglionic sympathetic neuron then may reenter the spinal nerve or exit the ganglion by a special sympathetic nerve and travel to the effector organ. Preganglionic sympathetic neurons may also send axons up or down the sympathetic ganglion chain, where they synapse with postganglionic sympathetic neurons at different levels. In this way, the sympathetic nervous system maintains communication between its different levels up and down the cord. This anatomy makes possible unitary activation of the sympathetic system so that all sympathetic end organs are stimulated maximally at the same time. This type of activation is important for the total sympathetic response involved in flight or fight. In contrast, the parasympathetic system is more diffuse, with more indirect communication between vagal parasympathetic centers in the brain

and sacral parasympathetic centers, and relative independence of activity.

Axons of preganglionic parasympathetic neurons leave the CNS by certain cranial or spinal nerves and travel to the effector organ. At or near the effector organ, they synapse with the postganglionic neuron, which innervates the effector organ.

Acetylcholine is the neurotransmitter synthesized by all preganglionic autonomic neurons—both sympathetic and parasympathetic (see Fig. 32-20, p. 709). Neurons that use acetylcholine as their neurotransmitter are called cholinergic neurons. When an action potential is conducted down the axon of a preganglionic autonomic neuron, acetylcholine is released into the synapse between the axon terminal and the membrane of the postganglionic neuron. It diffuses across the synapse and binds to nicotinic receptors on the membrane of the postganglionic neuron, depolarizing that membrane and possibly causing the postganglionic neuron to develop an action potential. The nicotinic acetylcholine receptors on the postganglionic neuron at its synapse with the preganglionic neuron are similar to, but slightly different from, the nicotinic acetylcholine receptors on the skeletal muscle membrane at the neuromuscular junction. This is why drugs such as vecuronium and tubocurarine are able to block the skeletal muscle nicotinic receptors without affecting ganglionic nicotinic receptors at normal doses.

Acetylcholine also is the neurotransmitter synthesized by the axons of postganglionic parasympathetic neurons (see Fig. 32-20, p. 709). An action potential conducted down the axon of a postganglionic parasympathetic neuron as the result of a strong depolarizing influence received from the preganglionic neuron causes acetylcholine to be released from the axon terminal into the synapse. The acetylcholine diffuses across the synapse and binds to muscarinic acetylcholine receptors on the parasympathetic end organ. These receptors cause changes in the end organ cell that result in smooth muscle contraction, glandular secretion, hyperpolarization of the sinoatrial node of the heart (causing a decrease in heart rate), or slowing in the speed of conduction in the atrioventricular node of the heart. As noted previously, the activity of the acetylcholine is terminated by acetylcholinesterase, an enzyme in the synapse. Because muscarinic receptors are structurally different from nicotinic receptors, although both can bind acetylcholine, drugs have been developed that affect only muscarinic receptors and not nicotinic receptors. An example of such a drug is atropine, which blocks muscarinic receptors and prevents the binding of acetylcholine. This and similar drugs are called muscarinic antagonists (also known as anticholinergics), and their effects are opposite to those of acetylcholine at muscarinic receptors.

Most postganglionic sympathetic neurons synthesize norepinephrine, also called noradrenaline. For this reason, they and other neurons that use norepinephrine as their neurotransmitter are called noradrenergic neurons. When an action potential is conducted down a postganglionic sympathetic neuron because of a strong depolarizing influence received from the preganglionic neuron, norepinephrine is released from the axon terminal into the synapse. It diffuses across the synapse and binds to receptors on the cell membrane of the effector organ. These receptors may be α or β receptors; α receptors may be α_1 or α_2 receptors, and β receptors may be β_1 or β_2 receptors. The heart has mostly β_1 receptors, and the smooth muscle of the arteries and veins has mostly α_1 and α_2 receptors. The sympathetic nervous system innervates organs with α_1, α_2, and β_1 receptors, and norepinephrine activates these receptors to cause changes in the effector organs that have them. For instance, activation of β_1 receptors in the sinoatrial node by norepinephrine released from sympathetic axon terminals results in depolarization of the sinoatrial node and an increase in heart rate. Activation of α_1 or α_2 receptors in the arteries results in increased contraction by arteriolar smooth muscle and an increase in blood pressure.

The adrenal medulla is innervated by the sympathetic nervous system through preganglionic (cholinergic) sympathetic neurons. When an action potential is conducted down their axons, these neurons release acetylcholine from their axon terminals into their synapses. The acetylcholine diffuses across the synapse and binds to nicotinic receptors on the cell membranes of the adrenal medullary cells, triggering the release of some norepinephrine but mostly epinephrine from the adrenal cells into the bloodstream. Circulating norepinephrine and epinephrine can both bind to α and β receptors on sympathetic effector organs, similar to synaptic norepinephrine that is released from sympathetic nerve terminals. However, there is one important difference between norepinephrine and epinephrine. As mentioned, β_2 receptors are not innervated by the sympathetic nervous system, and norepinephrine does not bind or activate β_2 receptors to any degree. However, epinephrine is a powerful stimulator of β_2 receptors, which it reaches through the bloodstream after being secreted by the adrenal medulla. Thus, dilation of bronchiolar smooth muscle and dilation of blood vessels in skeletal muscles are important effects of β_2 receptors that are mediated by circulating epinephrine rather than by norepinephrine released from sympathetic nerve terminals or the adrenal medulla.

Although the sympathetic nerves originate in the thoracic and high lumbar cord, and parasympathetic nerves originate with nuclei that send axons down various cranial nerves or sacral spinal nerves, inputs into the patterns of autonomic function are regulated or triggered by centers in the hypothalamus, medulla, and bulboreticular formations. These centers in the CNS send impulses along descending fibers to the appropriate preganglionic autonomic neuron. In the cord, such fibers travel by special descending tracts in the white matter until they reach the appropriate level of the cord. Thus, any interruption of these descending fibers (eg, transection of cervical tracts) impedes or prevents stimulation of preganglionic autonomic neurons in the thoracic, lumbar, and sacral regions of the cord.

Inputs into the centers in the brainstem and hypothalamus that regulate sympathetic or parasympathetic outflow come from diffuse areas throughout the brain, including visual or auditory centers and areas of the brain associated with conscious thought or planning. Therefore, when we see an alarming sight, such as a car bearing down on us, or hear a frightening noise, sympathetic centers are stimulated and sympathetic outflow increases. Conversely, if we smell food, parasympathetic outflow might increase to prepare the digestive glands for secretion.

In many organ systems, the sympathetic and parasympathetic systems are antagonistic. For instance, the sympathetic system increases the rate of firing of the sinoatrial node and increases the speed of conduction in the atrioventricular node of the heart, whereas the parasympathetic system does

the opposite. The parasympathetic system activates the gastrointestinal tract, whereas the sympathetic system inhibits it. Although this is a recurring theme, it is not an absolute rule. Thus, sympathetic stimulation constricts blood vessels (through α receptors), but blood vessels are not innervated by the parasympathetic system, so an opposite effect is not produced by the parasympathetic system. The sympathetic system stimulates cardiac ventricular contractility (through β_1 receptors), but the ventricles are not innervated by the parasympathetic system, so contractility is not affected by it. The two systems actually work together in the male genitalia, where the parasympathetic system mediates erection while the sympathetic system mediates ejaculation.

Both the sympathetic and parasympathetic systems are tonically active most of the time. One or the other may be more active at a given time, but it is rare that either of them is completely silent. Therefore, a person's heart rate at any given time is a summation of the positive effects of the sympathetic system and the negative effects of the parasympathetic system. At rest, parasympathetic influence is strongest, and the heart rate is slow. With exertion or strong emotion, sympathetic activation increases, and the heart rate speeds up.

▲ Reflexes

Basically, a reflex is a motor response to a sensory input. Reflexes have three components. There is a sensory component, which may consist of only one sensory input or multiple inputs. There is an integrative CNS component that processes the sensory component and "decides" whether it is strong enough to warrant a motor response. Finally, the motor component executes the response. The motor component can consist of one motor nerve and one muscle, or several motor nerves and several muscles. The three components together constitute a "reflex arc." Reflexes are mediated by lower areas of the brain or by the spinal cord, so that they happen without conscious thought. We become aware of the sensory input and the motor response when they are communicated to our cortex, but by then, the reflex is over. However, if we know that a reflexive action is likely, such as when we see someone about to strike our knee with a reflex hammer, we can often suppress a reflex by willing ourselves not to perform the motor action. This capability illustrates that the cortex has input into the integrative CNS component of the reflex. If higher centers are damaged, the reflex still occurs. For example, people with spinal cord transections still have reflexes in areas supplied by spinal nerves below the transection. Of course, they are unaware that these reflexes are taking place because they cannot receive sensory input to their cortex from below the level of the transection.

Brain Reflexes

Brain reflexes include those involving the cardioregulatory and vasomotor centers of the medulla, plus the pupillary adjustment center, which involves the midbrain. Additional reflexes mediated by brain centers include the gag reflex, blink reflex, vomiting, and swallowing.

Because of its importance to critical care, the baroreceptor reflex will be used as an illustration of a brain reflex. The sensory components of the baroreceptor reflex are stretch receptors in various arteries, the most important of which are in the carotid sinuses and the aortic arch. These stretch receptors are actually specialized dendrites of sensory nerves that sense the stretch in the arterial wall produced by the pulse. If the blood pressure is high, the stretch receptors are highly stimulated, whereas if the blood pressure is low, the stretch receptors are not stimulated very much. The stretch receptors send nerve impulses down their dendrites to sensory ganglia near the brain in their respective nerves (cranial nerve IX—the glossopharyngeal nerve—in the case of the carotid sinuses, and cranial nerve X—the vagus nerve—in the case of the aortic arch) that are proportional to the degree of stretch. This information is communicated by sensory axons to autonomic centers in the medulla that process the information and compare it with a "set point" that represents the degree of stimulation they should receive if blood pressure were normal. If the medullary centers receive too little stimulation from the baroreceptors, they send impulses to sympathetic centers to increase sympathetic outflow. This stimulates sympathetic nerves supplying the heart to increase their release of norepinephrine, which binds to β_1 receptors in the sinoatrial node to increase heart rate and β_1 receptors in the ventricles to increase contractility. Sympathetic nerves supplying the veins release norepinephrine, which binds to α receptors, causing constriction of the veins, which increases venous return to the heart. Sympathetic nerves supplying the arteries release norepinephrine, which binds to α receptors, causing constriction of the arteries, which raises the blood pressure. The combination of increased venous return to the heart, increased heart rate, and increased contractility raises the cardiac output, which also increases the blood pressure. Finally, sympathetic nerves supplying the juxtaglomerular apparatus in the kidney release norepinephrine, which binds to β_1 receptors there, stimulating renin release. Through a series of events, renin stimulates the formation of angiotensin II, which is a potent arterial constrictor, increasing blood pressure directly; it also acts on the kidney (through aldosterone) to cause sodium and water retention. Increased retention of sodium and water further increases venous return to the heart, causing additional increased cardiac output and blood pressure. Therefore, activation of the sympathetic nervous system in response to decreased stimulation of the baroreceptors produces many consequences at the effector organs, all of which separately and together cause a rise in blood pressure. If the stretch on the baroreceptors is too high (according to the normal set point), the sympathetic nervous system is inhibited, sympathetic outflow decreases, and consequences at the effector organs are diminished.

Spinal Cord Reflexes

In one type of cord reflex, the sensory component of the reflex is the sensory neurons that send their axons to the cord through one of the spinal nerves, the CNS integrative component is the spinal cord, and the motor component is the motor neurons that supply skeletal muscles. Deep tendon reflexes belong in this classification, as does the withdrawal reflex (Fig. 32-25A). These reflexes are present at each level of the spinal cord, bilaterally.

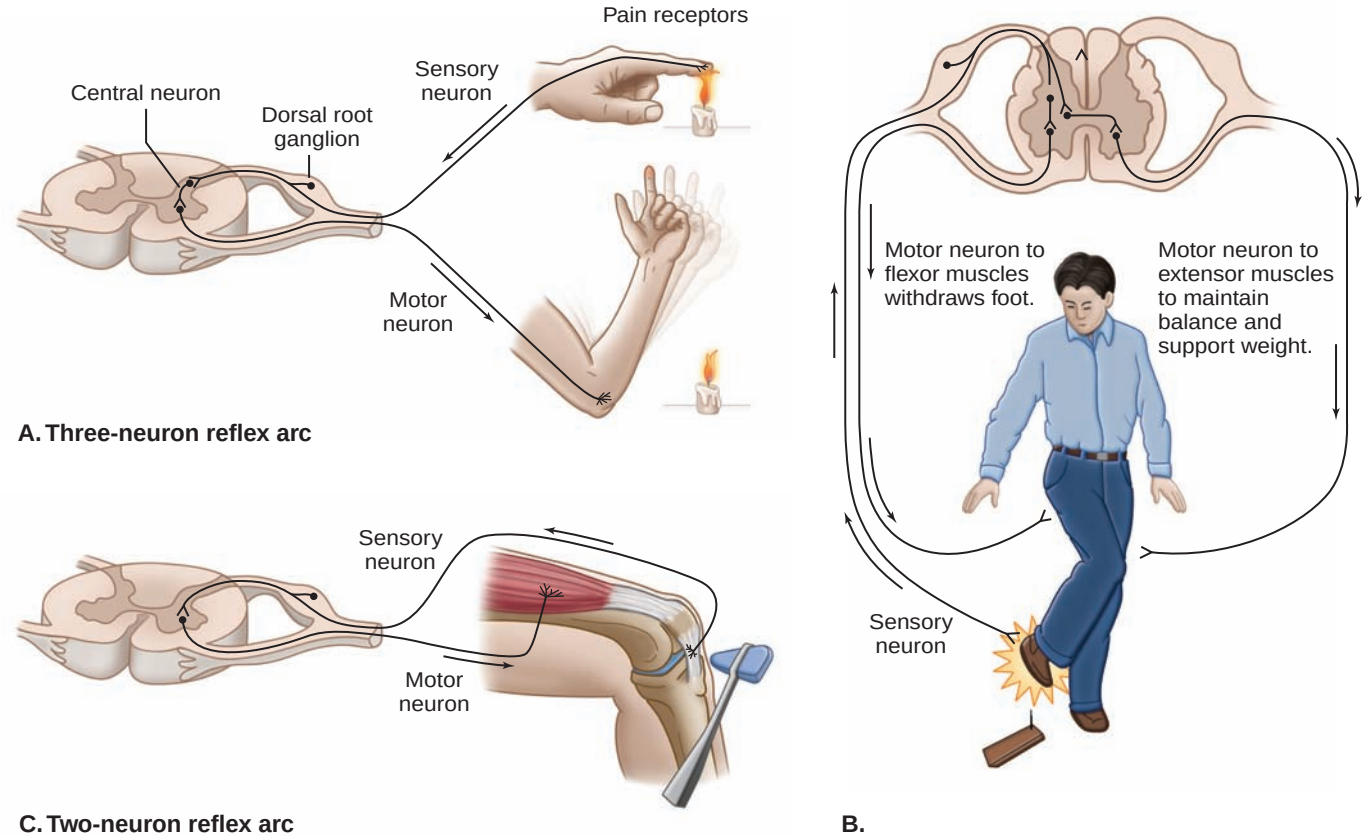


FIGURE 32-25 ▲ Reflex arcs showing pathways of impulses in response to a stimulus. **A**: The withdrawal reflex involves a three-neuron reflex arc: sensory, central, and motor neurons. **B**: The flexor and crossed extensor reflexes. **C**: Example of a stretch reflex, involving only a two-neuron reflex arc: sensory and motor neurons.

The sensory component of the withdrawal reflex is pain that originates in nociceptors, specialized dendrites of sensory neurons. Impulses are conducted through dendrites of the sensory neurons to the dorsal root ganglia next to the spinal cord and from there along sensory axons into the cord. These impulses stimulate cord interneurons, which, if the sensory input is strong enough, stimulate motor neurons whose axons innervate skeletal muscles, causing contraction. When contracted, the skeletal muscles produce withdrawal of the body part from the painful stimulus. The withdrawal reflex depends on the appropriate anatomical connections between sensory neurons, interneurons, and motor neurons in the cord. If these become nonfunctional (eg, spinal shock or physical trauma), this and other spinal reflexes do not occur.

The withdrawal reflex of one foot is associated with another reflex, the crossed extensor reflex (see Fig. 32-25B). This reflex involves stimulation of various extensor muscles in the opposite leg so that a person's weight is fully supported by the other leg while one lower extremity is withdrawn from a painful stimulus. Such a reflex is complex and involves many levels of the cord. Any imbalance, however slight, during the operation of this reflex in a normal person triggers the occurrence of additional reflexes involving the bulboreticular formation, cerebellum, and various muscles of arms and trunk to maintain balance and posture.

Another cord reflex is the stretch reflex or deep tendon reflex, most commonly illustrated by the clinical test of the knee jerk response (see Fig. 32-25C). In the deep tendon reflex, the sensory component is a specialized sense organ, the muscle spindle, which sends its signals along a spinal nerve to the dorsal horn of the spinal cord. The CNS component is a single synapse of the sensory axon terminal with the motor nerve cell body. The motor component is the motor axon supplying the skeletal muscle. In the knee jerk test, a reflex hammer blow stretches the quadriceps tendon, which stretches the muscle spindle, which sends impulses through the dendrite and axon of its nerve cell to release neurotransmitter from the axon terminal. This causes the motor neuron cell body in the spinal cord to depolarize. If the depolarization is strong enough, an action potential is conducted down the axon of the motor neuron to depolarize the muscle through release of acetylcholine into the synapse at the neuromuscular junction, as discussed previously. This causes contraction of the quadriceps, which causes the lower leg to kick forward. Other deep tendon reflexes of clinical importance are the ankle jerk and the biceps and triceps reflexes. All work similarly to the knee jerk.

An important feature of all cord reflexes involving skeletal muscles is reciprocal inhibition, which occurs in the antagonist muscle of the one stimulated. For example, when a flexor reflex stimulates the biceps, it also inhibits its antagonist, the triceps, and provides for more efficient performance of motor activities in the upper arm.

Spinal cord activities also include autonomic reflex circuits, which aid in the control of visceral functions of the body. Sensory input arises from visceral sensory receptors and

is transmitted to the spinal cord, where reflex patterns appropriate to the sensory input are determined. The signals are then transmitted to autonomic motor neurons in the gray matter of the spinal cord, which send impulses to the sympathetic nerves innervating visceral motor end organs.

A most important autonomic reflex is the peritoneal reflex. Tissue damage in any portion of the peritoneum results in the activation of this reflex, which slows or stops all motor activity in nearby viscera, such as the intestine. Other autonomic cord reflexes are capable of modifying local blood flow in response to cold, pain, and heat. This vascular control by autonomic reflexes in the spinal cord can operate as a backup mechanism for the usual brainstem control patterns in patients with transectional injuries at the brainstem. Alternatively, because the autonomic reflexes arising lower in the cord of a patient with a cervical transectional injury are not modulated by brainstem centers as they are in patients without a transection, sensory input to autonomic centers in the cord can cause extreme motor responses, similar to the development of clonus with unmodulated deep tendon reflexes. However, these motor reflexes are sympathetic, and their out-of-control state in spinal injury patients is called autonomic hyperreflexia.

Also included in the autonomic reflexes of the spinal cord are those causing the emptying of the urinary bladder and the rectum. These reflexes are mediated by the sacral parasympathetic system. When the bladder or bowel becomes distended, sensory signals from stretch receptors in the bladder or bowel wall are transmitted by sensory neurons to the internuncial neurons of the upper sacral and lower lumbar segments of the cord. These neurons in turn stimulate parasympathetic motor neurons innervating the smooth muscle in the wall of the bladder or bowel, and their respective internal smooth muscle sphincters also are reflexively inhibited by the internuncials. The result is a reflex contraction of bladder or bowel and an opening of the respective smooth muscle sphincter, thereby permitting micturition or defecation.

In addition to their smooth muscle sphincters, both the bladder and bowel have skeletal muscle sphincters that are controlled by motor neurons. Descending motor fibers from the cortex synapse with the motor neurons, and, in toilet-trained people, keep the skeletal muscle sphincters in a state of contraction, inhibiting the reflex emptying of bladder or bowel at times or places deemed inappropriate. When an appropriate time and place is reached, the person can consciously relax the skeletal muscle sphincter and either void or defecate reflexively. Toilet training of infants must await the functional maturation of these descending motor fibers. Cord transection or other damage above the level of the cord housing the neurons for the bladder or bowel evacuation reflexes interrupts some or all of these descending fibers. This produces a condition in which the patient cannot consciously control (prevent) the emptying of the bladder or bowel, or both. As long as the sacral cord and associated spinal nerves are functioning, voiding or defecation proceeds reflexively in such a patient. Damage to or interrupted function of the level of the cord housing the anatomical neuronal connections for these reflexes (as in, eg, spina bifida, spinal shock, or severe injuries to the lower sacral or lumbar cord) or damage to the spinal nerves supplying the bladder or rectum prevents reflex evacuation of bladder or bowel, or both. Such a patient may exhibit retention with overflow and does not possess any effective mechanism for emptying the bladder or bowel.

▲ Pain

The sensation of pain warrants special consideration because it plays such an important protective role. Whenever there is tissue damage, pain receptors, called nociceptors, are stimulated and send impulses back to the spinal cord. These impulses are transmitted up to the brain, where they are perceived, as previously explained. Stimulation of the nociceptors is caused by the release of substances from damaged tissue and from activation of the inflammatory response. Damaged cells release potassium and hydrogen ion, both of which can stimulate nociceptors. However, the inflammatory response that is evoked in response to tissue damage is responsible for much of the stimulation of nociceptors. For instance, histamine can stimulate nociceptors and prostaglandins, and leukotrienes can sensitize nociceptors to other stimuli. All of these substances are released by inflammatory cells (macrophages, neutrophils, and other white blood cells) that are attracted to the area of tissue injury. In addition, activated platelets participating in clot formation in response to tearing of blood vessels release serotonin, which also stimulates nociceptors. Finally, the nociceptor itself may release substance P when it is stimulated, which sensitizes it to other activating substances. Thus, pain may be due to actual tissue injury or to the inflammatory response evoked by the injury.

Pain Pathways and Their Modulation

The sensation of pain is transmitted to the spinal cord and up to the brain in the same manner as previously described for sensations in general. To review, the nociceptor is actually a specialized dendrite of a sensory neuron, whose cell body is in the dorsal root ganglion of the spinal nerve. When the nociceptor is stimulated enough to mount an action potential, the impulse travels to the dorsal root ganglion, and then down the sensory nerve's axon into the dorsal horn, where it synapses with one or more projection neurons. The projection neurons carry the pain message to the thalamus, where pain is first perceived. The projection neurons synapse in the thalamus with neurons that carry the message to the sensory cortex, where the pain is perceived as a localized sensation.

However, there is an important difference in how pain messages are transmitted to the thalamus and cortex compared with other sensations. This difference is in the way the pain impulse can be modulated by spinal influences before it ascends the cord. In brief, gating mechanisms exist in an area in the gray matter called the substantia gelatinosa at all levels of the dorsal cord. These mechanisms are capable of regulating the number of pain impulses that can enter the ascending tracts and travel to the brain.

To regulate ascending pain impulses, an area of the brainstem called the periaqueductal gray sends axons to the nucleus raphe magnus in the medulla. These axons synapse with neurons that send axons from the nucleus raphe magnus back down to all levels of the cord in the substantia gelatinosa. These neurons regulate the ability of pain-stimulated sensory

neurons to stimulate projection neurons of the spinothalamic tract. Thus, the descending fibers of the substantia gelatinosa control the entry of pain impulses into the spinal pain conduction system at the level of the cord where the particular sensory neuron enters (Fig. 32-26). Sensory stimuli cannot be conducted and, at least to the thalamus, cannot be perceived.

How do the descending fibers modulate stimulation of the projection neurons by the sensory neurons? When researchers answered this question, we also obtained the answer to another perplexing question: How do the opioid drugs relieve pain? The neurons that descend from the nucleus raphe magnus in the medulla, as well as the modulating internuncial neurons, use previously undiscovered small protein neurotransmitters collectively referred to as the endogenous opioid peptides:[7]

- Leucine-enkephalin
- Methionine-enkephalin
- β-Endorphin
- Dynorphin
- α-Neoendorphin

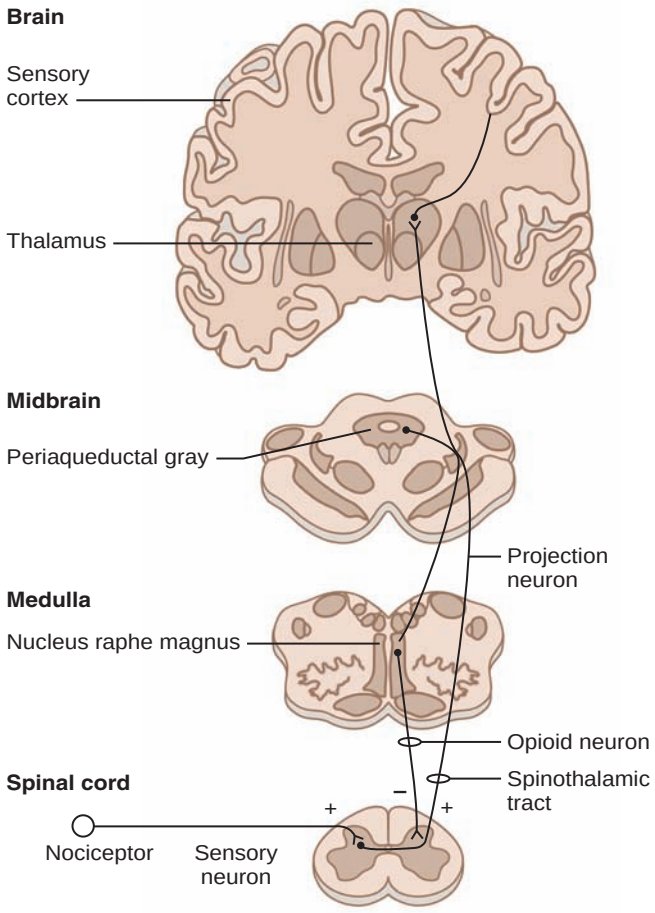


FIGURE 32-26 ▲ Modulation of ascending pain impulses by descending opioid neurons with origins in the nucleus raphe magnus and input from the periaqueductal gray. The sensory neuron's influence on the projection neuron is stimulatory (depolarizing), designated by the plus sign, but the influence of the opioid neuron is inhibitory (hyperpolarizing), designated by the minus sign. Therefore, if the strength of impulses descending in the opioid neuron is high, the projection neuron will experience fewer action potentials and send fewer pain impulses up to the thalamus.

When the endogenous opioid peptides are released into a synapse and bind to their receptors on the postsynaptic cell, they produce hyperpolarization of the postsynaptic cell. As explained previously, that makes the postsynaptic cell less likely to be able to conduct an action potential along its axon. Therefore, the descending fibers that synapse with the projection neurons can produce hyperpolarization in the projection neurons, lessening or perhaps even eliminating the pain messages that would otherwise be conducted upward by the projection neurons. Therefore, the endogenous opioid neurotransmitters, by binding to their receptors on the projection neurons, lessen the perception of pain in the thalamus and cortex. Under extreme circumstances, these descending pathways may be so inhibitory to projection neurons that they eliminate all ascending pain messages, producing complete analgesia to pain. This phenomenon is sometimes seen in victims of automobile crashes or in wounded soldiers, who continue to function, oblivious to their wounds.

What stimulates areas of the periaqueductal gray and nucleus raphe magnus to send these descending inhibitory messages to the substantia gelatinosa, resulting in the release of opioid neurotransmitters and diminution of ascending pain signals? Unfortunately, very little is known about this, but it is possible that acupuncture and electrical stimulation devices for pain control are stimulating these pathways, causing inhibition of the projection neurons by the descending neurons' release of opioid neurotransmitters.

The opioid drugs work in the same way as the endogenous opioid neurotransmitters. They bind to opioid receptors on the projection or internuncial neuron, producing hyperpolarization and a decrease in the amount of pain stimulus reaching the thalamus and the cortex. There are neurons that use the endogenous opioids as neurotransmitters in the brain as well, and opioid drugs also bind to the receptors that these neurons supply. These effects may increase the analgesic effects of the drug, or they may be responsible for other effects, such as the somnolence or dizziness that opioid drugs produce. In addition, there are opioid receptors in the intestinal tract that are stimulated by endogenous opioids and by opioid drugs. These receptors inhibit peristalsis in the intestinal tract, and this effect is responsible for the constipation and nausea often seen with opioid drugs.

Pain is a complex sensation. There is great variation in pain thresholds among different people and within the same person at different times. These variations can partly be explained by the modulation of pain pathways by endogenous opioid neurotransmitters. In addition, the amount of tissue injury and presence of chemical mediators can increase the pain experience qualitatively, quantitatively, temporally, and spatially. However, pain perception is also influenced by expectations and by cultural influences. It is helpful to remember that pain is a perception and that we have to take a person's word in describing that perception to us. It is impossible to judge a patient's pain by his or her appearance or actions, or physical or laboratory signs. The complexity of the pain pathways can make the clinical management of pain difficult, but every patient's description of his or her pain should be taken seriously. Opioid addiction is practically unknown in patients without a history of drug abuse who receive opioids for pain relief. Pain medication should be administered to most patients based on their own self-report of their pain.

Referred Pain

Referred pain is pain perceived as arising from a site that is different from its true point of origin. The "true point of origin" for this type of pain usually is some visceral organ or deep somatic structure, and the "point of reference" is some area of the body surface. Well-known examples include the referring of pain from severe cardiac ischemia to the left arm or the referring of diaphragmatic pain to the neck and shoulder.

The most generally accepted theory for referred pain is that the two sensory neurons, one from the region of the true point of origin and one from the point of reference, enter the same segment of the spinal cord and synapse with the same projection neuron. There is no way for the cortex to know whether a given projection neuron was originally stimulated by pain from the true point of origin or from the referred area. In localizing the source of the pain stimulus, the cortex relies on prior experience regarding the person's geographical knowledge of his or her own body. Because surface areas are more familiar to a person than the locations of the visceral or deep somatic structures, the referred locale is used preferentially over the more unfamiliar but true point of origin (Fig. 32-27).

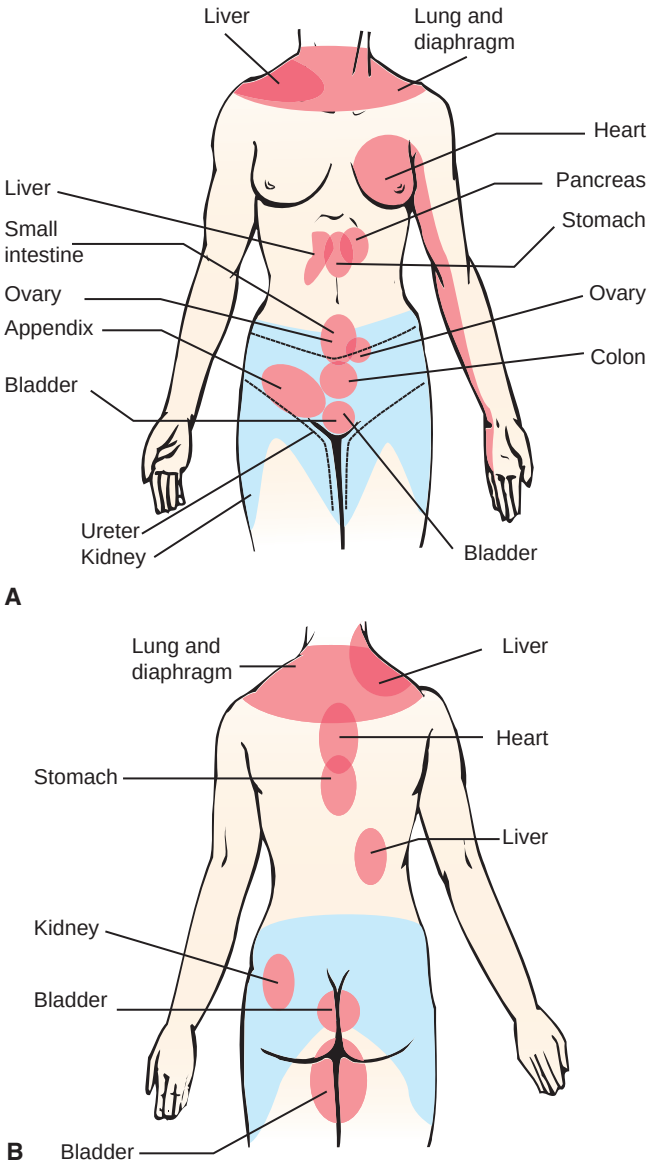


FIGURE 32-27 ▲ Areas of referred pain. **A**: Anterior view. **B**: Posterior view.

▲ The Neurohormonal Stress Response

Homeostasis

In the middle of the 19th century, the French physiologist Claude Bernard (1813–1878) coined the term milieu intérieur to mean the internal environment to which body cells are exposed. He stated that to maintain proper cell functioning, the milieu intérieur must be constant and proper.[1,2]

> The living body, though it has need of the surrounding environment, is nevertheless relatively independent of it. This independence which the organism has of its external environment, derives from the fact that in the living being, the tissues are in fact withdrawn from direct external influences and are protected by a veritable internal environment which is constituted, in particular, by the fluids circulating in the body.
>
> *Opening lecture in general physiology given to the* College de France, *as quoted in Scultz SG: The internal environment. In Johnson LR (ed): Essential Medical Physiology, 2nd ed. New York, Raven Press, 1998.*

Although Bernard thought the blood constituted the milieu intérieur, we now realize that each tissue and cell type probably has its own environment that may be different from that of other tissues or cell types. However, the idea of a constant and appropriate internal environment remains valid to this day. Bernard was the first to advance the concept that bodily processes are constantly responding to the external environment with mechanisms that maintain constancy of the milieu intérieur: "The constancy of the internal environment requires such a perfection in the organism that external variations are instantly compensated for and balanced."[1] The concept of a constant internal environment was expanded by Walter Canon in the early 20th century to include all bodily process and structures and was termed homeostasis.

Homeostasis is defined as the situation in which attributes of the body, such as blood pressure, level of alertness, and muscle tone, remain constant or change appropriately for different situations. The constant nature of these attributes is due to the right balance between stimulatory and inhibitory neuronal and hormonal influences. In addition, structural components, such as the composition of blood, composition of extracellular fluid, and bones, tendons, muscles, and internal organs, remain essentially constant even though their components are being constantly degraded and resynthesized. Again, this structural constancy is maintained through a combination of neural and hormonal mechanisms. Homeostasis is a dynamic equilibrium—dynamic because of the constant degradation and renewal, or stimulation and inhibition, and an equilibrium because there is a balance between these opposing processes such that conditions remain constantly the same. When stressors disturb the dynamic equilibrium, compensatory mechanisms return it to its previous steady state.

Disruption of Homeostasis

Although each of us is continually exposed to psychological and physiological stressors, we are usually able to maintain a state of emotional and physical health through compensatory mechanisms that maintain homeostasis. In fact, it has been shown that some level of stress is beneficial to health. However, in the event of stress that overwhelms compensatory mechanisms, we can become ill. Overwhelming stressors may become so because of their large magnitude over time, or because of their sudden onset. People who present as patients in critical care settings have usually experienced recent, severe physiological stressors, but these are superimposed on their level of chronic physiological and psychological stress. In addition, the patient is exposed to continuous stressors that are by-products of the critical care setting, such as monitoring devices, invasive procedures, and the continual presence of devices for intravenous access, endotracheal intubation, and others.[3]

Although others, especially Walter Cannon (who also coined the term homeostasis), presented some of these theories earlier, Dr. Hans Selye[5,6] was the most prolific researcher and author on the topic of stress. Stress is "any external or internal factor that affects the normal state of dynamic equilibrium in an individual." These factors, or stressors, can be either physical or psychological. Regardless of whether the person consciously perceives a threat, the body responds with certain intrinsic reactions. When the effects are realized by the conscious mind, the person perceives a stressful experience. The stress response is how a person reacts to the stressors that he or she encounters (ie, the person's physiological adaptation or psychological coping mechanisms). One person may have compensatory mechanisms with a large capacity to handle stressors, whereas another person may not. Therefore, exposure to a given stressor does not elicit the same response in all people. This difference may be based on internal conditioning factors, such as age, sex, and genetics, and on external factors, such as culture, previous life events, exposure to this or similar stressors, diet and nutrition, and medications.

Stressors may be either psychological or physiological in origin. Psychological stressors are additive in nature (eg, life events, such as a job change, divorce, marriage) and have a physiological impact. Physiological stressors, such as injury or infection, disrupt homeostasis, eliciting the stress response in an attempt to restore it. If homeostasis is not restored quickly, illness results.

General Adaptation Syndrome

Selye and his collaborators noted commonalities in responses to different stressors in different individuals. They termed these commonalities the general adaptation syndrome. Although this term has fallen from favor, and the changes they noted are probably not as general as they thought, the concept of a generalized response to stress remains. They also defined the three basic stages of the stress syndrome as the alarm reaction, stage of resistance, and stage of exhaustion.

- *Alarm reaction.* During this initial stage, the threat is perceived, either consciously or subconsciously, and body processes are modified to counteract it. The sympathetic nervous system is stimulated by the stressor, and there is a subsequent response through the release of norepinephrine and epinephrine. Additionally, adrenocorticotropic hormone (ACTH) and ADH are released by the anterior and posterior pituitary. Stimulation of the sympathetic nervous system raises heart rate and blood pressure and stimulates the renin–angiotensin–aldosterone system, which results in sodium and water retention, increasing the blood pressure further. ACTH stimulates the release of cortisol by the adrenal cortex, which produces numerous adaptations to stress, outlined below. ADH raises blood pressure mainly by causing the kidney to retain water. These effects are additive to those elicited by the sympathetic nervous system and the renin–angiotensin–aldosterone system.
- *Stage of resistance.* During the second stage, the stress is being compensated for by increased activity of the stress responses evoked during the alarm phase. Cortisol secretion, the sympathetic nervous system, and other mechanisms triggered in the alarm reaction may continue to be activated at a lower, more constant level. This phase may continue for a long time, even years, if the increased levels of stress response mechanisms are maintained. However, the increased levels of stress response mechanisms come at a price—the use of additional resources of energy and nutrients. It is during this stage that symptoms of disease may become chronic if the compensatory responses are not adequate to control them.
- *Stage of exhaustion.* The ability to mount a stress response has limits. Therefore, the stress response can be activated only for a finite time or to a finite degree. If the stressor is not removed or adaptation does not occur, the person is no longer able to resist the stressor. At this point, homeostasis is no longer achievable. A shock state may occur (see Chapter 54), and without appropriate intervention, organ failure and death may rapidly ensue.

The Stress Response

ACUTE STRESS. Consider a prehistoric man walking on the African veldt. Suddenly, a lion springs from behind some vegetation. The man's eyes capture an image of the lion, which is communicated to the visual areas in his occipital cortex. From there, the image is sent to and processed by his prefrontal and frontal cortex and perceived as a threat. This information of threat is communicated to many centers in his brain, including those supplying the ANS. Immediately, sympathetic outflow is greatly increased by activation of brainstem sympathetic centers and through unitary activation of the sympathetic nervous system (remember the autonomic ganglia that communicate with each other at all levels of the spinal cord), increasing the rate of firing of his sympathetic nerves, releasing norepinephrine into sympathetic synapses. At the same time, parasympathetic outflow is greatly inhibited, decreasing activity of the gastrointestinal system and the need of those organs for blood. The man's heart rate and cardiac contractility (β_1 receptors) are greatly increased, both of which increase his cardiac output, and his resistance arterioles constrict (α receptors). Increased cardiac output and increased arteriolar resistance raise his blood pressure. At the same time, veins also constrict (α receptors), increasing venous return to his heart and increasing cardiac output still more. The adrenal medulla is stimulated by sympathetic outflow (neuronal nicotinic receptors) to secrete some norepinephrine, but mostly

epinephrine, into the bloodstream. The epinephrine stimulates α and β_1 receptors all over the man's body, causing the same effects as norepinephrine does at those receptors. It also stimulates β_2 receptors on his bronchiolar smooth muscle, causing relaxation and dilation of the bronchioles, enabling him to inspire and expire greater volumes of air. In addition, it stimulates β_2 receptors on arterioles in his skeletal muscle beds, causing profound dilation, increasing the capacity of these beds for blood flow. Because arterioles to digestive and other internal organs are constricted by virtue of α-receptor activation, the greatly increased cardiac output is directed to skeletal muscle beds, supplying the muscle cells with increased amounts of oxygen and glucose to sustain increased contraction. Blood flow to the brain is also greatly increased by the increased cardiac output because arterioles supplying the brain have few α receptors, so they remain fully dilated. Stimulation of β_1 receptors in the juxtaglomerular apparatus of the kidney by norepinephrine (from sympathetic nervous system nerve terminals and the adrenal medulla) and epinephrine (from the adrenal medulla) causes the release of renin, which activates the renin–angiotensin–aldosterone system. Renin acts on circulating angiotensinogen to angiotensin I, which is further converted to angiotensin II by angiotensin-converting enzymes. Angiotensin II causes additional arteriolar constriction, increasing blood pressure still further. It also causes the release of aldosterone, which causes the kidney to retain sodium and water. Sodium and water retention increases the preload to the heart, increasing cardiac output still further.

In addition, communication of the threatening sight to the hypothalamus activates many neurohormonal mechanisms controlled by the pituitary. We consider only three of these. First, the hypothalamus increases its synthesis and release of ADH from the posterior pituitary. ADH causes the kidney to retain water, further increasing the preload and thereby the cardiac output. Second, the hypothalamus increases its secretion and release of corticotropin-releasing hormone, which causes the anterior pituitary to secrete additional ACTH, which in turn causes the adrenal cortex to release increased quantities of cortisol. Cortisol has far-reaching effects on many organs that increase their ability to respond to stress. Third, through increased synthesis and release of growth hormone–releasing hormone by the hypothalamus, growth hormone (GH) synthesis and release by the anterior pituitary is increased. Like cortisol, GH has many far-reaching effects on many organs, but its overall effect is to increase the activity of tissue repair mechanisms and utilization of nutrients.

Finally, the man's immune system is activated by the stress response. This activation is achieved by multiple influences, only a few of which are mentioned here. First, GH increases the ability of many cells of the immune system, such as neutrophils and T and B lymphocytes, to carry out their functions, including phagocytosis, antigen presentation, and antibody production. In addition, the physiologically high levels of cortisol affect the immune system's ability to respond to foreign antigens. (Levels produced by pharmacological doses of corticosteroids are much higher than those produced by stress and are immunosuppressive.) The effects of physiologically elevated levels of cortisol in response to stress on immune function are complex and may involve effects on the ability of immune cells to exit the circulation and go to sites of tissue injury, or their ability to respond to antigen presentation. Finally, catecholamines also affect the immune system's ability to respond to tissue injury or foreign antigens. The net response of the immune system to acute influences of the stress response is generally considered to be an increased ability to mount an inflammatory response and respond to a foreign antigen. Figure 32-28 summarizes the effects on various body systems.

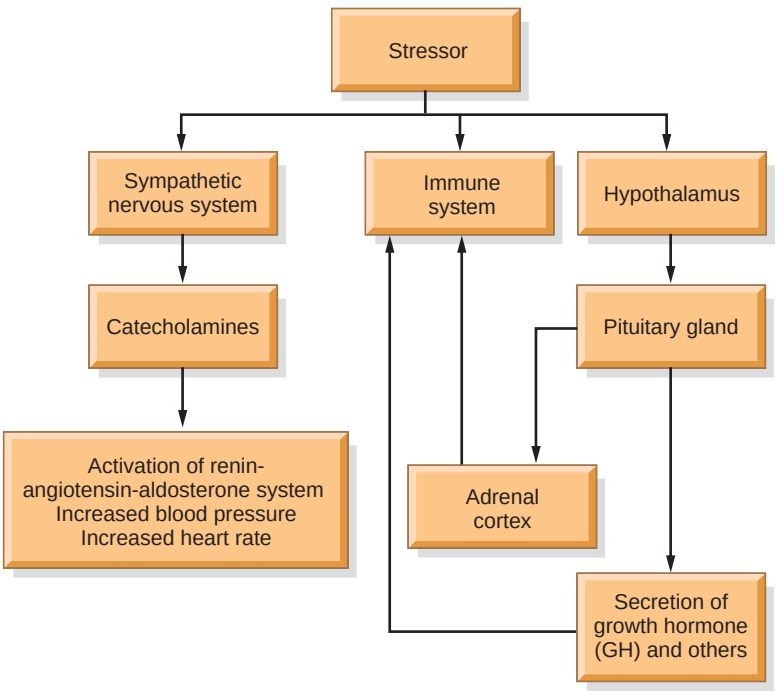


FIGURE 32-28 ▲ The stress response induces increased activity in the sympathetic nervous system, including the adrenal medulla, which activates the renin–angiotensin–aldosterone system and increases the blood pressure and heart rate, among other things. The stress response also activates the pituitary gland, which secretes increased growth hormone; the adrenal cortex, which secretes increased cortisol; and the immune system.

Returning to our prehistoric man threatened by a lion, the changes produced in his body by the threatening sight of a lion all have the effect of increasing the ability of his muscles to help him run faster or fight harder to escape the lion and increasing his ability to respond to tissue injury that might result from his encounter with the lion. These changes constitute the alarm reaction characterized by Selye. The lion threat represents an acute stressor that would end very quickly, because the man either escaped or was killed. If the man was successful in escaping the lion, the stress response would extinguish itself very rapidly, and his physiological state would gradually return to normal.

The question that arises for modern humans is, "How are the changes of the alarm reaction beneficial for stresses encountered in modern times?" If one is running to get away from a dangerous situation or to catch a bus, is injured in an automobile crash, or contracts an acute illness, the acute stress response is very likely still beneficial. However, if one undergoes an acute emotional stress, such as an intense argument or the death of a loved one, the alarm reaction is invoked, the same as it would be for a more physiologically oriented stress. In such circumstances, we do not need increased ability to fight or run away, decreased activity of our gastrointestinal system, or increased ability for tissue repair. Therefore, in these circumstances, the acute stress response is at best superfluous and at worst consumes resources and unnecessarily creates wear and tear on body systems.

CHRONIC STRESS. Selye's "stage of resistance" describes our ability to handle chronic stress. Chronic stressors that affected prehistoric humans included starvation or extreme heat or cold. Responses to these stressors included some of the same mechanisms as outlined earlier for acute stress but at diminished levels that are more sustainable over a long period. However, responses to these stressors also included additional mechanisms that conserve body stores of nutrients and energy. These responses include cortisol, but also insulin, glucagon, and GH. The chronic stress response in prehistoric times probably did not include response to diseases. Because disease processes could not be treated, affected people quickly died during the acute stress response. The mechanisms that evolved prehistorically to handle chronic stress may or may not be appropriate to handle modern stressors such as chronic disease or the chronic emotional stress associated with constant deadlines or commuting in heavy traffic. Moreover, with modern medical care, we now have the ability to maintain people with extremely high levels of chronic stress due to disease, their emotional state, and the stress induced by our interventions. In intensive care units, we measure many aspects of homeostasis, such as electrolyte levels, blood cell counts, cardiac functioning, and hormonal levels, and adjust our care to preserve homeostasis (a proper milieu intérieur).

In many such situations, the chronic stress responses as they have been handed down to us from prehistoric times may actually be counterproductive and decrease our ability to maintain homeostasis. In addition, increased activity of stress responses may increase the likelihood of degenerative diseases of the circulatory system, such as atherosclerosis, leading to vascular events in the heart, periphery, or brain; disorders of glucose metabolism that lead to type 2 diabetes; or, perhaps, disorders of the immune system that lead to inflammatory diseases. These types of diseases are all the result of a complex interplay between a person's genetic makeup, his or her life experiences (including exposure to antigens and infectious diseases), and level of both physiological and psychological stress.

▲ Age-Related Changes

Because of age-related changes in the nervous system, older people are at higher risk for injury and have less chance for survival after a severe injury. Impairment of sensation, proprioception, gait, vision, and hearing and delayed response time are a few factors that predispose older people to injury. Considerations for the older patient are given in Box 32-1.

BOX 32-1 CONSIDERATIONS FOR THE OLDER PATIENT

Anatomical and Physiological Changes in the Nervous System That Occur With Aging

- Cerebral atrophy results in a decrease in total brain weight and volume, especially in the frontal and temporal lobes; enlargement of the ventricles; and a loss of gray matter.
- Cerebral atrophy causes the dura mater and bridging veins to become tightly adherent to the skull; thus, they are easily torn with significant movement of the cranial contents, leading to subdural hematoma formation.
- Cerebral atrophy creates more space for intracranial blood to be concealed, so the older patient may manifest only subtle symptoms, which may lead to a delay in diagnosis.
- Axonal loss or decreases in myelination result in a loss of white matter.
- There is atrophy of the hippocampus, which correlates with a decline in learning and memory and cognitive impairments.
- A decreased number of neuronal cells and degeneration of dendrites and dendritic spines in cortical pyramidal cells lead to declining synaptic transmission and slowed impulse conduction.
- There is decreased production, release, and metabolism of neurotransmitters.
- Altered circulation in the inner ear and fewer functional cochlear cells lead to reduced hearing.
- A decreased number of olfactory cells in nasal mucosa lead to a reduced sense of smell.
- There is an increase in wakefulness and arousal from sleep and a decrease in slow-wave sleep, leading to changes in sleep patterns.
- The odontoid process in the cervical spine is most commonly fractured because of osteoporosis and degenerative joint disease.
- Central cord syndrome occurs more frequently because of spinal stenosis.
- The patient is more prone to severe brain injury and may have less reserve to survive a severe injury.
- The incidence of dementia somewhat increases. Those who develop dementia have a decline in cognitive and emotional abilities, which can affect memory, language, visuospatial skills, complex cognition, emotion, and personality.

▲ Clinical Applicability Challenges

SHORT ANSWER QUESTIONS

1. A 55-year-old man is brought to the emergency department after having a seizure. A CT scan of the brain reveals a large left frontoparietal mass. The patient is neurologically intact. The patient is taken to the operating room for resection of the mass. Explain the symptoms this patient could experience postoperatively.

2. A 38-year-old female is brought to the emergency department after developing the worst headache of her life. CT scan of the brain revealed a subarachnoid hemorrhage. CT angiography (CTA) of the brain confirmed the presence of a left middle cerebral aneurysm (MCA). The patient was taken to the interventional radiology suite for coiling of the MCA aneurysm. After the procedure, the patient is neurologically intact. On day 3, the patient becomes lethargic and restless. Explain what complications this patient could be experiencing. How would these complications best be treated?

3. A 21-year-old male is brought to the emergency department after diving into shallow water. Now he is unable to move his extremities. The patient is found to have a C5 fracture/dislocation resulting in traumatic quadriplegia. The patient is experiencing bradycardia, hypotension, and hypothermia. Explain why neurogenic shock occurs and differentiate it from spinal shock.

References

1. Conti F: Claude Bernard: Primer of the second biomedical revolution. Nat Mol Cell Biol 2:703–708, 2001
2. Conti F: Claude Bernard's Des Fonctions du Cerveau: An ante litteram manifesto of the neurosciences? Nat Neurosci 3:979–985, 2002
3. Daube JR, Rubin DI: Clinical Neurophysiology: Contemporary Neurology Series, 3rd ed. Oxford: Oxford University Press, 2009
4. Hickey JV: The Clinical Practice of Neurological and Neurosurgical Nursing, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009
5. Selye H: The general adaptation syndrome and the diseases of adaptation. J Clin Endocrinol 6:117, 1946
6. Selye H: Stress Without Distress. Philadelphia, PA: JB Lippincott, 1974
7. Young PA, Young PH, Tolbert DL: Clinical Neuroscience, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

33 Patient Assessment: Nervous System

Genell Hilton

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Perform a comprehensive neurological assessment.
2. Describe abnormal assessment findings consistent with neurological compromise.
3. Analyze assessment findings and identify potential nursing diagnoses.
4. Evaluate the effect of neurological dysfunction on the patient.
5. Discuss preprocedure and postprocedure nursing interventions appropriate to selected neurodiagnostic tests.

Assessment and care of a patient with a neurological problem constitute one of the biggest challenges for critical care nurses. Basic nursing education and critical care courses may not address the assessment of the nervous system to the depth or complexity observed with other body systems. In addition, a comprehensive neurological assessment involves the use of techniques not commonly performed in the assessment of other body systems. Therefore, it is not uncommon for even the experienced nurse to feel uncertain when gathering data about the nervous system.

There are four major objectives in the nursing assessment of a patient with a real or potential neurological problem. The first objective is to gather data about the functioning of the nervous system in an unbiased and orderly manner, avoiding inconsistencies in data collection or inadequate data collection. It is essential that examination results be recorded clearly so that changes in findings can be easily identified. A standard neurological check sheet should be used by all the nursing staff, with clearly defined grading scales or terms listed as appropriate.

The second objective of neurological assessment is to follow the data over time, discovering correlations and trends. For such correlations to be of value, it is necessary to interrelate the results of history, physical assessment, and diagnostic tests. Use of a patterned format helps establish medical and nursing diagnoses and guides the nurse in choosing and evaluating therapy.

The third objective of neurological assessment is to analyze the data to develop a list of potential or actual diagnoses. Minor changes in neurological status may be the first indication that the patient's physical condition is worsening. The nurse providing care to the patient is responsible to recognize these changes, correlate these findings to the pathophysiological process, and intervene appropriately.

The fourth objective of the neurological nursing assessment is to determine the effect of dysfunction on the patient's daily living and ability to perform self-care. Up to this point, the goals of physicians and nurses in the care of a patient with a neurological problem have been similar. Each discipline uses many of the same questions and techniques to determine normal and abnormal nervous system functioning. The focus of nursing is to help patients cope with real or potential changes in daily living and self-care.

These objectives of neuroassessment are the same for all patients. In older patients, it is necessary to take the normal changes of aging into account when assessing for neurological problems. Older adults are at increased risk for certain medical conditions that predispose them to neurological problems. These same medical conditions or their prescribed treatment may also alter neurological assessment findings. Special considerations for older adults are given in Box 33-1.

▲ History

Neurological assessment begins with the first patient encounter. Conversation with the patient and family is a vital source of data needed to evaluate overall functioning. The nurse ascertains the reason for the patient's visit and obtains data regarding symptoms and evaluates the patient's past medical history, family history, and personal and social history. A comprehensive review of systems is also performed as part of the initial assessment (Box 33-2).

▲ Physical Examination

A comprehensive neurological evaluation of the critically ill patient includes assessment of mental status, motor function, pupillary response, cranial nerve function, reflexes, and sensation. Findings are correlated with the vital signs.

BOX 33-1 CONSIDERATIONS FOR THE OLDER PATIENT

Neuroassessment

When assessing an older adult, it is necessary to ascertain the person's previous level of functioning to adequately assess the person's status. The following should be taken into consideration when the nurse assesses an older adult's neurological function:

- Motor function may be affected by decreased strength, alterations in gait, changes in posture, and increased tremors.
- Vision may be decreased, pupils may be less reactive, color discrimination may be decreased, gaze may be impaired, and night vision may be diminished.
- Hearing may be diminished and changes in Rinne test findings may be noted. The nurse should bear in mind that an undetected hearing impairment can lead to the erroneous assumption that a person has more neurological deficits than he or she actually has.
- Changes in sensory function may include decreased reflexes, decreased vibratory and position sense, and decreased two-point discrimination.
- Older adults are at increased risk for depression, nutritional abnormalities, stroke, transient ischemic attacks, and dementia.
- Older adults may have impaired sleep patterns.

Mental Status

The mental status examination includes tests to evaluate level of consciousness and arousal, orientation to the environment, and thought content. The quality of a patient's level of consciousness is the most basic and critical parameter requiring assessment. Level of consciousness evaluates the functioning of the cerebral hemispheres as well as that of the reticular activating system, which is responsible for arousal. The degree of a patient's awareness of, response to, and interaction with the environment is the most sensitive indicator of nervous system dysfunction. Responsivity may be categorized according to the patient's arousal to external stimuli, and gradations of response include terms such as lethargic, stuporous, and semicomatose (Box 33-3).

Orientation to the environment involves assessing not only the patient's ability to respond but also the content of his or her response. This is assessed by asking the patient

BOX 33-2 HEALTH HISTORY for Neurological Assessment

Chief Complaint

- One-sentence description, in patient's own words, of why the patient is seeking care

History of Present Illness

- Complete analysis of the following signs and symptoms (using the NOPQRST format; see Chapter 17, Box 17-1, p. 207)
- Dizziness, syncope, or seizures
- Headaches
- Vision or auditory changes, including sensitivity to light and tinnitus
- Difficulty swallowing or hoarseness
- Slurred speech or word finding difficulty
- Confusion, memory loss, or difficulty concentrating
- Gait disturbances
- Motor symptoms, including weakness, paresthesia, paralysis, decreased range of motion, and tremors

Past Health History

- Relevant childhood illnesses and immunizations: febrile seizures, birth injuries, physical abuse or trauma, meningitis
- Past acute and chronic medical problems, including treatments and hospitalizations: tumors, traumatic head injuries, hypertension, thrombophlebitis or deep venous thrombosis, coagulopathies, sinusitis, meningitis, encephalitis, diabetes, cancer, psychiatric disorders
- Risk factors: diabetes, smoking, hypercholesterolemia, hypertension, drug use, alcohol use, cardiovascular disease
- Past surgeries: peripheral vascular surgeries; carotid endarterectomy; aneurysm clipping; evacuation of hematoma; head, eyes, ears, nose, or throat (HEENT) procedures
- Past diagnostic tests and interventions: electroencephalography, brain scan, carotid Doppler, head and neck computed tomography, magnetic resonance imaging, thrombolytic therapy, cardiac catheterization
- Medications: anticonvulsants, anticoagulants, psychotropic agents, oral contraceptives, b-blockers, calcium channel blockers, antihyperlipidemics, hormone replacement therapy
- Allergies and reactions: contrast medium, medications
- Transfusions including type and date

Family History

- Health status or cause of death of parents and siblings: coronary artery disease, peripheral vascular disease, cancer, hypertension, diabetes, stroke, hyperlipidemia, coagulopathies, seizures, psychiatric disturbances

Personal and Social History

- Tobacco, alcohol, and substance use
- Family composition
- Occupation and work environment: exposure to chemicals and toxins
- Living environment: physical, verbal, and emotional abuse
- Diet
- Sleep patterns
- Exercise
- Cultural beliefs
- Spiritual and religious beliefs
- Coping patterns and social support systems
- Leisure activities
- Sexual activity
- Recent travel

Review of Systems

- HEENT: visual changes, tinnitus, headache
- Cardiovascular: hypertension, syncope, palpitations, intermittent claudication
- Respiratory: shortness of breath, infections, cough, dyspnea
- Gastrointestinal: weight loss, change in bowel habits, nausea/vomiting/diarrhea
- Genitourinary: change in bladder habits, painful urination, sexual dysfunction
- Musculoskeletal: sensitivity to temperature changes, varicosities, loss of hair on extremities, change in sensation

BOX 33-3 Clinical Terminology for Grading Responsiveness

Alert (full consciousness): normal
Awake: may sleep more than usual or be somewhat confused on first awakening, but fully oriented when aroused
Lethargic: drowsy but follows simple commands when stimulated
Obtunded: arousable with stimulation; responds verbally with a word or two; follows simple commands; otherwise drowsy
Stuporous: very hard to arouse; inconsistently may follow simple commands or speak single words or short phrases; limited spontaneous movement
Semicomatose: movements are purposeful when stimulated; does not follow commands or speak coherently
Comatose: may respond with reflexive posturing when stimulated or may have no response to any stimulus

questions such as, "What is your name? Where are you right now? What is the month/year/date/time?" An increase in the number of wrong answers indicates increasing confusion and possible deterioration in neurological status. Likewise, an increase in the number of correct answers may indicate neurological improvement.

In instances in which brain injury is suspected, the Glasgow Coma Scale (GCS) has proved a reliable tool for assessing arousal and level of consciousness (Box 33-4). The GCS allows the examiner to record objectively the patient's response to the environment in three major areas: eye opening, verbalization, and movement. In each category, the best response is scored. The GCS uses two responses, best eye-opening response and best verbal response, to assess arousal and level of consciousness. Best eye-opening response is scored from 1 to 4, with 1 as no response and 4 as spontaneous eye opening. Best verbal response addresses orientation and ranges from 1 to 5, with 1 again indicating no response and 5 indicating a fully oriented patient. The intubated patient is usually noted to have a verbal score of 1T, which should be added into the total score. In this way, recognition is given to the patient's inability to speak secondary to the presence of the endotracheal tube. Best motor responses ranges from 1 to 6, with 1 indicating no motor response and 6 representing a patient with movement of all extremities to command.

BOX 33-4 The Glasgow Coma Scale

Best Eye-Opening Response	Score
Spontaneously	4
To speech	3
To pain	2
No response	1
Best Verbal Response	**Score**
Oriented	5
Confused conversation	4
Inappropriate words	3
Garbled sounds	2
No response	1
Best Motor Response	**Score**
Obeys commands	6
Localizes stimuli	5
Withdrawal from stimulus	4
Abnormal flexion (decorticate)	3
Abnormal extension (decerebrate)	2
No response	1

A total score of 3 to 8 suggests severe impairment, 9 to 12 suggests moderate impairment, and 13 to 15 suggests mild impairment.

The maximal total score for a fully awake and alert person is 15. A minimal score of 3 is consistent with complete lack of responsiveness. An overall score of 8 or below is associated with coma. If maintained over time, a low GCS score may be a predictor of poor functional recovery. This scoring system was designed as a guide for rapid evaluation of the acutely ill or severely injured patient whose status may change quickly. It is not useful as a guide for evaluation of patients in long-standing comas or during prolonged recovery from severe brain injury.

More complex information about nervous system functioning can be obtained by gathering data about the patient's ability to integrate attention, memory, and thought processes (Table 33-1). Such a mental status examination also may uncover clues about additional problems affecting the patient's lifestyle. The Mini-Mental State Examination (MMSE) is a widely used cognitive assessment tool that is easy and rapid to administer and has good interrater reliability. It is frequently used to monitor disease progression in patients with dementia or other progressive disease states. The MMSE is composed of questions related to orientation, recall/memory, attention, calculation, language, and spatial insight. Points are assigned for correct answers, with a maximum of 30 points. Scores of less than 20 may indicate neurological disease. Examples of specific deficits are presented in Table 33-2.

When gathering such a wealth of data, assessment of the patient's ability to communicate becomes paramount. Use of language requires comprehension of verbal and nonverbal symbols and the ability to use those symbols to communicate with others. Evaluation of the patient's understanding normally is accomplished through the spoken word. However, speech dysfunctions can make such evaluations exceedingly difficult (Table 33-3). Use of an interpreter to ensure accurate assessment of orientation may be indicated in patients whose primary language is other than English.

Motor Function

Evaluation of motor function includes assessment of motor response to stimuli, as well as motor strength and coordination. Assessment of motor response involves evaluating the type of stimuli necessary to elicit a motor response. This gives the health care team information regarding the level of awareness necessary to obtain a motor response as well as the patient's ability to follow commands. Evaluation of motor strength and coordination assesses motor neuron pathways within the brain, from the primary motor cortex to the spinal cord, as well as multiple other areas involved in coordination, such as the cerebellum and basal ganglia.

Motor Response to Stimuli

The nurse first attempts to elicit a motor response by asking the patient to move an extremity against gravity. If no response is

Table 33-1 Mental Status Examination

Functions	Test	Implications
Orientation	*Time:* state year, month, date, season, day of week *Place:* indicate state, county, city of residency; state hospital name, floor or room number	May be altered by a multitude of neurological conditions
Attention	Digit span; serial 7's; recitation of months of the year in reverse order	May be impaired in delirium, frontal lobe damage, and dementia
Memory	*Short-term:* recall of three items after 5 min *Long-term:* recall of such items as mother's maiden name, events of previous day	May be impaired in conditions such as dementia, cerebrovascular accident, and delirium
Language	*Naming:* point to three objects and have patient name them *Comprehension:* give simple and complex commands *Repetition:* repeat phrases such as "no if's, ands, or buts" *Reading:* have patient read and explain a short passage *Writing:* have patient write a brief sentence	Requires integration of visual, semantic, and phonological aspects of knowledge Dysfunction may be associated with lesions of Broca's area; may be dependent on educational level
Spatial/perceptual	Copy drawings such as cross or square; draw a clock face Point out right and left side of self Demonstrate such actions as putting on a coat or blowing out a match	May be associated with parietal lobe lesions

Table 33-2 Selected Deficits in Higher Intellectual Function

Type	Characteristics
Anomia	Inability to name objects or recognize written or spoken names of objects
Phonemic paraphasia	Substitutes parts of words (eg, pan opener instead of can opener)
Semantic paraphasia	Substitutes whole words (eg, apple for orange)
Dyslexia	Inability to recognize and comprehend written words
Alexia	Reading letter by letter instead of whole words
Neglect dyslexia	Omissions or substitutions of letters confined to initial part of the word
Surface dyslexia	Difficulty reading words with irregular spelling
Dysgraphia	Difficulty with writing
Central dysgraphia	Affects both written and oral spelling
Neglect dysgraphia	Misspelling the initial part of the word
Agnosia	Failure to recognize objects despite intact sensory input; may be visual, auditory, or sensory
Prosopagnosia	Inability to recognize familiar faces
Achromatopsia	Inability to discriminate colors
Acalculia	Inability to read, write, and comprehend numbers

forthcoming, the patient may be unable to comprehend or to respond to verbal commands. In this instance, noxious stimuli should be used to elicit a motor response. When noxious stimuli are needed to evoke a response, the nurse pays careful attention to where the painful stimulus is applied. Noxious stimuli can involve either central or peripheral stimulation. Central stimulation involves pinching of the trapezius muscle, pressure on the supraorbital ridge, or a sternal rub while peripheral stimulation response may be elicited by compression of the nail bed. However, be aware that a misplaced examiner's hand may cause serious skin or tissue injury. Areas to avoid include the skin of the nipples and genital area. When stimulating the supraorbital ridge, one should take care not to compress the eye itself.

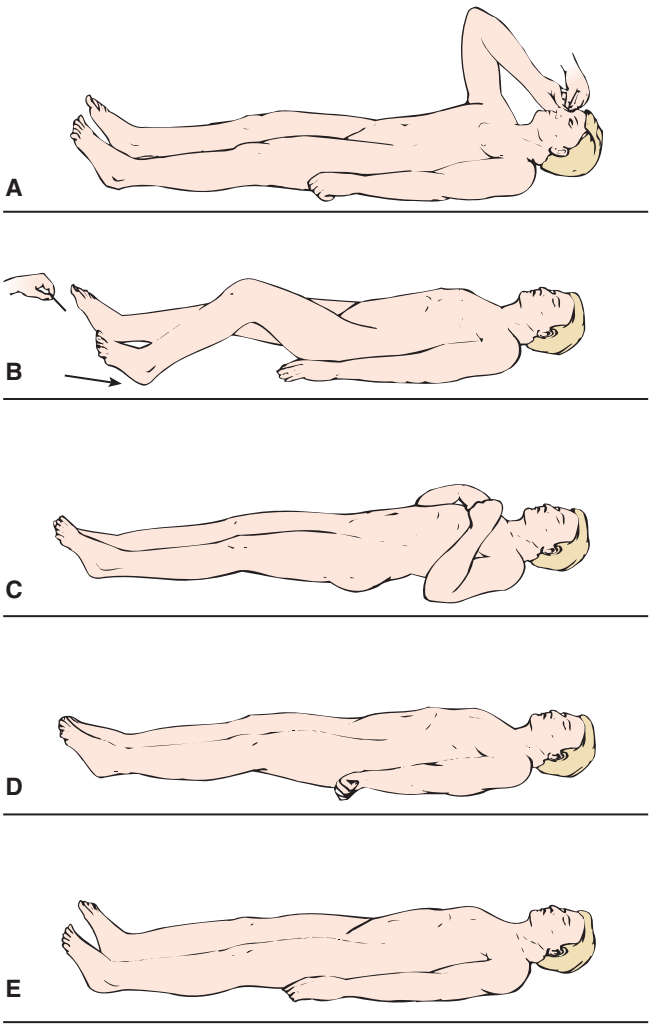
Localization to painful stimuli is characterized by an organized attempt to remove the stimulus, which entails movement of the extremity across midline. This contrasts to withdrawal, in which the patient simply pulls away from the noxious stimulus, rather than attempting to remove it (Fig. 33-1). Appropriate responses, such as localization or withdrawal, infer that the sensory and corticospinal pathways are functioning (see Fig. 33-1A,B). There may be monoplegia or hemiplegia, indicating that the corticospinal pathways are interrupted on one side.

Inappropriate responses include decorticate rigidity and decerebrate rigidity. Flexion of the arms, wrists, and fingers; adduction of the upper extremities; and extension, internal rotation, and plantar flexion of the lower extremities characterize decorticate rigidity (see Fig. 33-1C). Such rigidity results from lesions of the internal capsule, basal ganglia, thalamus, or cerebral hemisphere, interrupting corticospinal pathways.

Table 33-3 Patterns of Speech Deficits

Type	Deficit Locations	Speech Patterns
Fluent dysphasia	Left parietal–temporal lobes (Wernicke's area)	• Fluent speech that lacks coherent content • Impaired understanding of spoken word despite normal hearing • May have normal-sounding speech rhythm but no intelligible words • May use invented, meaningless words (neologism), word substitution (paraphasia), or repetition of words (perseveration, echolalia)
Nonfluent dysphasia	Left frontal area (Broca's area)	• Slow speech with poor articulation • Inability to initiate sounds • Comprehension usually intact • Usually associated with impaired writing skills
Global dysphasia	Diffuse involvement of frontal, parietal, and occipital areas	• Nonfluent speech • Inability to understand spoken or written words
Dysarthria	Corticobulbar tracts; cerebellum	• Loss of articulation, phonation • Loss of control of muscles of lips, tongue, palate • Slurred, jerky, or irregular speech but with appropriate content

Decerebrate rigidity consists of extension, adduction, and hyperpronation of the upper extremities and extension of the lower extremities, with plantar flexion of the feet (see Fig. 33-1D). Many times, the person also has clenched teeth. Injury to the midbrain and pons results in decerebration. At times, the inappropriate responses of decortication and decerebration may switch back and forth. If there is no response to noxious stimuli or only very weak flexor



(A) Localizing pain. An appropriate response is to reach up above shoulder level toward the stimulus. Remember, a focal motor deficit such as hemiplegia may prevent a bilateral response.

(B) Withdrawal. An appropriate response is to pull the extremity or body away from the stimulus. As brainstem involvement increases, your patient may respond by assuming one of the following postures. Each one shows more advanced deterioration.

(C) Decorticate posturing. One or both arms in full flexion on the chest. Legs may be stiffly extended.

(D) Decerebrate posturing. One or both arms stiffly extended. Possible extension of the legs.

(E) Flaccid. No motor response in any extremity.

FIGURE 33-1 ▲ Motor responses to pain. When a painful stimulus is applied to an unconscious patient's supraorbital notch, the patient responds in one of these ways.

responses (ie, flaccidity), the patient likely has extensive brainstem dysfunction (see Fig. 33-1E). Additional abnormal motor responses in a comatose patient include tonic contraction, which is consistent muscular contraction, and clonus, which is alternating muscle spasticity and relaxation.

Motor Strength and Coordination

The second component of the motor assessment addresses strength and coordination. Muscle weakness is a cardinal sign of dysfunction in many neurological disorders. The nurse tests extremity strength by offering resistance to various muscle groups, using his or her own muscles or gravity. As a quick test to detect weakness of the upper extremities, have the patient hold the arms straight out with palms upward and eyes closed and observe for any downward drift or pronation of the forearms. This is referred to as pronator drift. A similar test for the lower extremities involves having the patient lie in bed and raise the legs, one at a time, straight off the bed against the examiner's resistance. Weakness noted in any of these tests can indicate damage to the motor neuron pathways of the pyramidal system, which transmits commands for voluntary movement. Motor function for each extremity is reported as a fraction, with 5 as the denominator, as shown in Box 33-5.

Muscle groups are assessed individually, initially without resistance and then against resistance, to obtain a thorough evaluation. Upper extremity muscle strength is evaluated by asking the patient to shrug the shoulders (trapezius and levator scapulae muscles), raise the arms (deltoid muscle), flex the elbow (biceps muscle), extend the arm (triceps muscle), and extend the wrist (extensor carpi radialis longus muscle). Lower extremity muscle strength is evaluated by asking the patient to raise the leg (iliopsoas muscle), extend the knee (quadriceps muscle), dorsiflex and plantarflex the foot (anterior tibialis and gastrocnemius muscles, respectively), and flex the knee (hamstring muscle group consisting of the biceps femoris, semitendinosus, and semimembranosus muscles).

Assessment of movement and strength in a patient who cannot follow commands or is unresponsive can be difficult because participating in muscle strength testing against gravity requires the patient's understanding and cooperation. Unless the patient is able to mount a motor response secondary to painful stimuli, the nurse may not have the opportunity to test for muscle strength in any sort of reliable manner. Therefore, for comatose patients, it is important to note what, if any, stimuli initiate a response and to describe or grade the type of response obtained.

BOX 33-5 A Motor Function Scale

Score	Interpretation
0/5	No muscle contraction
1/5	Flicker or trace of contraction
2/5	Moves but cannot overcome gravity
3/5	Moves against gravity but cannot overcome resistance of examiner's muscles
4/5	Moves with some weakness against resistance of examiner's muscles
5/5	Normal power and strength

The nurse may also assess each extremity for size, muscle tone, and smoothness of passive movement. Abnormal responses may indicate problems in the basal ganglia (also called the extrapyramidal system). These pathways normally suppress involuntary movements through controlled inhibition. Assessment findings may include the "clasp-knife" phenomenon, in which initially strong resistance to passive movement suddenly decreases. Alternatively, "lead-pipe" rigidity may be present, which is steady, continuous resistance to passive movement and is characteristic of diffuse hemispheric damage. "Cogwheel" rigidity, which is a series of small, regular, jerky movements felt on passive movement, is characteristic of Parkinson's disease. The nurse also should be alert to involuntary movements, from mild fasciculation (muscle twitching) to violent, flailing movement of an extremity. Descriptive terms for involuntary movements are given in Box 33-6.

Hemiparesis (weakness) and hemiplegia (paralysis) are unilateral symptoms resulting from a lesion contralateral to the corticospinal tract. Paraplegia results from a spinal cord lesion below the first thoracic vertebrae or from peripheral nerve dysfunctions. Quadriplegia (also known as tetraplegia) is associated with cervical spinal cord lesions, brainstem dysfunction, and large bilateral lesions in the cerebrum.

The cerebellum is responsible for smooth synchronization, balance, and ordering of movements. It does not initiate any movements, so a patient with cerebellar dysfunction is not paralyzed. Instead, ataxia, dysmetria, and lack of synchronization of movement are common manifestations. Some of the more common tests for cerebellar synchronization of movement with balance include the following:

- **Romberg test.** This test is performed by having the patient stand with his or her feet together, first with the eyes open, then with the eyes closed. The nurse looks for sway or direction of falling and is prepared to catch the patient if necessary.

BOX 33-6 Types of Voluntary Movements

Tremor	Purposeless movement
Resting	Lesion in basal ganglia
Intention	Lesion in cerebellum
Asterixis	Metabolic derangement
Physiological	Due to fatigue or stress
Fasciculation	Twitching of resting muscles due to peripheral nerve or spinal cord lesion or to metabolic influences such as cold or anesthetic agents
Clonus	Repetitive movement; elicited with stretch reflex and implies lesion of the corticospinal tracts
Myoclonus	Nonrhythmic movement; single jerk-like movements; symmetrical; unknown etiology
Hemiballismus	Flailing movement of extremity; violent movement; not present during sleep; lesion in subthalamic nuclei of basal ganglia
Chorea	Irregular movements; involves limbs and facial muscles; asymmetrical movements at rest; involuntary movements may increase when purposeful movement is attempted
Athetosis	Slow, writhing movements

- **Finger-to-nose test.** This test is performed by having the patient touch one finger to the examiner's finger, then touch his or her own nose. Overshooting or past-pointing the mark is called dysmetria. Both sides are tested individually.
- **Rapidly alternating movement (RAM) test.** The patient's ability to perform RAMs is checked on each side by having the patient oppose each finger and thumb in rapid succession or by performing rapid pronation and supination of the hand on the leg. Inability to perform RAMs is termed adiadochokinesia; performing RAMs poorly or clumsily is termed dysdiadochokinesia.
- **Heel-to-shin test.** This test is performed by having the patient extend the heel of one foot down the anterior aspect of the shin, moving from the knee to the ankle.

Pupillary Changes

Assessment of pupillary response is an important component of the neurological examination. Pupils are examined for size (specified in millimeters) and shape (Fig. 33-2). The patient focuses on a distant point in the room. To isolate the eye being examined, the examiner places the edge of one hand along the patient's nose. A bright light is directed into one eye, and the briskness of pupillary constriction (direct response) is noted. The other pupil also should constrict (consensual response). The procedure is then repeated with the other eye. Anisocoria (unequal pupils) is normal in a small percentage of the population but can also indicate neural dysfunction. If it is a normal variant, the difference in pupil size should be less than 1 mm.

Pupil reactivity is also assessed with respect to accommodation. To test accommodation, an object is held 8 to 12 inches in front of the patient's face. The patient focuses on the object as the examiner moves it toward the patient's nose. The pupils should constrict as the object gets closer, and the eyes turn inward to maintain a clear image. The normal response to testing is documented as PERRLA, or **p**upils **e**qual, **r**ound, **r**eactive to **l**ight and **a**ccommodation.

Some important pupillary abnormalities are shown in Figure 33-3. Causes of small, reactive pupils include metabolic abnormalities and bilateral dysfunction in the diencephalon. Large, fixed pupils (5 to 6 mm) that may show slight rhythmic constriction and dilation when stimulated may indicate midbrain damage. Midposition, fixed pupils (4 to 5 mm) also may indicate midbrain dysfunction, involving interruption of the sympathetic and parasympathetic pathways. Pinpoint, nonreactive pupils are seen after damage to the pons area of the brainstem (thus the phrase "pontine pupils are pinpoint"), with selected eye medications, and with opiate administration. A unilaterally dilated, nonreactive ("blown") pupil is seen with third cranial (oculomotor) nerve damage when the uncal portion of the temporal lobe herniates through the tentorium. When structures are compressed around the opening in the tentorium or fold of dura that separates the cerebrum from the cerebellum and brainstem, loss of functioning of the parasympathetic nerves to the pupil on that side results in ipsilaterally (same side) dilated pupils. A quick guide to changes in pupil size is given in Box 33-7.

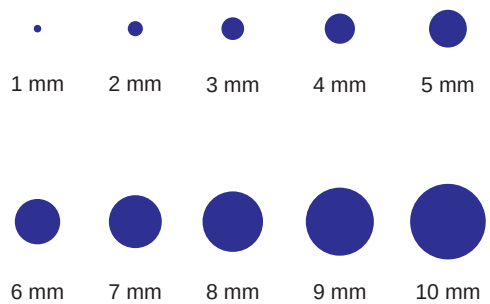


FIGURE 33-2 ▲ Pupil size chart.

The assessment of pupillary response for comatose patients is the same as for conscious patients. Pupil reactivity to light, by direct and consensual response, is easily obtained. It may be impossible to ascertain reactivity to accommodation because the patient may be unable to cooperate.

Cranial Nerve Function

The performance of a cranial nerve assessment varies depending on whether the patient is conscious or unconscious. Assessment of the cranial nerves in the unconscious patient is important because it provides data regarding brainstem function, however many components may need to be eliminated or adapted. For specific physiological information about the cranial nerves, see Chapter 32.

Cranial Nerve I (Olfactory Nerve)

The first cranial nerve contains sensory fibers for the sense of smell. This test usually is deferred unless the patient complains of an inability to smell. The nurse tests the nerve, with the patient's eyes closed, by placing aromatic substances near the nose for identification. Items that have a distinct smell (eg, soap, coffee, or cinnamon) should be used. Ammonia should not be used because the patient will respond to irritation of the nasal mucosa rather than to the odor. Each nostril is checked separately by closing off one nostril at a time. Loss of smell may be caused by a fracture of the cribriform plate or a fracture in the ethmoid area. The patient may also have anosmia (loss of sense of smell) from a shearing injury to the olfactory bulb after a basilar skull fracture or from cerebrospinal fluid (CSF) leak.

Cranial Nerve II (Optic Nerve)

Assessment of the optic nerve involves evaluation of visual acuity and visual fields. Gross visual acuity is checked by having the patient read ordinary newsprint, noting the patient's preinjury need for corrective lenses. Visual fields are tested by having the patient look straight ahead with one eye covered. The examiner moves a finger from the periphery of each quadrant of vision toward the patient's center of vision. The patient should indicate when the examiner's finger is seen. This is done for both eyes, and the results are compared with the examiner's visual fields, which are assumed to be normal

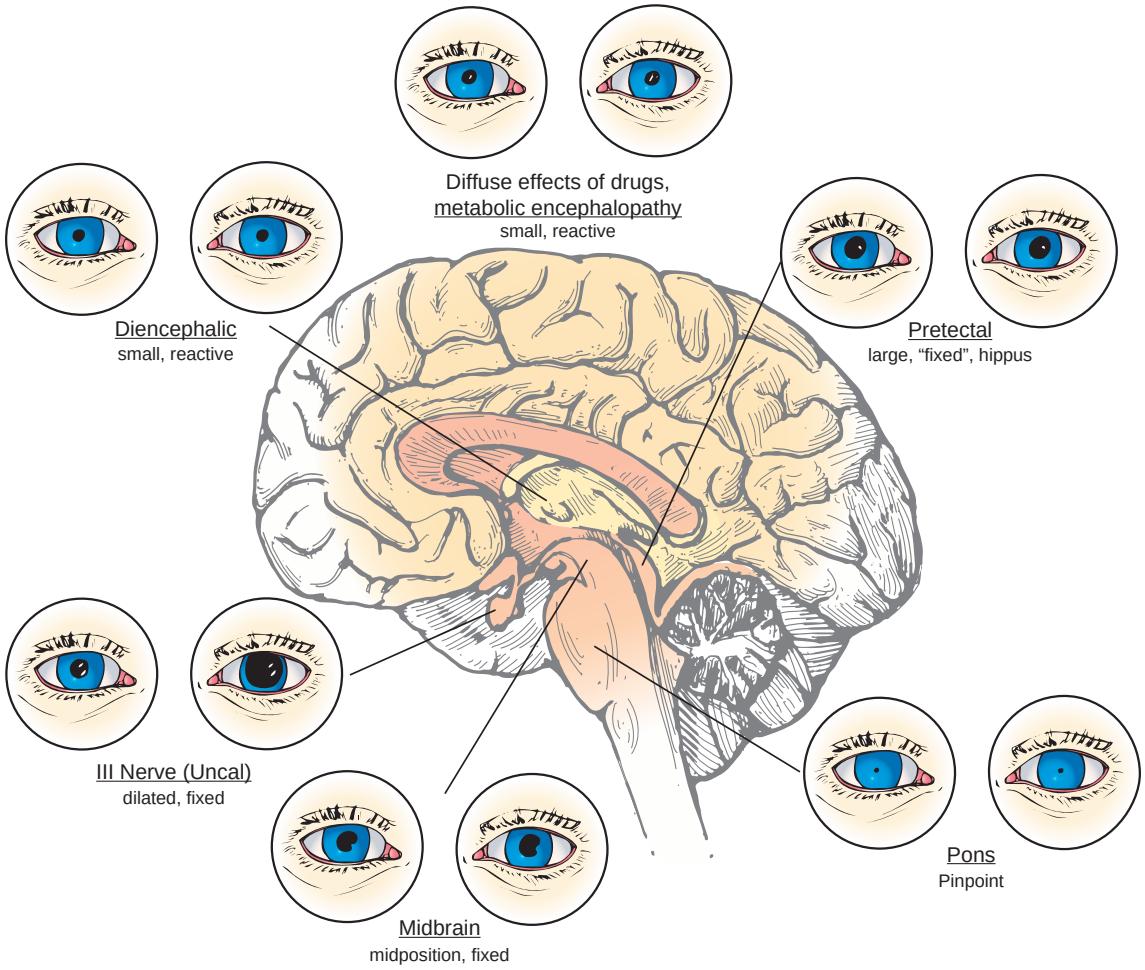


FIGURE 33-3 ▲ Abnormal pupils. (Adapted from Saper C: Brain stem modulation of sensation, movement, and consciousness. In Kandel ER, Schwartz JH, Jessel TM (eds): Principles of Neural Science, 4th ed. New York, NY: McGraw-Hill, 2000, pp 871–909, with permission from McGraw-Hill.)

(Fig. 33-4). Damage to the retina produces a blind spot. An optic nerve lesion produces partial or complete blindness on the same side. Damage to the optic chiasm results in bitemporal hemianopsia, blindness in both lateral visual fields (Table 33-4). Pressure on the optic tract can cause homonymous hemianopsia, half-blindness on the opposite side of the lesion in both eyes. A lesion in the parietal or temporal lobe may produce contralateral blindness in the upper or lower quadrant of vision, respectively, in both eyes (quadrant deficit). Damage in the occipital lobe can cause homonymous hemianopsia with central vision sparing.

BOX 33-7 Quick Guide to Causes of Pupil Size Change

Pinpoint Pupils
- Drugs: opiates
- Drops: medications for glaucoma
- "Nearly dead": damage in the pons area of the brainstem

Dilated Pupils
- Fear: panic attack, extreme anxiety
- "Fits": seizures
- "Fast living": cocaine, crack, phencyclidine

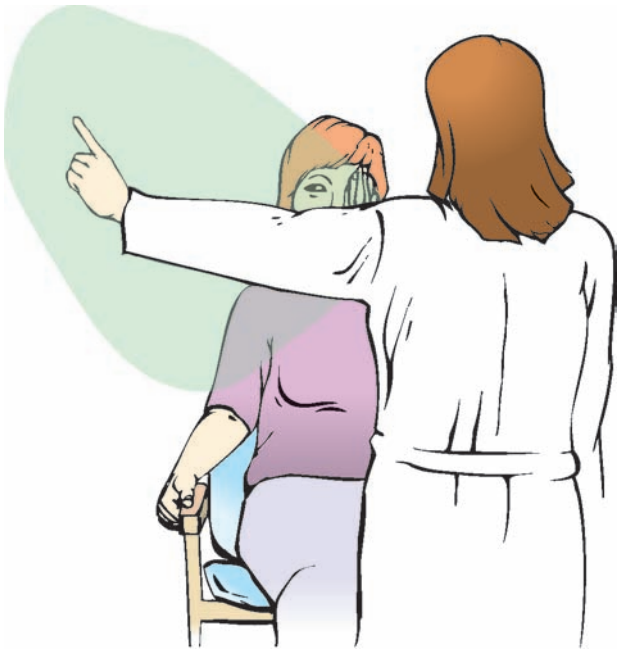


FIGURE 33-4 ▲ Confrontational method of testing visual fields.

Table 33-4 Visual Field Defects Associated With Defects of the Visual System

Visual Field Defect	Left	Right	Description
Anopsia			Blindness in one eye; due to complete lesion of the right optic nerve
Bitemporal hemianopsia (central vision)			Blindness in both lateral visual fields; due to lesions around the optic chiasm such as pituitary tumors or aneurysms of the anterior communicating artery. Affected fibers originate in the nasal half of each retina.
Homonymous hemianopsia			Half-blindness involving both eyes with loss of visual field on the same side of each eye; due to lesion of temporal or occipital lobe with damage to the optic tract or optic radiations (blindness occurs on the side opposite the lesion; here, the lesion occurred in the right side of the brain, resulting in loss of vision in the left visual field of both eyes)
Quadrant deficit			Blindness in the upper or lower quadrant of vision in both eyes, resulting from a lesion in the parietal or temporal lobe

Cranial Nerves III (Oculomotor Nerve), IV (Trochlear Nerve), and VI (Abducens Nerve)

Cranial nerves III, IV, and VI are assessed together because they all innervate extraocular muscles involved in eye movement. The parasympathetic fibers of the oculomotor nerve are responsible for lens accommodation and pupil size through control of the ciliary muscles. This is the nerve tested when a nurse elicits a pupillary response. The motor fibers of the oculomotor nerve innervate the muscles that elevate the eyelid and those that move the eyes up, down, and medially. These include the superior rectus, inferior oblique, inferior rectus, and medial rectus muscles. The trochlear nerve innervates the superior oblique muscle to move the eyes down and in. The lateral rectus muscle moves the eyes laterally and is innervated by the abducens nerve. Diplopia, nystagmus, conjugate deviation, and ptosis may indicate dysfunction of these cranial nerves. In the conscious patient, these nerves are tested by having the patient follow the examiner's finger as he or she moves it in all directions of gaze (Fig. 33-5).

Ocular position and movement are among the most useful guides to the site of brain dysfunction in the comatose person. When observing the eyes at rest, it is not uncommon to note a slight divergence of gaze. If both eyes are conjugately deviated to one side, there is possible dysfunction either in the frontal lobe on that side or in the contralateral pontine area of the brainstem. Downward deviation suggests a dysfunction in the midbrain.

Although the unconscious patient cannot participate in the examination by voluntarily moving the eyes through fields of gaze, the examiner still can test the range of ocular movement by assessing the oculocephalic ("doll's eyes" test) and oculovestibular (caloric ice-water test) reflexes.

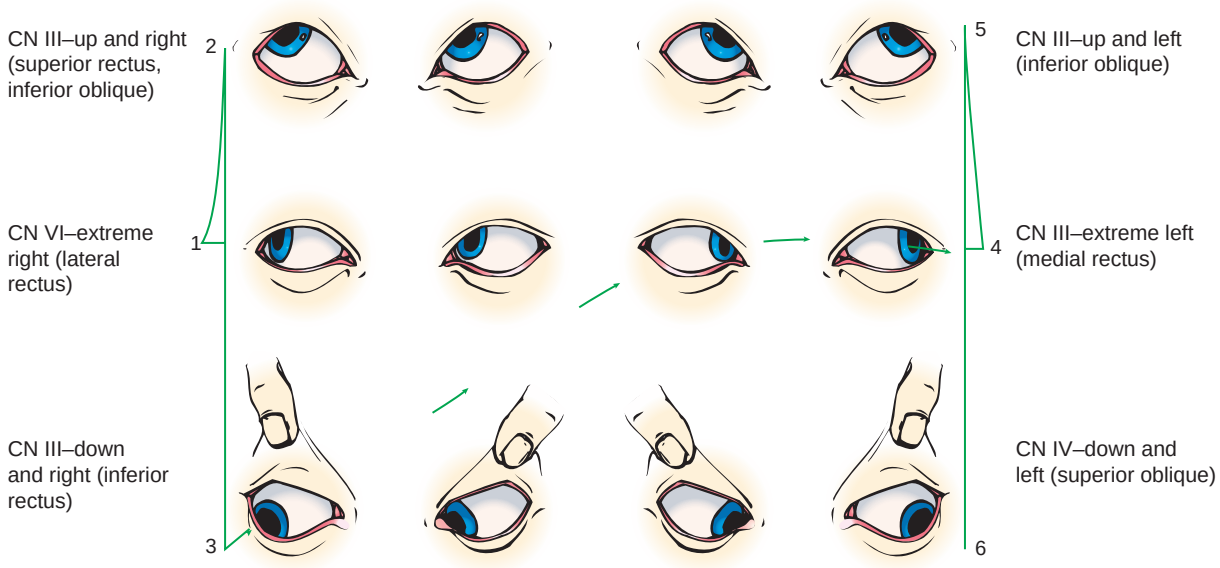


FIGURE 33-5 ▲ Muscles used in conjugate eye movements in the six cardinal directions of gaze. Lead the patient's gaze in the sequence numbered 1 through 6. CN III, oculomotor nerve; CN IV, trochlear nerve; CN VI, abducens nerve.

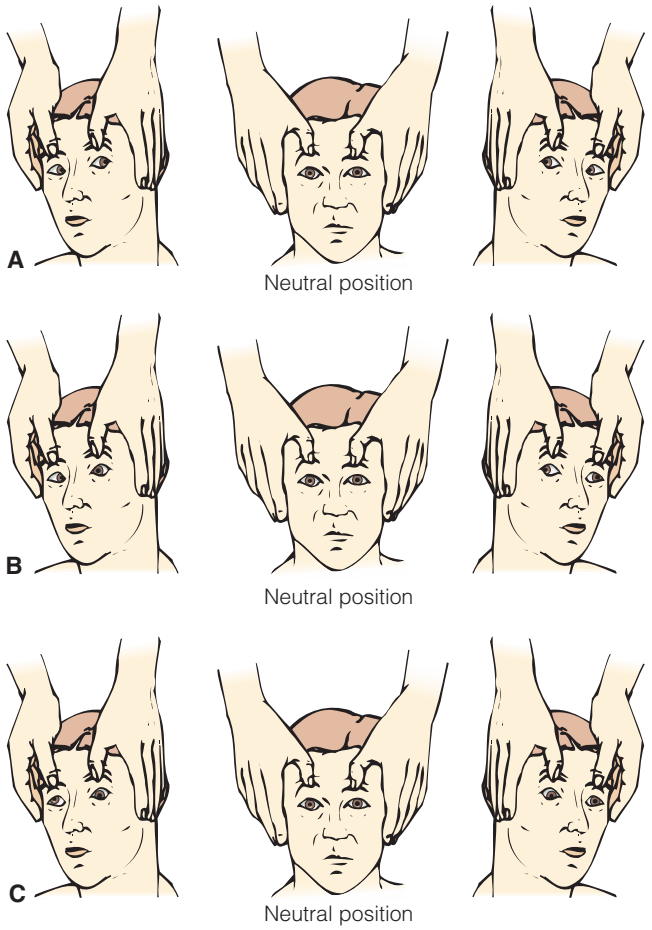


FIGURE 33-6 ▲ Test for oculocephalic reflex response (doll's eyes phenomenon). **A:** Normal response—when the head is rotated, the eyes turn together to the side opposite to the head movement. **B:** Abnormal response—when the head is rotated, the eyes do not turn in a conjugate manner. **C:** Absent response—as head position is changed, eyes do not move in the sockets.

The oculocephalic reflex can be assessed by quickly rotating the patient's head to one side and observing the position of the eyes (Fig. 33-6). This maneuver must never be performed in a person with possible cervical spine injury. A normal response consists of initial conjugate deviation of the eyes in the opposite direction, then, within a few seconds, smooth and simultaneous movement of both eyes back to midline position. This response indicates an intact brainstem. An abnormal reflex response occurs when one eye does not follow the normal response pattern. Absence of any ocular movement when the head is rotated briskly to either side or up and down indicates an absent reflex and portends severe brainstem dysfunction.

The examiner tests the oculovestibular reflex by elevating the patient's head 30 degrees and irrigating each ear separately with 30 to 50 mL of ice water (Fig. 33-7). This test should never be performed in a patient who does not have an intact eardrum or who has blood or fluid collected behind the eardrum. Also, the external ear canal should be unobstructed by cerumen or debris. In an unconscious patient with an intact brainstem, the eyes exhibit horizontal nystagmus with slow, conjugate movement toward the irrigated ear followed by rapid movement away from the stimulus. When the reflex is absent, both eyes remain fixed in midline position, indicating midbrain and pons dysfunction.

Cranial Nerve V (Trigeminal Nerve)

Cranial nerve V has three divisions: ophthalmic, maxillary, and mandibular. The sensory portion of this nerve controls sensation to the cornea and face. The motor portion controls the muscles of mastication. This nerve is partially tested by checking the corneal reflex; if it is intact, the patient blinks when the cornea is stroked with a wisp of cotton or when a drop of normal saline is placed in the eye. Care must be taken not to stroke the eyelash because this can cause the eye to blink regardless of a corneal reflex. Facial sensation can be tested by comparing light touch and pinprick on symmetrical sides of the face. The ability to chew or clench the jaw also is observed.

Cranial Nerve VII (Facial Nerve)

The sensory portion of cranial nerve VII is concerned with taste on the anterior two thirds of the tongue. The motor portion controls muscles of facial expression (Fig. 33-8). Testing is performed by asking the patient to raise the eyebrows, smile, or grimace. With a central (supranuclear) lesion, there is muscle paralysis of the lower half of the face on the side opposite the lesion. The muscles around the eyes and forehead are unaffected. With a peripheral (nuclear or infranuclear) lesion, there is complete paralysis of facial muscles on the same side as the lesion.

The most common type of peripheral facial paralysis is Bell's palsy, which consists of ipsilateral facial paralysis. There is drooping of the upper lid with the lower lid slightly everted. Facial lines on the same side are obliterated, with the mouth drawn toward the normal side.

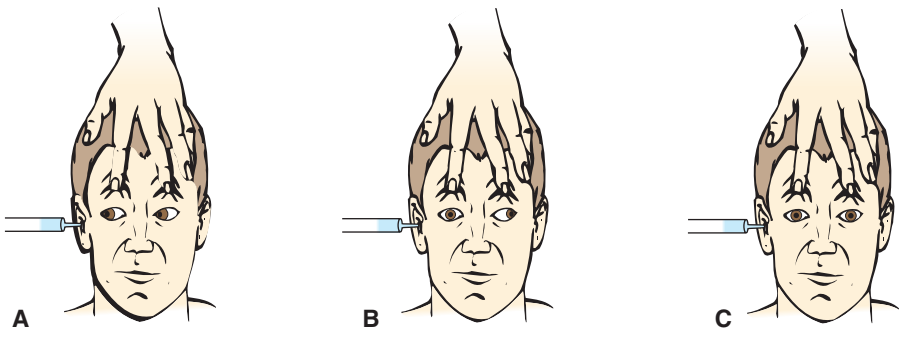


FIGURE 33-7 ▲ Test for oculovestibular reflex response (caloric ice-water test). **A:** Normal response—ice-water infusion in the ear produces conjugate eye movements. **B:** Abnormal response—infusion produces disconjugate or asymmetrical eye movements. **C:** Absent response—infusion produces no eye movements.

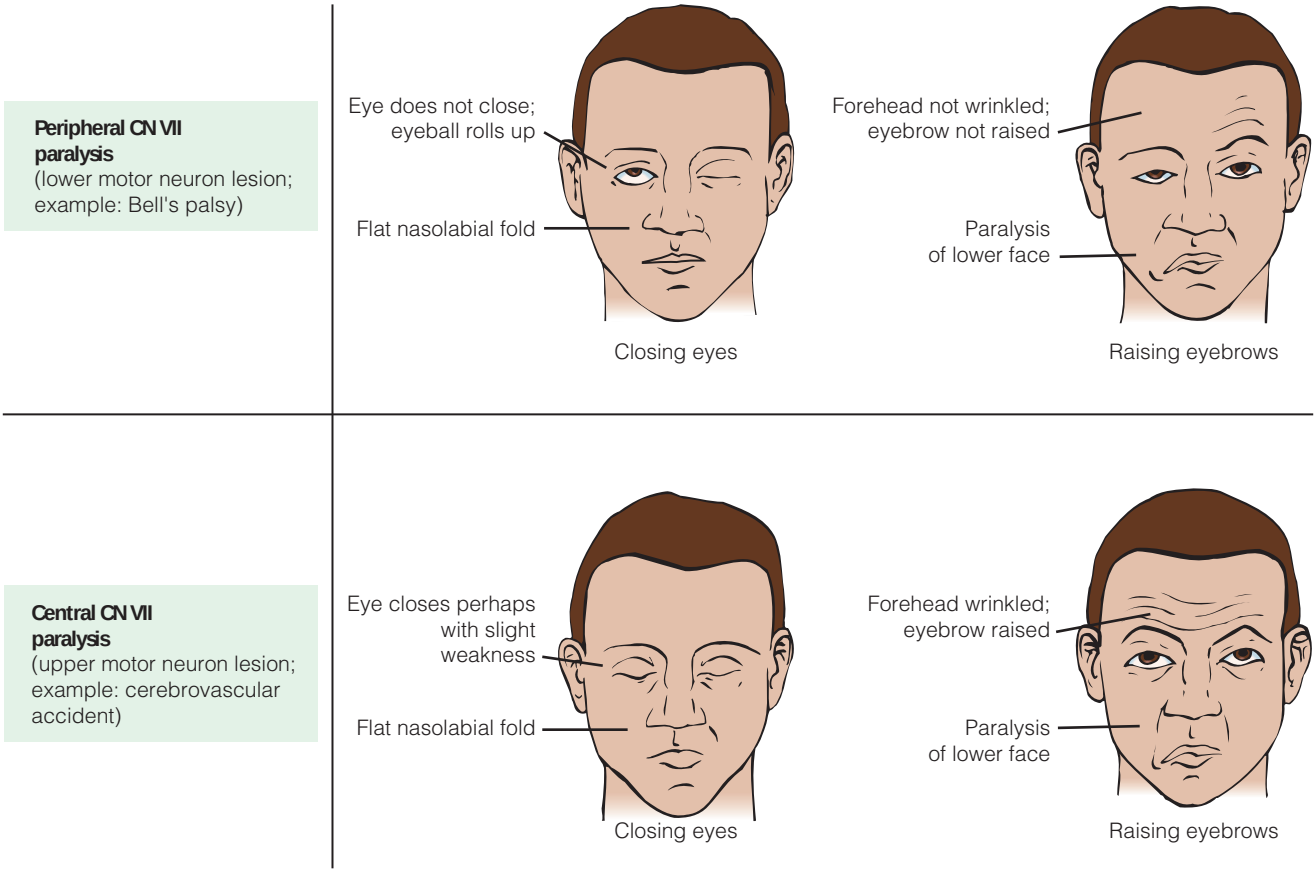


FIGURE 33-8 ▲ Facial movements with upper and lower motor neuron facial paralysis. CN VII, facial nerve.

In the comatose patient, motor function of the facial muscles and jaw can be ascertained by observing spontaneous muscle activity such as yawning, grimacing, or chewing. Symmetry of movement may be assessed, and facial droops may be observed.

Cranial Nerve VIII (Acoustic Nerve)

Cranial nerve VIII is divided into the cochlear and vestibular branches, which control hearing and equilibrium, respectively. The cochlear nerve is tested by air and bone conduction. There are two distinct auditory tests—the Weber and Rinne test. For the Rinne test, a vibrating tuning fork is placed on the mastoid process and the patient is asked to listen to the sound and to indicate when it disappears. The tuning fork is then placed in front of the ear and a normal test occurs when the patient can still hear sound transmitted through air. The Weber test involves placement of the tuning fork on the patient's forehead. A normal response would involve equal hearing of the sound in both ears. The patient may complain of tinnitus or decreased hearing if this nerve is damaged. The vestibular nerve may not be evaluated routinely. However, the nurse should be alert to complaints of dizziness or vertigo from the patient.

Cranial Nerves IX (Glossopharyngeal Nerve) and X (Vagus Nerve)

Cranial nerves IX and X usually are tested together. The glossopharyngeal nerve supplies sensory fibers to the posterior third of the tongue and the uvula and soft palate. The vagus nerve innervates the larynx, pharynx, and soft palate and conveys autonomic responses to the heart, stomach, lungs, and small intestine. These nerves can be tested by eliciting a gag reflex, observing the uvula for symmetrical movement when the patient says "ah," or observing midline elevation of the uvula when both sides are stroked. Inability to cough forcefully, difficulty with swallowing, and hoarseness may be signs of dysfunction. Autonomic vagal functions usually are not tested because they are checked during the general physical examination.

Cranial Nerve XI (Spinal Accessory Nerve)

Cranial nerve XI controls the trapezius and sternocleidomastoid muscles. The examiner tests this nerve by having the patient shrug the shoulders or turn the head from side to side against resistance.

Cranial Nerve XII (Hypoglossal Nerve)

Cranial nerve XII controls tongue movement. This nerve can be checked by having the patient protrude his or her tongue. The examiner checks for deviation from midline, tremor, and atrophy. If deviation is noted secondary to nerve damage, it will be to the side of the cerebral lesion.

Testing cranial nerve function completely is time consuming and exacting. A partial, quicker screening assessment may be performed, focusing on nerves in which

Table 33-5 A Quick Screening Test for Cranial Nerve Function

	Nerve	Reflex	Procedure
II III	Optic Oculomotor	Pupil constriction (protection of the retina)	Shine a light into each eye and note if the pupil on that side constricts (direct response). Next, shine a light into each eye and note if the opposite pupil constricts (consensual response).
V VII	Trigeminal Facial	Corneal reflex (protection of the cornea)	Approaching the eye from the side and avoiding the eyelashes, touch the cornea with a wisp of cotton. Alternatively, a drop of sterile water or normal saline may be used. A blink response should be present.
IX X	Glossopharyngeal Vagus	Airway protection	Touch the back of the throat with a tongue depressor. A gag or cough response should be present.

dysfunction may indicate serious problems or interfere with activities of daily living (Table 33-5). The cranial nerves of primary importance in a screening examination are the optic, oculomotor, trigeminal, facial, glossopharyngeal, and vagus nerves.

Reflexes

A reflex occurs when a sensory stimulus evokes a motor response. Cerebral control and consciousness are not required for a reflex to occur. Superficial and deep reflexes are tested on symmetrical sides of the body and compared by noting the strength of contraction elicited on each side.

Cutaneous, or superficial, reflexes occur when certain areas of skin are lightly stroked or tapped, causing contraction of the muscle groups beneath. Such reflexes are graded simply as normal, abnormal (pathological), or absent. An example is the plantar reflex. A sensory stimulus is applied by briskly stroking the outer edge of the sole and across the ball of the foot with a dull object, such as a tongue blade or key. The normal motor response is downward or plantar flexion of the toes. An abnormal response (Babinski's sign) is upward or dorsiflexion of the big toe, with or without fanning of the other toes. A positive Babinski's sign can indicate a lesion in the pyramidal tract. It should be noted however that a positive Babinski sign is normal in children under the age of 2.

Muscle stretch reflexes, also called deep tendon reflexes, are elicited by a brisk tap with a reflex hammer on the appropriate tendon insertion site. The target for this sensory stimulus is a stretched tendon of a muscle group. Deep tendon reflexes are tested on the biceps, brachioradial, triceps, patellar, and Achilles tendons. The desired motor response is contraction of the stimulated muscle group. Deep tendon reflexes are commonly graded on a scale of 0 to 4:

4+: A very brisk response; evidence of disease, electrolyte imbalance, or both; associated with clonic contractions
3+: A brisk response; possibly indicative of disease
2+: A normal response
1+: A response in the low-normal range
0: No response; possibly evidence of disease or electrolyte imbalance.

Hyperreflexia is associated with upper motor neuron disease, whereas areflexia (absence of reflexes) is associated with lower motor neuron dysfunction, such as spinal cord lesions. Reflexes can be tested on the comatose patient. It is anticipated that, depending on the severity and location of the neuronal damage, either hyperreflexia or areflexia will be present.

Sensation

The last component of the neurological examination involves a sensory assessment. Normal sensory findings depend on an intact spinal cord, sensory pathways, and peripheral nervous system. The primary forms of sensation are tested first. These include perception of touch (cotton wisp), pain (pinprick), temperature (hot, cold), proprioception (limb position), and vibration. With the patient's eyes closed, multiple and symmetrical areas of the body are tested, including the trunk and extremities.

The nurse assesses the perception of touch by asking the patient to close the eyes and identify when and where he or she feels a cotton wisp or cotton swab on the skin. Pain is assessed with the use of a pin or the sharp edge of a cotton swab, moving in a head-to-toe direction on both sides of the body. Temperature, if tested, uses glass tubes of hot and cold water and proceeds in the manner described previously. Two-point discrimination may also be tested and refers to the patient's ability to distinguish between two closely located points. Discrimination of sharp versus dull is also a commonly used test.

Proprioception is tested by asking the patient, again with the eyes closed, to identify the direction of movement (eg, moving a finger upward and then asking the patient if the finger is up or down). The same test is performed on the other hand, as well as both lower extremities. The nurse assesses vibration using a tuning fork placed over a bony prominence. The patient is asked to identify when vibration is felt.

The patient's ability to perceive the sensation is noted, with distal areas compared with proximal areas and right and left sides compared at corresponding points. The nurse also determines whether sensory change involves one entire side of the body. Abnormal results may indicate damage somewhere along the pathways of the receptors in the skin, muscles, joints and tendons, spinothalamic tracts, or sensory area of the cortex (Table 33-6).

Cortical forms of sensation also should be tested. When primary sensation is intact, but interpretation of the sensory input is altered, then damage to the parietal lobe may be anticipated. Problems with discriminative sensation include those involving stereognosis, graphesthesia, and point localization. The ability to recognize and identify objects by touch is called stereognosis and is a function of the parietal lobe. The inability to recognize objects by touch, sight, or sound is termed agnosia. This may be tested by placing an object in

Table 33-6 Testing Superficial and Deep Sensations

Sensation	Stimuli	Dysfunction
Spinothalamic Tracts Carry Impulses for		
Pain	Alternate sharp and dull ends of a pin, asking patient to discriminate between the two (superficial pain). Squeeze nail beds; apply pressure on the orbital rim; rub sternum (deep pain).	• Ipsilateral sensory loss implies a peripheral nerve lesion. • Contralateral sensory loss is seen with lesions of the spinothalamic tract or in the thalamus.
Light touch	Use a wisp of cotton on skin and ask patient to identify when it touches.	• Bilateral sensory loss may indicate a spinal cord lesion. • Paresthesia is an abnormal sensation, such as itching or tingling.
Temperature	Use test tubes filled with hot and cold water or use small metal plates of varying temperatures. (Test only if pain and light touch sensations are abnormal.)	• Causalgia is a burning sensation that can be caused by peripheral nerve irritation.
Posterior Columns Carry Impulses for		
Vibration	Apply a vibrating tuning fork on bony prominences, and note patient's ability to sense and locate vibrations bilaterally.	• Ipsilateral sensory loss may be due to spinal cord injury or to peripheral neuropathy.
Proprioception	Move the patient's finger or toe up and down and ask patient to identify final resting position.	• Contralateral loss may occur from lesions of the thalamus or of the parietal lobes.

a patient's hand and asking him or her, with the eyes closed, to identify the object solely based on touch. Identification of an object by the sense of sight is a function of the parieto-occipital junction. The temporal lobe is responsible for identification of objects by sound. Each of these senses should be tested separately. For example, a patient may not be able to identify a whistle by its sound but may recognize it immediately if he or she holds it or looks at it.

Graphesthesia is the ability to recognize numbers or letters traced lightly on the skin. Bilateral sides are compared. Point localization refers to the ability to locate the precise spot on the body touched by the examiner. One version of dysfunction in this area is called extinction phenomenon, the inability to recognize bilateral sensations when the examiner simultaneously touches two symmetrical areas on opposite sides of the body.

In a comatose patient, it is impossible to perform a complete test for sensation because patient cooperativeness is required. However, use of painful stimuli to elicit a response gives a gross indication that some degree of sensory function remains intact. More detailed data would be unavailable, though.

Vital Signs

Vital sign assessment is crucial to the neurological examination. Changes in temperature, heart rate, and blood pressure are considered late findings in neurological deterioration. Changes in respiratory rate, on the other hand, can indicate progression of neurological impairment and are frequently seen early in neurological deterioration.

Respirations

Variations in respiratory pattern are commonly associated with neurological injury. Shallow, rapid respirations can indicate a problem with maintenance of a patent airway or the need for suctioning. Snoring respirations or stridor can also indicate a partially obstructed airway. The inability to maintain an effective airway may be associated with a high cervical spinal cord lesion or progressive diaphragmatic paralysis (seen with neurodegenerative diseases), or it may be seen with a decreasing level of consciousness.

Changes in respiratory pattern can also be a direct indication of increasing intracranial pressure (ICP) (see Chapter 36, Fig. 36-6, p. 814). Cheyne-Stokes respirations (crescendo–decrescendo respirations alternating with periods of apnea) are frequently noted in neurological disease.

Hypoventilation after cerebral trauma can lead to respiratory acidosis. As the blood carbon dioxide increases and blood oxygen decreases, cerebral hypoxia and edema can result in secondary brain injury, thereby extending the degree of damage. Hyperventilation after cerebral trauma produces respiratory alkalosis with decreased blood carbon dioxide levels. This causes vasoconstriction of cerebral vessels, contributing to decreased cerebral blood flow.

Temperature

Normal regulation of temperature occurs in the hypothalamus. Diffuse cerebral damage can result in alterations in temperature. Central nervous system (CNS) fevers may be very high and differentiate themselves from other causes of fever by their resistance to antipyretic therapy. Hypothermia occurs with metabolic causes, pituitary damage, and spinal cord injuries.

Pulse

Variations in heart rate and rhythm may also be associated with neurological injury. An increase in ICP may lead to episodes of tachycardia and can predispose the patient to alterations in electrocardiogram pattern, such as ventricular or atrial dysrhythmias. As the ICP increases, bradycardia results, and the combination of the two is indicative of impending herniation.

Blood Pressure

Blood pressure is controlled at the level of the medulla. Therefore, specific damage to this area or encroaching edema secondary to injury in other areas results in alterations in blood pressure. Hypotension is not normally associated with neurological injury except in instances of impending cerebral herniation. On the other hand, hypotension must be avoided in the postinjury stage because it can lead to decreased cerebral perfusion, hypoxia, and extension of the initial injury.

Hypertension is much more commonly seen. In the intact brain, the mechanism of cerebral autoregulation maintains constant blood flow to the brain, despite wide variations in systemic pressure. However, after injury, autoregulatory mechanisms fail, and cerebral blood flow varies dramatically with variations in systemic pressure. As blood pressure increases, cerebral blood flow increases, resulting in an increase in ICP. Likewise, as blood pressure decreases, cerebral blood flow decreases, resulting in ischemia.

Signs of Trauma or Infection

Signs of trauma or infection may be evident on examination:

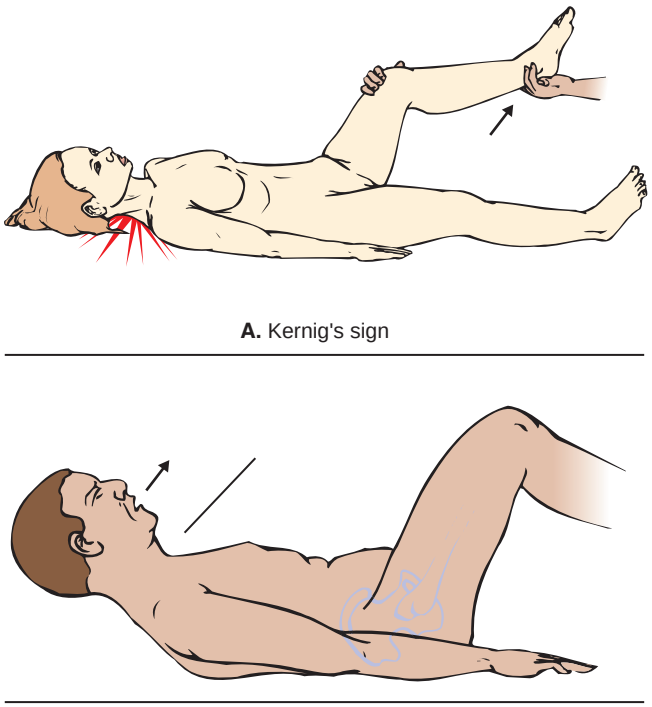
- **Battle's sign** (bruising over the mastoid areas) suggests a basilar skull fracture.
- **Raccoon's eye** (periorbital edema and bruising) suggests a frontobasilar fracture.
- **Rhinorrhea** (drainage of CSF from the nose) suggests fracture of the cribriform plate with herniation of a fragment of the dura and arachnoid through the fracture.
- **Otorrhea** (drainage of CSF from the ear) usually is associated with fracture of the petrous portion of the temporal bone.
- **Signs of meningeal irritation** include nuchal rigidity (ie, pain and resistance to neck flexion), fever, headache, and photophobia. A positive Kernig's sign (ie, pain in the neck when the thigh is flexed on the abdomen and the leg is extended at the knee) also may be present. Brudzinski's sign (involuntary flexion of the hips when the neck is flexed toward the chest) is another indication of meningeal inflammation. Kernig's sign and Brudzinski's sign are shown in Figure 33-9.

Signs of Increased Intracranial Pressure

The prevention of increased ICP, or intracranial hypertension, is of key importance to the nurse's role when caring for a patient with a neurological injury. It is first essential for the nurse to establish a baseline neurological assessment on the patient, on which further deterioration can be based. In general terms, increased ICP is manifested by deterioration in all aspects of neurological functioning.

Level of consciousness decreases as ICP rises. Initially, the patient may present with evidence of restlessness, confusion, and combativeness. This then decompensates into lower levels of consciousness, ranging from lethargy to obtundation to coma. Pupillary reactions begin to diminish, with sluggishly reactive pupils and eventually fixed, dilated pupils. Frequently, because of the potential for injury to be ipsilateral, one pupil dilates before the other one does, resulting in unequal pupils.

Motor function also declines, and the patient begins to show abnormal motor activity. For example, the patient who initially may have localized to painful stimuli now shows either abnormal flexion or extension. Changes in vital signs are considered a late finding. Variations in respiratory patterns occur, eventually resulting in complete apnea. Cushing's triad is considered a sign of impending herniation (see Chapter 36). This triad consists of an increased systolic pressure (resulting in an increased pulse pressure), bradycardia, and decreased irregular respirations.



A. Kernig's sign

B. Brudzinksi's sign

FIGURE 33-9 ▲ Two signs of meningeal irritation.

Evaluation of Dysfunction in the Patient's Living Patterns

Neurological nursing assessment would be incomplete if the process consisted solely of gathering data and identifying abnormal functions. Nursing expertise should expand the scope to include an evaluation of the impact of dysfunction on the patient's living patterns and ability to care for self. For example, diplopia (double vision) is an abnormal finding and may be an indicator of problems with the ocular muscles or with the nervous system; however, it also may be a clue suggesting difficulty in carrying out daily activities.

▲ Neurodiagnostic Studies

Many diagnostic tests are available to help diagnose neurological and neurosurgical problems. Such neurodiagnostic testing is performed in conjunction with a thorough neurological examination. The availability and diagnostic accuracy of current technology benefit the patient in an acute setting by shortening the time required to arrive at a diagnosis and institute therapy. The choice of which investigative test to perform should be based on the examiner's

ability to integrate the findings with neurological assessment and locate the cause of the abnormality.

The nurse's role in neurodiagnostic testing involves patient and family preparation and monitoring the critically ill patient for potential complications during and after the procedure. Although there has been a definite increase in the number of tests that can be performed at the bedside, many still require that the patient be transported to the imaging department or even out of the institution, further expanding the role of the critical care nurse. Table 33-7 summarizes some of the diagnostic tests and outlines nursing implications.

Neuroradiological Techniques

Conventional radiographs of the skull and spine are used to identify fractures, dislocations, and other bony anomalies, especially in the setting of acute trauma. In addition, radiographs may be diagnostic when displacement of the calcified pineal gland is visible, which is an immediate clue to the presence of a space-occupying lesion. Air inside the skull also suggests an open skull fracture, such as a frontal or basilar skull fracture, that may not be readily apparent externally. However, the use of plain films has decreased in recent years

Table 33-7 Neurodiagnostic Tests

Diagnostic Test	Description	Information Obtained	Nursing Considerations and Interventions
Computed tomography, or CT scan (invasive and noninvasive)	A scanner takes a series of radiographic images all around the same axial plane. A computer then creates a composite picture of various tissue densities visualized. The images may be enhanced with the use of IV contrast dye.	CT scans give detailed outlines of bone, tissue, and fluid structures of the body. They can indicate shift of structures due to tumors, hematomas, or hydrocephalus. A CT scan is limited in that it gives information only about structure of tissues, not about functional status.	Instruct the patient to lie flat on a table with the machine surrounding, but not touching, the area to be scanned. Patient also must remain as immobile as possible; sedation may be required. The scan may not be of the best quality if the patient moves during the test or if the x-ray beams were deflected by any metal object (ie, traction tongs, intracranial pressure (ICP) monitoring devices).
Magnetic resonance imaging (MRI)	A selected area of the patient's body is placed inside a powerful magnetic field. The hydrogen atoms inside the patient are temporarily "excited" and caused to oscillate by a sequence of radiofrequency pulsations. The sensitive scanner measures these minute oscillations, and a computer-enhanced image is created.	An MRI scan creates a graphic image of bone, fluid, and soft tissue structures. It gives a more defined image of anatomical details and may help one diagnose small tumors or early infarction syndromes.	Risk factors for this technique are not well identified. This test is contraindicated in patients with previous surgeries where hemostatic or aneurysm clips were implanted. The powerful magnetic field can cause such clips to move out of position, placing the patient at risk for bleeding or hemorrhage. Other contraindications include cardiac pacemakers, prosthetic valves, bullet fragments, and orthopedic pins. Inform patient that the procedure is very noisy. Use caution if patient is claustrophobic. The patient (and care givers) must remove all metal objects with magnetic characteristics (eg, scissors, stethoscope).
Positron emission tomography (PET); single-photon emission computed tomography (SPECT)	The patient either inhales or receives by injection radioactively tagged substances, such as oxygen or glucose. A gamma scanner measures the radioactive uptake of these substances, and a computer produces a composite image, indicating where the radioactive material is located, corresponding to areas of cellular metabolism.	These diagnostic tests are the only ones to measure physiological and biochemical processes in the nervous system. Specific areas can be identified as to functioning and nonfunctioning. Cerebral metabolism and cerebral blood flow can be measured regionally. PET and SPECT scans help diagnose abnormalities (tumors, vascular disease) and behavioral disturbances, such as dementia and schizophrenia, that may have a physiological basis.	The patient receives only minimal radiation exposure because the half-life of the radionuclides used is from a few minutes to 2 h. Testing may take a few hours. Procedure is very expensive. Inform the patient that remaining very still and immobile will produce best test results.

(continued on page 738)

Table 33-7 Neurodiagnostic Tests (continued)

Diagnostic Test	Description	Information Obtained	Nursing Considerations and Interventions
Cerebral angiography (invasive)	This is a radiographic contrast study in which radiopaque contrast medium is injected by a catheter into the patient's cerebral arterial circulation. The contrast medium is directed into each common carotid artery and each vertebral artery, and serial radiographs are then taken.	The contrast medium illuminates the structure of the cerebral circulation. The vessel pathways are examined for patency, narrowing, and occlusion, as well as structural abnormalities (aneurysms), vessel displacement (tumors, edema), and alterations in blood flow (tumors, arteriovenous malformations).	In preparation for this test, inform the patient as to the location of the catheter insertion (femoral artery is a common site) and that a local anesthetic will be used. Also warn that a warm, flushed feeling will occur when the contrast medium is injected. After this procedure, assess the puncture site for swelling, redness, and bleeding. Also check the skin color, temperature, and peripheral pulses of the extremity distal to the site for signs of arterial insufficiency due to vasospasm or clotting. A large amount of contrast medium may be needed during this test, with resulting increased osmotic diuresis and risk for dehydration and renal tubular occlusion. Other complications include temporary or permanent neurological deficit, anaphylaxis, bleeding or hematoma at insertion site, and impaired circulation to the extremity used for injection.
Digital subtraction angiography (invasive)	In this test, a plain radiograph is taken of the patient's cranium. Then, radiopaque contrast medium is injected into a large vein, and serial radiographs are taken. A computer converts the images into digital form and "subtracts" the plain radiograph from the ones with the contrast medium. The result is an enhanced radiographic image of contrast medium in the arterial vessels.	Extracranial circulation (arterial, capillary, and venous) can be examined. Vessel size, patency, narrowing, and degree of stenosis or displacement can be determined.	There is less risk to the patient for bleeding or vascular insufficiency because the injection of contrast medium is intravenous rather than intra-arterial. The patient must remain absolutely motionless during the examination (even swallowing will interfere with the results).
Radioisotope brain scan (noninvasive)	In this test, radioactive isotope is usually injected intravenously. The scanning device produces films of areas of concentration of the isotope within the patient's head.	Because damaged brain tissue absorbs more isotope, the presence of an intracranial lesion can be diagnosed as well as cerebral infarction or contusion. Lack of uptake of the isotope may indicate brain death.	Minimal patient preparation is required. The isotope may not be readily available within the institution. Movement will make the test difficult to interpret. This test is less commonly used than CT scan or MRI.
Myelography (invasive)	A myelogram is a radiographic study in which a contrast medium (either air or dye) is injected into the lumbar subarachnoid space. Fluoroscopy, conventional radiographs, or CT scans are used to visualize selected areas.	The spinal subarachnoid space is examined for partial or complete obstructions due to bone displacements, spinal cord compression, or herniated intervertebral disks.	Instruct the patient as for a lumbar puncture. In addition, advise that a special table will tilt up or down during the procedure. Postprocedure care is determined by the type of contrast medium used. Oil-based contrast dye: • Flat in bed for 24 h • Force fluids • Observe for headache, fever, back spasms, nausea, and vomiting Water-based contrast dye: • Head of bed elevated for 8 h • Keep patient quiet for first few hours • Do not administer phenothiazines • Observe for headache, fever, back spasms, nausea, vomiting, and seizures

(continued on page 739)

Table 33-7 Neurodiagnostic Tests (continued)

Diagnostic Test	Description	Information Obtained	Nursing Considerations and Interventions
Electroencephalogram, or EEG (noninvasive)	An EEG is a recording of electrical impulses generated by the brain cortex that are sensed by electrodes on the surface of the scalp.	Analysis of the resulting tracings helps detect and localize abnormal electrical activity occurring in the cerebral cortex. It aids in seizure focus detection, localization of a source of irritation such as a tumor or abscess, and diagnosis of metabolic disturbances and sleep disorders.	Reassure the patient that he or she will not feel an electrical shock or pain during this test. The nurse also may need to clarify for the patient that the machine cannot "read minds" or indicate the presence of mental illness. The patient's scalp and hair should be free of oil, dirt, creams, and sprays because they can cause electrical interference and thus an inaccurate recording. Inform the EEG technician of electrical devices around the patient that may cause interference during the procedure (eg, cardiac monitor, ventilator).
Cortical evoked potentials (noninvasive) Somatosensory evoked potentials Brainstem auditory evoked response Visual evoked potentials	In this test, a specialized device senses central or cortical cerebral electrical activity by skin electrodes in response to peripheral stimulation of specific sensory receptors. The sensory receptors stimulated can be those for vision, hearing, or tactile sensation. The signals are graphically displayed by a computer and characteristic peaks, and the intervals between them, are measured.	Cortical evoked potentials provide a detailed assessment of neuron transmission along particular pathways. It has value in determining the integrity of visual, auditory, and tactile pathways in patients with multiple sclerosis and spinal cord injury. This test also may be used in the assessment of a sensory pathway before, during, and after surgery.	This test may be used in conscious as well as unconscious patients and can be performed at the bedside. The patient must be as motionless as possible during some phases of this test to minimize musculoskeletal interference. Depending on the sensory pathway being tested, the patient may be instructed to watch a series of geometric designs or listen to a series of clicking noises.
Transcranial Doppler sonography	This is a test in which high-frequency ultrasonic waves are directed from a probe toward specific cerebral vessels. The ultrasonic energy is aimed through cranial "windows," areas in the skull where the bony table is thin (temporal zygoma) or where there are small gaps in the bone (orbit or foramen magnum). The reflected sound waves are analyzed for shifts in frequency, indicating flow velocity.	The speed or velocity at which blood travels through cerebral vessels is an indicator of the size of the vascular channel and the resistance to blood flow. An approximation of cerebral blood flow may be determined. Cerebral autoregulation can be monitored by observing the response of intracranial vessels to changes in arterial carbon dioxide and to the partial occlusion of the proximal vessels, as may occur in vasospasm.	The test is noninvasive and may be performed at the bedside by the physician or ultrasound technician in 30–60 min. There are no known adverse effects, and the procedure may be repeated as often as necessary. The testing is accomplished with the patient initially supine, and later on his or her side, with the head flexed forward.
Lumbar puncture (invasive)	A hollow needle is positioned in the subarachnoid space at L3–L4 or L4–L5 level, and cerebrospinal fluid (CSF) is sampled. The pressure of the CSF also is measured. Normal pressure varies with age from 45 mm H_2O in full-term newborns to 120 mm H_2O in adults.	The CSF is examined for blood and for alterations in appearance, cell count, protein, and glucose. The opening pressure is roughly equivalent to the ICP for most patients, if the patient is recumbent and no block is present.	This test is contraindicated in patients with suspected increased ICP because a sudden reduction in pressure from below may cause brain structures to herniate, leading to death. In preparation for this test, position the patient on side with knees and head flexed. Explain to the patient that some pressure may be felt as the needle is inserted and not to move suddenly or cough. After this procedure, keep the patient flat for 8–10 h to prevent headache. Encourage liberal fluid intake.

as computed tomography (CT) and magnetic resonance imaging (MRI) have proved to be better diagnostic tools.

Spinal films may still be used as an initial screening in suspected vertebral or spinal cord trauma; however, designated trauma centers rely increasingly on cervical spine CT scans. If plain films are to be taken, visualization of the cervical spine through C7 is indicated to rule out a cervical spine injury. Further, visualization of C1–C2 may require an odontoid or Waters' view. This film is taken through the open mouth of the patient and necessitates patient cooperation. The procedure for plain films of the skull and spine requires careful patient positioning and is relatively painless. The nurse's role involves monitoring the patient and attendant equipment during the procedure and being alert for complications related to patient position and the length of the procedure. In the spinal cord injured–patient, care should be taken to ensure stabilization of the neck by a hard cervical collar and logrolling during testing.

Computed Tomography

CT scans have been in use in the United States since 1973. CT scanning uses intersecting x-ray beams through the brain and skull to measure the density of tissues through which the x-ray beams pass. The denser the material (ie, skull), the whiter it appears on the film (Fig. 33-10). The less dense the material (ie, air), the darker it appears on the film. With mathematical reconstruction, multiple views or slices of the brain can be seen, which allows for a very precise, detailed picture of the brain and its contents.

The CT scan permits more refined measurement of the density of tissues, blood, and bone in the body compared with that afforded by conventional radiographs. For example, cerebral edema appears less dense and therefore is of a lighter color than normal tissue. The value of this technique is illustrated best in the trauma setting, where the ability to image rapidly and accurately the intracranial contents and position of vertebrae and spinal cord has dramatically changed the treatment of neurological patients. CT scans are recommended in the initial workup of seizures, headache, and loss of consciousness and for the diagnosis of suspected hemorrhage, tumors, and other lesions. CT scanning can reliably detect conditions such as skull fractures, tissue swelling, hematomas, tumors, and abscesses. However, it has been noted that some vascular lesions are not as reliably documented using CT scan as they are with MRI. Therefore, MRI is indicated if these lesions are suspected.

Use of a contrast medium can enhance a CT scan. Using radiographic contrast material allows better visualization of vascular areas and enhances lesions previously seen on noncontrast films. Serial CTs may allow the health care team to follow neurological progression and therefore to intervene in a rapid manner. Care should be taken in the patient with renal failure or renal insufficiency because contrast medium clearance by the kidneys may be impaired.

Sometimes two technologies are used in combination, such as myelography or angiography with CT scanning, to provide a more refined image of anatomical structures. With current technology, a routine scan now takes less than 5 minutes to survey the patient, analyze the data, and display a finished image.

Nursing management focuses on patient education to obviate any potential complications, such as poor patient tolerance. The patient should be aware that he or she must lie very still during the procedure and that he or she may experience feelings of claustrophobia. In addition, the nurse ascertains whether the patient has any preexisting allergies, particularly if contrast medium is to be used. The nurse may need to remain with the patient during the procedure to continue to monitor neurological status and vital signs.

Magnetic Resonance Imaging

MRI, known in the past as nuclear MRI, has become widely available in medium and large medical centers. This modality uses nonionizing forms of radiation to produce computerized cross-sectional images in much the same fashion as a CT scan. However, it provides more finely detailed images that look remarkably like anatomical slices of the body. MRI is superior to a CT scan in the early diagnosis of cerebral infarction and the detection of demyelinating disorders, such as multiple sclerosis. It is also helpful in diagnosing small lesions, such as tumors and hemorrhages, that might not

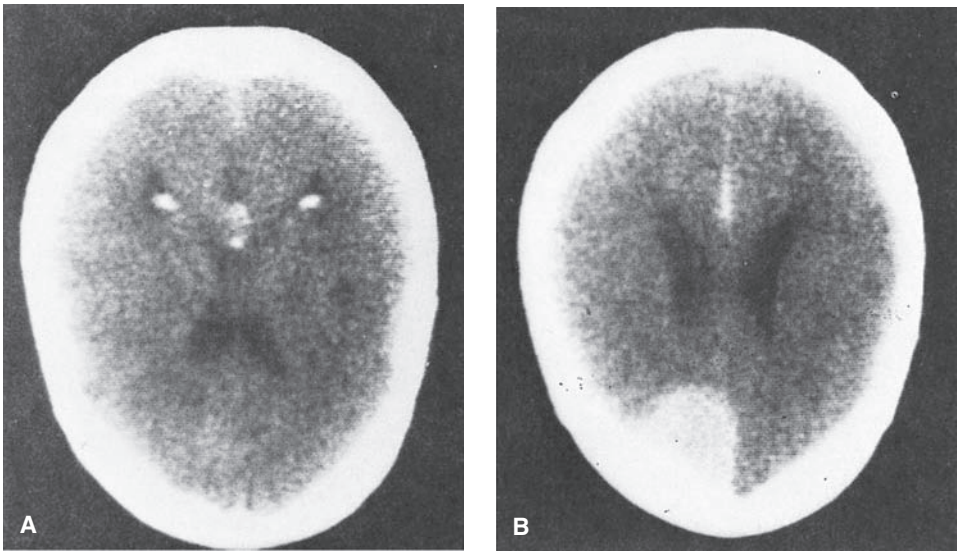


FIGURE 33-10 ▲ Computed tomography scan of the brain. **A:** Normal scan. **B:** Scan showing a large mass in the left frontal lobe. (Reprinted from Hickey J: The Clinical Practice of Neurological and Neuroscience Nursing, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 92, with permission.)

appear on a CT scan or in evaluation of ligamentous injury of the spinal cord. However, traditional CT scanning is superior for scanning for bony abnormalities, which are visualized poorly on MRI.

Although superior in many ways to CT scanning, MRI has its limitations. Its powerful magnetic fields interfere with the functioning of devices such as cardiac pacemakers. Patients with surgical clips and prosthetic implants made of ferrous metal cannot be scanned. It is also difficult to study patients on life-support equipment because most ventilators and monitors are constructed in part of ferrous metal. If emergency therapy is needed, the patient must be removed from the scanning chamber and the imaging suite before resuscitation can begin.

Positron Emission Tomography and Single-Photon Emission Computed Tomography

Positron emission tomography (PET) is a process in which molecules labeled with radioactive isotopes are located in the brain and recorded by radiation-sensitive detectors outside the head. PET has the capacity to measure cerebral blood flow and cerebral metabolism as the isotope-labeled glucose or oxygen is used in the body. It is superior to previous technologies that could image structure only, not function. It currently assists in diagnosing Alzheimer's disease, which shows a characteristic pattern of glucose consumption, as well as in Parkinson's disease, Huntington's disease, and Tourette's syndrome. However, the complexity of the testing, the comparatively high cost per scan, and the need to have a cyclotron nearby to produce the short-lived radioactive isotopes make this modality impractical and unwieldy in the clinical setting.

Single-photon emission computed tomography (SPECT) combines the imaging ability of conventional nuclear medicine scanners with the technology of transaxial CT scanning to overcome some limitations. Using more stable radioisotopes, SPECT scanning can detect diminished perfusion in an area of stroke before there is conventional CT evidence of infarction. SPECT can also detect alterations in regional blood flow in patients with Alzheimer's disease.

Angiography and Digital Subtraction Angiography

Cerebral angiography remains the study of choice for evaluating cerebrovascular problems (Fig. 33-11). It is the only test that can reveal large and small aneurysms and arteriovenous malformations and their relationship to adjacent structures and vessels. It involves the passage of a radiographic catheter through a large artery (usually femoral) to each of the arterial vessels bringing blood to the brain and spinal cord. Radiopaque contrast (a dye-like medium) is then injected into each vessel. A rapid sequence of images is taken after the contrast agent has passed through small arterial branches and capillaries and into the venous circulation. In this way, the vessel lumen and size and the presence of any occlusions can be visualized. Cerebral angiography has been used before surgery to help decide the appropriateness of medical versus surgical management. It has also been combined with balloon angioplasty in instances of vascular occlusion or coiling in the treatment of aneurysms.

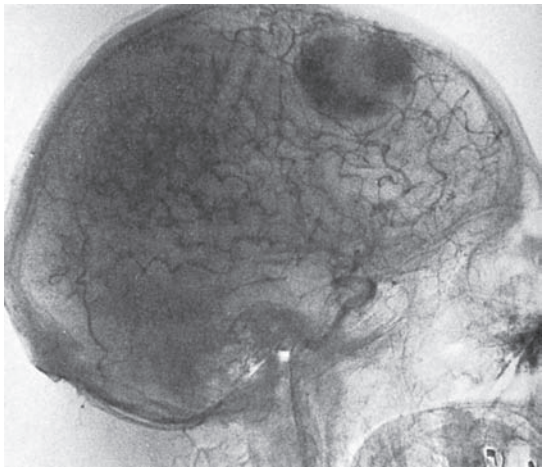


FIGURE 33-11 ▲ Cerebral angiogram showing an abnormal, large, space-occupying lesion at 1 o'clock. (Reprinted from Hickey J: The Clinical Practice of Neurological and Neuroscience Nursing, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 103, with permission.)

Digital subtraction angiography makes use of radiographic contrast to illuminate the cerebral circulation, but in considerably smaller quantities than required for conventional angiography. The contrast medium may be injected into the arterial or the venous systems. Films are taken before and after the injection and converted into digital information in the accompanying computer. The images are "subtracted" from each other, removing all images in common. The resultant image displays only the enhanced circulatory system, free of other anatomical distortion.

The major complications associated with angiography include stroke, vasospasm, or renal failure secondary to the contrast load. Contraindications to angiography include identified allergies to the contrast medium, anticoagulant therapy, and kidney and liver disease.

Cerebral Blood Flow Studies

In the diagnostic setting, cerebral blood flow is evaluated most commonly by a radioisotope brain scan. A radioactive isotope is injected intravenously. In unusual circumstances, the isotope can also be administered orally or intra-arterially. The brain is then scanned to determine which areas show an accumulation of the radioactive substance. If there is blood flow to the brain, damaged areas absorb more of the isotope than areas without damage. A newer technique, perfusion CT, involves scanning before, during, and after infusion of the contrast agent to obtain "real time" data with respect to blood flow. Cerebral blood flow studies are indicated in the detection of either increased or decreased blood flow during surgical procedures or to assess for vasospasm. They may also be used after carotid endarterectomy. The test may be used to determine brain death, which is evidenced if there is no flow to the cerebral hemispheres. In certain disorders, such as carbon monoxide poisoning, there may be increased blood flow to the brain, yet anoxic brain death may still occur. The measurement of cerebral flow assists in decision making regarding treatment and in the identification of complications.

Myelography

Myelography is a contrast study of the spinal cord and surrounding structures. It involves the introduction of water-soluble material into the CSF through a lumbar or cisternal puncture, performed under fluoroscopy, after about 10 mL of CSF has been removed. Myelography is indicated in evaluating herniated intervertebral disks, spinal cord tumors, and congenital problems and in assessing spinal cord trauma. It allows for better visualization of nerve roots and surrounding structures because it uses a contrast agent that is lighter than CSF. However, the agent disperses rapidly into the subarachnoid space, which means that the patient's position cannot be adjusted. The contrast agent does not require removal; therefore, the patient should be kept well hydrated to facilitate the agent's excretion. A heavier, oil-based contrast agent is sometimes used, and it must be removed at the end of the procedure.

The contrast agents are potentially toxic to cerebral tissue and may cause grand mal seizures. Thus, the patient must remain with the head up at least 30 to 45 degrees, and phenothiazine medications, which increase the toxic symptoms, must be avoided.

Ultrasonography and Noninvasive Cerebrovascular Studies

Transcranial Doppler ultrasonographic studies are a noninvasive means of monitoring intracranial hemodynamics at the bedside. The examination is performed through cranial "windows," areas in the skull where the bone is relatively thin, such as the temporal area, or where there are small spaces between bones, such as the orbit. The ultrasonic probe transmits sound waves at certain frequencies to a specified depth. The resultant reflected signal from blood traveling through cerebral vessels is interpreted for speed or velocity. As resistance or vascular size changes, it is reflected as a change in blood flow velocities. The data may be used to monitor therapy, aid in determining prognosis, and provide early recognition of cerebral vasospasm in patients after subarachnoid hemorrhage or severe head injury. Serial Doppler studies in patients with an aneurysm provide data regarding postoperative vasospasm and alleviate the need for repeat angiograms.

Carotid and vertebral artery duplex scans provide anatomical imaging of blood vessels combined with hemodynamic information. Doppler studies at the cranial window provide information about direction of flow, pulsatile rhythmicity, and resistance to flow of the cerebral vasculature. Carotid duplex scans are routinely used as a screening tool in patients at risk for atherosclerotic disease. The nurse should be aware of whether the patient has any history of dysrhythmias or cardiac disease because these may alter the hemodynamic profile and findings of the test.

Electrophysiological Studies

Electroencephalography

Using electroencephalography (EEG), a record is made of the brain's electrical activity. Small plate electrodes are placed in specific locations on the patient's scalp, and 16 to 21 channels transcribe the electrical potentials generated by the brain. Waveforms are classified in terms of voltage and amplitude. EEG is most valuable in the diagnosis and treatment of patients with seizures. In addition, it may help localize structural abnormalities, such as tumors and abscesses, and aid in the differentiation of structural and metabolic abnormalities. It also may provide confirmatory criteria in the diagnosis of brain death. In recent years, a modified form of EEG has been used at the bedside in critical care to monitor the effects of pharmacological agents that reduce cerebral blood flow and hence reduce electrical activity. This is termed continuous EEG monitoring and is rapidly becoming a standard of care in many facilities. It is intended to detect subclinical or nonconvulsive seizure activity in patients who are taking medications that suppress electrical activity.

A computerized technique that dramatically compresses standard EEG data and converts them into a more easily interpreted and colorized form is compressed spectral array. This technique is also seen at the bedside in neurological intensive care units to monitor patients with severe head injuries.

Evoked Potentials

An evoked potential is an electrical manifestation of the brain's response to an external stimulus: auditory, visual, somatic, or a combination of these. The measurement of such a response provides an assessment of the function of neuropathways from the periphery through the spinal cord and brainstem and finally to cortical structures. This technique has been most helpful in diagnosing multiple sclerosis and Guillain-Barré syndrome and in determining the prognosis for reversibility of coma in the brainstem-injured patient. It also may be used during surgery to monitor potential injury during manipulations of spinal nerves and structures.

The three most frequently used techniques in head trauma evaluation are somatosensory evoked potentials (SSEPs), which use electrical shock as a stimulus; brainstem auditory evoked response (BAERs), which uses click or sound stimulus; and visual evoked potentials (VERs), which use light stimulus. SSEPs assess neurological function in specific neural pathways postinjury and detect further CNS insults from secondary processes, such as hypoxia and hypertension.

Lumbar Puncture for Cerebrospinal Fluid Examination

A lumbar puncture for CSF analysis may be performed to help diagnose autoimmune disorders or infections. Occasionally, it is performed to verify subarachnoid hemorrhage, although a CT scan is the procedure of choice and is safer for such a patient. CSF is obtained by the insertion of an 18- to 22-gauge needle between the vertebrae at the L3–L4 or L4–L5 levels. The fluid is sent for content analysis and for culture, sensitivity, and other serological tests (Table 33-8). Pressure readings may also be obtained for diagnostic use.

If a lumbar puncture is performed in a patient with elevated ICP, herniation can be a life-threatening complication. Complications that can result from a CSF leak include a postprocedure headache, nuchal rigidity, fever, and difficulty voiding. Treatment involves the injection of blood into the dura, called a blood patch, to stop the leak.

Table 33-8 Normal and Abnormal Values for Cerebrospinal Fluid

Characteristic	Normal	Abnormal
Color	Clear, colorless	Cloudy often due to presence of WBC or bacteria Xanthochromic due to presence of RBC
White blood cell (WBC)	0–5/mm^3, all mononuclear	Elevated count accompanies many conditions (tumor, meningitis, subarachnoid hemorrhage, infarct, abscess)
Red blood cell (RBC)	None	Presence may be due to traumatic tap or subarachnoid hemorrhage
Chloride	120–130 mEq/L	Low concentration associated with meningeal infection and tuberculous meningitis Elevated level not neurologically significant
Glucose	50–75 mg/100 mL	Decreased level associated with presence of bacteria in CSF Elevated level not neurologically significant
Pressure	70–180 mm H_2O	Low pressure associated with inaccurate placement of needle, dehydration, or block along subarachnoid space or at foramen magnum Elevated pressure associated with benign intracranial hypertension; cerebral edema; central nervous system tumor, abscess, or cyst; hydrocephalus; muscle tension or abdominal compression; subdural hematoma
Protein	14–45 mg/100 mL	Decreased level not neurologically significant Increased level associated with demyelinating or degenerative disease, Guillain-Barré syndrome, hemorrhage, infection, spinal block, tumor

From Cammermeyer M, Appeldorn C (eds): Core Curriculum for Neuroscience Nursing, 4th ed. Chicago, IL: American Association of Neuroscience Nurses, 1996.

▲ Clinical Applicability Challenges

CASE STUDY

Mr. S. is a 25-year-old male involved in a motor vehicle accident. Witnesses upon the scene indicate that he had a 5-minute loss of consciousness. Upon arrival in the emergency department, it is noted that his GCS is 14 (E4/V4/M6). Pupils are 3 mm bilaterally and reactive. He demonstrates repetitive questioning. His blood pressure is 134/72, his pulse rate is 68, and his respiratory rate is 22. His cervical spine is immobilized, and intravenous fluids are infusing. Within moments of arrival, your assessment reveals decreasing responsivity. Upon reassessment, you note that his GCS is now 8 (E2/V2/M4), and his pupils are now unequal and sluggishly reactive.

1. What diagnostic tests are recommended?
2. If Mr. S.'s condition deteriorates, what neurological changes might you anticipate?
3. What nursing interventions would be appropriate at this time?

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

34 Patient Management: Nervous System

Mona N. Bahouth and Karen L. Yarbrough

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Define intracranial pressure (ICP) and intracranial hypertension.
2. Discuss several physiological principles affecting ICP, including the Monro-Kellie doctrine, compliance, autoregulation, and cerebral perfusion.
3. Discuss indications for ICP monitoring.
4. Describe currently available methods of monitoring ICP.
5. Explain three possible complications associated with ICP monitoring and discuss troubleshooting strategies.
6. Identify various strategies to manage increased ICP.
7. Discuss three general principles when selecting a sedating agent for use in the patient with neurological injury.

Caring for a critically ill patient with neurologic injury requires an understanding of three general themes: (1) the neurological examination as compared with baseline; (2) concepts of intracranial pressure (ICP) and factors that influence ICP; and (3) medications strategies in this population of patients.

ICP is defined as the pressure in the cranial vault relative to atmospheric pressure. Understanding general principles regarding the concepts of ICP provides the critical care nurse with a framework that he or she can then apply to multiple neurological conditions. In addition, a working knowledge of the pharmacological agents used in neurological emergencies, such as steroids, antihypertensive agents, diuretics, analgesics, sedatives, barbiturates, and anticonvulsants, better prepares the nurse to handle these situations.

▲ Physiological Principles

Intracranial Dynamics

Concepts of ICP management and intervention strategies are based on the principle that the skull is a rigid box, a nonexpansile, noncontractile space. Its contents are divided into three intracranial sections: blood maintained in the blood vessels, cerebrospinal fluid (CSF), and brain parenchyma. The brain's ability to self-regulate is based on the Monro-Kellie doctrine of fixed intracranial volume. This doctrine states that the volume of the intracranium is equal to the volume of the intravascular cerebral blood (3% to 10%); plus the volume of the CSF (8% to 12%); plus the volume of brain tissue, which consists of more than 80% water. As long as the total intracranial volume remains the same, ICP remains constant. To maintain equilibrium, there cannot be any increase in volume of one of these components without a compensatory decrease in the other two. Any alterations in the volume of any of these three components within the cranial vault, without a response from the other two components, may lead to a change in ICP. A normal ICP measurement ranges between 0 and 15 mm Hg. An ICP measurement greater than 15 mm Hg is considered intracranial hypertension or increased ICP. Basic physiological responses to illness in any of the three components in the intracranial vault can cause increased ICP (Table 34-1).

Cerebral Blood Flow

Autoregulation is defined as the ability of an organ to maintain consistent blood flow despite marked changes in arterial circulatory and perfusion pressures. The normal brain has the ability to autoregulate cerebral blood flow (CBF). Normally, autoregulation ensures a constant blood flow through the cerebral vessels over a range of perfusion pressures by changing the diameter of vessels in response to changes in arterial pressures. This mechanism is the brain's protective device against the constantly fluctuating changes of blood pressure. When autoregulation is impaired, the CBF fluctuates in direct correlation with the systemic blood pressure. In patients with impaired autoregulation, any activity that causes an increase in blood pressure, such as coughing, suctioning, or restlessness, can cause an increase in CBF that could also increase ICP.

The first of three components that may undergo changes as the body attempts to maintain a consistent intracranial

Table 34-1 Potential Causes of Increased Intracranial Pressure

Contributing Physiology	Intracranial Component Involved	Potential Cause	Potential Treatment
Overproduction of CSF	CSF space	Choroid plexus papilloma	Surgical removal, diuretics
Inadequate CSF reabsorption (communicating hydrocephalus)	CSF space	Subarachnoid hemorrhage, infection	Drainage of CSF from lumbar intrathecal site, shunt placement
Blockage of CSF circulation (obstructive hydrocephalus)	CSF space	Posterior fossa tumor, brain injury, birth defects (spina bifida)	Ventricular drainage, surgical removal of obstruction
Edema (vasogenic, cytotoxic)	Brain tissue	Tumor, infection, infarction, hypoxia, arteriovenous malformation	Drainage of CSF, removal of lesion, adequate oxygenation
Expansile mass	Brain tissue	Tumor, abscess, intracerebral hemorrhage	Surgical removal, steroids
Vasospasm	Intracranial circulation	Subarachnoid hemorrhage	Hypervolemia, hypertensive therapy, calcium channel antagonists
Vasodilation	Intracranial circulation	Elevated $PaCO_2$, systemic vasodilators (α-adrenergic agents)	Hyperventilation, removal of offending agent

volume is the CBF. Normal CBF is provided by a cerebral perfusion pressure (CPP) in the range of 60 to 100 mm Hg. The brain receives approximately 750 mL/min of arterial blood (15% to 20% of total cardiac output when at rest). For autoregulation to be functional, carbon dioxide levels must be in an acceptable range, and hemodynamic pressure targets must be within the following ranges: CPP greater than 60 mm Hg, mean arterial pressure (MAP) less than 160 mm Hg, systolic pressure between 60 and 140 mm Hg, and ICP less than 30 mm Hg.[1] Factors that alter the ability of the cerebral vessels to constrict or dilate, such as hypoxia, hypercapnia, and brain trauma, also interfere with autoregulation. Carbon dioxide is a potent vasodilator of cerebral vessels, causing increased CBF and increased volume, leading to increased ICP.

Cerebrospinal Fluid Circulation

Cerebrospinal fluid (CSF) also contributes to fluctuations in intracranial hemodynamics. CSF is a clear fluid produced predominantly in the choroid plexus in the lateral, third, and fourth ventricles. It fills the ventricles and subarachnoid space and protects the brain and spinal cord from injury. Circulation of CSF occurs in a closed system; it is predominantly reabsorbed by the arachnoid villi located in the subarachnoid space and dispersed into the venous system through the superior sagittal sinus (see Chapter 32, Fig. 32-6). Along the entire CSF cycle, potential disturbances in production, circulation, and absorption can contribute to changes in ICP. For instance, overproduction of CSF in the choroid plexus overwhelms the circulatory system. Obstruction of CSF circulation through the ventricles leads to dilation of the ventricular system (obstructive hydrocephalus). Marked slowing of absorption in the arachnoid villi caused by blood or infectious debris interferes with the reabsorption of CSF, thereby leading to systemic overload (communicating hydrocephalus).

Parenchyma

The third and most difficult intracranial component to manipulate without surgical intervention is the brain parenchyma. However, the brain tissue does respond to increased ICP and changes within the other two intracranial components. The brain can accommodate or compensate for minimal changes in volume by partial collapse of the cisterns, ventricles, and vascular systems, in turn decreasing production and increasing reabsorption of CSF. Compensatory mechanisms to maintain normal ICP include the following:

- Shunting of CSF into the spinal subarachnoid space
- Increased CSF absorption
- Decreased CSF production
- Shunting of venous blood out of the skull

During the compensatory period, ICP remains fairly constant. However, when these compensatory mechanisms have been exhausted, pressure increases rapidly until shifting of brain tissue toward open spaces in the skull occurs, and the blood supply to the medulla is cut off. The ability of the intracranial content to compensate depends on the location of the lesion, the rate of expansion, and cranial compliance.

Volume–Pressure Curve

The intracranial volume–pressure curve, also called a pressure–volume index (PVI), demonstrates the relationship between changes in intracranial volume and changes in ICP. An awareness of the patient's position on this curve is useful in monitoring and selecting appropriate interventions. The rate at which ICP increases in response to a change in intracranial volume depends on the compliance of the brain. *Compliance* is a change in volume resulting from a change in pressure. When compliance in the intracranial compartment is low, a small volume change causes a large increase in ICP. In Figure 34-1, the curve illustrates compliance, as the compensatory mechanisms maintain ICP in the normal range during increases in intracranial volume. Little change occurs in ICP during the initial increase in volume because the volume added to the cranium is compensated for by volume displacement. As the compensatory mechanisms become exhausted, the volume added becomes greater than the volume displaced, and there is a larger increase in ICP with any incremental

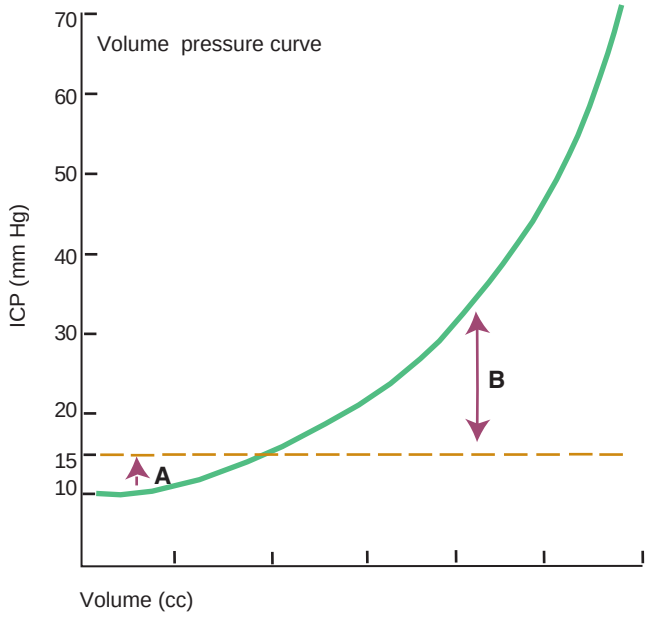


FIGURE 34-1 ▲ Volume–pressure curve. Volume–pressure response (VPR), also referred to as the pressure–volume index (PVI), provides a method of estimating the compensatory capacity of the intracranial cavity. Note that the intracranial pressure (ICP) remains within the normal limit of 0 to 15 mm Hg as long as compliance is normal and fluid can be displaced by the additional volume (**A**). Once the compensatory system is exhausted, a small additional volume causes a greater increase in pressure (**B**). Acute changes can cause serious and sometimes fatal neurological deterioration.

volume increase. Free communication of CSF between the lateral ventricles and infratentorium is lost as a result.

A major reason for controlling and decreasing ICP is the maintenance of cerebral oxygenation by adequate CBF, which is estimated clinically by the measurement of CPP. Many factors contribute to increased ICP along the volume–pressure curve. Drastic increases in ICP may result from hypercarbia, hypoxia, rapid eye movement sleep, pyrexia, or the administration of certain anesthetics. Additionally, ICP is affected by environmental stimulation and increased metabolic rates.[2]

Cerebral Perfusion Pressure

Cerebral perfusion pressure (CPP) is the blood pressure gradient across the brain. CPP is calculated by subtracting the mean ICP from the mean arterial pressure (MAP): CPP = MAP − ICP. When the CPP is greater than 100 mm Hg, there is a potential for hyperperfusion and increased ICP. When the CPP is less than 60 mm Hg, blood supply to the brain is inadequate, and neuronal hypoxia and cell death may occur. If MAPs and ICPs are equal, CPP is zero, indicating no CBF. CBF may also cease totally at pressures somewhat greater than zero. Patients with hypotension, such as postcardiac resuscitation or trauma patients, with normal ICPs (0 to 15 mm Hg), may have impaired CPP.

The autoregulation system for maintenance of constant blood flow does not function at pressures less than 40 mm Hg. Because an acutely injured brain requires a higher CPP than a normal brain, a minimum CPP of 70 mm Hg is required for maintenance of adequate cerebral perfusion and potentially improved outcomes in patients with brain injuries.[2] When CPP decreases, the cardiovascular system responds by increasing systemic pressure.

When brain damage is severe, as with widespread brain edema or when blood flow has been arrested in the brain, CBF may be reduced at relatively normal levels of CPP. The cause is impedance to the flow of blood across the cerebrovascular bed. If autoregulation is impaired, CBF may not increase despite increases in CPP. Increased ICP leads to ischemia, anoxic injury, decreased compliance, and possible herniation.[3]

▲ Increased Intracranial Pressure

Cushing's Triad

Cushing's triad is the classic syndrome of increased ICP and includes increased pulse pressure, decreased pulse, and change in respiratory pattern with pupillary changes. This syndrome usually occurs only in association with posterior fossa lesions and seldom with the more commonly observed supratentorial mass lesions, such as subdural hematomas. When these classic signs do accompany a supratentorial lesion, they are associated with a sudden pressure increase and usually herald a state of decompensation. Brain damage usually is irreversible if prolonged, and death is imminent without rapid intervention.

Cerebral Edema

Cerebral edema leading to increased ICP is a process common to multiple neurological illnesses including brain trauma, central nervous system (CNS) infections, brain tumors, stroke, etc. Its presence leads to secondary complications related to the expansion of brain tissue within the closed space of the cranium including impaired circulation leading to secondary hypoxia. Independently, cerebral edema can cause marked increases in ICP and must be treated aggressively. In general, once edema begins, its progression is rapid and difficult to control.

Treatment of cerebral edema may include using corticosteroids as well as osmotic diuretics directed at reducing ICP. These agents work by increasing plasma osmolarity, which draws fluid out of the brain tissue and into the circulating blood. The goal of therapy is to maintain plasma osmolarity up to 320 mOsm/L. See Chapter 36 for further information about cerebral edema.

Vasogenic Edema

The most common type of cerebral edema is vasogenic edema, which is characterized by a disruption in the blood–brain barrier and the inability of the cell walls to control movement of water in and out of the cells. Capillary permeability is affected, and fluid and protein are allowed to leak from the plasma into the extracellular space, resulting in increased extracellular fluid volume predominantly in the white matter. Common processes leading to vasogenic edema include brain tumors, cerebral abscess, and both ischemic and hemorrhagic stroke.

Cytotoxic Edema

Cytotoxic edema is characterized by swelling of the individual neurons and endothelial cells, which increases fluid

in the intracellular space and reduces available extracellular space, affecting the gray matter. Eventually, the cell membrane cannot maintain an effective barrier, and both water and sodium enter the cell, causing swelling and loss of function. Cytotoxic edema occurs after injuries such as anoxia or hypoxic injury.

Herniation

Herniation is defined as the displacement of tissue through structures within the skull and is the result of increased ICP. Transtentorial or uncal shifting of brain tissue through rigid openings in the skull or dura leads to displacement of midline brain structures and compression of structures in the CNS, causing traditional clinical herniation syndromes. See Chapter 36 for more information about herniation syndromes.

▲ Intracranial Pressure Monitoring

Monitoring ICP provides information that facilitates earlier interventions to prevent secondary cerebral ischemia and brainstem distortion. For ICP monitoring to be safe and effective, the indications for monitoring, methods of monitoring, and ethical considerations for patient care and nursing practice must be taken into account for each patient. Factors that affect patient selection include the potential benefit from invasive ICP monitoring and therapy, the patient's diagnosis and prognosis, and the availability of the appropriate level of critical care. ICP monitoring helps improve patient outcome by providing information about the likelihood of cerebral herniation and facilitating calculation of CPP. It is also helpful in guiding the use of potentially harmful treatments, such as hyperventilation, mannitol, and barbiturates.

Indications for Intracranial Pressure Monitoring

ICP monitoring is primarily used for guiding therapy. General guidelines exist to provide direction in therapy for patients at risk for and with increased ICP. Potential diagnostic indications for ICP measurement include brain injury, stroke, brain tumors, cardiac arrest, and surgery. The decision to use ICP monitoring should be based on clinical and radiographic evaluation as well as computed tomography (CT) diagnosis.

Currently, ICP monitoring is not indicated for patients with mild to moderate brain injury, defined as a Glasgow Coma Scale (GCS) score of 9 to 15. However, ICP monitoring may be appropriate for comatose patients or patients with severe brain injury with or without abnormalities on a CT scan of the head. Severe brain injury is defined as a GCS score of 3 to 8 with abnormal CT scan findings such as hematoma, contusion, edema, or compressed basal cisterns. ICP monitoring is also appropriate for patients who have brain injury with a normal CT scan and who have two or more of the following criteria: age greater than 40 years, any motor posturing, or systolic blood pressure less than 90 mm Hg.[4]

The upper limit of normal ICP is defined typically as 15 mm Hg. Although no prospective, randomized trial has been completed, a summary of the literature suggests that ICP monitors are beneficial in:

- Limiting indiscriminate use of therapies with potentially harmful consequences
- Reducing ICP through CSF drainage, thereby improving CPP
- Assisting in determining a prognosis
- Possibly improving outcomes[4]

Nontraumatic neurological disorders that may benefit from ICP monitoring are subarachnoid hemorrhage, intracerebral hemorrhage, large territory ischemic infarction, infection, hydrocephalus, and, rarely, brain tumors with associated edema or with significant lesion volume. Coagulopathy, systemic infection, CNS infection, and infection at the site of device insertion are relative contraindications to the placement of ICP monitors.

Intracranial Pressure Monitoring Devices

Various devices, such as intraventricular catheters, fiberoptic devices, and epidural monitors, are used to monitor ICP. A chosen ICP device should have pressure range capability of 0 to 100 mm Hg, accuracy within the ICP range of 0 to 20 ± 2 mm Hg, and a maximal error of 10% in the range of 20 to 100 mm Hg of ICP.[4] The type of monitor used depends on several clinical factors, the type of neurological process, and the patient's symptoms on presentation (Fig. 34-2). A variety of advantages and disadvantages exist with each of the devices; therefore, an awareness of potential complications is essential in the bedside management of the patient undergoing such monitoring (Table 34-2).

IVCs provide accurate, low-cost, and reliable ICP monitoring, and they are widely used. The catheter is a tubular instrument that is placed inside fluid-filled cavities in the ventricles. CSF is synthesized in these cavities and flows out to circulate over the surface of the brain. IVCs allow for simultaneous monitoring and treatment of ICP by intermittently draining CSF (Fig. 34-3). IVCs can be inserted

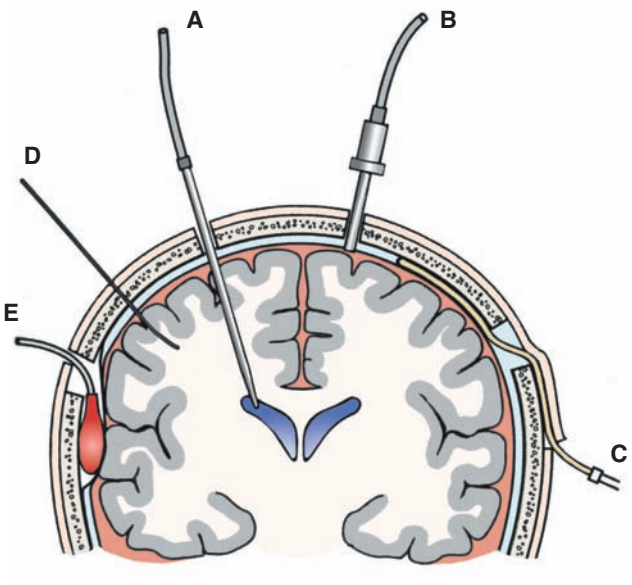


FIGURE 34-2 ▲ ICP monitoring systems: intraventricular (**A**); subarachnoid (**B**); subdural (**C**); parenchymal (**D**); and epidural (**E**).

Table 34-2 Advantages and Disadvantages of Intracranial Pressure Monitoring Devices

Monitoring Site	Advantages	Disadvantages
Intraventricular (ventriculostomy)	• Very accurate • True central direct measure of ICP • Can withdraw CSF to decrease ICP or measure compliance • Ease of CSF sampling	• Need transducer repositioned with every change in head position • High risk for serious infection • Difficult insertion in patients with small or displaced ventricles • Risk for intracerebral bleeding or edema along the cannula track
Intraventricular (fiberoptic catheter)	• Versatile; may be placed in ventricle or subarachnoid space • No adjustment of transducer with head movement	• Separate monitoring system required • Fragile catheter • Unable to recalibrate once device is placed
Intraparenchymal	• Ease of insertion • True brain pressures	• Infections rare, but serious
Lumbar/subarachnoid	• Simple-to-do single readings • No penetration of the brain parenchyma • Decreased risk for infection • Can sample CSF • Direct pressure management	• Contraindicated with evidence of increased ICP • Requires intact skull • Transducer repositioned with head movement
Subdural	• Ease of insertion	• Risk for serious infection
Epidural	• Low risk for infections • No transducer adjustment with head movement	• Imperfect correlation with intradural pressure (sensing through dura) • Operating room for placement • Unable to recalibrate once device is placed • Unable to drain CSF

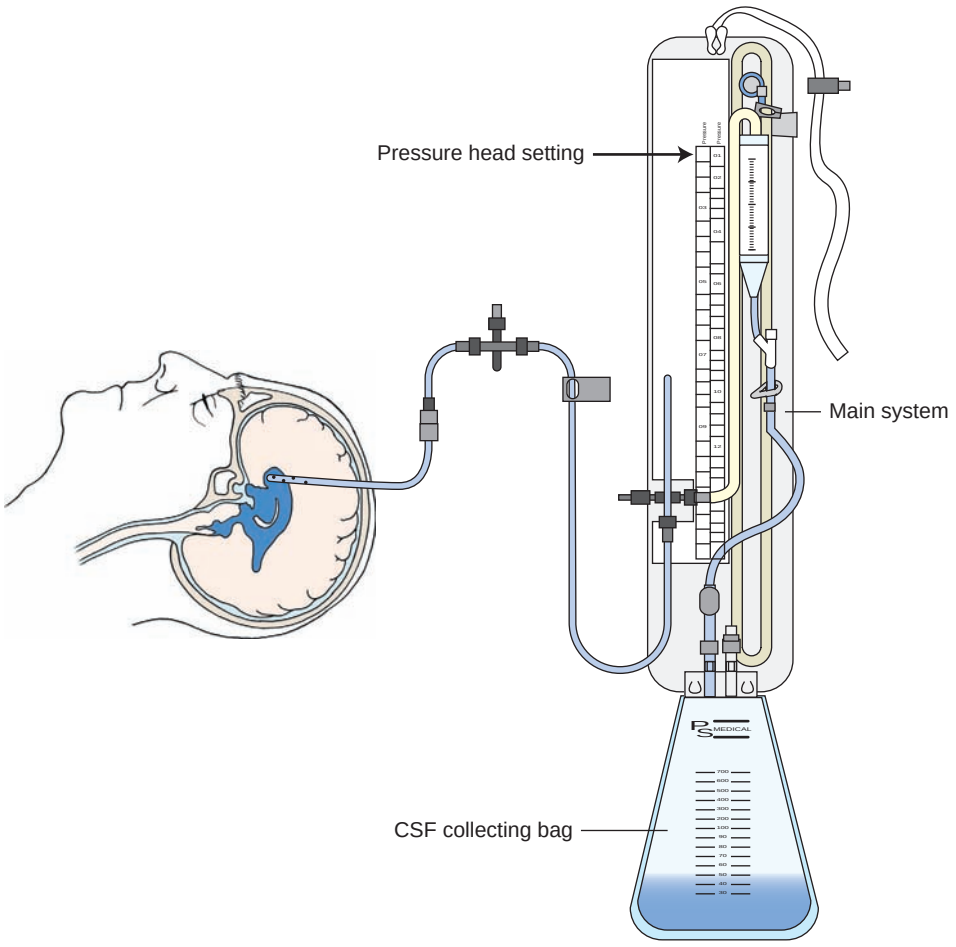


FIGURE 34-3 ▲ Intraventricular catheter system. (Courtesy of Medtronic Neurologic Technologies, Goleta, CA.)

under sterile conditions at the bedside in the intensive care unit (ICU) or in the operating room during surgery. Unlike parenchymal monitors, the IVC can be recalibrated *in situ*.

Fiberoptic monitors use fiberoptic technologies to measure ICP. The tip of the fiberoptic probe has a transducer, which is inserted into brain parenchyma, the ventricles it surrounds, or the subdural space. Fiberoptic monitors are easily inserted, and their use is increasing. Fiberoptic ventricular catheters provide benefits similar to those of IVCs, but at a higher cost. Of similar precision are parenchymal ICP monitors with fiberoptic or strain-gauge catheter-tip transduction; however, these devices are subject to potential measurement drift.

Subarachnoid, subdural, and epidural monitors are less accurate and less frequently used than the other types of monitors.[5] The subarachnoid bolt or screw is inserted through a twist drill hole to the level of the subarachnoid space and secured to a saline-filled pressure tubing transducer system. Epidural monitors are placed into the epidural space between the inner surface of the skull and the dura mater to monitor ICP.

Complications of Intracranial Pressure Monitoring Devices

Each type of monitoring system has potential complications. To ensure accurate measurements and reduce morbidity, the nurse must be alert to problems associated with ICP monitoring systems that could cause incorrect ICP measurements and complications. When the monitor indicates a change in ICP, the nurse must first determine whether the reading is accurate. If the reading is accurate, an attempt is then made to determine the reason for the pressure change. Table 34-3 provides a guide to troubleshooting ICP lines.

As with any invasive procedure, complications may occur. In critically ill patients with neurological problems, the risk/benefit ratio for any therapy must be considered before the implementation of that therapy. For instance, IVCs carry the potential risks for catheter misplacement, obstruction, infection, and hemorrhage. Use of antibiotic-impregnated IVCs is on the rise as a promising technology for reducing device-related infection rates. There are few published data about this practice to date; however, studies are ongoing.

Because drainage holes responsible for the collection of excessive CSF are very small, it is easy for the catheter to become obstructed; the nurse must monitor for this complication evidenced by either poor drainage of the system or change in the patient's neurological status. Malfunction or obstruction occurs at a rate of 6% to 10% in patients with an IVC and is significantly higher in patients with parenchymal or ventricular fiberoptic catheter-tip devices, at a rate of 9% to 40%.[6] Higher rates of obstruction have been correlated with malignant elevations of ICP greater than 50 mm Hg. A reduction in catheter infections has been reported with recent changes in insertion techniques, antibiotic prophylaxis, and improved CSF sampling methods.[6] Hemorrhage associated with IVC placement is poorly described in the literature, prompting the reporting of a 1.1% to 2.8% risk for hematoma formation. The hemorrhage rate is highly dependent on the choice of device.

Intracranial Pressure Waveforms

Waveforms of ICP provide an index of ICP dynamics, such as changes in intracerebral compliance. The appearance of ICP waveforms varies according to the measurement technique being used, the patient's pathological status and activities, interventions, or environmental changes. Hemodynamic and respiratory oscillations can be observed in ICP traces. Computerized systems are being developed to analyze waveforms and integrate ICP, CPP, and other relevant parameters.

Sometimes, the waveforms closely resemble arterial pressure waveforms; at other times, they resemble central venous pressure waveforms. To varying degrees, oscillations corresponding to intracranial arterial pulsations with retrograde venous pulsations are seen with each heartbeat (Fig. 34-4). In patients with ICP less than 20 mm Hg, a slower waveform, synchronous with respiration and caused by changes in intrathoracic pressure, can be seen (see Fig. 34-4, middle). Alterations in arterial driving force, disturbance of venous outflow, and cerebral vasodilation correlate with changes in waveform appearances. At times, a small "a" wave is superimposed on diastole, reflecting right arterial pressure.

Some patients exhibit waveform variation, most commonly A, B, and C waves. A waves, also known as plateau waves, are spontaneous, rapid increases of pressure ranging from 50 to 200 mm Hg, occurring at variable intervals (see Fig. 34-4, bottom). They tend to occur in patients with moderate elevations of ICP, last 5 to 20 minutes, and fall spontaneously. Plateau waves usually are accompanied by a temporary increase in neurological deficit.

Although the mechanism of A waves has not been established firmly, it is thought that they indicate decreased intracranial compliance; therefore, these waveforms should be identified and treated quickly. They may result from an increase in blood volume with a simultaneous decrease in blood flow. The sudden reversal of high pressure may be caused by increased CSF absorption. Falls in CPP with intact autoregulation and low intracranial compliance have been correlated with the initiation of plateau waves. Plateau waves may also be set off by a vasodilating stimulus or by nonspecific stimuli, such as hypoventilation or hyperventilation, pain, and aroused mental activities.

B waves are small, sharp, rhythmic waves with ICPs up to 50 mm Hg, occurring at a frequency of 0.5 to 2 per minute. They correspond to changes in respiration, providing clues to periodic respiration related to poor cerebral compliance or pulmonary dysfunction. B waves often are seen with Cheyne-Stokes respirations (see Chapter 36). They may precede A waves and increase as compliance decreases. At times, they occur in patients with normal ICP and no papilledema. They may be secondary to oscillations of cerebral blood volume.

C waves are small, rhythmic waves with ICPs up to 20 mm Hg, occurring at a rate of approximately six per minute. They are related to blood pressure. Like A waves, they indicate severe intracranial compression, with limited remaining volume residual in the intracranial space.

Intracranial Pressure Measurements

Normal measurements of ICP range between 0 and 10 mm Hg, with an upper limit of 15 mm Hg. During coughing or straining, a normal ICP may increase to 100 mm Hg. A patient's tolerance of a change in ICP varies with the acuteness of its onset. A patient with a slower buildup of ICP (eg, as the result of an expanding brain tumor) is typically more tolerant of elevations in ICP than a patient whose ICP increases rapidly (eg, as the result of an acute subdural

Table 34-3 Troubleshooting Intracranial Pressure Lines

Problem	Cause	Nursing Considerations and Interventions
No ICP waveform	Air between the transducer diaphragm and pressure source	Eliminate air bubbles with sterile saline solution.
	Occlusion of intracranial measurement device with blood or debris	Flush intracranial catheter or screw as directed by physician: 0.25 mL sterile saline solution is often used.
	Transducer connected incorrectly	Check connection, and be sure the appropriate connector for amplifier is in use.
	Fiberoptic catheter bent, broken	Replace fiberoptic catheter.
	Incorrect gain setting for pressure or patient having plateau waves	Adjust gain setting for higher pressure range.
	Trace turned off	Turn power on to trace.
False high-pressure reading	Transducer too low	Place the venting port of the transducer at the level of the foramen of Monro. For every 2.54 cm (1 inch) the transducer is below the pressure source, there is an error of ~2 mm Hg.
	Transducer incorrectly balanced	With transducer correctly positioned, rebalance. Transducer should be balanced every 2–4 h and before the initiation of treatment based on a pressure change.
	Monitoring system incorrectly calibrated	Repeat calibration procedures.
	Air in system: air may attenuate or amplify pressure signal	Remove air from monitoring line.
High-pressure reading	Airway not patent: an increase in intrathoracic pressure may increase $PaCO_2$	Suction patient. Position. Initiate chest physiotherapy.
	Ventilator setting incorrect	Check ventilator settings.
	PEEP	Draw arterial blood gases because hypoxia and hypercarbia cause increases in ICP.
	Posture	Head should be elevated 15–30 degrees unless contraindicated by other problems, such as fractures.
	Head and neck	The head should be positioned to facilitate venous drainage.
	Legs	Limit knee flexion. Avoid acute hip flexion.
	Excessive muscle activity during decerebrate posturing in patients with upper brainstem injury may increase ICP.	Muscle relaxants or paralyzing agents sometimes are indicated.
	Hyperthermia	Initiate measures to control muscle movement, infection, and pyrexia.
	Excessive muscle activity	
	Increased susceptibility to infection	
	Fluid and electrolyte imbalance secondary to fluid restrictions and diuretics	Draw blood for serum electrolytes, serum osmolality. Note pulmonary artery pressure. Evaluate input and output with specific gravity.
	Blood pressure: vasopressor responses occur in some patients with elevating ICP.	Use measures to maintain adequate continuous positive pressure.
	Low blood pressure associated with hypovolemia, shock, and barbiturate coma may increase cerebral ischemia.	
False low-pressure reading	Air bubbles between transducer and CSF	Eliminate air bubbles with sterile saline.
	Transducer level too high	Place the venting port of the transducer at the level of the foramen of Monro. For every 2.54 cm (1 inch) the transducer is above the level of the pressure source, there will be an error of approximately 2 mm Hg.
Low-pressure reading	Zero or calibration incorrect	Rezero and calibrate monitoring system.
	Collapse of ventricles around catheter	If ventriculostomy is being used, there may be inadequate positive pressure. Check to make sure a positive pressure of 15–20 mm Hg exists. Drain CSF slowly.
	Otorrhea or rhinorrhea	These conditions cause a false low-pressure reading secondary to decompression. Document the correlation between drainage and pressure changes.
	Leakage of fluid from connections	Eliminate all fluid leakage.
	Dislodgment of catheter from ventricle into brain	Contact physician regarding appropriate diagnostic studies and intervention. Use soft catheter designed for intraventricular measurement.
	Occlusion of the end of a subarachnoid screw by the necrotic brain	In most cases, remove screw.

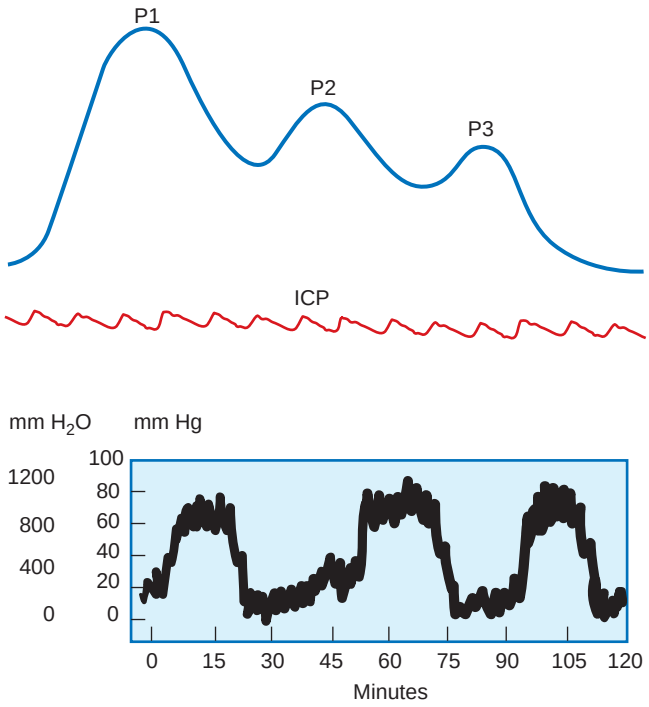


FIGURE 34-4 ▲ Intracranial pressure (ICP) waveforms. **Top:** A normal ICP pulse waveform may demonstrate three or more descending peaks. P1, the pressure wave, originates from choroid plexus pulsations. P2, the tidal wave, is more variable in shape and amplitude and ends on the dicrotic notch. P3, the dicrotic wave, follows the dicrotic notch and tapers down to the diastolic position unless retrograde venous pulsations cause a few more peaks. The P2 portion of the waveform most directly reflects the state of intracerebral compliance. As mean ICP rises, P2 progressively elevates, causing the pulse wave to appear more rounded. When a state of decreased compliance exists, the P2 component is equal to or higher than P1. **Middle:** An ICP waveform demonstrating hemodynamic and respiratory oscillations. Note the vascular pressure-type notches in the waveforms and the baseline variations that reflect respirations. **Bottom:** "A," or plateau, waves, associated with decreased intracranial compliance, may be secondary to an increase in blood volume with a simultaneous decrease in blood flow.

hematoma). Uncontrolled ICP between 20 and 25 mm Hg is considered extremely dangerous for the patient with brain injury. Sustained ICP greater than 60 mm Hg usually is fatal. ICP may rise to the level of the MAP. The greater the variations in the mean ICP, the more nearly exhausted are the compensatory mechanisms for intracranial volume increases.

CPP is the main indicator of the circulatory system's ability to infuse the brain. However, CPP has limitations; it measures only a single parameter that influences oxygen delivery and the neurons' ability to sustain injury. Neuronal demand for oxygen is governed by the cell's metabolic needs, which increase during neuronal activity or injury. Therefore, to understand the metabolic status of the neuron, both CBF and oxygen content in the blood must be measured. The equation CBF × OEF × SaO_2 is commonly used to calculate the cerebral metabolic rate for oxygen ($CMRO_2$). The oxygen extraction fraction (OEF), which is measured using both the arterial and venous oxygen content, describes how much oxygen is extracted. SaO_2 represents the oxygen saturation in the arterial blood. Necessary information can be obtained using multimodality monitoring, including blood flow studies, such as jugular venous bulb oximetry, as well as positron emission tomography and single-photon emission CT.

Jugular Venous Bulb Oximetry

Jugular venous bulb oximetry is an invasive technique that involves placing a sampling catheter in the internal jugular vein, with the tip of the jugular venous bulb at the base of the brain. Blood samples from this location measure the mixed venous oxygen saturation (SjO_2) of blood leaving the brain. This is normally 50% to 75%. The SjO_2 decreases when there is an imbalance between oxygen consumption and delivery. If the SjO_2 decreases to less than 50% (without a decrease in SaO_2), this implies either a decrease in CBF or an increase in oxygen utilization (higher $CMRO_2$). If CPP is maintained, a decrease in CBF is due to an increase in cerebrovascular resistance (CVR). Vascular spasm and an increase in CVR are very common after brain injury and are significantly worsened by hyperventilation; therefore, this technique is discouraged. An increase in SjO_2 greater than 85% implies either a hyperemia with an increase in CBF, shunting of blood away from neurons, or a decrease in $CMRO_2$ (impending cell death or brain death). It should be emphasized that SjO_2 is a measure of global cerebral oxygenation and is not sensitive to small areas of focal ischemia. However, SjO_2 may be of assistance in guiding some therapies for the patient with brain injury, including barbiturate-induced cerebral metabolic suppression and the rare use of induced hyperventilation.

Transcranial Doppler Ultrasound

Transcranial Doppler is a noninvasive method of assessing the state of the intracranial circulation. The velocity of flow can be measured in the middle, anterior, and posterior cerebral arteries, the ophthalmic artery, and internal carotid artery. Flow cannot be measured from velocity because the cross-sectional area of the arteries cannot be measured directly. However, the Doppler shift measured is inversely proportional to the diameter of the vessel, so that if all other factors remain constant, vascular narrowing leads to an increase in flow velocity. Doppler waveform analysis can provide further information about the state of blood flow, but the value and utility of these and other multimodality techniques are as yet unknown.

▲ Management of Increased Intracranial Pressure

In the stage between the onset of increased ICP and herniation, many treatments are available to reduce ICP and maintain adequate cerebral perfusion. No single management routine is appropriate for all patients. In addition to clinical pathways and nursing care protocols, algorithms for the incremental application and weaning of ICP management have been developed. First-tier therapy includes ventricular CSF drainage (as discussed previously), mannitol administration, respiratory support, and sedation and analgesia. Second-tier therapy includes hypothermia, barbiturate coma, optimized hyperventilation, hypertensive CPP therapy, and decompressive craniectomy.

Treatment goals for the patient with increased ICP are as follows: reduce ICP, optimize CPP, maintain adequate tissue

oxygenation, and avoid brain herniation. Most management techniques are oriented toward control of cerebral blood volume and CSF circulation, the two major mechanisms responsible for the regulation of ICP. Measures to reduce ICP are usually initiated when the patient's ICP increases to approximately 15 mm Hg.

Clinical Management

Hyperosmolar Therapy

Several options exist for the management of intracranial hypertension: hypertonic saline and mannitol administration.

HYPERTONIC AGENTS. Hypertonic saline is a mainstay for the treatment of patients with intracranial edema. Induced hypernatremia has been demonstrated to both increase cerebral perfusion pressures and decrease intracerebral pressure in the setting of multiple pathologies (stroke, subarachnoid hemorrhage, mass lesions). Timing of the initiation of such therapies continues to be debated and clear recommendations have yet to be provided. Therefore, treatment with hypertonic saline varies both in the concentration of the hypertonic solution and in the method of delivery. Induced hypernatremia can be achieved with solutions ranging from 2% to 23.4%. Additionally, agents can be administered via bolus dosing versus continuous infusion.

Peripheral intravenous access can be used if administering 2% hypertonic saline. However, giving a more hyperosmolar agent (>3%) requires central venous access to avoid infusion phlebitis or regional necrosis. A 250 mL bolus of 3% hypertonic saline can expected to raise serum sodium by nearly 5 mEq/L.[7] Central pontine myelinolysis remains a theoretical concern and has yet to be proven in the literature as a reality.

MANNITOL ADMINISTRATION. Mannitol, a hypertonic crystalloid solution that decreases cerebral edema, is also used as first-tier therapy for reducing ICP after brain injury. It is typically administered as a bolus intravenous (IV) infusion over 10 to 30 minutes in doses ranging from 0.25 to 2 g/kg body weight. Studies have demonstrated the effect of mannitol on ICP, CPP, CBF, and brain metabolism and have also shown a beneficial effect on long-term neurological outcome. The immediate plasma-expanding effect of mannitol, which reduces blood viscosity, increases CBF and cerebral oxygen metabolism, permitting cerebral arterioles to decrease in diameter. This lowers cerebral blood volume and ICP, while maintaining constant CBF. Use of hypertonic saline follows a similar pharmacodynamic effective in the treatment of elevated ICP and may provide an alternative therapy.[8–10]

Mannitol is excreted in the urine. If it is administered in large doses and serum osmolarity is greater than 320 mOsm, there is a significant risk for acute tubular necrosis and renal failure. Therefore, it is customary to measure serum osmolarity every 6 to 8 hours to a target of less than 320 mOsm.

A Foley catheter must be inserted when mannitol is administered. When mannitol is used during the early resuscitation phase in hypovolemic patients with brain injuries, crystalloid solutions are infused simultaneously to correct hypovolemia. Adjunct crystalloid fluid administration facilitates rapid renal excretion of mannitol, preventing renal failure. In the early phases of acute brain injury, mannitol is recommended as monotherapy. When mannitol is combined with furosemide, there is a risk for excessive diuresis, causing depletion of intravascular volume and electrolytes.

Respiratory Support

There are several considerations when managing the respiratory status of a critically ill patient with neurological injury. Mean airway pressure is the leading factor affecting ICP in the patient who is receiving ventilation therapy. Positive airway pressure is transmitted to the intracranial cavity through the mediastinum by preventing jugular venous outflow. Therefore, any condition decreasing pulmonary compliance or use of positive end-expiratory pressure (PEEP) increases the mean airway pressure and decreases the MAP and CPP.

Normocapnia is essential for maintaining stable ICP because carbon dioxide directly affects the degree of vasodilation in the cerebral blood vessels. Hyperventilation remains controversial in the management of increased ICP. Hyperventilation decreases the arterial carbon dioxide tension ($PaCO_2$) and results in cerebral vasoconstriction. As a result, the CBF is reduced because of the strong vasoconstrictive effect of hypocarbia on the cerebral arteries. The $PaCO_2$ should be lowered gradually to avoid a rebound effect of vasodilation from overcorrection. When hyperventilation is discontinued, ventilation rates should be gradually returned to normal. In the absence of a malignant increase in ICP, hyperventilation therapy ($PaCO_2$ < 25 mm Hg) should be avoided after a traumatic brain injury. Also, the use of prophylactic hyperventilation therapy ($PaCO_2$ < 35 mm Hg) during the first 24 hours after a traumatic brain injury should be avoided because it can compromise cerebral perfusion during a time of critically reduced CBF.[4,11] Severe, prolonged hyperventilation has been conclusively shown to worsen the outcome of patients with a severe brain injury and should be reserved for those cases in which all other therapies have failed. Severe hyperventilation is defined as a $PaCO_2$ of less than 25 mm Hg by jugular venous oxygen saturation monitor. Extreme hyperventilation is believed to cause secondary ischemia by constricting cerebral vasculature.[4]

Increasing intrathoracic pressure directly increases ICP, and therefore suctioning should be approached thoughtfully. Limiting the duration of passes of the suction catheter to no more than 5 to 10 seconds avoids hypoxia. And limiting the number of passes to one or two avoids overstimulation of the cough reflex and decreases the incidence of increased intrathoracic pressure and ICP.

Pharmacological Therapy

Analgesics, Sedatives, and Paralytics

In patients with a severe brain injury (GCS score < 8), pain medications and sedatives are used to:

- Reduce agitation, discomfort, and pain
- Facilitate mechanical ventilation by suppressing coughing
- Limit responses to stimuli, such as suctioning, which may increase ICP

Before starting patients on analgesics or sedatives, every effort should be made to implement nonpharmacological management techniques for pain, agitation, anxiety, and confusion. When medication is required, the agent should be selected based on both desired mechanism of action as well as half-life of the medication because restoration of the neurological function is critical for the care of these patients (Table 34-4). The treatment of pain lowers energy expenditures and thereby facilitates healing. Further, analgesics and sedatives may potentiate each other, allowing patients to achieve comfort and sedation.[12]

Analgesics

Opioid narcotics primarily affect the CNS. Fentanyl and morphine are two of the most frequently used opiate narcotics for brain-injured patients (see Table 34-4). They:

- Limit pain caused by injuries and nursing interventions
- Facilitate mechanical ventilation
- Potentiate the effect of sedatives[12]

Potentially life-threatening adverse effects of narcotics include respiratory depression, depression of the cough reflex, mood changes, nausea, and vomiting. Vital signs and pulse oximetry values must be monitored diligently when a patient receives IV pain medication. When preparing for such complications, the critical care nurse should ensure that intubation equipment is readily available in case respiratory depression occurs. Naloxone, which reverses CNS depression that can occur with the administration of fentanyl and morphine, may be required. With proper dosing and diligent nursing observation, narcotic analgesics can be used effectively in the critically ill patient.

The basic principles of narcotic administration are adequate pain relief and safe administration. It is especially important to begin with the lowest possible dose when treating a patient with neurological illness because the injured brain's response to medication is unpredictable. When a patient with a brain injury also has severe pain caused by multiple traumatic injuries, a continuous infusion of fentanyl or morphine is indicated and can be titrated every 15 to 30 minutes until pain control is achieved. For a patient with moderate pain, a 24-hour regimen of opiate narcotic administration has proved to provide increased pain relief versus an "as-needed" schedule of opiate dosing.

For the patient who can communicate, a verbal pain scale is used to assess pain. Standardized rating scales, such as a 1 to 10 pain scale, should be used to quantify and evaluate pain status and response to therapy. In addition to location, quality, and duration of pain, the nurse must document the effectiveness of analgesics every hour for 4 hours with initial administration, then every 4 hours, and 15 minutes after each dose change.[12] All hospitals have a pain management protocol that identifies analgesics that should be used, dosing titration guidelines, and documentation requirements. In addition, most protocols address sedation recommendations.

For the patient who is unable to communicate, physiological parameters are used to determine the effectiveness of pain management. The nurse assesses heart rate, respiratory rate, use of accessory muscles for breathing, and blood pressure. Adequate pain management leads to less patient movement, such as thrashing, which increases metabolic activity. The following clues can be used to determine whether adequate pain relief has been obtained for the patient who cannot communicate:

- Ease of breathing
- Mechanically ventilated patient whose breathing is synchronous with the ventilator
- Possible decrease in heart rate
- Less agitation as indicated by restful sleep state
- Cooperation with nursing interventions without excessive physical activity

Also, narcotics decrease gastric motility and may cause constipation. Patients with neurological illness in the critical care setting are often immobilized or confined to bed and are especially prone to constipation. All patients on narcotic therapy should be on a bowel regimen to avoid this complication, and the quality and frequency of bowel movements should be monitored closely. Straining increases ICP and could possibly be avoided with strict bowel regimens. Patients may also be susceptible to nausea and vomiting with the administration of narcotics. Protection of the airway is especially important in the patient with a neurological diagnosis. Mechanically ventilated patients require the insertion of a nasogastric or orogastric tube to decompress the stomach and prevent vomiting.

Sedatives

The most commonly used sedatives in the ICU are benzodiazepines, which cause little change in CBF, ICP, and cerebral metabolic rate, and potentiate the effects of analgesic agents. Midazolam, diazepam, and lorazepam are used frequently for sedation before ICU procedures and as needed to treat anxiety (see Table 34-4). Lorazepam is frequently used for alcohol withdrawal and anticonvulsant therapy. Midazolam, in combination with fentanyl, is most often used for sedation before procedures to produce amnesia of immediate events. Side effects of sedatives include respiratory depression, hypotension, and somnolence. It is mandatory that resuscitation equipment be available at all times when IV benzodiazepines are used. Benzodiazepines should be administered at the lowest possible dose that produces effective sedation, without causing somnolence. As with analgesic agents, frequent vital signs must be obtained with the administration of sedatives. Recommended minimal documentation of vital signs should be every hour for 4 hours, then every 4 hours, and 15 minutes after every dosage change.[12]

Various scales may be used to document the patient's response to sedation. The target sedation level for a patient with a critical neurological illness is one that allows easy arousal of the patient with light touch or voice.[12] For the patient who cannot communicate (as discussed earlier with analgesic use), the assessment of physiological parameters can be used to determine the patient's response to sedation in order to achieve maximal comfort. Pharmacological management of sedation is only one strategy to treat anxiety. Nursing measures to provide comfort must be offered in addition to medications.

Anesthetics

Propofol is a fat-soluble anesthetic that is administered as a continuous infusion to decrease agitation in the critically ill patient. Studies have shown that propofol may decrease

Table 34-4 Major Classes of Pharmacological Medications Used to Treat Neurological Emergencies

Class	Medication	Mechanism of Action/Dosage	Comments
Direct vasodilators	Sodium nitroprusside Nitroglycerin Hydralazine	Directly dilate the peripheral vasculature and lower vascular resistance Sodium nitroprusside: start at 0.3 mcg/kg/min, maximal continuous IV infusion 10 mcg/kg/min Nitroglycerin: start at 5 mcg/min, then titrate as needed upward q5min (max 100 mcg/min) Hydralazine: 10–20 mg IV, may be repeated as needed	• Dilate the cerebral vasculature • Increase cerebral blood volume and ICP, decrease MAP and CPP • Sodium nitroprusside: at high doses may cause cyanide toxicity, check thiocyanate levels, protect infusion from light • Nitroglycerin: venous dilation, caution with high doses; may cause hypotension • Hydralazine: used in hypertensive emergency, may cause hypotension and headache
β-Adrenergic antagonists	Metoprolol Esmolol	β-Adrenergic receptor antagonist Metoprolol: 5 mg IV q3–5 min in three doses, followed by PO administration 25–50 mg PO twice daily and may increase to 50–100 mg PO twice daily Esmolol: for hypertensive emergency, bolus dose 500 mcg/kg over 1 min, then 50–200 mcg/kg/min	• Do not affect CBF • Use with caution with Cushing's response, can potentiate bradycardia
Mixed α- and β-adrenergic antagonists	Labetalol	Selective α- and nonselective β-adrenergic receptor antagonist Labetalol: for hypertensive emergency, start 20 mg IV over 3–5 min, followed by 40–80 mg IV every 10 min PRN up to 300 mg, or IV infusion 0.5–2 mg/min	• Reduce systemic vascular resistance (SVR) • Improve CPP and do not increase ICP • May slow heart rate
Calcium channel antagonists	Verapamil Diltiazem Nicardipine	Prevent transport of calcium ions in vascular smooth muscle, resulting in vasodilation, decreased myocardial contractility, decreased heart rate Verapamil: for supraventricular tachycardia, 5–10 mg IV over 2 min, followed by 40–80 mg three to four times a day Diltiazem: 20 mg over 2 min, continuous infusion 5–15 mg/h Nicardipine: Start 5 mg/h; increases 2.5 mg/h every 5–15 min; Max dose 15 mg/h	• Use with caution; may cause cerebral vasodilation with increased ICP • Contraindicated in patients with tumors and cerebral edema
Angiotensin-converting enzyme (ACE) inhibitors	Lisinopril	Shift the upper and lower limits of blood brain autoregulation by inhibiting angiotensin II–mediated vascular tone in large cerebral arteries, while small vessels vasoconstrict Lisinopril: start 10 mg/d, titrate to 80 mg/d PRN	• Preserve CBF after single dose • Increase CBF with chronic treatment • Have the potential to increase ICP in patients with intracranial hypertension by increasing CBF
Osmotic diuretic	Mannitol	Indicated for intracranial hypertension Mannitol: 0.25–2 g/kg IV over 30–60 min	• If infused too quickly, may contribute to renal insufficiency
Nondepolarizing muscle blockading agents	Pancuronium	Skeletal muscle paralysis Bolus 0.04–0.1 mg/kg Continuous infusion	• May cause tachycardia and dysrhythmias • Patient must be mechanically ventilated
	Atracurium	Skeletal muscle paralysis Bolus 0.4–0.5 mg/kg IV Continuous infusion 4–12 mcg/kg/min IV	• Rare occurrence of prolonged weakness after discontinuation of drug infusion • Patient must be mechanically ventilated • Use in patients with hepatic and renal impairment • Neuromuscular blockade should be preceded by adequate sedation and analgesia, which should be maintained during course of blockade
	Cisatracurium	Skeletal muscle paralysis Bolus 0.1–0.2 mg/kg Continuous infusion 2.5–3 mcg/kg/min IV	• Rare incidence of prolonged weakness after continuous IV infusion • Patient must be mechanically ventilated • Use in patients with hepatic and renal impairment

(continued on page 755)

Table 34-4 Major Classes of Pharmacological Medications Used to Treat Neurological Emergencies (continued)

Class	Medication	Mechanism of Action/Dosage	Comments
Sedatives	Diazepam Lorazepam Midazolam	Benzodiazepines are sedatives and hypnotics that induce anterograde amnesia Diazepam and lorazepam are also used to stop seizure activity Diazepam: 10–20 mg IV no faster than 2 mg/min, then repeat q4h Lorazepam: 0.05–0.2 mg/kg/dose, up to 8 mg, may repeat every 15 min, continuous infusion 1 mg/h and titrate to goal, not to exceed 8 mg/h Midazolam: 0.1–0.3 mg/kg bolus, no faster than 4 mg/min, continuous infusion 0.05 mg/kg/h up to 1.0 mg/kg/h	• May cause somnolence, hypotension, delirium, hallucinations, respiratory depression • Titrate to goal using sedation scale • Lorazepam/midazolam: use with caution in patients with hepatic/renal failure, contraindicated in patients with acute narrow-angle glaucoma or in shock, and in older patients
Anticonvulsants	Phenytoin Carbamazepine	Indicated for tonic-clonic seizures and partial seizures Phenytoin: loading dose 15–20 mg/kg for status epilepticus, no faster than 50 mg/kg; maintenance dose 200–500 mg daily or in divided doses three times a day Carbamazepine: 200 mg twice daily, maintenance 200–400 mg three times a day	• May cause ataxia, lethargy, movement disorders, rash, coarse facies, lymphadenopathy • May decrease theophylline, oral contraceptive, and warfarin levels • Phenytoin levels may increase with methsuximide and alcohol • Phenytoin levels may decrease when used with valproic acid and Tegretol • Contraindicated with alcohol ingestion, sinus bradycardia, heart block
Analgesics	Morphine Fentanyl Hydromorphone	Opiate analgesics blunt the pain response by interfering with central and peripheral pain pathways Morphine: bolus 1–4 mg, continuous infusion 0.07–0.5 mg/kg/h Fentanyl: bolus 50–100 mcg over 1–2 min, continuous infusion 0.7–10 mcg/kg/h Hydromorphone: continuous infusion 7–15 mcg/kg/h	• May cause respiratory depression; must have naloxone readily available • Have resuscitation equipment nearby at all times • Use the lowest dose that provides adequate pain relief • Use a 1–10 pain scale to assess effectiveness of pain relief • For severe pain, around-the-clock administration or a continuous infusion of pain medication provides more efficient pain relief than PRN dosing • Use lower doses in older patients • Use nonpharmacological approaches to pain management • Side effects: decreased gastric motility; nausea/vomiting, tremulousness
Sedation/Anesthetic	Propofol Precedex	Propofol is an IV general anesthetic agent that can be used to treat agitation in the ICU Propofol: bolus not necessary, continuous infusion 5–50 mcg/kg/min Precedex: 0.2–0.7 mcg/kg/h; start 1 mcg/kg then titrate to effect	• Patient must be mechanically ventilated (propofol) • Must have dedicated IV line • Must use aseptic technique • May cause hypotension • Must provide analgesics and sedation • Use for the shortest period possible • Can extubate patient while Precedex is still infusing • Watch for atrial fibrillation in patients prone to A fib
Barbiturates	Phenobarbital Pentobarbital	Used to produce CNS depression and reduce the spread of an epileptic focus Phenobarbital: loading dose 6–8 mg/kg IV; maintenance dose 1–3 mg/kg/24 h IV Pentobarbital: loading dose 3–10 mg/kg over 30 min; maintenance dose 0.5–3.0 mg/kg/h	• May cause respiratory depression and cardiac depression • Patient must be mechanically ventilated • Continuous EEG monitoring for barbiturate-induced coma

CBF, ICP, CPP, and cerebral metabolic function.[13] Propofol is easily titrated based on patient response. It also has a short half-life and can be discontinued for neurological assessments. Propofol can decrease the level of consciousness in 2 minutes. A common side effect is hypotension; therefore, frequent blood pressure monitoring must be performed, especially if the patient has increased ICP. Since this anesthetic agent causes loss of respiratory drive, diligent airway protection must be provided for the patient receiving propofol. To prevent respiratory depression, the patient must be intubated and mechanically ventilated when propofol is administered. For these reasons, the patient who receives a continuous infusion of propofol must be cared for in the ICU, with constant surveillance by the critical care nurse. In most states, an anesthesia provider may administer an IV bolus dose of propofol.

Propofol infusion syndrome is a rare but significant adverse drug event associated with prolonged propofol use (>48 hours) at high doses (>4 mg/kg/h). This syndrome is characterized by hypotension, severe metabolic acidosis, rhabdomyolysis, hyperkalemia, renal failure, hepatomegaly, and cardiovascular collapse. Therefore, duration of propofol therapy should be monitored and limited when possible.

Other considerations with propofol are related to the handling of the drug. Propofol is manufactured by using a fat emulsion, making it a powerful medium for bacterial growth. Propofol must be handled meticulously to prevent the risk for bacterial or fungal infection associated with its use. The fat calories provided by propofol should be included when calculating nutritional supplementation for the patient receiving this infusion. It may be necessary to monitor the triglyceride level for patients receiving propofol longer term.

Dexmedetomidine (Precedex) is an alpha-adrenergic receptor agonist that provides sedation, antianxiolysis, and analgesia without respiratory depression.[14] This IV medication may be a good alternative in the critical care setting since patients can be weaned from the ventilator and extubated while receiving this medication. The most common side effect is hypotension.

Neuromuscular Blockade

Neuromuscular blockading (NMB) agents (see Table 34-4, p. 754) are used to induce muscle paralysis and remain a "last resort" therapy. An NMB agent blocks the transmission of acetylcholine at the motor end plate, producing skeletal muscle paralysis. Reversal of NMB agents is provided by acetylcholinesterase inhibitors such as neostigmine, edrophonium, and pyridostigmine. The use of an NMB agent requires mechanical ventilation with full support. Resuscitation equipment must be present at all times when a patient is treated with an NMB agent. For a conscious patient, the inability to move and communicate is frightening; therefore, concurrent administration of analgesia and sedation is mandatory.[15] Analgesia and sedation provide the added benefit of producing amnesia.

Complications common with most NMB agents are tachycardia, hypotension, and dysrhythmias. Cardiac medications such as antidysrhythmics, diuretics, and calcium channel and β-blockers can potentiate the action of NMB drugs. Certain antibiotics, such as aminoglycosides and clindamycin, can potentiate the action of paralytic agents. Alterations in body temperature or acid–base balance and electrolyte disturbances also alter the action of NMB agents.[15]

A troubling complication of paralytic therapy is prolonged polymyopathy. A condition known as acute quadriplegic myopathy syndrome, or postparalytic quadriparesis, is one of the most devastating complications caused by prolonged use of an NMB agent.[15] This condition is manifested by prolonged weakness of the upper and lower extremities. The extraocular motor muscles are usually spared in this condition. A patient may also experience painful muscle fasciculations. To avoid these complications, the smallest dosage of an NMB agent should be used to obtain adequate respiratory support.

Peripheral nerve stimulation monitoring is mandatory with NMB therapy;[15] its use every 4 hours, with a dosage change as necessary, may help prevent complications associated with NMB therapy. A peripheral nerve stimulator is a small hand-held device used in the ICU to monitor the depth of NMB in the patient receiving prolonged paralytic therapy. This device delivers a small jolt of energy to the ulnar surface of the wrist, causing the thumb to twitch. The train-of-four method is used to measure the efficacy and depth of NMB. The peripheral nerve stimulator delivers four 2-Hz stimuli of 0.2 millisecond delivered at intervals of 0.5 second to the ulnar nerve at the wrist. Normally, the thumb twitches four times when the peripheral nerve stimulator is activated. The nurse observes thumb movements after peripheral nerve stimulation. If the thumb twitches two or three times, NMB dosing is usually sufficient. If four thumb twitches occur, paralysis is ineffective. If no twitches occur, paralysis is excessive, and the dose of the NMB agent must be reduced.

Excellent nursing care also can help prevent some of the complications caused by NMB agents. A rigid patient repositioning schedule must be maintained to prevent the development of pressure ulcers. Aspiration precautions and excellent pulmonary toilet must be maintained at all times to avoid pneumonia. Also, deep venous thrombosis prophylaxis must be implemented before induction of paralysis.

Barbiturate Coma

For the patient with severe and refractory elevated ICP, an induced barbiturate coma may be attempted to decrease systemic metabolic activity in an attempt to preserve brain function. It must be understood that induction of a barbiturate coma is a rare therapy and is used only as a last resort to save brain function. Criteria for inducing a barbiturate coma includes a GCS score of less than 7, ICP greater than 25 mm Hg at rest for 10 minutes, and failed maximal interventions, including drainage of CSF, mannitol, analgesia, and sedation. Barbiturate coma is typically used for less than 72 hours.

Barbiturates suppress seizure activity and reduce cerebral metabolic activity and cerebral oxygen demand (see Table 34-4, p. 754). Barbiturates affect CBF, metabolic demand, electroencephalographic (EEG) activity, and systemic hemodynamics. CBF may decrease by 50%. Barbiturate therapy appears to have a direct restrictive effect on cerebral vasculature by diverting small amounts of blood from well-perfused areas to ischemic areas, thereby improving cerebral pressure.

Before the administration of barbiturates, the following must be provided for the patient: a secure airway with mechanical ventilation; ICP, blood pressure, cardiac, and pulmonary artery monitoring; and continuous EEG monitoring. An EEG is obtained before administration of a barbiturate so that spontaneous electrocortical activity is documented.

The EEG pattern of burst suppression is the most common method to establish barbiturate dosing. The barbiturate dose is adjusted until EEG burst suppression is achieved. The initial dose may be supplemented by an IV bolus to achieve burst suppression. Barbiturate serum levels alone are poor measures of therapeutic efficacy and systemic toxicity.

Barbiturates should be discontinued with any of the following clinical findings:

- ICP less than 15 mm Hg for 24 to 72 hours
- Systolic blood pressure less than 90 mm Hg despite the use of vasopressors
- Progressive neurological impairment, as evidenced by deterioration of brainstem auditory evoked responses
- Cardiac arrest

At the time of discontinuation, the barbiturate dosage is gradually reduced over 24 to 72 hours. Arousal is gradual and prolonged, even after blood levels have been zero for several days. The patient must be weaned slowly and carefully from mechanical ventilation because residual muscle weakness may occur. Patients may experience facial weakness for several days after the barbiturate has been discontinued. Occasionally, the patient may experience dysarthria, related to weakness of the muscles of speech. During the first 24 hours of barbiturate withdrawal, slow, abnormal muscle movements may be observed.

Blood Pressure Management

The regulation of blood pressure is an important aspect of managing the patient with increased ICP. Blood pressure is directly related to cerebral blood volume, perfusion pressure, ischemia, and compliance. For patients with brain injuries, the preservation of CPP and maintenance of systemic oxygenation are two important goals. Also, patients with the most severe brain injuries are at risk for secondary injury caused by hypotension and hypoxia. Patients with brain injury may have increased metabolic oxygen consumption, mild hypertension, and increased cardiac indices. Invasive blood pressure monitoring is routinely used to provide continuous and accurate blood pressure measurement during the acute management phase of patients with brain injuries. MAP is the parameter used for evaluating CPP and the efficacy of antihypertensive or vasopressor therapies.

Patients with brain injuries must be monitored continuously for any adverse effect from drug therapies. In addition to the MAP, the cardiac output may be monitored in the acute management of patients with head injuries. See Chapter 17 for further description of hemodynamic monitoring. Drug therapies to manage blood pressure may cause a precipitous increase or decrease in cardiac output. When the cardiac output is low, patients with neurological injuries are in jeopardy of further ischemic injury since the protective mechanisms of autoregulation are disturbed. The cardiac index is usually maintained at 3 L/min/m^2 because patients with head injuries frequently have increased metabolic needs. The pulmonary artery capillary wedge pressure is usually maintained at 12 to 15 mm Hg in patients with head injuries. In addition, noninvasive continuous pulse oximetry and arterial blood gas measurement are used to determine arterial oxygen content.

Different classes of antihypertensive medications can be used to treat systemic hypertension in the critically ill patient.[16] To maintain CPP, avoidance of hypotension is critical for patients with neurological injury. Moreover, any dysrhythmias must be managed aggressively to avoid secondary hypotension. As with any medication, the patient must be assessed for the development of adverse effects associated with cardiovascular medications, including bradycardia, hypotension, myocardial ischemia, tachycardia, and decreased cardiac output.

For patients with acute ischemic stroke, acute hypertension is defined as a systolic blood pressure greater than 185 mm Hg and a diastolic blood pressure greater than 110 mm Hg. These patients are especially dependent on systemic blood pressure to maintain perfusion through a partially occluded intracranial vessel. IV hydralazine or labetalol are frequently used for elevated blood pressure and should be titrated slowly to avoid sudden episodes of hypotension. If the patient remains hypertensive after hydralazine or labetalol administration, nicardipine or nitroprusside may be used (see Table 34-4, p. 754). Both nicardipine and nitroprusside are potent vasodilators and can lower the blood pressure quickly. They are administered as continuous infusions, are easily titrated, and have relatively short half-lives. These medications must be used in the ICU or emergency department where continuous blood pressure monitoring can be provided. Hypotension may occur if the infusion is increased too quickly.

For patients with hemorrhagic brain injury, a more rigorous blood pressure range is maintained. An MAP greater than 110 mm Hg is avoided in these patients.[17] Angiotensin-converting enzyme (ACE) inhibitors and β-blockers are often used to treat hypertension in the head-injured patient with systemic hypertension. β-Blockers are most often used because of their safe side effect profile, although they may cause bradycardia. Calcium channel blockers are usually avoided in head-injured patients because of their potential to exacerbate cerebral edema. See Table 34-4 for additional information.

Seizure Prophylaxis

Often, patients with neurological injury are prone to seizure activity. Seizure activity markedly elevates the cerebral metabolic rate and CBF and may lead to hypoxia. In patients with traumatic brain injury, use of antiepileptic agents for 7 days has been shown to decrease the incidence of early seizure activity but does not prevent development of later seizures. Anticonvulsant therapy may be used to prevent early posttraumatic seizure, especially in patients with a reduced seizure threshold (eg, brain tumor; temporal lobe pathologies). Phenytoin, levetiracetam, and carbamazepine are considered treatment agents for preventing early (<7 days) posttraumatic seizure activity. Levetiracetam may be more convenient to administer as no blood drug levels need to be followed.

The treatment of choice for acute-onset seizures (such as tonic–clonic seizures) in the critically ill patient remains diazepam. The patient should be positioned on his or her side and an oxygen mask applied. Attempting to restrain a seizing patient may lead to joint dislocation or fracture and this should be avoided. When seizure activity has subsided, the nurse should obtain a serum glucose analysis to determine whether hypoglycemia is a contributing cause. An EEG may also be ordered to determine whether the patient is continuing to experience subclinical seizures and to determine seizure focus.

Table 34-5 Nursing Considerations for Patients at Risk for Increased Intracranial Pressure

Problem	Nursing Action	Rationale
Adequate ventilation	• Assess respiratory patterns and rate	• Indicates neurological changes, pain status, and patency of airway
	• Suctioning: Preoxygenate with 100% O_2, one or two catheter passes, no more than 10 s per catheter insertion	• Prevents increased CO_2 (vasodilator that increases ICP); decreases coughing stimulation and increased intrathoracic pressure
	• Monitor continuous pulse oximetry and blood gases	• Alerts nurse to airway problems; good indicator of hemodynamics of respiration
Neurological assessment	• Evaluate patient baseline neurological status at beginning of shift (preferably with previous shift RN)—mental status; pupil shape, size, and response; motor function	• Subtle changes from baseline indicate deterioration and the need for early intervention
	• Assess vital signs—note trends (review ordered parameters for notification of physician)	• MAP directly correlates with ICP in patient with loss of autoregulation
	• Review nursing actions and emergency algorithm for neurologic deterioration (available medications—mannitol, hyperventilation, etc.)	• Ensures optimal benefit to patient and decreases secondary injury from prolonged ICP
Positioning	• Place head of bed flat or at 30 degrees elevation per orders	• Promotes cerebral perfusion or facilitates venous drainage; orders based on physiological process
	• Maintain head in neutral position	• Promotes jugular outflow
	• Avoid hip flexion	• Decreases intrathoracic pressure
	• Assess agitation in restrained patients	• Increases ICP
	• Turn patient every 2h, instructing patient to exhale with turn	• Prevents skin breakdown and avoids Valsalva maneuver during repositioning
	• Carry out passive range-of-motion exercises	• Prevents contractures while avoiding Valsalva-inducing isometric contractions
	• Avoid clustering of patient activities (eg, turning, bathing, suctioning)	• Produces prolonged ICP spikes
	• Use therapeutic interventions for emotional upset—speak with soft voice, use caution with unpleasant conversations, decrease noxious stimuli (noise), use therapeutic touch	• Causes elevations in ICP; comatose patients still respond to unpleasant environmental stimuli
Transport of patient with invasive ICP monitor	• Confirm time of test or possibility of completing as portable study	• Avoids excessive delays in uncontrolled and potentially overstimulating environment
	• Prepare respiratory therapy and other assistants during transport	• Adequate oxygenation remains a priority; multiple lines necessitate additional manpower
	• Gather transport supplies (sedation if ordered, transport monitor, antihypertensives)	• Prepare for intervention with any adverse patient response during travel specific to contributors of increased ICP
	• Assist with transfer of patient to diagnostic table with RN at head of bed monitoring device	• Ensures patient protection and provides for monitor equipment recalibration for accuracy of monitoring
	• Monitor and record hemodynamics and ICP dynamics during study	• Monitors patient response to procedure
Temperature control	• Frequent temperature checks (oral or rectal preferred if no contraindications)	• Cerebral metabolic rate increases with elevated body temperature
	• Confirm orders for early treatment of fever and aggressively treat	• Increased CBF increases ICP
	• Provide gradual cooling with cooling blanket, closely monitored	• Shivering increases ICP
Glycemic control	• Monitor serum glucose and fingersticks as ordered (every 4–6h)—adhere closely to sliding scale protocols in nondiabetic patients	• Alterations in glucose can produce neurological changes (ie, changes in metabolic rate)
	• Maintain euvolemia with normal saline	• Hypotonic glucose IV solutions should be avoided
Bowel and bladder regimens	• Administer daily stool softeners as ordered	• Reduces risk for straining and increased intra-abdominal pressure, which increases ICP
	• Avoid enemas	• Prevents Valsalva maneuver
	• Assess patency of Foley catheters	• Important to monitor amount of diuresis, especially in patients treated with osmotic diuretics
	• Document strict intake and output	• Important to maintain euvolemia
Seizure precautions	• Seizure precautions per hospital protocol (padding, etc.)	• Prevents injury in high-risk patients
	• Monitor serum anticonvulsant drug levels	• Maintains therapeutic levels

Other Management Methods

Hypothermia

Hypothermia continues to be explored as a means for reducing the brain's metabolic demands during peak times of cerebral edema and brain injury. One difficulty is cooling the patient adequately to achieve optimal neuroprotection. To date, the degree of coolness has not been established, although it continues to be investigated in multicenter clinical trials. However, control of fever is essential and is being more aggressively addressed using different types of cooling devices (both surface cooling and intravascular cooling devices).[18]

Decompressive Craniectomy

Another strategy used for managing refractory intracranial hypertension is decompressive craniotomy. This surgery is based on the theory that ICP can be reduced through surgical release of the rigid skull. Although surgical decompression remains an option for patients with uncontrollable ICPs, studies of patients suffering massive cerebral edema and refractory intracranial hypertension after ischemic stroke have shown varying results when comparing postdecompression outcomes and outcomes after best medical management.[19–28] However, the procedure remains widely used for patients with malignant cerebral edema after traumatic brain injury. Further studies are in progress to evaluate the risks and benefits of craniectomy for patients with traumatic brain injury. Evaluation of long-term morbidity and mortality as well as the best timing for this procedure continues.[19–27]

Patient Care Considerations

Nursing care activity can compound primary and secondary intracranial insults, contributing to rapid deterioration in the unstable patient who has lost intracranial compliance, autoregulation, and vasomotor tone.[24] Patient positioning, agitation, pain, hemodynamic and respiratory status, and seizures can all contribute to a patient's elevated ICP. The following sections describe a few patient management strategies for reducing ICP (Table 34-5).

Positioning

Primary positioning strategies for the patient with impending or active increased ICP include placement of the head and neck in a neutral position. Extreme neck flexion, extension, or rotation restrict venous drainage from the head through the internal jugular venous system and the vertebral venous plexus, increasing the total intracranial content. Decerebrate or decorticate posturing may also increase ICP. In addition, head-of-bed elevation has been shown to promote venous drainage and decrease ICP. The head is elevated 15 to 30 degrees, unless contraindicated by spine or limb fractures.

Tracheostomy ties and cervical collars are frequently checked for proper fit. Flexion of the hips greater than 90 degrees is avoided because it contributes to intra-abdominal and thoracic pressures and also impairs venous outflow.

Environmental Stimuli

Environmental stimuli contributing to pain, stress, or anxiety can increase cerebral metabolic rates and blood flow, confounding the management of increased ICP. Pain control and sedation are essential to reduce environmental overload, with consideration for the need for serial neurological assessments in the critically ill patient. Anxiety and discomfort in the ICU cannot be underestimated and should be considered in the patient who is neurologically impaired. Periods of uninterrupted sleep and rest should be provided between activities. Only essential interventions are performed during times of poor intracranial compliance, and activities are spaced to avoid a cumulative effect. Also, avoiding unnecessary painful procedures, such as frequent blood draws, is helpful.

▲ Clinical Applicability Challenges

CASE STUDY

Mr. H., a 56-year-old retired man, is brought by ambulance to the emergency department after having fallen down. At that time, he had speech problems and right arm and leg weakness. His neurological baseline examination and following workup suggests a large middle cerebral artery (MCA) territory ischemic infarction.

Past medical/surgical history: Hypertension and hyperlipidemia

- Allergies: none
- Social history: retired military computer analyst, married with supportive wife, three biological children, ages 28, 26, and 25, and all healthy; social ingestion of alcohol; smoking history 1 pack per day for 35 years
- Medications: lisinopril (Zestril), 20 mg/d; atorvastatin (Lipitor), 10 mg/d

Mr. H. is admitted to the neuroscience ICU for acute stroke management, monitoring, and treatment of potential complications. Because of his large left MCA stroke, he is at risk for cerebral edema, seizure, and worsening of his stroke. During the first 24 hours, neurological checks are completed hourly, and findings remain unchanged compared with baseline examination findings.

(continued on page 760)

CASE STUDY *(Continued)*

On day 2, neurological examination shows a decrease in level of consciousness, a decrease in heart rate to 55 beats/min, and an increase in blood pressure to 200/110 mm Hg. He requires immediate intubation for airway protection. The nurse suspects that Mr. H. is experiencing increased intracranial pressure (ICP) resulting from either cerebral edema or hemorrhagic conversion of the ischemic stroke territory. Blood samples are sent to the laboratory for analysis; computed tomography (CT) scan is obtained. The neurology specialist and neurosurgeon are notified of the rapid change in Mr. H.'s status.

The CT scan confirms massive cerebral edema with midline shift (no hemorrhage noted). Foley catheter is placed and Mr. H. is given his first dose of mannitol. The neurosurgeon places an intraventricular drainage device under sterile conditions at the bedside with initial ICP recorded at 32 mm Hg. Mr H's nurse activates strict interventions to avoid increased ICP including: maintaining a quiet environment, limiting nursing activities, and avoiding flexion and extension of the neck. The patient's blood pressure decreases, and he does not require antihypertensive medications.

On day 3, Mr. H.'s nurse observes a 30-second tonic–clonic seizure, which is treated with diazepam and a phenytoin load, with daily phenytoin ordered. Again, CT scan is negative for hemorrhage. On day 4, neurological examination findings and vital signs are stable for 24 hours and the intraventricular catheter is removed.

On day 5, Mr. H. is extubated. He is alert enough to understand what is being said to him and begins aggressive physical, occupational, and speech therapy.

Mr. H. is at risk for complications of immobility related to this stroke and therefore receives prophylaxis for stress ulcers and deep venous thrombosis, as well as aspirin for stroke prevention. He remains on lisinopril for hypertension. Diagnostic studies to determine the stroke etiology include a transthoracic echocardiogram, which reveals a normal ejection fraction, no right-to-left shunting, and no valvular abnormalities or vegetation. A carotid duplex scan does not reveal significant carotid stenosis.

Until discharge, Mr. H.'s vital signs and clinical status remain stable. Mr. H. is transferred to a stroke rehabilitation facility for aggressive treatment of persistent hemiparesis and dysarthria.

1. Describe other noninvasive approaches that the nurse might incorporate into Mr. H.'s care in order to reduce ICP.

2. Describe the potential complications that Mr. H. might experience while the intraventricular catheter for ICP monitoring is in place.

3. Does the neurological worsening that Mr. H. experiences on day 2 occur at a "typical" time point for patients with neurological injury? Explain your answer.

References

1. March K: Intracranial pressure monitoring and assessing intracranial compliance in brain injury. Crit Care Nurs Clin North Am 12(4): 429–436, 2000
2. Ng I, Lim J, Wong HB: Effects of head posture on cerebral hemodynamics: It's influences on intracranial pressure, cerebral perfusion pressure, and cerebral oxygenation. Neurosurgery 54:593–598, 2004
3. Brady KM, Lee JK, Kibler KK, et al: The lower limit of cerebral blood flow autoregulation is increased with elevated intracranial pressure. Anesthesia & Anelgesia 108 (4):1278–1283, 2009
4. Brain Trauma Foundation, American Association of Neurological Surgeons, Congress of Neurological Surgeons: Guidelines for the management of severe traumatic brain injury. J Neurotrauma 24(Suppl 1):S14–20, 2007
5. Brain Trauma Foundation: Guidelines for the Management of Severe Traumatic Brain Injury, 3rd ed. New York, NY: McGraw-Hill, 2007
6. McCarthy PJ, Patil S, Conrad SA, et al: International & specialty trends in the use of antibiotics to prevent infectious complications after insertion of external ventricular drainage devices. Neurocrit Care 12 (2): 220–224, 2010
7. Torre-Healy A, Marko NF, Weil RJ: Hyperosmolar therapy for intracranial hypertension. Neurocritical Care 2011
8. Knapp JM: Hyperosmolar therapy in the treatment of severe head injury in children. AACN Clin Issues 16(2):199–211, 2005
9. Roberts I, Schierhout G, Wakai A: Mannitol for acute traumatic brain injury. Cochrane Database 2:CD001049, 2003
10. Himmelscher S: Hypertonic salne solutions for treatment of intracranial hypertension. Curr Opin Anaesthesiol 20:414–426, 2005
11. Curley G, Kavanagh BP, Laffey JG: Hypocapnia and the injured: more harm than benefit. Crit Care Med 38(5):1348–1359, 2010
12. Jacobi J, Fraser G, Coursin D: Clinical practice guidelines for the sustained use of sedatives and analgesics in the critically ill adult. Crit Care Med 30(1):119–141, 2002
13. McKeage K, Perry CM: Propofol: A review of its use in intensive care sedation of adults. CNS Drugs 17(4):235–272, 2003
14. Changani S, Papadakos P: The use of dexmedetomidine for sedation in patients with traumatic brain injury. Anesthesiology B20, 2002.
15. Murray MJ, Cowen J, DeBlock H, et al: Clinical practice guidelines for sustained neuromuscular blockade in the adult critically ill patient. Crit Care Med 30(1):142–156, 2002
16. Joint National Committee on Prevention, Detection, and Evaluation and Treatment of High Blood Pressure: The Seventh Report of the Joint National Committee on Prevention, Detection, and Evaluation and Treatment of High Blood Pressure. US department of Health & Human Services; National Institutes of Health publication No 04-5230, 2004
17. Broderick JP, Connolly S, Feldman E, et al: Guidelines for the management of spontaneous intracerebral hemorrhage in adults. Stroke 38:1–23, 2007
18. Harris OA, Colford JM, Good MC, et al: The role of hypothermia in the management of severe brain injury: A meta-analysis. Arch Neurol 59(7):1077–1083, 2002
19. Albanese J, Leone M, Alliez J, et al: Decompressive craniectomy for severe traumatic brain injury: Evaluation of the effects at one year. Crit Care Med 31(10):2535–2538, 2003
20. Figaji A, Fieggen A, Peter J: Early decompressive craniotomy in children with severe traumatic brain injury. Childs Nerv Syst 19(9):666–673, 2003

21. Jaeger M, Soehle M, Meixensberger J: Effects of decompressive craniectomy in brain tissue oxygen in patients with intracranial hypertension. J Neurol Neurosurg Psychiatry 74(4):513–515, 2003
22. Vahedi K, Hofmeijer J, Juettler E, et al: Early decompressive surgery in malignant infarction of the middle cerebral artery: A pooled analysis of three randomised controlled trials. Lancet Neurol 6(3):215–222, 2007
23. Vahedi K, Vicaut E, Mateo J, et al: Sequential-design, multicenter, randomized controlled trial of early decompressive craniectomy in malignant middle cerebral artery infarction (DECIMAL trial). Stroke 38: 2506–2517, 2007
24. Cho DY, Chen TC, Lee HC: Ultra-early decompressive craniectomy for malignant middle cerebral artery infarction. Surg Neurol 60(3): 227–232, 2003
25. Juttler E, Schwab S, Schmidek P, et al: Decompressive surgery for the treatment of malignant infarction of the middle cerebral artery (DESTINY): A randomized, controlled trial. Stroke 38(9): 2518–2525, 2007
26. Morik K, Nakao Y, Yamamoto T, et al: Early external decompressive craniectomy with duraplasty improves functional recovery in patients with massive hemispheric embolic infarction: Timing and indication of decompressive surgery for malignant cerebral infarction. Surg Neurol 62(5):420–429, 2004
27. Merenda A, DeGeorgia M: Craniectomy for acute ischemic stroke: How to apply the data to the bedside. Curr Opin Neurol 23:3–58, 2010
28. Unterberg A, Juttier E: The role of surgery and ischemic stroke. Curr Opin Crit Care 13:175–179, 2007

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

35 Common Neurosurgical and Neurological Disorders

Richard Arbour

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Discuss the surgical management of the patient with a brain tumor.
2. Explain the care for the patient with a cerebral aneurysm or arteriovenous malformation.
3. Compare and contrast the classifications of stroke.
4. Discuss the nursing management of a patient who has experienced a stroke.
5. Differentiate between partial and generalized seizures.
6. Review the current antiepileptic drugs used to manage epilepsy.
7. Describe the clinical manifestations and management of the patient with Guillain-Barré syndrome.
8. Describe the clinical manifestations and management of the patient with myasthenia gravis.

During the course of illness, many patients with neurological diseases require critical care management. Routine neurosurgical procedures may involve a short intensive care unit (ICU) admission for monitoring in the immediate postoperative period. Complications of tumor or related treatments may necessitate readmission. ICU admission may follow use of thrombolytic therapy for stroke and may be required to treat the patient with stroke complicated by increased intracranial pressure (ICP). Patients with myasthenia gravis or Guillain-Barré syndrome may require ICU level of care for cardiorespiratory consequences of their disease. The critical care nurse is better prepared to manage the acute and chronic needs of this patient population when armed with understanding of the course of the disease, as well as available medical and surgical tools. This chapter provides an overview of the etiology, clinical manifestations, diagnostic tests, and current management of the neurosurgical and neurological disorders most often encountered in the ICU environment.

▲ Neurological Surgery

Surgery is indicated for several neurological disorders. Neurological surgery is a common and integral part of the management of patients with intracranial tumors, arteriovenous malformations (AVMs), and aneurysms. Craniotomy is the most common procedure performed for these problems. The following section reviews the etiology and pathophysiology of these conditions and describes surgical approaches used for the management of these patients.

Brain Tumors

A brain tumor is broadly described as any neoplasm arising within the intracranial space. Tumors may originate in the brain (primary) or seed in the brain from other organs (metastatic).[1,2] Pathological examination of a tumor is used to classify the tumor by cell type. Tumors are further graded based on the degree of malignancy. Classification and grade are used to predict patient outcome.[1,2] Table 35-1 outlines the most common brain tumors designated by the World Health Organization (WHO) system.[3] Other predictors of outcome include patient age and general health, early detection, and tumor location.[2,4]

Although many brain tumors are low grade or "benign," their location may impede surgical removal and cause brain edema as well as shifting of the surrounding structures. This causes increased ICP. Untreated ICP can lead to brain herniation and can be fatal. Early diagnosis, symptom management, and histological diagnosis are important prognostic factors.[1,2,4]

Etiology

The cause of most brain tumors is still unknown. As research advances in the area of genetics, there is increased interest in identifying chromosomal abnormalities in many types of cancer, including brain tumors. Cytogenetic studies of glioblastoma multiforme, the most common primary brain tumor, have shown multiple chromosomal changes, with both gain and loss of certain chromosomes.[3,4] It is hypothesized that this information will, at the very least, aid in the development

Table 35-1 Classification and Grading of Brain Tumors (Most Common Intracranial Tumors)*

Classification/Grade	Description	Symptoms	Treatment/Prognosis
Neuroepithelial (approximately 50% of primary tumors)			
Gliomas			
Astrocytic			
WHO grade I—pilocytic astrocytoma	Pediatric; 85% cerebellar; slow growing; well circumscribed; cystic; benign	Increased ICP; focal neurologic signs	Curable with surgery (craniotomy for tumor removal)
WHO grade II—astrocytoma	Infiltrative; slow growing	Seizures; acute or subtle onset of symptoms	Radiation therapy (RT) for residual tumor; may withhold RT after gross total resection; young age is good prognostic factor
WHO grade III—anaplastic astrocytoma	Hypercellular; anaplasia	May have acute onset of symptoms	RT with or without chemotherapy; high recurrence rate; age and overall health affect prognosis
WHO grade IV—glioblastoma multiforme	Poorly differentiated, with high mitotic rate; highly malignant; most common glioma in adults	Rapid onset of symptoms; increased ICP or focal signs	Infiltrative nature: complete removal of all cells not possible; RT with chemotherapy; experimental protocols; recurrence in virtually all cases; median survival: 12–18 mo
Oligodendroglioma	Well differentiated; calcified; infiltrative; slow growing; some tumors are malignant (anaplastic)	Seizures; headaches; subtle onset of symptoms	RT with residual tumor; may withhold after gross total resection; RT with or without chemotherapy for anaplastic oligodendroglioma
Mixed glioma (oligoastrocytoma)	May behave more or less aggressively, depending on features	Dependent on location and degree of malignancy	Variable outcome
Ependymoma	Pediatric and young adult patients; originates from lining of the ventricles; frequently in posterior fossa; usually benign	May present with hydrocephalus; symptoms related to location	RT for residual or recurrent disease; craniospinal RT for evidence of spinal disease only; good prognosis
Embryonal (primitive neuroectodermal tumor) medulloblastoma, most common	Primarily pediatric; malignant; occurs mainly in posterior fossa; CSF metastasis in 33% of patients	Symptoms by location; hydrocephalus common	Craniospinal RT; poor prognosis, particularly with CSF dissemination
Peripheral Nerve Tumors (approximately 8% of primary brain tumors)			
Vestibular schwannoma (acoustic neuroma)	Cerebellopontine angle; benign; encapsulated; seen in association with neurofibromatosis, type 2	Decreased hearing; tinnitus; balance problems; may have other cranial nerve deficits	Curable with surgery; excellent prognosis; cranial nerve deficits may be permanent or temporary; affect quality of life
Meningeal Tumors (approximately 30% of primary brain tumors)			
Meningioma	Composed of arachnoid cells; attached to dura; usually benign; well circumscribed; may be vascular; common locations: falx convexity; olfactory groove; sphenoid ridge; parasellar region; optic nerve	Headaches may occur from dural stretching; seizures and focal neurologic signs	Degree of resection (and recurrence) associated with location; excellent prognosis with gross total resection; atypical and malignant meningiomas have more aggressive features and less favorable outcomes

(continued on page 764)

Table 35-1 Classification and Grading of Brain Tumors (Most Common Intracranial Tumors)* (continued)

Classification/Grade	Description	Symptoms	Treatment/Prognosis
Lymphomas and Hematopoietic Tumors (approximately 3% of primary brain tumors)			
Malignant CNS lymphoma	Arise in CNS without systemic lymphoma; commonly suprasellar; diffuse brain infiltration; may be periventricular and may involve leptomeninges; solitary or multiple	Neurologic or neuropsychiatric symptoms	Diagnosis commonly via stereotactic biopsy or CSF cytology; steroids may decrease or temporarily obliterate lesion on CT or MRI; RT with or without chemotherapy; high-dose methotrexate used as single medication; some studies defer RT; increasing incidence in immunocompetent persons; decreasing in patients with AIDS; possible improved survival with newer treatments
Germ Cell Tumors (approximately 1% of primary brain tumors)			
	Developmental tumors—from gonads and extragonadal sites; germinoma (solid, enhancing on MRI) and teratoma (cystic, with fat and calcification) most common	Symptoms are location dependent; germinomas are often suprasellar—diabetes insipidus	RT for germinomas; curable; teratoma has less favorable prognosis; gross total resection means improved survival; chemotherapy in some cases
Sellar Tumors (approximately 7% of primary brain tumors)			
Pituitary adenoma	6.3% of sellar tumors; benign; originate from adenohypophysis; classification by hormonal content; microadenoma 1 cm or less; macroadenoma 1 cm or more	Hypersecretion • Prolactin: amenorrhea, galactorrhea • Growth hormone: acromegaly • Adrenocorticotropic hormone: Cushing's syndrome • Thyroid-stimulating hormone: hyperthyroidism (rare) Hyposecretion caused by compression of the pituitary gland Visual field deficits (bitemporal hemianopia); headache; pituitary apoplexy: acute hemorrhage or infarct of gland—emergency treatment indicated	*Surgical:* transsphenoidal for approximately 95% of surgical cases; *medical:* appropriate in some cases of prolactin-secreting and growth hormone–secreting tumors; RT for recurrence or for hypersecretory tumors, when medical management has failed
Craniopharyngioma	Benign, calcified, cystic tumors	Endocrine abnormalities; visual impairment; cognitive and/or personality changes; may have increased ICP	Gross total resection affects prognosis; RT for residual tumor
Metastatic Tumors (approximately 150,000 new cases yearly; occur in 20%–40% of cancer patients)			
	Originate from primary systemic tumors; discrete, round, ring-enhancing; 50% are solitary; lung and breast are most common primary sites	Symptoms are location dependent	Prognosis dependent on number of tumors, tumor location, systemic disease, and patient age; improved prognosis with gross total resection and RT

*For all tumors, biopsy or craniotomy for tumor removal is necessary to establish a definitive diagnosis.

of individualized therapies for patients with primary brain tumors. Some hereditary diseases, such as neurofibromatosis and polyposis, are associated with the development of certain types of brain tumors.

Environmental factors currently being studied for their association with intracranial tumors include electric and magnetic fields, foods (particularly those that are broken down in the stomach or bladder to form *N*-nitroso compounds), occupational exposure, and chemical exposure.[2–4]

Ionizing radiation has been shown to increase the occurrence of some brain tumors (nerve sheath tumors, meningiomas, and gliomas) when given in high doses.[2–4] Low-dose radiation exposure is a topic of discussion and controversy.

Epidemiology

Recent statistics indicate that there are approximately 14 cases per 100,000 person-years of newly diagnosed primary brain tumors in the United States each year.[2] The prevalence of primary high-grade or malignant brain tumors is 29.5 per 100,000 persons.[2] There is a significantly higher incidence of metastatic brain tumors diagnosed in the United States each year.

Epidemiological studies have confirmed certain patterns of brain tumor incidence. There has been a significant increase in the incidence of brain tumors in developed countries over the past several decades. Some of this increase can be attributed to improved diagnostic techniques, access to medical care, and an increasing elderly population. However, it is suspected that some of these increases are also attributable to environmental and lifestyle factors, as previously discussed.

Other patterns of incidence have been documented by age, ethnicity, and sex.[2,4] For example, the average age of onset for glioblastomas and meningiomas is approximately 60 years. Gliomas are diagnosed more often in the white population and in men. African Americans and women have higher rates of meningiomas.

Pathophysiology

The brain has its own distinct protective mechanism in the form of the blood–brain barrier. Studies undertaken in the mid-19th century showed that dyes injected intravenously were not observed in brain tissue as they were in other body organs.[1] However, when injected directly into the cerebrospinal fluid (CSF), they were absorbed into the brain but not disseminated through the brain's vascular system to the rest of the body. These studies confirmed the restricted permeability between the blood and the brain and between the blood and the CSF, but not between the brain and CSF.[1] Tumors are able to disrupt this blood–brain barrier, as evidenced by computed tomography (CT) and magnetic resonance imaging (MRI) scans, which show contrast uptake at the site of many tumors.[1,4] Disruption of the blood–brain barrier may relate to increasing permeability of the tumor blood supply at the capillary level with higher levels of malignancy.

Vasogenic edema, caused by increased capillary permeability and commonly seen in association with brain tumors and other brain lesions, is the direct result of blood–brain barrier disruption.[1,4,5] As outlined in the Monro-Kellie doctrine, the contents of the cranial vault—brain, CSF, and blood—have a fixed volume. Any addition to this volume must be balanced by reduction in one of the other components. When

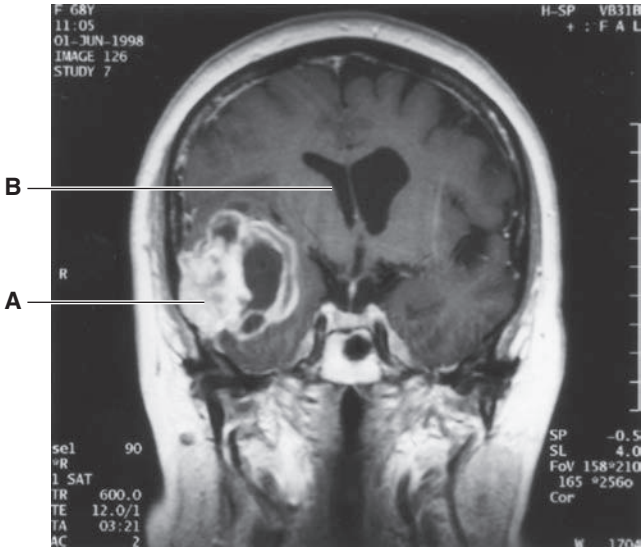


FIGURE 35-1 ▲ Coronal MRI view of ring-enhancing glioblastoma multiforme (**A**) with evidence of mass effect (**B**). (Courtesy of Henry Brem, MD, Johns Hopkins University, Baltimore, MD.)

this compensatory mechanism can no longer function, edema develops and ICP increases.[1,5] In Figure 35-1, an MRI scan shows brain edema and mass effect (shifting of brain structures) caused by a glioblastoma multiforme. Some slow-growing tumors (eg, meningiomas) can become quite large as a result of this compensatory mechanism and the brain's plasticity. This plasticity allows the brain to accommodate to slow tumor growth over a long period of time.

Clinical Manifestations

The patient with a brain neoplasm may present with one or more signs of tumor growth. The signs may be general or focal. The most common general signs of brain tumors are headaches, seizures, or mental status changes. These are related to increasing ICP.[1,2,4,5]

The triad of symptoms associated with increased ICP includes headache, nausea with or without vomiting, and papilledema (swelling of the optic disks). Symptoms are typically treated with corticosteroids, which are discussed later in this chapter.[1,2,5] Clinical evidence of herniation (shifting of brain tissue by masses, increased ICP, or both) often requires critical care management using fluid restriction, hyperventilation, osmotic drugs, and diuretics. Some situations necessitate the use of CSF drainage through an intraventricular catheter (see Chapter 34).

The frequency of seizures depends on tumor location as well as tumor histology. More than 60% of patients experience seizures over the course of their disease.[1,4] Seizure activity is more common in patients with low-grade tumors. Tumors in the cerebral hemispheres are much more likely to cause seizure activity than posterior fossa tumors. Seizures may be focal or generalized, as discussed later in this chapter.

Mental status changes occur as the result of mass effect on the brain caused by increased ICP or hydrocephalus. Patients may become drowsy and mentally slower as ICP increases. Cognitive changes occur in the form of problems concentrating, memory difficulties, personality changes, confusion, or disorientation. Although mental status changes are

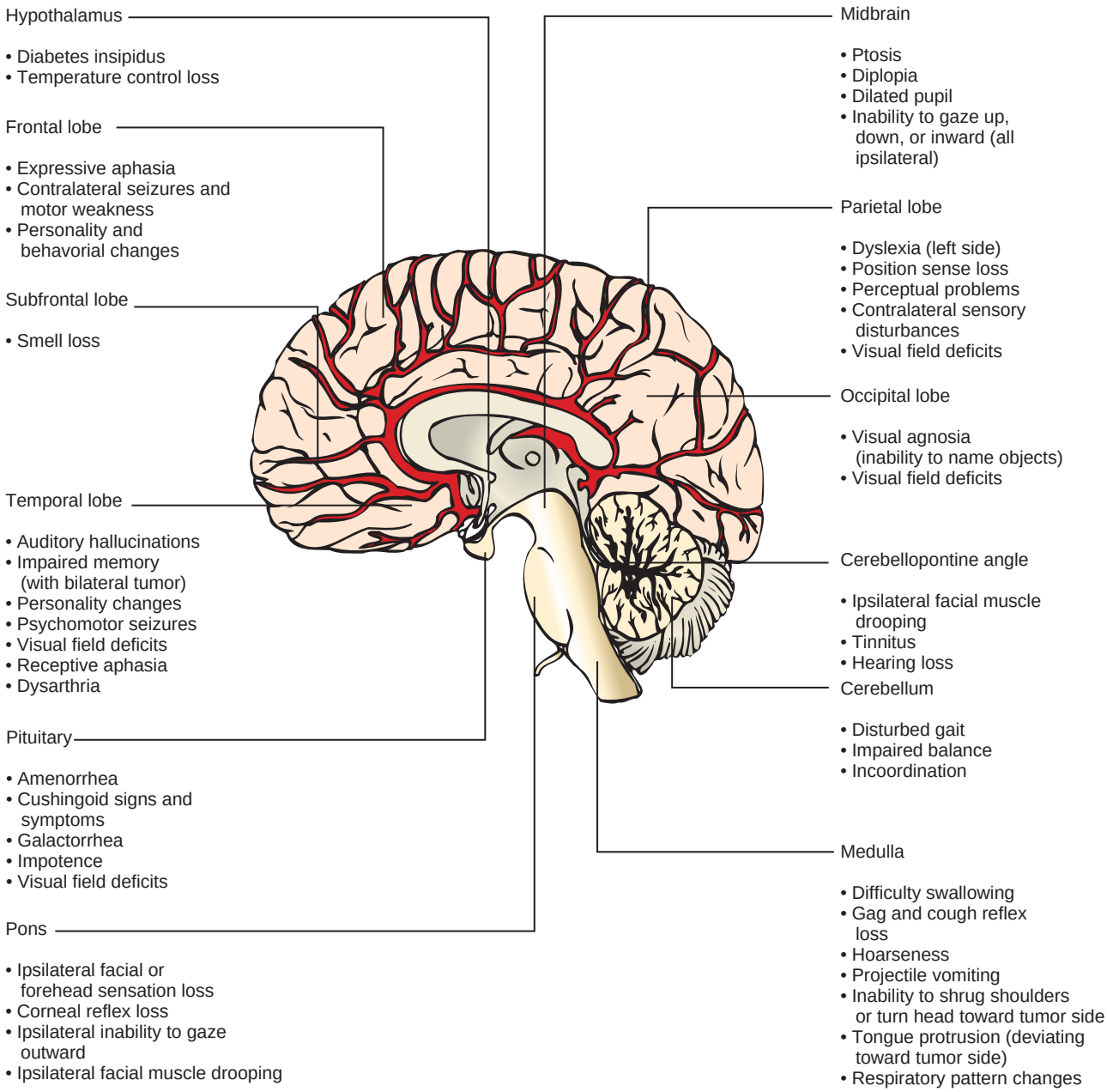


FIGURE 35-2 ▲ Site-specific signs and symptoms of brain tumors.

associated with frontal lobe tumors, they are also the result of increased ICP.[1]

Focal neurological deficits may be the temporary result of tumor compression, or may be permanent, as a result of tumor destruction. These deficits are directly related to tumor location. Figure 35-2 outlines site-specific signs and symptoms of brain tumors.

Diagnosis

History taking is a key element in the process of diagnosing a brain neoplasm. The duration, frequency, and severity of symptoms are ascertained. It is important to assess whether there is a particular time of day or series of activities that initiate symptoms. Are symptoms intermittent or continuous? Do they resolve with pharmacological management? Morning headaches and headaches that increase with Valsalva maneuver or are associated with nausea and vomiting are seen in patients with tumor-induced increased ICP.[1,2,4] The physical examination aids in further localizing the lesion. Patients may minimize or are often unaware of subtle neurological deficits. Family involvement in this discussion is useful.

Imaging studies, such as CT scans and MRI, are typically ordered to localize the lesion and assess the amount of edema and mass effect on surrounding structures. CT scans are often performed to establish a differential diagnosis when a patient is seen in the emergency department.[1,4] Because MRI shows tumors in three dimensions (axial, coronal, and sagittal), it is the preferred diagnostic tool. An electroencephalogram (EEG) is used to confirm the presence of seizure discharges, which may be useful in determining whether anticonvulsants

are needed. Magnetic resonance angiography (MRA) images the vascular anatomy and vessels that feed certain tumors. It can be a noninvasive alternative to the angiogram, which is an invasive study that may be needed to identify and perform embolization of feeding vessels to the tumor with the use of glue preparations. In some cases, angiography and embolization are performed within 24 to 48 hours of surgery for large tumors such as meningiomas.

Functional MRI (fMRI) is a type of imaging used for tumors in the dominant hemisphere or motor strip. It is believed that increased cerebral blood flow (CBF) is recognized as an increased signal on the fMRI. The patient performs a particular task, and the fMRI indicates which part of the brain is activated. This noninvasive procedure is currently used in some centers as part of the preoperative assessment of language, motor, and sensory function in relation to tumor location. Positron emission tomography (PET) uses radionuclides to measure CBF and brain metabolism. It is used to differentiate low-grade from high-grade (and more metabolically active) tumors. PET is also used to differentiate radiation necrosis from high-grade tumors in previously treated patients. Magnetic resonance spectroscopy (MRS) is a noninvasive radiographic technique that measures metabolite levels in brain tumors. Biological compounds, such as choline, can be quantitated in brain tumors. Because MRS is obtained at the same time as MRI, the anatomic and metabolic characteristics of the tumor are obtained with little additional inconvenience to the patient.

Clinical Management

Once the differential diagnosis of brain tumor is obtained through history, physical examination, and imaging studies, decisions are made regarding appropriate treatment modalities. A medical and surgical plan of care is discussed with the patient and family members.

Pharmacological Management

Tumors and tumor treatments are known to cause increased ICP, which is treated with corticosteroids.[4,5] Corticosteroids such as dexamethasone reduce brain edema by reducing the permeability of tumor capillaries and possibly by shifting some of the fluid into the ventricular system. A dose of 16 mg daily is standard in the perioperative period. It is usually given in two to four divided doses, spaced over the course of the day. Resolution of symptoms can be quite rapid. Steroids also increase the safety of the surgical procedure. They are, however, associated with significant side effects, as outlined in Table 35-2.[4,5]

Type 2 histamine receptor (H_2) blockers are often prescribed for the patient taking steroids. They are used to prevent gastrointestinal (GI) symptoms that can be associated with prolonged steroid use. Steroids used alone have a low risk for causing peptic ulcers and GI bleeding, but the risk increases in patients taking nonsteroidal anti-inflammatory drugs (NSAIDs).[1,3–5]

Anticonvulsant therapy is initiated when the patient presents with a seizure. Many surgeons also use prophylactic antiepileptic drugs (AEDs) during the perioperative period. Because studies have shown little difference in the occurrence of postoperative seizures in patients receiving AEDs compared with control groups without such drugs, some physicians are limiting the use of postoperative and prophylactic AED therapy. However, 70% of members of the American Association of Neurological Surgeons who responded to a survey stated that they continue to prescribe prophylactic AEDs for their patients with brain tumors. Seizures are extensively discussed later in this chapter.

Surgical Management

Clinical and radiographic evaluations are useful in obtaining a differential diagnosis. However, pathologic examination of tumor tissue produces the definitive diagnosis. There are two

Table 35-2 Complications of Corticosteroid Therapy

System	Complications
Neurological	*Common:* Behavior changes, insomnia, myopathy, hallucinations, hiccups, tremor, cerebral atrophy *Uncommon:* Psychosis, dementia, seizures, dependence, paraparesis (epidural lipomatosis)
General	Weight gain, cushingoid features (moon facies, buffalo hump, centripetal obesity), opportunistic infections (eg, candidiasis, *Pneumocystis carinii* pneumonitis), night sweats, hypersensitivity reactions, peripheral edema *Note:* Steroid taper may cause recurrence of preexisting conditions (eg, arthritis, allergic reaction)
Cardiovascular	Hypertension, atherosclerosis, increased cardiovascular and cerebrovascular disease
Dermatological	Thin skin, ecchymoses, purpura, acne, striae, inhibited wound healing, hirsutism
Endocrinological	Hyperglycemia, hypokalemia, hyperlipidemia, fluid retention
Gastrointestinal	Increased appetite, abdominal bloating, GI bleeding, peptic ulcers, pancreatitis, liver hypertrophy
Genitourinary	Polyuria, menstrual irregularities, infertility
Hematological	Neutrophilia, lymphopenia
Ophthalmological	Visual blurring, cataracts, glaucoma, uveitis
Rheumatological	Osteoporosis, avascular necrosis

Modified from Hickey JV: The Clinical Practice of Neurological and Neurosurgical Nursing. Philadelphia, PA: Wolters Kluwer/Lippincott Williams & Wilkins, 2009; and Stummer W: Mechanisms of tumor-related brain edema. Neurosurg Focus 2007. Accessed June 9, 2010 from http://www.medscape.com/viewarticle/559000.

distinct surgical approaches to diagnosing and treating brain tumors. Stereotactic biopsy is used to obtain small samples of tumor tissue under CT or MRI guidance. Craniotomy is performed when tumor removal is feasible and provides both a pathological diagnosis and surgical resection of the lesion.[2–4] These approaches are discussed in the "Surgical Approaches" section of this chapter (see pages 778–779).

During the past several decades, improvements in anesthesia, microsurgical equipment, intraoperative monitoring techniques, and pharmacological management have significantly improved intraoperative mortality rates. Postoperative morbidity has also significantly decreased. The perioperative management of the patient with a brain tumor involves a multidisciplinary team approach, as outlined in Table 35-3.

Despite substantial improvements in the management of the patient with a brain tumor, surgical complications may be severe and require critical care monitoring and management. The most common complications include brain edema, infection, hyponatremia or other electrolyte imbalances, hemorrhage, venous thromboembolism (including deep venous thrombosis [DVT] and pulmonary embolism [PE]), and seizures (Table 35-4).[2]

Radiation Therapy

Many brain tumors are treated with adjuvant therapies, either because they cannot be surgically resected or because of their aggressive nature. For most brain tumors, radiation therapy (RT) is the first-line treatment after biopsy or craniotomy. The energy produced by radiation damages tumor DNA at the time of cell division.[3,4,6] Three-dimensional conformal radiation is generally used to treat those tumor cells that are not surgically resectable. This form of RT treats the shape and the volume of tumor while normal brain tissue is protected. A standard dose of up to 6,000 centigray (cGy; also referred to as radiation absorbed dose, or rad) is administered to primary brain tumors 5 days a week over a period of 6 weeks. Multiple metastatic tumors receive a dose of approximately 3,000 cGy divided over 10 treatments. Some metastases may be treated with higher radiation doses with or without a boost of focused radiation.[4,6,7]

There are other approaches to applying RT. Intensity-modulated radiation therapy (IMRT) modifies the radiation beam so that a more focused dose can be given, without exposing surrounding brain tissue. Stereotactic radiosurgery (SRS) (eg, gamma knife and linear accelerator) is applied under MRI guidance. A three-dimensional image is obtained, and radiation is given in one large dose to residual tumor, sparing normal brain tissue.[4,7–9] Brachytherapy uses radioactive isotopes in seeds or liquid-filled balloons inserted into residual tumor.[4] Radiosensitizers are agents given in addition to RT. It is postulated that some substances increase oxygen delivery to hypoxic tumors. The oxygen enhances the effects of radiation. Hyperthermia is also being applied with the same goal of increasing oxygen to tumors to maximize the effects of radiation.[3]

Chemotherapy

Malignant brain tumors require multiple treatment modalities. Chemotherapy is given in conjunction with radiation or at the time of tumor recurrence. Drugs may be administered orally or intravenously but can cause systemic toxicities and have difficulty crossing the blood–brain barrier in sufficient amounts to provide benefit. An approach using RT in conjunction with temozolomide chemotherapy has shown survival benefit in the most malignant primary brain tumor, glioblastoma multiforme.[1,4]

Chemotherapy can also be placed in the tumor resection cavity at the time of craniotomy. A biodegradable polymer wafer that delivers a continuous infusion of carmustine (BCNU) chemotherapy over a period of 2 to 3 weeks is being used for primary malignant and metastatic brain tumors. The wafer is surgically implanted at the time of initial diagnosis or when the tumor recurs.[4]

Research Initiatives

Radiation and chemotherapy are targeted at damaging cell DNA, thereby killing the cell or preventing it from dividing. Other approaches have been used in an effort to address different aspects of tumor growth:

- Glioma cells are deficient in a protein that suppresses tumor growth (−p53). Gene therapy is being explored using adenoviruses, for example, which are engineered to replicate in and inactivate these cells. They are designed not to replicate in normal (+p53) cells.[4]
- Antiangiogenic factors are used to prevent tumors from forming their own blood supply, crucial to tumor growth.[3,4,10]
- Immunotherapy is designed to stimulate the immune system to eliminate tumors. Targeted toxins are injected into and infiltrate tumor cells. Cytokines, such as interferon and interleukin, are proteins produced by the body to stimulate an immune response. They are being combined with inactivated tumor cells as the basis for tumor vaccines.[4]
- Cancer cells have enzymes that resist chemotherapy. Certain drugs called resistance modifiers are being used to inhibit these enzymes and to increase the effectiveness of chemotherapy.[4]
  - Surgical techniques, such as neuroendoscopic approaches and fluorescence-guided resection, are increasingly being used in managing malignant gliomas.[10]
  - Genetic research has refined glioblastoma classification into four distinct molecular entities and may lead to new treatments as may use of cancer stem-like cells.[10]

Nursing Management

ASSESSMENT. Some tumors are cured with surgery, whereas others have a protracted course involving adjuvant therapies and treatment for recurrent disease. As part of the multidisciplinary team, the nurse plays a central role in patient care and family support throughout the course of the patient's illness. The nurse participates in the diagnosis, treatment, and follow-up care of the patient with a brain tumor.[1,2] One additional significant factor to consider in patient assessment is the potential for mental status changes, however subtle, that may compromise decision-making capacity in the patient with a brain tumor. This patient population must frequently make medical and surgical care decisions. As such, ongoing assessment of medical decision-making capacity is an appropriate part of care.[11]

PLAN. Careful history taking and symptom evaluation contribute to the accurate diagnosis of a brain tumor. Once

Table 35-3 Multidisciplinary Management Guide for the Patient With a Brain Tumor

Stage	Management Team	Interventions	Nursing Considerations
Preoperative			
• History and physical	• Neurosurgeon, nurse practitioner, RN	• Baseline history and physical examination; neurological evaluation: mental status, cranial nerves, motor and sensory function, coordination, reflexes	• Preoperative teaching to begin • Involve family as much as possible
• Medications	• Physician, pharmacist, nurse practitioner, RN	• Steroids; histamine type 2 receptor (H_2) blockers, as needed; anticonvulsants for supratentorial lesions • Prescribe new medications; medication review; discuss interactions or contraindications	• Anticoagulants, NSAIDs to be discontinued (with consent of prescribing physician)
• Diagnostic testing	• Neuroradiologist	• Baseline MRI or CT scan • ECG, chest x-ray • Other diagnostic studies as indicated	• Most preoperative testing performed within 1 wk of surgery
• Preoperative teaching	• By specialty: RN, neurosurgeon, neuroanesthesiologist	• Informed consent • Obtain all test results before admission day	• A written teaching pamphlet is recommended for patient and family use
• Hospital admission	• Admitting office, OR staff	• Obtain/confirm demographics	• Most patients are admitted the day of surgery
Intraoperative			
• Stereotactic biopsy	• OR team, surgeon, anesthesiologist, radiologist, pathologist	• Stereotactic frame placed; samples taken through catheter inserted under MRI/CT guidance • Histological evaluation	• May be performed in radiology suite or OR • Teaching regarding stereotactic frame • May also be done as a frameless procedure
• Craniotomy	• OR team, surgeon, anesthesiologist, pathologist	• Tumor tissue obtained for biopsy; tumor resected • Histological evaluation	• Teaching regarding general anesthesia
Postoperative			
• Critical care unit	• Critical care staff, neurosurgeon	• Hemodynamic monitoring; frequent neurological evaluations	• If possible, it is useful for patient and family to see the unit before surgery
• Nursing unit	• Floor nurses, surgeon, consulting physicians, rehabilitation medicine, pharmacist, clergy, nutritionist	• Postoperative care to include vital signs, neurological exam, wound care, cough, and deep breathing; increase activity as tolerated; advance diet as tolerated • Rehabilitation medicine consult • Evaluate for deficits and complications and provide consultations, as indicated	• More family participation in care when possible • Patients are usually out of bed within 24 h of surgery • Recently, short hospital stays cause increased family involvement and responsibility; begin teaching while patient is on the nursing unit
Discharge Planning	• Social worker, nursing staff, consulting physicians, radiation oncologist, medical oncologist (when indicated)	• Inpatient/outpatient rehabilitation as needed (occupational therapy, physical therapy, speech, cognitive) • Outpatient therapies as needed (eg, radiation, chemotherapy) • Hospice care (inpatient or home hospice) may be indicated, particularly in cases of recurrent malignant gliomas, refractory to conventional treatments	• Ideally, planning begins as soon as the patient arrives on the nursing unit • Family takes on greater role because patient is often discharged from the hospital 2–3 d postoperatively, particularly in cases of highly malignant tumors where home hospice is indicated

Adapted from Bohan E, Macenka DG: Surgical management of patients with brain tumors. Semin Oncol Nurs 20(4):240–252, 2004.

Table 35-4 Critical Care Management of the Patient With Brain Tumor Complications

Diagnosis	Management
Increased ICP	• Corticosteroids and antacids or histamine type 2 receptor (H_2) blockers • IV fluids: Avoid hypotonic solutions • Elevate head of bed and maintain adequate body alignment • Avoid hypotension and control hypertension; arterial line useful; if ICP monitor is available, titrate fluid therapy and vasoactive/inotropic drugs as ordered and clinically appropriate to maintain MAP between 70 and 80 mm Hg to maintain CPP. Elevated ICP may require higher MAP to maintain CPP 60–70 mm Hg. • Keep well oxygenated; may need to intubate • Judicious use of osmotic therapy: mannitol to expand plasma volume and draw fluid out of the brain; may be used in conjunction with furosemide • Sedation to reduce activity and decrease hypertension • Intraventricular catheter may be necessary to monitor ICP and drain CSF • Cautious use of hyperventilation for short periods only to reduce arterial carbon dioxide pressure (PCO_2) (6–24 h) • May require surgical intervention for hematoma
Wound infection, intracranial abscess, or bone flap infection	• Blood work, including complete blood count and blood cultures • CT scan, MRI, and in some cases MRS to identify abscess • Surgical removal of abscess or bone flap, when feasible • Appropriate wound cultures, when possible • Antibiotic therapy • Infectious disease consultation for appropriate drug, dose, and duration
Hyponatremia or hypernatremia	• Possible diabetes insipidus, salt-wasting syndrome, or syndrome of inappropriate antidiuretic hormone secretion • For hyponatremia: fluid restriction, hypertonic saline • For hypernatremia: fluids, vasopressin
Intracranial hemorrhage	• Immediate CT scan to evaluate for early signs of a bleed • Monitor BP • Check laboratory values: prothrombin time, partial thromboplastin time, platelets • Management of increased ICP, as described above • May need to intubate and ventilate • Surgery may be necessary to remove blood clot
Thromboembolism: DVT and pulmonary embolus (PE)	• Diagnosed through TCD study or ventilation-perfusion scan • Heparinization *only after* CT scan has ruled out intracranial blood • Alternatively, vena cava filter (Greenfield filter) may be used • Large PEs require ICU care for further medical treatment
Seizures	• Potential for status epilepticus • Protect patient from injury • Titrate antiepileptics to therapeutic levels

medications are prescribed, patient education includes discussion of dose, side effects, and contraindications. The results of tests leading to a differential diagnosis are reviewed with the patient and family. When a decision has been made to proceed with surgery, extensive teaching is required in the perioperative period. Table 35-3 outlines the nurse's role in the plan of care for this patient population. Patient teaching is better retained if it is both verbal and written. Teaching sheets are useful and can be referred to at different times during the treatment period.

Although surgery and follow-up therapies are disruptive to patient and family activities, it is important to encourage a return to normalcy as soon as possible. Continuing with activities of daily living that encourage a positive and motivated attitude contributes to recovery.

Hospice

Multiple surgical procedures and adjuvant therapies are often successful in containing malignant brain tumors for a period of time. Inevitably, these tumors recur and become resistant to all treatment modalities. In addition, patient quality of life may be so compromised that further therapy is not feasible. Palliative care services are now available in many hospitals. Home and inpatient hospice are available in most communities and provide dedicated and supportive end-of-life care.

Aneurysms

An aneurysm is a weakening in the arterial wall that causes either a ballooning effect or overall distention of the affected vessel. Aneurysms may be congenital or degenerative arterial lesions. Concern arises if the outpouching of the vessel wall ruptures or becomes large enough to exert pressure on surrounding brain structures.[12–14]

Approximately 10% to 30% of patients die from the initial bleeding of their aneurysm before reaching medical care.[14] An additional 50% die within 1 month from the

initial hemorrhage.[12] Rebleeding is the leading cause of death in patients with a history of ruptured aneurysm. Of those who survive the initial bleeding, 25% die within 24 hours, and 40% to 49% die within 3 months.[13] Some recent data suggest that the overall mortality rate from aneurysmal subarachnoid hemorrhage (SAH) is as high as 65%.[13] Rebleeding most often occurs around the seventh day after the original bleed. Predictors of good recovery by 1 month after the bleed include a high score on the admission Glasgow Coma Scale (GCS) and an absence of blood on the first CT scan.[12–14]

Etiology

The etiology of aneurysms is unclear but is probably a combination of congenital and degenerative factors. Carmichael described the combination hypothesis of aneurysm formation. Of the three layers of an artery—tunica intima (innermost), tunica media (middle), and tunica adventitia (outer layer)—he found that congenital focal defects of the tunica media are common. However, degenerative changes are also necessary for the formation of aneurysms. Histological investigation of the normal vessel wall into the aneurysmal sac shows that the tunica media usually ends at the neck of the aneurysm and the internal elastic lamina becomes fragmented as it enters the sac. Consistent with this hypothesis, aneurysms are rare in childhood but are common into late adulthood.[12–14]

Although the exact cause of intracranial aneurysms is not understood, there is evidence to support that acquired and genetic factors contribute to their development. Genetic factors include heredity and genetically transmitted diseases. Acquired factors include traumatic brain injury, sepsis, cigarette smoking, and hypertension.[12–14] Multiple concurrent clinical conditions including Marfan syndrome, coarctation of the aorta, polycystic kidney disease, and systemic lupus erythematosus are associated with cerebral aneurysms, supporting a genetic component to aneurysm development.[12–14]

Epidemiology

Intracranial aneurysms are common lesions, with autopsy reports indicating a prevalence of 1% to 6% of the adult population. Most of these aneurysms are small and do not rupture during the patient's lifetime, and some people have more than one aneurysm.[12] In the United States, approximately 30,000 new cases of SAH each year are attributed to aneurysmal rupture.[14] The incidence of SAH is 8 to 10 cases per 100,000 persons.[14] The annual risk for rupture is about 1.34% per year.

Aneurysm may also be associated with other pathological processes, such as polycystic kidney disease.[12–14] Screening with MRI is warranted for patients with two immediate relatives with a history of intracranial aneurysm and for all patients with autosomal dominant polycystic kidney disease. The incidence of aneurysms increases with age, with peak incidence occurring between 55 and 60 years.[12–14] They occur more often in women than in men and, as noted earlier, can be linked to cigarette smoking. Seasonal variability is another epidemiological feature. Clinicians observed that there may be a seasonal variability, with increases occurring in spring and fall.

The Cooperative Study of Intracranial Aneurysms and Subarachnoid Hemorrhage reported that in 32% of cases, the hemorrhage occurred during physical activity. The study also reported that a similar proportion occurred during sleep. In summary, the incidence of SAH can be roughly divided into thirds: sleeping, active, and at rest.

Pathophysiology

Arterial vessels are composed of three layers: endothelial lining, smooth muscle, and connective tissue. A defect in the smooth muscle layer, or tunica media, allows the endothelial lining to bulge through, creating an aneurysm. Most aneurysms arise from larger arteries around the anterior section of the circle of Willis. The most frequent sites of occurrence in the anterior circulation are the anterior communicating artery, the posterior communicating artery (PCA), the middle cerebral artery (MCA) bifurcation, and the internal carotid artery bifurcation.[12–14] In the posterior circulation, the most common locations are the basilar artery apex and the posterior inferior cerebellar artery.[12–14]

As the intimal layers of the vessel weaken, high-velocity blood begins to flow to create a whirlpool effect, thus stretching the wall of the vessel. This creates an abnormal pocket or sac of blood. As the wall of the vessel expands, it begins to weaken and may eventually rupture. Compression by the sac on surrounding brain structures may result in focal neurologic deficits. Rupture of the aneurysm may result in subarachnoid, intracerebral, or intraventricular hemorrhage.[12–14]

Aneurysms may be classified according to shape. *Saccular* aneurysms are also known as "berry" because of a well-defined stem and berry-like outpouching of the medial layer of the arterial wall. Berry aneurysms are usually located on major cerebral arteries at the apex of branch points, which is where maximal hemodynamic stress occurs in the vessel (Fig. 35-3). *Fusiform* aneurysms are another type of aneurysm that occur more commonly in the vertebrobasilar system. Fusiform aneurysms are dilated circumferentially and usually occur secondary to atherosclerosis. A third type of aneurysm is known as *mycotic*, which is due to infection.[12–14]

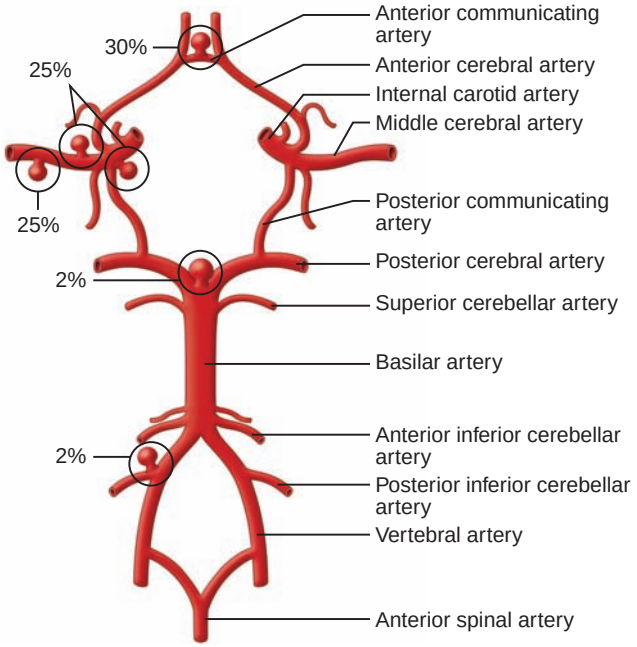


FIGURE 35-3 ▲ Circle of Willis with common aneurysm sites.

Aneurysms may also be categorized according to size. A small aneurysm is smaller than 10 mm, a large one is 10 to 20 mm, and a giant one is larger than 25 mm. Giant aneurysms are a cause for great concern because they may compress surrounding brain tissue or compromise the existing circulation to that area. Controversy exists about size and relationship to rupture. Seven millimeters is the average size of a ruptured aneurysm, but smaller lesions routinely hemorrhage.[12–14] Hemorrhage from an aneurysm usually occurs in the subarachnoid space because aneurysm-forming vessels usually lie in the space between the arachnoid layer of the meninges and the brain. The force of the rupturing vessel can be so great that it can push blood through the pia mater and into the brain substance, causing an intracerebral hemorrhage. It can also push through the arachnoid into the subdural space, causing a subdural hemorrhage.[12,13]

Clinical Manifestations

Many aneurysms are silent and never cause a problem but may be discovered on postmortem examination. An aneurysm that does cause problems typically does so in patients between ages 35 and 60 years. Use of a grading scale can enhance the ability to determine clinical outcome. Aneurysmal SAHs are graded according to their severity on the Hunt and Hess scale. In this grading system, grade 0 is the unruptured aneurysm, and grade V is a hemorrhage with severe neurological sequelae (Table 35-5). However, the Hunt and Hess scale is not the only grading system that has been developed for SAH. The World Federation of Neurological Surgeons grading scale is also used for SAH. The Fisher scale is another grading scale that is used to estimate the density of subarachnoid blood on CT scan at the time of a patient's admission to the hospital (Table 35-6).[12–14] A score of 3 or 4 on the Fisher scale has been found to increase the likelihood of poor clinical outcomes. A score of 1 to 2 has not been shown to increase mortality rates.

Table 35-5 Hunt and Hess Grading Scale for Aneurysms

Grade 0	Unruptured aneurysm
Grade I	Asymptomatic Minimal headache Slight nuchal rigidity (neck stiffness)
Grade II	Moderate to severe headache Nuchal rigidity Cranial nerve deficits
Grade III	Lethargy Mental confusion Mild focal neurological deficit
Grade IV	Stupor Moderate to severe motor deficit Possible posturing
Grade V	Deep coma Posturing Declining appearance

Data from Brisman JL, Soliman E. Cerebral aneurysm. Updated May 22, 2009; accessed May 16, 2010 from http://www.emedicine.medscape.com/article/252142-print; Leibskind DS. Cerebral aneurysms. Updated May 10, 2009; accessed from http://emedicine.medscape.com/article/1161518-print; Alexander S, Gallek M, Prescuitti M, et al: Care of the patient with subarachnoid hemorrhage: AANN Reference Series for Clinical Practice. Am Assoc Neurosci Nurses 2007.

Table 35-6 Fisher Grading Scale

Fisher Group	Blood on CT
1	No subarachnoid blood detected
2	Diffuse or vertical layers <1 mm thick
3	Localized and/or vertical layers 1 mm or more
4	Intracerebral or intraventricular clot with diffuse or no SAH

Amount of blood on CT scan is a predictor of vasospasm.
Data from Brisman JL, Soliman E. Cerebral aneurysm. Updated May 22, 2009; accessed May 16, 2010 from http://www.emedicine.medscape.com/article/252142-print; Leibskind DS. Cerebral aneurysms. Updated May 10, 2009; accessed from http://emedicine.medscape.com/article/1161518-print; Alexander S, Gallek M, Prescuitti M, et al: Care of the patient with subarachnoid hemorrhage: AANN Reference Series for Clinical Practice. Am Assoc Neurosci Nurses 2007.

Approximately half of patients have some warning signs before an aneurysm ruptures. These signs may include headache, lethargy, neck pain, a "noise in the head," and optic, oculomotor, or trigeminal cranial nerve dysfunction.[12–14]

After an aneurysm has bled or ruptured, the patient usually complains of a horrific headache. The classic description is "the worst headache of my life," which is often abbreviated as WHOL in the medical record. Other symptoms that may accompany SAH or aneurysms that present with mass effect are nausea, vomiting, focal neurologic deficits, or coma. Aneurysms presenting with mass effect typically show symptoms associated with increased ICP. With an SAH, there are also signs of meningeal irritation, such as a stiff and painful neck, photophobia, blurred vision, irritability, fever, positive Kernig's sign, and positive Brudzinski's sign. Exactly which deficits are present depends on the location of the aneurysm, the subsequent hemorrhage, and the severity of the bleeding.[12–14]

Bleeding stops because ICP in the subarachnoid space reaches mean arterial pressure (MAP) quickly, resulting in a tamponade effect that stops the bleeding long enough for the rupture to seal. If this does not occur, the patient dies.[12–14]

When there is blood in the subarachnoid space, it irritates the brainstem, causing abnormal activity in the autonomic nervous system, often with cardiac dysrhythmias and hypertension. Hypertension can also result from elevated ICP. Another complication of blood in the subarachnoid space is hydrocephalus. Blood in the subarachnoid space impedes reabsorption of CSF by the arachnoid villi. Hydrocephalus results in enlargement of the lateral and third ventricles.[12–14]

Diagnosis

The diagnosis of a cerebral aneurysm usually is made on the basis of history, physical examination, CT scan, lumbar puncture, and cerebral angiogram. When the nurse takes the patient's history, he or she identifies risk factors such as genetic predisposition, hypertension, and cigarette

smoking. Patients with an SAH may present with a headache, neck discomfort, or both without any neurological signs. The headache may range in severity from mild to severe. A CT scan reveals hemorrhage in most cases when it is obtained within 24 hours of the hemorrhage. It has the most sensitivity when obtained within 24 hours of onset.[12–14] There is a steady decline over ensuing days, with approximately 50% of CTs being positive 5 days after an SAH occurs.

If the results of a CT scan are negative and there are signs and symptoms indicating that a patient has experienced an SAH, a lumbar puncture is typically performed to confirm the diagnosis. After a positive lumbar puncture, a cerebral angiogram is obtained to determine the source of the SAH. Different types of angiography may be used for this purpose, including CT angiography, MRA, or digital subtraction angiography (DSA). Although all of these studies can determine vascular anatomy, DSA is the gold standard if surgery is planned. Transcranial Doppler (TCD) ultrasonography can also be used to diagnose and treat vasospasm, a common complication of an SAH.[12–14] Examples of nursing diagnoses for the patient with a cerebral aneurysm or AVMs nursing (discussed later) are presented in Box 35-1.

Clinical Management

Before repair, the management of a patient with a ruptured or leaking aneurysm focuses on minimal stimulation of the patient. Some institutions initiate "aneurysm precautions" as precautionary measures to prevent rebleeding. These measures include providing a quiet environment, establishing a bowel regimen to prevent straining (Valsalva maneuver), and limiting visitors.[14]

Pharmacological Management

Antihypertensive medications may be used to manage blood pressure (BP) before procedures or surgery. Plasma volume should not be allowed to fall. Frequently, abnormalities of electrolytes, notably hyponatremia, exist. Hyponatremia, usually associated with cerebral salt wasting rather than syndrome of inappropriate antidiuretic hormone, must be managed with sodium replacement and euvolemia.[12–15]

Stool softeners are used in managing patients with an aneurysm to prevent straining. Mild analgesics can be used to relieve headaches. An antipyretic, usually acetaminophen, and hypothermia blankets can be used to manage fever typically caused by blood in the subarachnoid space. Acetaminophen can be used without masking neurological signs. Judicious use of narcotics is appropriate in these patients.[12–14]

BOX 35-1 EXAMPLES OF NURSING DIAGNOSES

For the Patient With Cerebral Aneurysm or Arteriovenous Malformations

- Risk for Ineffective Cerebral Tissue Perfusion related to interruption in CBF or intracranial hypertension
- Acute Pain related to meningeal irritation
- Excess Fluid Volume related to hypervolemia used to treat vasospasm
- Deficient Fluid Volume Risk for fluid volume deficit related to fluid restriction and use of osmotics to control intracranial hypertension

Surgical Management

CLIPPING. Surgical clipping may be considered if the aneurysm is in an accessible area. The goal of surgery is complete obliteration of the aneurysm. Aneurysms of the vertebrobasilar system often present the problem of surgical inaccessibility. The accepted surgical treatment is the placement of a clip across the neck of the aneurysm. These clips, which are made of titanium, come in a variety of shapes and sizes. For larger or wider aneurysms, more than one clip can be used to ensure complete occlusion of the neck of the aneurysm. Once clipped, the aneurysm may be punctured to allow it to collapse and relieve mass effect if present.[12–14]

Some aneurysms may be wrapped in a gauze-like material or coated with an acrylic substance that gives the aneurysm support. Although wrapping or coating should not be the goal of surgery, there may be conditions, as with a fusiform aneurysm, in which there is no other option.

In the past several years, controversy continued about when surgical intervention should occur. The current thinking is that surgery should occur sooner rather than later. Thus, surgery may generally be performed 24 to 48 hours after the rupture and initial bleed occur.[12–15] (Fig. 35-4).

After aneurysm clipping, the patient is managed in a critical care environment. Maintenance of an adequate airway is vital. If the patient is intubated and suctioning is needed, it is important that the suction catheter be passed and removed quickly to prevent oxygen desaturation as well as surges in ICP consequent to increased intrathoracic pressure resulting from stimulation of the cough reflex.

Signs of vasospasm, such as hemiparesis, visual disturbance, seizures, or a decreasing level of consciousness (LOC), should be noted and reported so that medical interventions can be rapidly implemented. Control of ICP is a collaborative effort. Nurses should keep the head of the patient's bed elevated and ensure that there is no neck flexion or severe rotation. Nursing care activities should be spaced to avoid causing a sharp rise in ICP.[12–14]

COILING. One of the most valuable recent developments in aneurysm management has been the technique of endovascular thrombosis of aneurysms with Guglielmi detachable coils (GDCs). These are thrombogenic platinum alloy microcoils. They are soft to allow the coil to conform to the shape of the aneurysm. Coils are available in various shapes, dimensions, lengths, and diameters to provide maximal occlusion.[12,13,16]

The procedure used to introduce the coil into the aneurysm is similar to that used for a cerebral angiogram, using the femoral artery and fluoroscopic equipment. In this procedure, a microcatheter is passed through the aorta, around the aortic arch, and then into the vessel specific to the aneurysm. Once the catheter is in position, the coil system is advanced through the catheter into the aneurysm sac. The coil is then in position, and if placement is satisfactory, a low-voltage current is applied. The current causes the coil to detach. If the coil is successfully placed, it occludes the aneurysm and separates it from the cerebral circulation. The number of coils placed is individualized to the patient. Through this

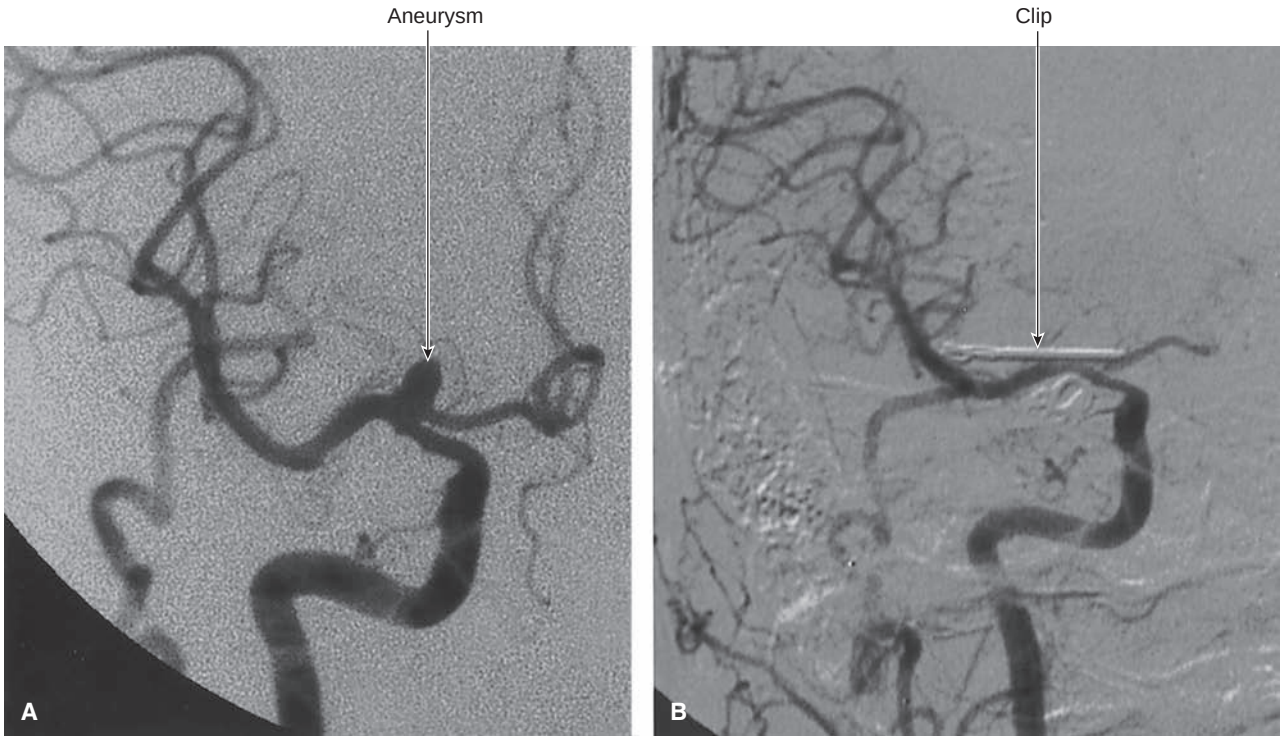


FIGURE 35-4 ▲ Preoperative (**A**) and postoperative (**B**) angiography: clipping of right internal carotid artery termination aneurysm. (Courtesy of Rafael Tamargo, MD, and Richard Clatterbuck, MD, Johns Hopkins University, Baltimore, MD.)

coiling procedure, the risk for hemorrhage or rehemorrhage is reduced.[12,13,16]

Complications of this treatment are embolic stroke, coil migration, failure to obliterate the aneurysm, and aneurysm rupture. Stroke may occur because the parent artery feeding the aneurysm becomes occluded or because of the introduction of air or particles into the catheter system. Coil migration occurs because of reintroduction of blood into the sac. The coils become repositioned outside the fundus of the sac and can then migrate to other anatomic areas of the brain, creating complications such as brain ischemia in areas supplied by the affected blood vessel. Failure to obliterate the aneurysm is also a possibility. If the coils fail to obliterate the aneurysm, the sac may grow larger, cause more symptoms, and may even rupture. Further intervention with another endovascular procedure or surgery is then warranted.[12,13]

Although clipping is the preferred treatment or gold standard for most aneurysms, GDC coiling is an option for patients who are considered to be surgically high risk because of medical instability, or those who would otherwise be treated conservatively. With the rapidly evolving technology, endovascular embolization of cerebral aneurysms is a safe alternative to surgical clipping in treating ruptured and unruptured cerebral aneurysms.[12,13,16] Long-term outcomes require further study. Some experts currently consider coiling to be the preferred treatment, although this is still a matter of controversy.

Medical Management of Complications

As previously noted, vasospasm can occur after, as well as before, surgery in the patient with an aneurysm. Angiographic vasospasm is seen early. Vasospasm may be recognized clinically through frequent neurological assessment. The patient's signs and symptoms can fluctuate but may include changes in LOC, headache, language impairment, hemiparesis, and seizures.[12–15]

Vasospasm usually occurs 3 to 12 days after an SAH. The peak incidence is between postbleed days 7 and 10. Although the aneurysm may have been clipped successfully, vasospasm can cause the development of a large area of ischemia or infarcted brain, with severe deficits. Vasospasm is of clinical significance because it decreases CBF, depriving brain tissue of oxygen and promoting accumulation of metabolic waste products, such as lactic acid. The reduced size of the vessel lumen restricts blood flow to the brain tissue, causing brain ischemia and possible permanent neurologic consequences.[12–15]

TCD imaging is a valuable noninvasive technique used to diagnose vasospasm. This technique, which can be performed at the bedside, measures the velocity of blood flow through segments of the arterial vessels. Monitoring trends in flow velocity allows prompt identification of early indications of vasospasm and patients at risk for developing vasospasm. The results of the neurological examination can be correlated with TCD findings for prompt diagnosis and treatment of vasospasm. This technique is highly reliable in predicting vasospasm of the middle cerebral and internal carotid arteries.[12–14]

The exact etiology of vasospasm is not clear. Apparently, there is a positive correlation between the size of the hemorrhage seen on CT scan and the subsequent development of spasm. Vasospasm is likely caused by inflammatory processes. There has been some success in using nimodipine

(Sular), a calcium antagonist, after an SAH to improve patient outcomes. It is recommended that nimodipine be used from onset through day 21. Nimodipine reduces the contraction of smooth and cardiac muscles without affecting skeletal muscles. The dose is 60 mg every 4 hours. The theory is that nimodipine improves outcomes by limiting collateral cerebral damage that is mediated by calcium.[12–15]

"Triple H" therapy is the standard for preventing and treating vasospasm. It consists of hypervolemic expansion, hemodilution, and induced hypertension in postoperative patients. Nimodipine is used with this therapy. These measures reduce smooth muscle spasm and maximize perfusion when spasm does occur.[12–15]

Hypervolemia is accomplished by volume expansion, using both intravenous (IV) colloid and crystalloid solutions. They are given to increase intravascular volume and decrease blood viscosity. Through hypervolemia, the cerebral vessels dilate and the MAP increases, thereby improving cerebral perfusion pressure (CPP). During this therapy, the patient should be monitored for pulmonary edema and heart failure. A pulmonary artery catheter helps monitor the patient's hemodynamic status.[12–15]

Hemodilution through the administration of IV fluids decreases blood viscosity, increases regional CBF, and may decrease infarction size and increase oxygen transport. The goal of hemodilution is to reduce the hematocrit by 15%. The patient's hematocrit level should be maintained between 30% and 33%, which helps improve CBF without causing hypoxia.[12–15]

Vasopressors are used to induce hypertension. The objective is to maintain systolic BP at greater than 20 mm Hg over normal. Vasopressors raise the patient's BP and brain perfusion to the point where neurological deficit improves.[12–15]

When conventional medical therapy is not effective, acute arterial vasospasm secondary to an SAH can be managed by balloon angioplasty in centers where this technology is available. Recent advances in microballoon technology now allow access to the cerebral vasculature with soft, flexible angioplasty balloons, which mechanically dilate and improve CBF through the major arterial segments affected by vasospasm. Balloon angioplasty allows direct widening of the stenotic segment.[12,13,15] Intra-arterial administration of nicardipine to selectively treat cerebral vasospasm is an additional technique to utilize. Increasing vasopressor dosing may be required to maintain systemic arterial pressure but was noted to be well-tolerated.[17] IV administration of magnesium sulfate in high doses has been shown effective in attenuating cerebral vasospasm and reducing cerebral ischemic events.[18]

Another complication after aneurysmal rupture is hydrocephalus. Hydrocephalus indicates an imbalance between the production and absorption of CSF. It may occur in patients who have experienced an SAH. When there is blood in the subarachnoid space, the red blood cell clots and possible brain edema can occlude the very small channels leading from one ventricle to another. If this occurs, an obstructive hydrocephalus develops and obstructs the normal flow of CSF, often between the third and fourth ventricles, or at the exits from the fourth ventricle. There is also the potential for a reabsorption problem, whereby red blood cells and their breakdown products occlude the arachnoid villi, impeding reabsorption and resulting in a communicating hydrocephalus. The patient may require a shunt. With a ventriculoperitoneal shunt, the proximal tip of the catheter is placed in a lateral ventricle, and the distal tip is placed in the peritoneum. The shunt drains CSF into the peritoneal cavity to treat the hydrocephalus and prevent dangerous ICP elevations.[12–14]

Seizures may occur from blood in the subarachnoid space acting as an irritant to neurons. Typically, patients receive an anticonvulsant to minimize seizure risk.[12–14]

Rebleeding is another complication in patients with an SAH if the aneurysm is not repaired. There is a 2% to 4% risk of aneurysmal rebleeding within the first 24 hours of initial hemorrhage. This increases to 15% to 20% risk of rebleeding within the next 2 weeks after the initial bleeding. The immediate mortality rate of rebleeding may be high—between 50% and 80%.[14]

Nursing Management

ASSESSMENT. One of the nurse's primary responsibilities is to obtain a baseline neurological assessment and perform subsequent assessments to monitor for changes. After surgery, the nurse must be alert to the development of new deficits or a worsening of preoperative deficits. The severity and duration of any postoperative disability depend largely on the location and extent of the vascular lesion and resultant ischemia. The patient must also be carefully monitored for the development of cerebral edema.[14]

PLAN. Before surgery, the nurse implements aneurysm precautions by providing a quiet environment with limited stimulation. The nurse does a bowel assessment and implements individualized interventions.

A patent airway is required. Management of the patient's fluid and electrolytes includes careful monitoring for hyponatremia, which can cause an increase in cerebral edema. Accurate intake and output measurements are imperative.[14]

The nurse also monitors vital signs to rapidly identify any changes in BP and initiate corrective action to maintain BP within the target range. Hypotension must be treated immediately to prevent a drop in cerebral perfusion. Cardiac dysrhythmias may be present, especially if there was bleeding into the subarachnoid space. Dysrhythmias require prompt management because they may precipitate a drop in cardiac output and a consequent drop in cerebral perfusion.[12–14]

A patent IV site is maintained for hydration. Maintaining two IV accesses may be appropriate as a matter of clinical judgment. Continuous fluids are a part of the management of vasospasm. The IV site is monitored frequently, and fluids are not interrupted for any reason. In the event of an infiltrate, the nurse restarts the line immediately.[14] Measures also need to be taken to manage increased ICP elevation if it develops. It would be noted initially as a decline in LOC. In the event the ICP elevation is severe enough to markedly reduce the LOC, endotracheal intubation and controlled ventilation should be initiated. Hyperventilation may be done as an initial emergent measure to lower dangerous ICP elevations pending initiation of additional therapies, such as osmotic diuretics and ventricular drainage, which are valuable tools in managing ICP elevations.

Emotional support is also a crucial part of the overall nursing care of the patient with a ruptured aneurysm. Because of an aneurysm's abrupt onset, the hospital admission cannot be planned. Often the rupture suddenly interrupts the patient's daily life and may leave the patient with neurological

impairment. The patient's support system needs to be organized to tend to daily activities and responsibilities in his or her absence. Families require assistance when confronting the financial, physical, and emotional burden of caring for a patient after an SAH. A social worker can be instrumental in helping to organize the support of friends and family.[14]

Patient Education and Discharge Planning

Smoking and hypertension are both preventable risk factors associated with intracranial aneurysm and SAH. Patients can be instructed that cessation of smoking and control of hypertension can reduce the incidence of aneurysm formation and rupture.

Patients who have undergone clipping of cerebral aneurysms should be carefully screened before MRI. Although titanium clips used after 1996 are "MRI friendly," it is necessary to determine the composition of the clip before the patient undergoes MRI.

If a patient has experienced a seizure and is being maintained on anticonvulsant therapy, instructions should be given about medication monitoring and the need for compliance. In addition, the patient should be instructed about seizure safety.

Patients who have experienced SAH face a lengthy recovery. Rehabilitation for specific deficits should begin early.[14] Also, family participation in the rehabilitation plan is encouraged. Members of the health care team, such as physical, occupational, and speech therapists, can help towards restoring the patient's independence, and such services can be coordinated either for inpatient or outpatient settings, depending on the extent of impairment and financial circumstances.

Arteriovenous Malformations

Arteriovenous malformations (AVMs) are lesions consisting of dilated arteries and veins without a capillary system. Arterial blood flows directly into the venous system. AVMs are usually described as a "tangle" of blood vessels with a well-defined nidus that does not involve brain parenchyma.[19–21] Presumably, AVMs are congenital, and they typically enlarge with age.[19] Although they are found throughout the central nervous system (CNS), approximately 90% of AVMs are located in the cerebrum. Of these, the most common locations are the frontal and temporal lobes, most often supplied by the MCA.

Epidemiology

AVMs are relatively uncommon brain lesions. Population-based studies have estimated an prevalence of approximately 0.01% of the general population.[19] There is no statistically significant predisposition by sex. AVMs are diagnosed most often in young adults, with the majority being diagnosed in patients younger than age 40.[19] In cases in which the patient has both an AVM and an aneurysm (7%), the symptomatic lesion is treated initially. In some instances, both can be surgically treated at the same time. Of the two, aneurysms are more likely to be the cause of hemorrhage.

Pathophysiology

AVMs develop as an atypical preservation of embryonic connections between arteries and veins, most likely between 4 and 8 weeks of embryo development. This is the period in which cells begin to differentiate and capillary components to the brain develop. AVMs are most likely to arise at this time because of the failure of the primitive vasculature to develop an adequate capillary system. Because blood is shunted directly from the arterial to the venous circulation without benefit of a capillary bed, there is less resistance, and AVMs receive significant blood flow. Arteries and veins enlarge to carry this increased flow, and their walls are characteristically quite thin. Arteries providing blood to the malformation and draining veins also become enlarged with increased flow volume in the lesion.[19–21]

Clinical Manifestations

Hemorrhage, the most common presenting sign of AVMs, occurs in 38% to 70% of affected patients according to population-based studies.[20] These hemorrhages may be intracerebral, subdural, or subarachnoid. Opinions differ regarding whether the size of the lesion affects the likelihood of bleeding. Small size may be associated with an increased risk for hemorrhage, which may be caused by higher flow and pressure from the feeding vessels. In addition, increased risk of hemorrhage is seen in AVMs located in the basal ganglia, posterior fossa, and lesions with deep venous drainage, having fewer or single draining veins as well as high pressures within the arterial vessels feeding the AVM.[20]

There is an approximate 10% mortality rate after a hemorrhage with a higher percentage of patients having significant morbidity following initial bleeding.[21] The risk for rebleeding is higher in the first year after the initial hemorrhage and declines over subsequent years.[19] Cerebral AVMs cause about 4% to 5% of SAHs. An SAH caused by an AVM is less lethal than one caused by an aneurysm rupture but is associated with significant neurological morbidity.

Seizures are another common presenting sign (15% to 40%) of an AVM.[20] The risk for a seizure increases with the size of the lesion. Patients are treated with AEDs after presentation with a seizure, but AEDs are not routinely used prophylactically for AVMs. Seizures are more likely to occur in patients who have large and more superficial AVMs.[20,21]

Other presenting signs include headache, increased ICP, neurological deficits (5% to 7%) referable to the location of the lesion, bruit, and visual symptoms.[19–21] Cognitive decline is seen particularly in older patients with large AVMs. This may be related to cerebral steal, which can cause ischemic changes by diverting arterial blood away from normal brain tissue to the AVM.[20]

AVMs are graded based on features, location, and venous drainage. The Spetzler-Martin grading system assigns 1 point for lesions less than 3 cm, 2 points for 3- to 6-cm lesions, and 3 points for lesions greater than 6 cm. If the AVM is located in an eloquent area of the brain (sensory, motor, speech, visual, brainstem), it is given 1 point. No points are given for noneloquent areas. Deep venous drainage associated with the malformation is allotted 1 point, with no points for superficial venous drainage. A low score is associated with better outcomes and a higher score with increased morbidity from surgery.[21]

Diagnosis

CT and MRI are used to evaluate the presence of an AVM. The lesion is differentiated from tumors and other brain

lesions by the presence of a hemosiderin ring around the lesion. Three-dimensional imaging is useful in establishing the malformation in relation to the surrounding anatomy. MRA is a noninvasive method of evaluating feeding and draining vessels in relation to the nidus of the AVM. Although MRA does provide useful information, it cannot consistently replace the more invasive angiography that is also used to evaluate feeding arteries and draining veins. Rarely, the AVM is not angiographically evident. This may be true of lesions that have bled, have small feeding and draining vessels, or have low flow. TCD, single-photon emission computed tomography (SPECT), and PET are also used to image blood flow changes. To identify the AVM in relation to eloquent areas of the brain, fMRI is useful.[19–21]

Clinical Management

AVMs can be managed in a number of ways based on the patient's age and medical condition, flow associated with the malformation, history of hemorrhage, other symptoms, and the location of the lesion. Table 35-7 outlines procedures, indications, outcomes, and possible complications.

Interventional, Surgical, and Radiosurgical Management

Endovascular embolization of feeding arteries is used for small, low-grade malformations. The cure rate is low (10% to 15%), and one or more procedures may be needed to occlude the abnormal vessels. Embolization is used as an adjunct to surgery and radiosurgery. In this procedure, particles, liquids (such as acrylic glue), balloons, or coils are inserted into the AVM nidus before surgery or radiosurgery.[19–22] Endovascular treatment of intracranial micro-AVMs can be accomplished immediately, with good patient selection, and may be an alternative to open operative resection of the lesion.[23]

SRS provides good outcomes for relatively small lesions. This can be accomplished by gamma knife, linear accelerator, or heavy ion radiation. When radiosurgery is used for lesions less than 3 cm in diameter, the cure rate is estimated at 65% to 85%. However, complete obliteration of the malformation takes 2 to 3 years, and there is a risk for hemorrhage during this time.[20] Very large cerebral AVMs may be successfully treated by staged radiosurgical procedures. Given the time required to achieve obliteration of the lesion, long-term follow-up remains necessary.[24]

Surgery is the preferred treatment for most AVMs. Surgery can reduce the risk for both hemorrhage and seizure. Brain mapping, fMRI, and intraoperative evoked potentials may be used in surgical planning for lesions in eloquent areas of the brain. Outcomes are positive, particularly in the case of Spetzler-Martin grade I and II lesions. Moreover, neurological complications are low; surgery also provides an immediate cure. Intraoperative angiography is recommended.[20,21]

Treatment approaches vary between surgery and radiosurgery for Spetzler-Martin grade III lesions. For large AVMs or lesions in eloquent areas of the brain, multimodality therapy with embolization, radiosurgery, or microsurgery is preferred. The most appropriate treatment approach for Spetzler-Martin

Table 35-7 Management of the Patient With Arteriovenous Malformation

Procedure	Indication	Outcome	Complications and Comments
Surgery	Surgically accessible AVM	Removal of lesion Decreased risk for bleeding Improved seizure control Preoperative propranolol believed to minimize postoperative bleeding and edema Labetalol used to keep MAP 70–80 mm Hg perioperatively	Inability to remove lesion Risks for surgery: cerebral edema; hemorrhage; neurological deficits Inpatient hospital stay Excellent results in Spetzler-Martin grade I–III lesions
Radiation: Stereotactic radiosurgery (SRS) (external beam radiation effective in only a small percentage of cases; gamma knife, proton beam, or linear accelerator used)	3 cm or smaller AVM Surgery not indicated	Reduction in size of lesion Noninvasive Outpatient, no recovery period	Lesion not removed May take years for complete obliteration May require multiple treatments Continued risk for bleeding for 2–3 y Used for small AVMs or as part of multimodality treatment
Embolization	Injection of substance to occlude the feeder vessels	Facilitates other therapies (surgery; radiation) by reducing AVM Useful for larger AVMs Short hospital stay	Does not typically cure AVM May need more than one procedure Need to wait days or weeks before surgery or SRS Risk for stroke or hemorrhage

Data from Friedlander RM: Arteriovenous malformations of the brain. N Engl J Med 356(26):2704–2712, 2007; Sen S, Webb SW, Selph J: Arteriovenous malformations. Updated May 18, 2010; accessed June 1, 2010 from http://emedicine.medscape.com/article/1160167-print; Altschul D, Smith M, Sinson GP: Intracranial arteriovenous malformation. Updated May 26, 2009; accessed June 10, 2010 from http://emedicine.medscape.com/article/252426-print; Guedin P, Gaillard S, Boulin A, et al: Therapeutic management of intracranial dural arteriovenous shunts with leptomeningeal venous drainage: Report of 53 consecutive patients with emphasis on transarterial embolization with acrylic glue. J Neurosurg 112(3):603–610, 2010; Andreou A, Loannidis I, Lalloo S, et al: Endovascular treatment of intracranial microarteriovenous malformations. J Neurosurg 109(6):1091–1097, 2008; Chung WY, Shiau CY, Wu HM, et al: Staged radiosurgery for extra-large cerebral arteriovenous malformations: Method, implementation and results. J Neurosurg 109(Suppl 6):65–72, 2008; and Geibprasert S, Peirera V, Krings T, et al: Hydrocephalus in unruptured brain arteriovenous malformations: Pathomechanical considerations, therapeutic implications and clinical course. J Neurosurg 110(3):500–507, 2009.

grade IV and V lesions is a matter of controversy. Some practitioners recommend the multimodality approach, and others opt for no treatment. In addition to intracranial hemorrhage as a presenting event of AVM, hydrocephalus may also occur. Hydrocephalus may be consequent to the AVM acting as a space-occupying lesion, interfering with CSF flow. Cerebral hydrodynamics may also be affected by mechanical obstruction by a draining vein associated with the lesion. In the event this causes acute hydrocephalus and ICP elevation, ventricular drainage may be necessary; if chronic, ventriculoperitoneal shunt may be necessary for long-term management. Definitive management should be prioritized at management of the AVM.[25]

Nursing Management

ASSESSMENT. The nursing management of the patient with an AVM is similar to that described for a patient with a cerebral aneurysm. Baseline and follow-up neurological assessments are necessary to monitor for subtle changes or evidence of hemorrhage.

PLAN. Careful evaluation of focal neurological signs or evidence of cerebral edema minimizes significant postoperative morbidity.

Patient Education and Discharge Planning

Patients who have experienced a hemorrhage or seizures secondary to an AVM are managed in much the same way as patients with aneurysms. Safeguards and family teaching include a discussion of signs of increased ICP, seizure control and safety, anticonvulsant therapy, postoperative complications, and side effects of radiation, when appropriate.

Surgical Approaches

Neurological surgery may be performed in a number of situations as follows:

1. To obtain tissue for pathological diagnosis
2. To remove an abnormal mass or space-occupying lesion (eg, tumor, cyst, hemorrhage) and, consequently, to reduce mass effect
3. To repair an abnormality (eg, aneurysm)
4. To place a device (eg, shunt, reservoir)

A number of factors are considered when making the appropriate surgical decision. Diagnostic studies are first performed to establish a differential diagnosis. Patient age, neurological status, and concurrent medical conditions are factors in the decision to proceed with surgery and the decision regarding the appropriate approach. The most commonly used surgical procedures are briefly described in the following sections.

Stereotactic Biopsy

A stereotactic biopsy is used to obtain tissue for definitive pathological diagnosis. It is often used when a tumor is suspected but the lesion is too small or deep for surgical removal. It is used for tumors in eloquent areas of the brain, lesions crossing the corpus callosum, and multiple lesions that are not resectable.[2,26,27] A stereotactic biopsy is also used to confirm the diagnosis of previously treated tumors—for example, when a malignant glioma has been treated with multiple therapies, tissue is obtained and analyzed to differentiate active tumor from treatment effect (ie, necrosis). In addition, some patients may have multiple medical problems and be too ill to proceed with a craniotomy. Others may opt to have the less invasive biopsy.[2]

The goal of stereotactic surgery is to locate a target using a trajectory. Stereotactic biopsies with a frame require placement of a rigid head frame to establish the appropriate coordinates. Next, a contrast-enhanced CT scan or MRI is obtained in the radiology suite by using the localizing frame. An axial image of the tumor is displayed, with a number of coordinates to indicate entry points. After the imaging is completed, the patient is usually transferred from the radiology suite to the operating room for completion of the procedure.[2] The biopsy can be performed under general or local anesthesia.[2] After the skin is shaved and prepared, a small hole (twist drill or burr hole) is made, a needle is passed to the lesion, and one or more biopsies are obtained and immediately evaluated by a pathologist. Once sufficient tissue or cyst fluid is obtained for diagnostic purposes, the procedure is complete. Because of the possibility of sampling error, it is not unusual for the neurosurgeon to take multiple specimens from various areas of the tumor or other abnormal area defined on neuroimaging, when feasible.[2,27]

A stereotactic biopsy may also be performed without a frame. Frameless stereotaxy is a navigational system used to generate a three-dimensional tumor image. A CT or MRI scan is taken before the procedure, and markers (fiducials) are placed on the scalp. The markers are evident on the scan and are used to determine the actual target. A computer image is then generated from the imaging data.[2,26]

Craniotomy

A craniotomy is performed to remove a space-occupying abnormality such as a tumor, cyst, or vascular malformation. This procedure may also be needed on an emergency basis to evacuate a hematoma or reverse a herniation syndrome. When appropriate, a craniotomy is used to clip an aneurysm.[1,2]

In this procedure, the surgeon makes a skin incision, elevates the bone flap, opens the dura, and obtains tissue from the lesion for biopsy or performs resection. The neurosurgical patient has quite distinct intraoperative pharmacological needs. The neuroanesthesiologist administers drugs that provide the needed anesthetic effect while minimizing risks for increasing ICP or lowering seizure threshold. Rapid reversibility is also particularly important in patients receiving a craniotomy because their postoperative neurological status needs to be assessed quickly.[1,2]

In addition to the equipment used during surgery to maximize safety and efficiency, specific tools for intraoperative monitoring may enhance the outcome for these patients. During the past 10 to 15 years, significant advances have taken place. Ultrasonography has been a standard of neurosurgical monitoring for some years because it can distinguish abnormal lesions from normal brain tissue and edema. Residual abnormal tissue may be identified before closing the surgical site. Frameless stereotaxy, as described previously, is also used during craniotomy. It is thought that this procedure enhances surgical safety and effectiveness by reducing craniotomy size, minimizing brain manipulation, and maximizing tumor resection.[2,26,27] Cortical mapping is used for masses in

eloquent areas of the brain. Somatosensory evoked potentials are recorded during surgery under general anesthesia to assess the relationship between the motor strip and the lesion to be resected. Direct cortical stimulation provides for localization of the sensorimotor cortex and is also used to minimize neurological deficits and maximize tumor removal. In some cases, greater seizure control is accomplished with these procedures. Direct cortical stimulation requires local anesthesia and the patient to be conscious during much of the procedure.[28]

Craniotomies have been enhanced by the use of microscopes, operating loupes, self-retaining retractors, high-speed drills, ultrasonic aspirators (using sound waves/high-frequency vibration and immediate aspiration of tissue from the lesion), and laser (using light beams). Bipolar coagulation is used to minimize bleeding.[2]

Postoperative management of the patient who has undergone a craniotomy or stereotactic biopsy for a brain tumor focuses on assessment of and intervention for a number of potential complications. In the immediate postoperative period, patients may be slow to respond because of the effects of general anesthesia. Temporary changes in mental status or new focal neurological signs should resolve rather quickly in this situation.[1,2] If there is a significant change from the baseline examination, radiographic documentation of hemorrhage or cerebral edema is performed. A CT scan or MRI is obtained to rule out postoperative complications. Edema is expected and can often be treated with corticosteroids. If significantly increased ICP occurs, the patient is medically managed in an intensive care environment under close observation. Occasionally, surgical intervention is required for acute postoperative hemorrhage.

Other postoperative monitoring includes ongoing evaluation of vital signs and neurological status; early ambulation to avoid pulmonary and cardiovascular complications; physical and occupational therapy evaluations; speech and cognitive assessment when indicated; DVT and PE prophylaxis; and wound evaluation and care.

Transsphenoidal and Transnasal Surgeries

Transsphenoidal and transnasal surgeries are being used in many centers to remove pituitary tumors and cysts. The transnasal and transsphenoidal approaches replace transcranial surgery, when appropriate. An estimated 75% to 95% of cases are treated in this way. The patient is positioned on the operating table under general anesthesia. The sphenoid sinus is opened, the sella is opened, and the tumor is removed using the surgical microscope. If there is evidence of a CSF leak at the time of surgery, the sellar cavity is packed with fat tissue, typically taken from the patient's abdomen. The mucosal incision is closed with reabsorbable sutures. Nasal septal splints are applied.[1,2]

This procedure is usually well tolerated. Postoperative care is aimed at increasing mobility, monitoring respiration, evaluating fluid and electrolyte balance, and observing for evidence of CSF leak. The major risks of intracranial surgery—cerebral edema and intracerebral hemorrhage—are avoided. Nasal splints are removed 2 to 4 days after surgery.[1,2]

Neuroendoscopy as a Surgical Tool

Endoscopic microsurgical techniques are being used with increasing frequency. This surgical tool improves visualization of normal anatomy and of abnormal lesions. It is most often used for smaller, avascular lesions of soft consistency. Colloid and choroid plexus cysts, ependymomas, some skull base tumors, and certain gliomas are amenable to this approach. The technique involves the use of an angled, flexible endoscope, thus enhancing the approach to tumor removal and aneurysm clipping.[2,29] Additionally, it allows for improved inspection during transsphenoidal surgery, providing closer evaluation of the pituitary tumor versus the normal gland.[2,29] Risks of neuroendoscopy approaches to pituitary tumor resection include persistent CSF leakage. Detailed surgical repair using autologous materials such as fat, bone, and fascia may be successfully done at the time of surgery.[29]

In selected endoscopic cases, frameless stereotaxy is used to increase accuracy and minimize brain trauma. It should be noted that the surgeon's experience and expertise are crucial to minimizing morbidity and maximizing the effectiveness of this approach.[2,26]

▲ Neurological Disorders

Common neurological disorders requiring critical care management are discussed in the following section. Spotlight on Genetics 35-1 contains information about Huntington's Disease, a neurodegenerative genetic disorder that affects muscle coordination and leads to cognitive decline and dementia.

Stroke

Cerebrovascular disease includes any pathological process that involves the blood vessels of the brain. It is the most frequent

SPOTLIGHT ON GENETICS 35-1

Huntington's Disease

- Huntington's disease affects an estimated 3 to 7 per 100,000 people of European ancestry and causes uncontrolled movements, emotional problems, and loss of thinking ability.
- Mutations in the HTT gene cause Huntington's disease. The HTT gene provides instructions for making a protein called huntingtin, which appears to play an important role of neuron coordination in the brain.
- The HTT mutation that causes Huntington's disease involves a DNA segment known as a CAG trinucleotide repeat. This segment is made up of a series of three DNA building blocks (cytosine, adenine, and guanine) that appear multiple times in a row. The CAG segment is repeated 10 to 35 times within the gene. In people with Huntington's disease, the CAG segment is repeated 36 to more than 120 times. People with 36 to 40 CAG repeats may or may not develop the signs and symptoms of Huntington's disease, while people with more than 40 repeats almost always develop the disorder.
- Genetic testing, such as targeted mutation analysis, is available.

Genetic Home Reference-http://ghr.nlm.nih.gov, accessed July 14, 2011.

Ross CA, Tabrizi SJ: Huntington's disease: from molecular pathogenesis to clinical treatment. *Lancet Neurol* 10(1):83–98, 2011.

neurological disorder that affects adults. Most cerebrovascular disease is caused by thrombosis, embolism, or hemorrhage. The mechanism of each of these etiologies is different, but the ultimate result is damage to a focal area of the brain.[30–32]

A stroke may be defined as a neurological deficit that has a sudden onset, results in permanent damage to the brain, and is caused by cerebrovascular disease. A stroke occurs when there is a disruption of blood flow to a region of the brain. Blood flow is disrupted because of an obstruction of a vessel, a thrombus or embolus, or the rupture of a vessel. The apparent clinical features depend on the location of the event and region of the brain perfused by the vessel.[30–32]

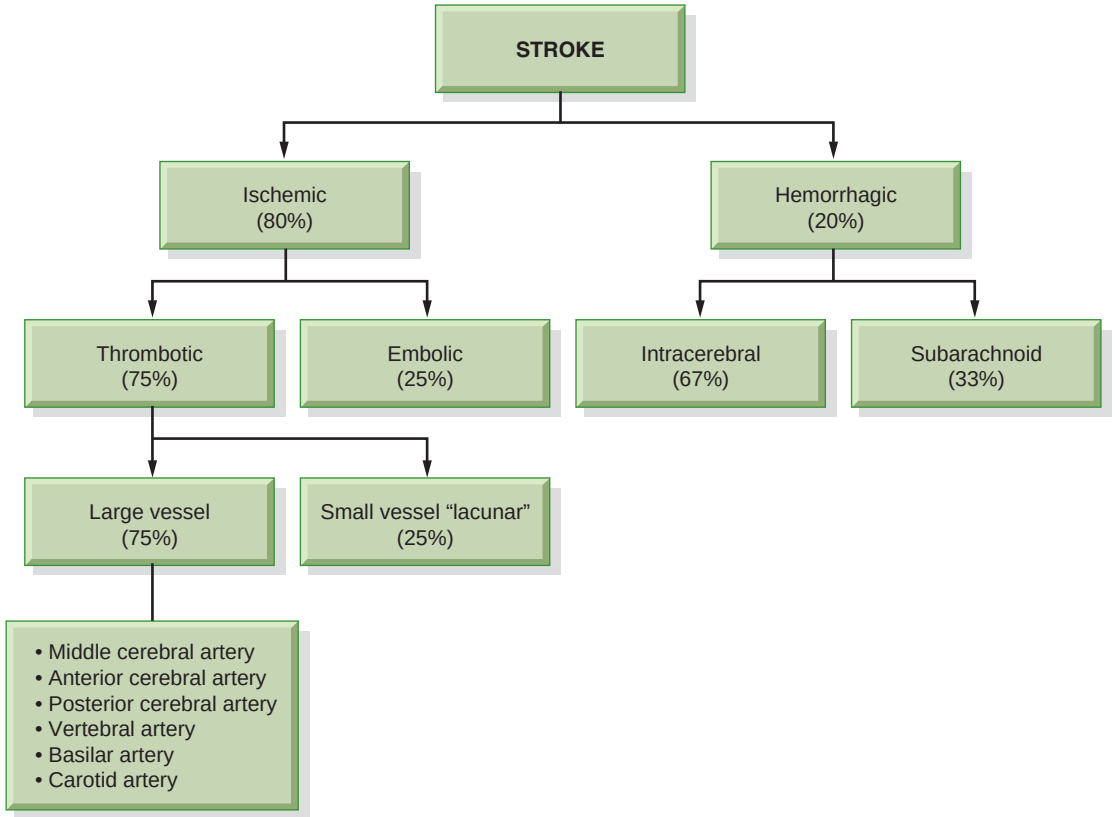
A stroke is now referred to as a "brain attack" to encourage health care professionals and the public to think about stroke with the same urgency as a "heart attack." A "brain attack" must be viewed as a medical emergency. To reverse cerebral ischemia, patients must be evaluated promptly. Ischemic brain injury occurs when arterial occlusion lasts longer than 2 to 3 hours. Delay in seeking medical care may eliminate the potential for tissue-saving therapy with thrombolytic drugs. Stroke is the fourth leading cause of death in the United States.[33] Even when stroke is not fatal, it can result in serious long-term disability.[30,34]

During the past several years, advances have been made in the treatment of stroke. Early recognition and prompt entry into the emergency medical system (EMS) are essential to reduce death and disability from stroke. Media campaigns have been launched to increase public awareness about the signs and symptoms of stroke so that care may be sought promptly.

A recent innovation in the way stroke care is delivered involves the establishment of the Joint Commission's certified stroke centers. The Joint Commission implemented a Disease-Specific Care Certification program in 2002, and stroke was one of the disease-specific categories in which program certification could be achieved. In this voluntary program, organizations have disease management programs reviewed by the Joint Commission. Criteria for the program include compliance with consensus-based national standards, effective use of established clinical practice guidelines to manage and optimize care, and an organized approach to monitor performance improvement activities. Achievement of Primary Stroke Center certification denotes clinical excellence in the management of stroke provided by a multidisciplinary team from several departments. This designation is attractive to many institutions that aspire to be recognized for their care of stroke patients.[34,35]

Etiology

Approximately three fourths of strokes in the United States are due to vascular obstruction (thrombi or emboli), resulting in ischemia and infarction. About one fourth of strokes in the United States are hemorrhagic, resulting from hypertensive vascular disease (which causes an intracerebral hemorrhage), a ruptured aneurysm, or an AVM.[30,32,34,35] Figure 35-5 outlines stroke classification.



Note: Generated/simplified data

FIGURE 35-5 ▲ Classification of stroke. (Courtesy of Eric Aldrich, MD, PhD, The Johns Hopkins University, Baltimore, MD.)

Epidemiology

Approximately 780,000 people have a new or recurrent stroke each year, with a mortality rate of 35%.[35] Even though the average age of stroke is 70 years, 40% of all strokes occur in people younger than 60 years. Women have overtaken men in stroke incidence, with about 60,000 more women than men experiencing strokes annually. With longer life expectancies for women, more women actually die of stroke, accounting for 61% of stroke deaths. It is estimated that there are 3 million stroke survivors and that stroke is a leading cause of disability and a leading diagnosis for long-term care.[35] Risk factors for stroke include smoking, hypertension, obesity, cardiac disease, hypercholesterolemia, diabetes, cancer, use of birth control pills, and patent foramen ovale with atrial septal aneurysm. Prevention efforts focus on lifestyle changes that can modify risk factors. In addition, the appropriate use of warfarin (Coumadin) in patients at risk for cardiac sources of emboli (eg, atrial fibrillation) and use of aspirin in patients at risk for thrombotic stroke constitute primary prevention.[30,32,34,35]

Pathophysiology

When blood flow to any part of the brain is impeded as a result of a thrombus or embolus, oxygen and substrate deprivation of the cerebral tissue begins. Deprivation for 1 minute can lead to reversible symptoms, such as loss of consciousness. Oxygen deprivation for longer periods can produce microscopic necrosis of the neurons. The necrotic area is then said to be infarcted.[30,32,34,35]

The initial oxygen deprivation may be caused by general ischemia (from cardiac arrest or hypotension) or hypoxia (from an anemic process or high altitude). If the neurons are ischemic only and have not yet necrosed, they may be saved. This situation is analogous to the focal injury caused by a myocardial infarction. An occluded coronary artery can produce an area of infarcted (dead) tissue. Surrounding the infarcted zone is an area of ischemic tissue, which has been marginally deprived of oxygen. This ischemic tissue, as in the brain, may be either salvaged with appropriate treatment or killed by secondary events.[30,32,34]

Cerebral ischemia is a complex process that depends on the severity and duration of the decline in CBF. The ischemic cascade begins within seconds to minutes after perfusion failure, creating a zone of irreversible infarction and a surrounding area of potentially salvageable "ischemic penumbra." The goal of acute stroke management is to salvage the ischemic penumbra, or the territory at risk. Without prompt intervention, the entire ischemic penumbra can eventually become an infarcted region.[32,34,35]

A stroke caused by an embolus may be a result of blood clots, fragments of atheromatous plaques, lipids, or air. Emboli to the brain most often have a cardiac source, secondary to myocardial infarction or atrial fibrillation. If hemorrhage is the etiology of a stroke, hypertension often is a precipitating factor. Vascular abnormalities, such as AVMs and cerebral aneurysms, are more prone to rupture and cause hemorrhage in the presence of hypertension.[35]

The most frequent neurovascular syndrome seen in thrombotic and embolic strokes is due to involvement of the MCA. This artery supplies mainly the lateral aspects of the cerebral hemisphere. Infarction to that area of the brain can cause contralateral motor and sensory deficits. If the infarcted hemisphere is dominant, dysphasia may be present. It is difficult to predict the amount of brain ischemia and infarction resulting from a thrombotic or embolic stroke. There is a possibility that the stroke will extend after the initial insult. There can be massive cerebral edema and an increase in ICP to the point of herniation and death after a huge thrombotic stroke. The area of the brain involved and the extent of the insult influence the prognosis. Because thrombotic strokes often are caused by atherosclerosis, there is risk for a future stroke in a patient who already has had one.[32,34,35] With embolic strokes, patients also may have subsequent episodes of stroke if the underlying cause is not treated. If the extent of brain tissue destroyed from a hemorrhagic stroke is not excessive and is in a nonvital area, the patient may recover with minimal deficits. If the hemorrhage is large or in a vital area of the brain, the patient may not recover; however, if the intracerebral hemorrhage is less massive, survival is possible. For the purposes of this discussion, the focus is on the diagnosis and management of ischemic stroke.

Clinical Manifestations

A stroke is usually characterized by the sudden onset of focal neurological impairment. The patient may experience signs such as weakness, numbness, visual changes, dysarthria, dysphagia, or aphasia. The manifestations of a stroke depend on the anatomical location of the lesion; an infarct in a certain portion of the brain results in loss of function of the body part that it controlled or skill for which it was responsible.[32,34,35] Table 35-8 presents the correlation of blood supply to symptomatology in a brain attack.

If symptoms resolve in less than 24 hours, the event is classified as a transient ischemic attack (TIA), which is defined as a "neurological deficit lasting less than 24 hours that is attributed to focal cerebral or retinal ischemia." Most TIAs last for only minutes to less than an hour, which further clouds recognition and prompt treatment. A significant proportion of people who experience a TIA will have a stroke within 5 years.[35] An aggressive workup following TIA is encouraged.

Diagnosis

Rapid diagnosis of a stroke is essential so that appropriate patients can receive thrombolytic therapy, the goal of which is to save damaged brain tissue and minimize permanent deficits. The patient should be taken to an emergency department where a neurologist can perform an initial screening and obtain appropriate neuroimaging studies.[30–32,34,35] The time of symptom onset to administration of thrombolytic therapy (or "time to needle") should be as quickly as possible. Formerly, the time limit for administration of thrombolytic therapy was to be within a 3-hour window from symptom onset. Recent evidence supports a time window of 4.5 hours from symptom onset for administration of thrombolytic therapy.[30,31,36] Emergency departments need to have services streamlined so that testing may be performed and treatment initiated promptly.

The patient's history helps determine what has happened to the person. It is important to obtain a description of the neurological event; how long it lasted; and whether the symptoms are resolving, completely gone, or the same as at the time of onset. The differential diagnosis of stroke includes ruling out intracerebral hemorrhage, SAH, subdural

Table 35-8 Anterior and Posterior Blood Supply

Artery	Brain Structure	Signs/Symptoms of Occlusion
Anterior Blood Supply		
Anterior choroidal	Globus pallidus, lateral geniculate body, posterior limb of internal capsule, medial temporal lobe	Contralateral hemiplegia Hemihypesthesia Homonymous hemianopia
Ophthalmic	Orbit and optic nerve	Transient mononuclear blindness or complete unilateral blindness
Anterior cerebral	Anterior three fourths of medial surface of cerebral hemispheres, caudate nucleus, globus pallidus, and the internal capsule	Contralateral sensory and motor deficits greater in leg than arm Incontinence Deviation of the eyes and head toward the lesion Contralateral grasp reflex Abulic symptoms Arm apraxia Expressive aphasia (in dominant hemisphere occlusion) Motor or sensory aphasia (distal occlusion)
Middle cerebral	Cortical surfaces of the parietal, temporal, and frontal lobes Basal ganglia and internal capsule	Complete: Spatial neglect and homonymous hemianopia Global aphasia (left lesion) Superior trunk: Contralateral hemiplegia and hemianesthesia in face and arm Ipsilateral deviation of eyes and head Broca's aphasia (usually left sided) Inferior trunk: Contralateral hemianopia or upper quadrantanopia Wernicke's aphasia (left lesion) Left visual neglect (right lesion)
Posterior Blood Supply		
Vertebral	Anterolateral parts of the medulla	Contralateral impairment of pain and temperature sensation
Posterior cerebral	Occipital lobe, medial and inferior surface of temporal lobe, the midbrain, third and lateral ventricles	Contralateral hemiplegia, sensory loss, and ipsilateral visual field deficits
Posterior inferior cerebellar	Medulla and cerebellum	Medial branch: Vertigo, nystagmus, ataxia, persistent dizziness Lateral branch: Unilateral clumsiness with gait and limb ataxia Inability to stand Sudden falling Vertigo, dysarthria, oculomotor signs
Anterior inferior cerebellar	Cerebellum and pons	Horner's syndrome and contralateral loss of pain and temperature sense of the arm, trunk, and leg
Superior cerebellar	Upper part of cerebellum, midbrain	Slurred speech and contralateral loss of pain and thermal sensation
Basilar	Pons and midbrain	Limb paralysis, bulbar or pseudobulbar paralysis of cranial nerve motor nuclei, nystagmus, coma, or locked-in syndrome

From Testani-Dufour L, Morrison CAM: Brain attack: Correlative anatomy. J Neurosci Nurs 29(1):213–224, 1997.

or epidural hematoma, neoplasm, seizure, or migraine headache.[30,32,34,35] Identifying the type of symptoms can help determine the diagnosis and locate a possible vascular distribution. Determination of risk factors for stroke, such as hypertension, chronic atrial fibrillation, elevated serum cholesterol, smoking, oral contraceptive use, or a familial history of stroke, also aids in diagnosis.

In the emergency department, some of the tests that are frequently used to evaluate the patient with acute ischemic stroke are a CT scan of the brain without contrast media, blood studies, neurological examination, and screening using the National Institutes of Health Stroke Scale (NIHSS).[30,32,34,35] This tool allows a score to be given for the severity of the stroke. Table 35-9 summarizes the NIHSS.

There is no definitive laboratory study currently available that determines whether a patient has experienced a stroke. Rather, the results are viewed in conjunction with the history, neurological examination, and neuroimaging studies. Laboratory tests, including complete blood cell count, electrolytes, glucose, and coagulation parameters, are obtained.

Table 35-9 National Institutes of Health Stroke Scale

1.a.	Level of consciousness (LOC)	Alert	0
		Drowsy	1
		Stuporous	2
		Comatose	3
1.b.	LOC questions: The patient is asked the month and/or his or her age. There is no partial credit for being "close" with an answer. Aphasic and stuporous patients who do not comprehend the question would score a "2."	Answers both correctly	0
		Answers one correctly	1
		Answers neither correctly	2
1.c.	LOC commands	Performs both correctly	0
		Performs one correctly	1
		Performs neither correctly	2
2.	Best gaze	Normal	0
		Partial gaze palsy	1
		Forced deviation	2
3.	Visual	No visual loss	0
		Partial hemianopia	1
		Complete hemianopia	2
		Bilateral hemianopia	3
4.	Facial palsy	Normal	0
		Minor paralysis	1
		Partial paralysis	2
		Complete paralysis	3
5.	Motor arm	No drift	0
		Drift	1
		Some effort against gravity	2
		No effort against gravity	3
		No movement	4
		Amputation, joint fusion explain:	9
6.	Motor leg	No drift	0
		Drift	1
		Some effort against gravity	2
		No effort against gravity	3
		No movement	4
		Amputation, joint fusion explain:	9
7.	Limb ataxia	Absent	0
		Present in one limb	1
		Present in two limbs	2
8.	Sensory	Normal	0
		Mild to moderate loss	1
		Severe to total loss	2
9.	Best language	No aphasia	0
		Mild to moderate	1
		Severe	2
		Mute	3

An urgent CT scan should be performed to rule out intracerebral hemorrhage. Ideally, the CT scan is obtained within 60 minutes of arrival in the emergency department so that treatment decisions can be made. A CT scan can be useful in differentiating between cerebrovascular and nonvascular lesions. For example, a subdural hemorrhage, brain abscess, tumor, SAH, or intracerebral hemorrhage is visible on the CT scan.[30,32,34,35] However, an area of infarction may not show on the CT scan for 24 to 48 hours.

Newer neuroimaging techniques also provide valuable information. MRI, including T1- and T2-weighted, fluid-attenuated inversion recovery (FLAIR), and diffusion-weighted techniques, has become widely available and is better at detecting infarction than a CT scan.[32,34] The earliest changes normally appear within the first 24 hours.

Other studies that may be performed, based on availability of the technology, are MRI diffusion-weighted imaging (DWI) and perfusion-weighted imaging (PWI). These techniques help identify the infarct core and penumbra, which is important because the presence of viable tissue directs interventions such as reperfusion. The ischemic penumbra surrounds the infarcted tissue. It is the marginally perfused area of the brain that has been damaged by the insult but is potentially salvageable. DWI detects acute infarction as early as a few hours after the onset of symptoms. It can reveal changes associated with infarcted tissue hours before

a CT scan or conventional MRI can detect any abnormality. It also differentiates acute from chronic ischemic changes. PWI shows the regional abnormalities of CBF. The difference between the diffusion defect and the perfusion defect represents the ischemic penumbra, or the "territory at risk."[32] DWI-PWI identifies patients who are ideal candidates for thrombolytic therapy.

Cerebral angiography has been the gold standard for evaluating cerebral vasculature. There is an estimated 1.5% to 2% associated risk for morbidity or mortality with this procedure. However, it can demonstrate an arterial occlusion or embolus. Because of the time that it takes to perform cerebral angiography, the window of opportunity to treat a patient with IV thrombolytics may be missed. However, angiography is necessary for intra-arterial thrombolysis in which tissue plasminogen activator (t-PA) or another thrombolytic is administered at the site of the clot by catheter into the artery.[31,34] The time window may be as long as 6 hours from symptom onset for intra-arterial thrombolysis, 8 hours from symptom onset for mechanical thrombolysis, and 24 hours for intravascular intervention for basilar artery stroke. The vasculature can be evaluated noninvasively by the use of TCD, ultrasonography, MRA, or CT angiography.

An electrocardiogram (ECG) should be obtained to assess for evidence of dysrhythmia or cardiac ischemia. The ECG helps determine whether a dysrhythmia is present, which may have caused the stroke. Atrial fibrillation is a dysrhythmia in which clots form in the heart and may travel to the brain (hence a cardioembolic etiology). Other changes that might be found on an ECG are an inverted T wave, ST elevation or depression, and QT prolongation. Transesophageal echocardiography and Holter monitoring may also be performed.[35]

In summary, prompt performance of a CT scan and subsequent interpretation are crucial to acute stroke management. Head CT provides vital information to allow the physician to make the decision to use thrombolytic therapy. An alternate approach is urgent MRI with PWI and DWI.

Clinical Management

The management of an ischemic stroke has four primary goals: restoration of CBF (reperfusion), prevention of recurrent thrombosis, neuroprotection, and supportive care. The timing of each element of clinical management should be implemented in a decisive manner.

Optimally, patients are initially evaluated at a center that has a stroke program, perhaps even a Joint Commission Primary Stroke Center. Decisions in the emergency department determine the patient's treatment plan. Emergency departments may have standardized orders, clinical pathways, or protocols that have been developed by a multidisciplinary team to guide care.[30,34,35]

The focus of initial treatment is to save as much of the ischemic area as possible. Three ingredients necessary to this area are oxygen, glucose, and adequate blood flow. The oxygen level can be monitored through arterial blood gas analyses or pulse oximetry, and oxygen can be given to the patient if indicated. Hypo/hyperglycemia can be evaluated with serial checks of blood glucose levels. Reperfusion may be accomplished by the use of IV t-PA.[30,34,35]

CPP is a reflection of the systemic BP and ICP. Regional perfusion is influenced by autoregulation in the brain, and MAP is influenced by cardiac output and heart rate (CPP = MAP − ICP). The parameters most easily controlled externally are the BP and cardiac rate and rhythm.[1] Dysrhythmias can reduce cardiac output and BP but usually can be corrected. There is a loss of autoregulation in the ischemic penumbra, so that reducing BP can further reduce blood flow in the penumbra and can lead to infarction.[32,34]

If the patient is a candidate for IV thrombolytic therapy, treatment with t-PA begins in the emergency department, and he or she is then moved to the ICU or other specialized monitored setting such as a neuroscience step-down or dedicated stroke unit for further monitoring. If the patient is not a candidate for thrombolytic therapy, the complexity of the patient's problems determines his or her placement in the ICU, medical unit, or stroke specialty unit.

Currently, two emergency treatments are available for stroke management: IV t-PA and the mechanical embolus removal for cerebral ischemia (MERCI) retriever. IV t-PA is an approved U.S. Food and Drug Administration (FDA) treatment. The MERCI retriever was approved by the FDA in 2004 for other uses and is currently being used in trials to determine its efficacy, but it is not approved as a treatment for stroke management.

Thrombolytic Drugs

Thrombolytic medications are exogenous drugs that dissolve clots. IV t-PA dissolves the clot and permits reperfusion of the brain tissue. IV thrombolytic therapy should be initiated as quickly as possible from symptom onset. The maximal time window is now expanded to 4.5 hours or less from the onset of neurological symptoms.[30,31,35,36] The clock begins for the patient from the time he or she was last seen well. For example, a patient retires to bed at 11:00 PM and awakens at 5:00 AM to go to the bathroom. As he attempts to rise from the bed, he feels weak and has difficulty standing up. As he calls out for his wife's help, his speech is garbled. The last time he was awake and functioning normally was 11:00 PM. Even if his symptoms started only a few minutes ago, the time he was last seen well was 6 hours ago. Therefore, he is already outside of the treatment window for IV t-PA.

Candidate selection for IV t-PA must be done carefully. The neurological examination, NIHSS score, and results of neuroimaging studies assist the physician with the decision to offer thrombolytic therapy. Box 35-2 outlines eligibility criteria for this treatment. The standards for the administration of IV t-PA to treat stroke are a result of the National Institute of Neurologic Disorders and Stroke t-PA Stroke Study. A dose of IV t-PA, 0.9 mg/kg (maximal dose, 90 mg), is administered as 10% of the total dose as a bolus over 1 to 2 minutes, with the remainder infused over 60 minutes. The t-PA activates plasminogen, a naturally occurring enzyme present in the intravascular endothelium that protects against excessive clotting. Activating plasminogen initiates the process of dissolving the clot through fibrinolysis. No other antithrombotic therapy should be given for the next 24 hours. A major risk of this therapy is intracerebral hemorrhage.[31,32,36] However, it is encouraging that this agent may prove effective in reversing a neurological deficit and improving quality of life after a stroke.

The direct administration of a thrombolytic into an artery is an alternative to IV t-PA. Such administration is effec-

BOX 35-2 Eligibility Criteria for Thrombolytic Therapy

Inclusion Criteria

1. Symptom onset of less than 4.5 hours
2. Clinical diagnosis of ischemic stroke with measurable deficit on the NIHSS
3. Older than 38 years
4. CT criteria: absence of high-density lesion consistent with intracerebral hemorrhage; absence of significant mass effect or midline shift; absence of parenchymal hypodensity; or effacement of cerebral sulci in more than 33% of the MCA territory

Exclusion Criteria

1. Stroke or serious head trauma within past 3 months
2. Systolic BP more than 185 mm Hg or diastolic BP more than 110 mm Hg refractory to aggressive pharmacological treatment, or BP readings that require aggressive treatment and yield only marginal BP control.
3. Conditions that could precipitate or suggest parenchymal bleeding (subarachnoid and intracerebral hemorrhage; recent-onset myocardial infarction; seizures at onset; major surgery within past 14 days; GI or urinary tract hemorrhage within previous 21 days; and arterial puncture of a noncompressible site or lumbar puncture within previous 7 days)
4. Glucose less than 50 mg/dL or greater than 400 mg/dL; INR greater than 1.7; platelet count less than 100,000/mm^3
5. Rapidly improving or deteriorating neurological signs or minor symptoms
6. Recent myocardial infarction
7. Recent treatment with IV or subcutaneous heparin within past 48 hours and elevated partial thromboplastin time
8. Women of childbearing age who have a positive pregnancy test result

Data from Saver JL, Kalafut M: Thrombolytic therapy in stroke. Updated April 15, 2010; accessed June 5, 2010 from http://emedicine.medscape.com/article/1160840-print; Becker JU, Wira CR, Arnold JL: Stroke, ischemic. Updated May 12, 2010, accessed June 15, 2010 from http://emedicine.medscape.com/article/793904-print; Pugh S, Mathiesen C, Meighan D, et al: Care of the Patient With Ischemic Stroke: AANN Reference Series for Clinical Practice. Am Assoc Neurosci Nurses 2009; and Summers D, Leonard A, Wentworth D, et al: Comprehensive overview of nursing and interdisciplinary care of the acute ischemic stroke patient. Stroke 40:2911–2944, 2009.

tive in acute ischemic stroke and can be given up to 6 hours after the onset of symptoms. A limiting factor is that the patient must be admitted to a specialty center in which localized intra-arterial infusion of thrombolytic drugs is possible. Through this approach, an occluded cerebral artery can be reopened. For intra-arterial therapy, a femoral arterial sheath is usually inserted, through which a microcatheter can be threaded, under fluoroscopy. The catheter tip is positioned into the clot and advanced as the clot dissolves. The femoral sheath usually remains in place for 24 hours in case of recurrent vessel occlusion. The advantage of this approach is that the medication can be delivered directly to its target.[30,32,34]

MERCI Retriever

The MERCI system (a mechanical clot retriever) can be used for up to 8 hours after stroke onset to remove blood clots from vessels. If a patient arrives at the hospital too late to receive IV t-PA, the MERCI device presents another treatment option if the patient meets strict criteria. Inclusion criteria include diagnosis of acute ischemic stroke; NIHSS greater than 8; and occlusion of internal carotid artery, basilar artery, or vertebral artery on cerebral angiography.[37] Some exclusion criteria include blood glucose level less than 50 mg/dL, excessive vessel tortuosity, hemorrhagic tendency, elevated international normalized ratio (INR), decreased platelets, sustained hypertension, CT revealing large areas of hypodensity, and arterial stenosis on angiography proximal to the embolus.[37]

The MERCI retriever works like a corkscrew to retrieve the clot. During cerebral angiography, an interventional radiologist passes the microcatheter into the femoral artery. It is advanced into the carotid until it reaches the clot. A wire is then pushed through the catheter, causing it to return to a corkscrew shape. It then snares the embolus.[37]

Potential hazards from use of the MERCI retriever include bleeding and vascular dissection or perforation. The patient needs to be closely monitored for the first 24 hours to detect adverse effects. The nurse, who plays a key role in postprocedure monitoring, performs neurological assessments and carefully monitors the patient for signs of intracranial hemorrhage, new stroke, or myocardial infarction.[37]

Anticoagulation

Aside from thrombolytic therapy and mechanical clot retrieval, secondary treatment options for stroke include anticoagulation with antithrombotic and antiplatelet drugs. If a patient experiences atrial fibrillation, anticoagulation with warfarin may be warranted. The patient will need instruction about bleeding precautions. Education also includes the purpose of the medication, information about moderate consumption of leafy green vegetables containing vitamin K, and the importance of having blood drawn regularly to monitor prothrombin time and the INR. In addition, for safety, patients should be instructed to obtain medical identification cards and bracelets so that they can be identified as taking an anticoagulant in the event of a medical emergency.[34,35]

Antiplatelet drugs include dipyridamole-ER, ticlopidine, clopidogrel, and aspirin. These drugs deter platelets from adhering to the wall of an injured blood vessel or other platelets and are given to prevent a future thrombotic or embolic event. The modified-release formula of dipyridamole increases the effect of specific factors that act as antiaggregates to reduce platelet aggregation. Ticlopidine inhibits platelet function by suppressing adenosine diphosphate–induced platelet aggregation and aggregation due to other factors. The recommended dose of ticlopidine is 250 mg twice a day. Neutropenia and thrombocytopenia are known serious adverse effects, so it is now rarely used. Clopidogrel also inhibits the activity of adenosine diphosphate but is not associated with an increased risk for neutropenia. Aspirin limits platelet adhesion and aggregation. The suggested dose of aspirin is 81 to 325 mg/d. The administration of these drugs plays a role in stroke prevention by decreasing the risk for future strokes.[30,34,35]

Control of Hypertension and Increased Intracranial Pressure

The control of hypertension, increased ICP, and CPP takes the efforts of the nurse and the physician. The nurse must assess for these problems, recognize them and their significance, and advocate for the patient, ensuring that medical interventions are initiated.

Patients with moderate hypertension usually are not treated acutely. If their BP decreases after the brain becomes accustomed to the hypertension needed for adequate perfusion, the brain's perfusion pressure falls along with the BP. If the diastolic BP is above approximately 105 mm Hg, it needs to be lowered gradually. This can be accomplished effectively with labetalol as well as calcium channel blockers.[32,34,35]

If ICP is elevated in a patient who has had a stroke, it usually occurs after the first day. Although this is a natural response of the brain to some cerebrovascular lesions, it is destructive to the brain. The usual methods of controlling increased ICP can be instituted: hyperventilation (in a patient receiving controlled ventilation in the short term only to control critical ICP elevations pending institution of additional therapies); fluid restriction; head elevation; avoidance of neck flexion or severe head rotation that would impede venous outflow from the head; and the use of osmotic diuretics (mannitol) to decrease cerebral edema (see Chapter 34 for more information).

Surgical Management

In patients with carotid stenosis, carotid endarterectomy may be performed to prevent a stroke. Carotid endarterectomy is a surgical procedure in which atherosclerotic plaque that has accumulated inside the carotid artery is surgically removed. Once the plaque is removed, blood flow is restored. The North American Symptomatic Carotid Endarterectomy Trial and the European Carotid Surgery Trial were designed to examine the benefit of surgery for patients with symptomatic carotid stenosis. These studies determined that carotid endarterectomy is justifiable in patients with high-grade stenosis (>70%) if the operation is performed by a skilled surgeon. The benefit of surgery increases for male patients with a prior history of stroke. Patients with less than 50% stenosis do not benefit from surgery.[35] During aggressive clinical management for stroke, brain edema and ICP elevation may become refractory to measures such as controlled/short-term hyperventilation and osmotic diuresis. A subset of patients may benefit from hemicraniectomy, allowing additional space for edematous brain to expand and relieving ICP elevations pending resolution of brain edema. Additionally, extracranial–intracranial bypass surgery, with careful patient selection in limited case series, has been effective in arresting stroke progression and has resulted in rapid improvement in neurological assessment findings.[38]

Nonsurgical Management

Although the gold standard for managing carotid artery stenosis has been carotid endarterectomy, another management option, carotid stenting, is available. This newer, minimally invasive procedure is attractive for patients in whom traditional surgery is contraindicated, such as those with severe cardiac or pulmonary disease. Carotid artery stenting opens vessels that have been narrowed by plaque accumulation. An interventional radiologist passes a catheter along the femoral artery to the narrowed artery. Once the catheter crosses the area of stenosis, a small filter may be deployed to catch any pieces of plaque that may be dislodged during the procedure. Angioplasty, in which plaque is pressed against the artery wall, may be performed, and a stent is placed in the artery. Recent research showed that relative risk of stroke, myocardial infarction, or death did not differ significantly between patients who underwent carotid artery stenting versus carotid endarterectomy.[39]

Following carotid stenting, there is a risk for stroke and hyperperfusion syndrome. Postprocedure, the nurse monitors the patient's neurological status and assesses the groin site for bleeding and hematoma formation.

Additional options under study and potentially on the treatment horizon for stroke patients include therapeutic hypothermia. Hyperthermia after a stroke has a direct correlation with additional neuronal death and further neurological deficits. Aggressive control of fever (aggressive normothermia) in the stroke patient may be accomplished using acetaminophen as well as convective and conductive cooling devices. Therapeutic hypothermia has been studied after cardiac arrest and is under study poststroke for neuroprotection. Pharmacological neuroprotective agents are being studied with no clear benefit obtained at this stage.[32,34]

Intracerebral Hemorrhagic Stroke

Intracerebral hemorrhagic (ICH) stroke is a potentially devastating consequence of cerebrovascular disease. It may occur, as noted earlier, consequent to cerebral aneurysm or AVM rupture. It may also occur consequent to prolonged hemodynamic stress within arterial vessels in the brain parenchyma. With vessel rupture, blood, under arterial pressure, flows from a high-pressure arterial system into the low-pressure system of the intracranial space and brain parenchyma. Expanding hematoma formation acts as a space-occupying lesion, displacing internal structures and promoting brain edema as an additional mechanism of injury. Additionally, if hemorrhage extends to the ventricles or causes obstruction of CSF pathways, obstructive or communicating hydrocephalus may result. Figure 35-6, a CT scan of the brain, shows a severe ICH stroke with resulting hematoma formation and progressive displacement of intracranial structures.

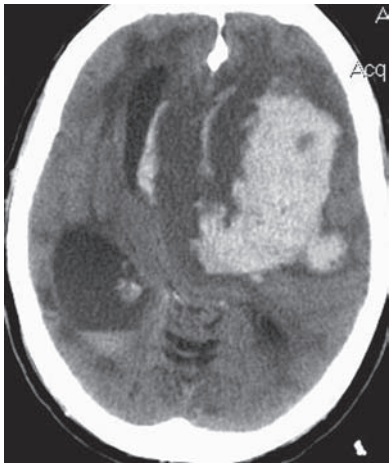


FIGURE 35-6 ▲ Large hypertensive hemorrhagic stroke. CT of head illustrating expanding hematoma with mass effect, brain edema, displacement of brain structures with midline shift and hemorrhage extending into the ventricular system. (Courtesy of Richard Arbour, MSN, RN, FAAN, Albert Einstein Healthcare Network, Philadelphia, PA.)

Intracerebral hemorrhage accounts for 10% to 15% of all strokes. The 30-day mortality rate for hemorrhagic stroke is 40% to 80% with approximately 50% of all deaths occurring within the first 48 hours. In addition to aneurysmal and AVM hemorrhage, ICH may be caused by coagulopathies, vasculitis, and abuse of cocaine or other sympathomimetic drugs.[40] Clinical assessment findings are determined by location of hemorrhage and affected vessel watershed within the brain as well as degree and rate of ICP elevation.[40] Diagnostics include clinical neurological examination and CT of the brain. Based on clinical state and LOC, airway control and ICU admission may be necessary for aggressive, mechanism-based care.[40]

Aggressive, mechanism-based care for managing ICH may include airway control and mechanical ventilation to prevent hypercarbia. Controlled ventilation may also be utilized for short-term hyperventilation ($PaCO_2$ 25 to 30 mm Hg) to modulate CBF in the short term only while other therapies for ICP control are being maximized. Osmotic diuresis with agents, such as mannitol or hypertonic saline solution, may be utilized to draw water from edematous brain tissue. Ventricular drainage may be utilized to remove CSF and decrease ICP as well as provide an ICP monitoring device. Metabolic suppression using drugs such as sedatives/analgesics, propofol or barbiturates decreases brain metabolism, blood flow and ICP in a dose-related manner.[40] Surgical intervention, as clinically appropriate, for clot removal as well as aggressive BP control may be utilized.[41,42] Aggressive BP control may slow hematoma expansion and preserve neurological function.[42]

Nursing Management

ASSESSMENT. A thorough neurological assessment is essential to identify deficits the patient is experiencing. As previously discussed, the NIHSS is a valuable tool that can be used in the emergency department to rate severity of the stroke and determine whether the patient is a candidate for t-PA (see Table 35-9). The brevity and reliability of the tool make it ideal for use in the emergency department. The NIHSS is also helpful for making subsequent assessments and should be performed in conjunction with the neurological examination.

As a member of a large multidisciplinary team, the nurse must be prepared to assume a critical role to assist with the administration of thrombolytic therapy, optimize acute patient care, and move the patient to rehabilitation quickly to maximize the patient's opportunity for an improved outcome. The nurse is in the unique position to identify problems and collaborate with the physician to initiate appropriate referrals to rehabilitation medicine specialists, social workers, speech-language pathologists, and dietitians. Because of the nature of the patient's problems, the multidisciplinary approach provides comprehensive care by addressing all needs.

In addition, the nurse must carefully monitor the patient for infection, changes in temperature, and changes in glucose level, all of which have potentially deleterious effects in people who have had a stroke. Hyperglycemia in acute stroke patients increases cerebral infarction size and worsens neurologic outcomes with and without preexisting diabetes mellitus. In a critical care unit, the upper limit of glycemic control should be 110 mg/dL. Strict glycemic control in the intensive care setting may be achieved with a continuous insulin infusion or sliding-scale regimen. Sliding-scale insulin regimens typically measure point-of-care blood glucose levels at intervals between 4 and 6 hours. Depending on the blood glucose level obtained, a specific insulin dose is ordered. For example, blood glucose between 150 and 200 mg/dL may be treated with 8 units of regular insulin. Correspondingly, a blood glucose level between 200 and 250 mg/dL may be treated with 14 units of regular insulin. The insulin dosing is increased or decreased based on patient response.

PLAN. The nurse plays a significant role in preventing complications associated with immobility, hemiparesis, or any neurological deficit produced by a stroke. Preventive measures are particularly important in the areas of urinary tract infections, aspiration, pressure ulcers, contractures, and thrombophlebitis. Patients in critical care units are at risk for DVT and its resultant complications. Mechanical prophylactic measures for DVT prevention include range-of-motion exercises, antiembolism stockings, and pneumatic compression devices. Additionally, pharmacological measures such as unfractionated heparin, low-molecular-weight heparin, or warfarin may be ordered to prevent blood coagulability. Effective interventions for treating acute stroke help lower the mortality rate and reduce the morbidity of patients who have had a stroke. The Collaborative Care Guide (Box 35-3)

BOX 35–3 *COLLABORATIVE CARE GUIDE for the Patient Who Has Experienced a Stroke*

Outcomes	Interventions
Oxygenation/Ventilation	
Adequate airway is maintained. Oxygen saturation (SpO_2) is maintained within normal limits. Atelectasis is prevented.	• Monitor breath sounds every shift. • Check oxygen saturation every shift. • Instruct to cough and deep-breathe and use incentive spirometry every 2 h while awake. • Assist with removal of airway secretions as needed.
Circulation/Perfusion	
Patient is free of dysrhythmias.	• Monitor vital signs closely. • Manage BP carefully; avoid sharp drops in BP that could result in hypotension and cause an ischemic event secondary to hypotension. • During cardiac monitoring, identify dysrhythmias. • Treat dysrhythmias to maintain adequate perfusion pressure and reduce chance of neurological impairment.

(*continued on page 788*)

BOX 35-3 COLLABORATIVE CARE GUIDE for the Patient Who Has Experienced a Stroke (continued)

Neurological	
Adequate perfusion pressure is maintained.	• Obtain vital signs and perform a neurological assessment to establish a baseline and to monitor for the development of additional deficits. • Use the NIHSS for detection of early changes suggesting cerebral edema or extension of stroke. • Position head of bed at 30 degrees to promote venous drainage.
Effective communication is established.	• Assess ability to speak and to follow simple commands. • Arrange for consultation with speech-language pathologist to differentiate language disturbances. • Use communication aids such as picture cards, pantomime, erase board, or computer to enhance communication. • Provide a calm, unrushed environment. Listen attentively to the patient. Speak in a normal tone.
Fluids/Electrolytes	
Electrolytes are within normal limits.	• Monitor laboratory results, especially glucose. • Monitor intake and output.
Mobility/Safety	
Safety is maintained. Complications of immobility are avoided.	• Initiate DVT precautions to include TED hose, sequential compressive devices, and subcutaneous heparin, as ordered. • Perform fall risk assessment. • Consult with physical therapy. • Provide active or passive range-of-motion exercises to all extremities every shift. • Establish splinting routine for affected limbs. • Instruct in use of mobility aids and fall prevention strategies. • For visual field cuts, teach scanning techniques.
Skin Integrity	
Skin is intact.	• Perform skin assessment using the Braden scale. • Provide pressure relief mattress as indicated by Braden scale. • Turn and reposition patient every 2 h. • Consult with wound nurse specialist for skin issues and concerns.
Nutrition	
Patient has adequate caloric intake and does not experience decrease in weight from baseline. Patient is free from aspiration.	• Obtain admission weight. • Perform cranial nerve assessment (including ability to swallow) to identify deficits. • Obtain consultation from speech-language pathologist to determine whether patient is safe to eat orally. • Provide proper diet and assist with feeding as needed. • Monitor calorie intake; implement calorie count, if necessary. • Obtain dietary consultation to obtain recommendation for nutritional supplements.
Psychosocial	
Support network is established.	• Use picture boards or aids to facilitate communication. • Assess for family support systems. • Screen for poststroke depression.
Teaching/Discharge Planning	
Risk factors are modified. Secondary preventive measures have been taken.	• Provide education about BP management. • Provide dietary instructions.

delineates the specific outcomes and interventions for the patient who has had a stroke.

EMOTIONAL AND BEHAVIORAL MODIFICATION. Patients who have experienced a stroke may display emotional problems, and their behavior may be different from baseline. Emotions may be labile; for example, the patient may cry one moment and laugh the next, without explanation or control. Tolerance to stress may also be reduced. A minor stressor in the prestroke state may be perceived as a major problem after the stroke. Families may not understand the behavior. Patients may show frustration or agitation with the nursing staff or their family members.

It is the nurse's role to help the family understand these behavioral changes. The nurse can help modify the patient's behavior by controlling stimuli in the environment, providing rest periods throughout the day to prevent fatigue, giving positive feedback, and providing repetition when the patient is trying to relearn a skill.

COMMUNICATION. Patients can demonstrate much frustration with their deficits. Probably no deficit produces more frustration for the patient and those trying to communicate with him or her than the one involving the production and understanding of language. Dysphasia can involve motor abilities, sensory function, or both. If the area of brain injury is in or near the left Broca's area, the memory of motor patterns of speech is affected. This results in an expressive or nonfluent dysphasia, in which the patient understands language but is unable to use it appropriately.

Receptive or fluent dysphasia usually is a result of injury to the left Wernicke's area, which is the control center for recognition of spoken language. The patient therefore is unable to understand the meaning of the spoken word (and usually the written word). Having both expressive and receptive dysphasia is referred to as global dysphasia. Box 35-4 summarizes differences between expressive and receptive problems.

It is important for the nursing staff to inform families that having dysphasia does not mean that a person is intellectually impaired. Communication at some level should be attempted, whether it is by writing, using picture boards, or gesturing.

Patient Education and Discharge Planning

Education provides information to patients about modifying risk factors and teaches people to recognize the signs and symptoms of a stroke. Information can be presented regarding medication and other lifestyle modifications to manage BP. Patients can be referred to smoking cessation programs. Teaching about glucose control, weight management, and exercise programs is also valuable. Compliance with medication regimens should be stressed as well.

Hospitals need to organize community outreach programs regarding stroke prevention, the recognition of signs and symptoms of a stroke, its emergent nature, and the need to contact 911 at the onset of symptoms. There must be public awareness about the signs and symptoms, such as sudden onset of numbness or weakness of the face, arm, or leg; confusion; trouble speaking or understanding; vision problems; dizziness; loss of balance; or severe headache. The urgency of immediate attention must be stressed. Emergency medical personnel must be able to identify the symptoms of a stroke and mobilize the patient to the nearest hospital with a full complement of stroke services from diagnosis to discharge.

In addition, a stroke is often a life-altering experience for the patient and family. Depending on the outcome, family members may require education about how to provide care for the patient at home. Instruction about mobility, nutrition, safety, sleep, and eliminative care must occur, along with referrals for home care, if appropriate. With support, the patient will be able to achieve maximal quality of life and reintegrate into the community.

Seizures

A seizure is an episode of abnormal and excessive discharge of cerebral neurons. It can result in altered sensory, motor, or behavioral activities and can be associated with changes in the LOC. Specific symptoms depend on the location of the discharge in the brain. The most common sites of seizure origin are the frontal and temporal lobes, particularly the hippocampus in the medial temporal lobe.[43] Some

BOX 35-4 Comparison of Expressive and Receptive Dysphasia

Expressive Dysphasia	Receptive Dysphasia
Hemiparesis is present because motor cortex is near Broca's area.	Hemiparesis is mild or absent because lesion is not near motor cortex.
	Hemianopsia or quadrantanopsia may be present.
Speech is slow, nonfluent; articulation is poor; speaking requires much effort. Total speech is reduced in quantity. Patient may use telegraphic speech, omitting small words.	Speech is fluent; articulation and rhythm are normal. Content of speech is impaired; wrong words are used.
Patient understands written and verbal speech.	Patient does not understand written and verbal speech.
Patient writes dysphasically.	Content of writing is abnormal. Penmanship may be good.
Patient may be able to repeat single words with effort. Phrase repetition is poor.	Repetition is poor.
Object naming is often poor, but it may be better than attempts to use spontaneous speech.	Object naming is poor.
Patient is aware of deficit, often experiencing frustration and depression.	Patient is often unaware of deficit.
Curses or other ejaculatory speech may be well articulated and automatic. Patient may be able to hum normally.	Patient may use wrong words and sounds.

seizures are so mild that only the patient is aware of them. Others are quite severe. The actual period of the seizure (the ictal period) may be followed by a postictal phase of lethargy and disorientation, which varies with the severity of the seizure.

Epilepsy is a condition in which seizures are spontaneous and recurrent. Status epilepticus is defined as a condition of either continued seizure activity or repetitive seizures without interictal recovery, over a period exceeding 30 minutes. Status epilepticus can be associated with tonic-clonic, complex–partial, or absence seizures. It is a neurological emergency and requires immediate treatment.[43–45]

Psychogenic nonepileptic seizures, which emulate epileptic seizures, are events that involve either motor activity or physical collapse.[44] Pseudoseizures can often be clinically differentiated from epilepsy because they may involve asymmetrical motor activity, side-to-side head movements, and purposeful activity. They also may be gradual in onset. The motor activity can last for many minutes, unlike epilepsy. There is usually a brief or no "postictal" phase. Patients, who are likely to have either emotional or psychological disorders, may require antidepressants, counseling, and psychiatric intervention. In about 20% of cases, patients also suffer from true epilepsy. Childhood abuse is not uncommon in these patients, although the episodes are usually signs of abnormal coping and can have many causes.

Etiology

Many generalized seizures may have a genetic basis and are termed "idiopathic" or primary seizures. They have no specific underlying cause. Symptomatic or secondary seizures have a known cause.[43–45]

Idiopathic seizures account for about 50% of all epileptic seizures. They occur most often in children younger than 10 years. Congenital and genetic causes of epilepsy are seen in approximately 10% of the population. Although inherited epilepsy is more often idiopathic, it is also associated with other conditions.[45]

Symptomatic seizures have numerous causes, including vascular disease, alcohol, cerebral tumors, trauma, infection or fever, metabolic disturbances, anoxia, and degenerative diseases. Developmental abnormalities, such as cortical dysgenesis (abnormal development of the cerebral cortex), are common causes of childhood-onset epilepsy.[43–45]

A number of other variables affect seizure frequency and intensity. Fatigue and sleep deprivation may lower the seizure threshold.[46] Emotional and physical stresses correlate with seizure onset but are difficult to quantify. Many women who keep records of seizure activity discover that they are cyclic, occurring more frequently or increasing in severity during menstruation, pregnancy, or menopause. Alcohol and drug abuse as well as electrolyte disturbances cause seizures and may provoke epileptic seizures.[43,45] Many medications have the effect of lowering seizure threshold, although most patients with controlled epilepsy on medications are not affected. Patients keeping a diary of seizure activity, warning signs, and any precipitating factors, such as sleep deprivation or emotional upset, may potentially lead to effective preventive treatment.[46]

Common causes of new-onset status epilepticus are cerebrovascular disease, brain tumors, intracranial infections, fevers, head trauma, and metabolic disorders. Status epilepticus is also associated with drug withdrawal, metabolic disturbances, or concurrent illness in patients with known epilepsy.[43,45]

Epidemiology

Approximately 1% to 3% of the population has a diagnosis of epilepsy.[45] Infants and young children are most likely to experience seizures, followed by elderly people.[43,45] Populations in developing countries are at greater risk for seizures or epilepsy, probably because of compromised hygiene, poor nutrition, increased risk for infection, and the high percentage of children. Single seizures are much less common than epileptic seizures (epilepsy or seizure disorder) (20 per 100,000 versus 50 per 100,000, respectively). Five percent of patients who develop epilepsy present with status epilepticus. Of epileptic seizures, partial seizures account for approximately 57%, and generalized seizures account for 40%. Medical treatment completely controls seizures in approximately 70% of patients.

Pathophysiology

Nerve cells (neurons) in the brain possess an electrical charge that reflects a balance between intracellular and extracellular charged ions. The electrical activity of the neuronal membrane is determined by the flow of ions between these spaces. Ions, such as sodium (Na^+), potassium (K^+), calcium (Ca^{2+}), and chloride (Cl^-), are regulated by receptor channels and flow across the membrane when receptors are activated by voltage changes and neurotransmitter modulation. If the permeability of the cells is altered, their excitability can change, making the neuron more likely to discharge. Hyperexcitability can result in increased random neuronal firing. When this is combined with a certain pattern of neuronal firing (synchronization), epileptogenic properties in the neuron exist.[43,44]

Although it is not known exactly how the mechanisms of abnormal neuronal excitation and synchronization lead to epileptiform activity, continued exploration of cell membrane activity, environmental variables, and pharmacological responses has led to increased understanding and management of epilepsy.

Clinical Manifestations

Clinical features of epilepsy are based on the location of the epileptiform discharge and the type of event.[43–45] Box 35-5 describes specific seizures and resulting clinical characteristics.

Diagnosis

The patient who presents with a seizure is initially evaluated to ascertain the cause of the seizure and establish a diagnosis of epilepsy.[43–45] History taking begins with a description of the event by the patient or witnesses. This description should include the following:

1. What the patient was doing at the time of the seizure
2. Duration of the episode
3. Unusual symptoms or behaviors before the seizure

BOX 35-5 Classification of Seizure Types

1. **Generalized:** involves both hemispheres; loss of consciousness; no local onset in the cerebrum
   a. *Tonic-clonic* (grand-mal)—loss of consciousness; stiffening; forced expiration (cry); rhythmic jerking
   b. *Clonic*—symmetrical, bilateral semirhythmic jerking
   c. *Tonic*—sudden increased tone and forced expiration
   d. *Myoclonic*—sudden, brief body jerks
   e. *Atonic* ("drop attacks")—sudden loss of tone; falls
   f. *Absence* (petit mal)—brief staring, usually without motor involvement
2. **Partial:** involves one hemisphere
   a. *Simple partial seizure*—no change in LOC, jacksonian
      i. Motor—frontal lobe
      ii. Somatosensory—parietal lobe
      iii. Visual—occipital lobe
      iv. May involve: autonomic (eg, respiratory changes, tachycardia, flushing); psychic (eg, déjà vu); cognitive (without change in LOC)
   b. *Complex partial seizures*—altered LOC; with or without automatisms: lip smacking, swallowing, aimless walking, verbalizations
      i. Simple partial followed by change in LOC
      ii. Starts with change in consciousness
      iii. Typically of temporal lobe origin
   c. *Partial with secondary generalization*
      i. Simple partial→ generalization
      ii. Complex partial → generalization
      iii. Simple partial → complex partial → generalization
      iv. Continuous EEG monitoring may be necessary to differentiate from generalized seizures
3. **Unclassified**

4. Specific features, including movements, sensations, sounds, tastes, smells, and incontinence
5. LOC during and after the event
6. Duration and description of symptoms after the seizure
7. Reporting of any similar previous episodes and age of onset

Inquiry should be made about the following:

1. Sleep patterns
2. Alcohol or drug abuse
3. History of illnesses or injuries
4. Family seizure history
5. Other possible variables: menstrual cycle, stress, fevers, metabolic disorders
6. If other seizures have occurred, similarities in symptoms, duration, frequency, and time of day

After the first seizure occurrence, a CT scan or MRI is obtained to assess for a structural lesion. An EEG is obtained to screen for interictal seizure discharges (electrical abnormalities present in between seizures) and to measure cerebral excitability. These techniques can help determine whether the seizures have focal origins or are more generalized. During the EEG, scalp electrodes are placed to measure neuronal membrane activity in the underlying cerebral cortex. Rhythms may be obtained when the patient is both awake and asleep. The EEG localizes the region from which the patient's seizure arises at one time point.[43–45]

If additional information regarding seizure patterns and characteristics is needed, an inpatient hospital stay may be recommended in an epilepsy monitoring unit, as discussed in the following section. Also, continuous EEG monitoring is now being used in the ICU, where it has helped identify subtle seizures in critically ill patients. Because it is not uncommon for these patients (eg, brain injured or comatose patients) to suffer nonconvulsive seizures, use of EEG is becoming more widespread in the ICU.[45,47]

Other diagnostic studies that may be obtained include PET to identify abnormal hypometabolic cortical areas that correlate with the epileptogenic area; SPECT to identify CBF and perfusion differences during and after a seizure; bilateral carotid amobarbital (Amytal) testing to assess speech dominance and memory; fMRI to localize seizure focus and identify its relationship to a structural lesion; and neurocognitive testing to obtain a baseline evaluation.[43,45]

Epilepsy Monitoring Unit

Patients who require more detailed seizure characterization or localization are admitted for video EEG monitoring to an appropriate unit, where scalp electrodes are placed. Video EEG monitoring is continuous and involves audiovisual observations. Monitoring occurs while the patient is awake and asleep. Medication dosages may be slowly reduced or withdrawn during these observations. Because video EEG monitoring captures ictal, postictal, and interictal data, seizures are documented and localized, and the patient's clinical symptoms are observed. Video EEG can often localize seizures for possible epilepsy surgery. It is also very helpful for identifying pseudoseizures and other disorders that are mistaken for epileptic disorders. Neuropsychological and psychiatric evaluations may be part of the assessment, particularly if surgery is being anticipated.[45]

In situations in which localization is not obtained or is questionable, a more invasive approach using surgical electrodes to localize seizure activity may be necessary. Three different types of electrodes can be implanted for intracranial recording.

Depth electrodes are typically placed bilaterally, targeting the hippocampus and other common seizure sites in the amygdala and frontal lobes. Multiple electrodes are placed under local or general anesthesia through twist drill or burr holes for simultaneous recording. The electrode cables exit the skull, and patients have continuous video monitoring over several days in the epilepsy monitoring unit. Hemorrhage, headache, and infection are possible complications. This procedure is most often used to show which regions are involved at seizure onset.[45]

Subdural and epidural electrodes are typically placed unilaterally, under general anesthesia. Strips are placed through burr holes. Grids require a craniotomy and allow for a larger region to be monitored (Fig. 35-7). They are secured to the dura, and the electrode leads exit through an incision for continuous monitoring. Infection, hemorrhage, and mass effect from cerebral edema are possible risks. The bone flap can be replaced at the end of the procedure or after the strips or grids are removed. Intracranial monitoring may be initiated before seizure surgery.[45]

Clinical and Pharmacological Management

In most cases, the patient with epilepsy can be medically treated. Some AEDs are known to be more appropriate for specific classes of seizures. Certain drugs are found most useful in the pediatric patient population. Some AEDs are preferred as monotherapy, and others are more appropriate as adjunctive therapy with

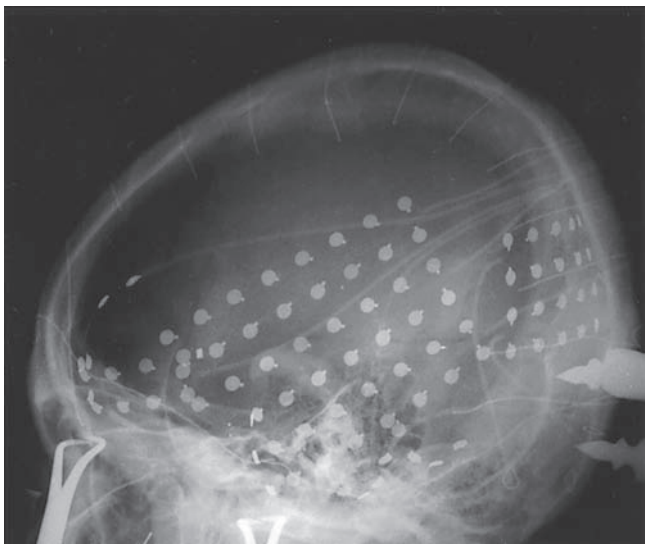


FIGURE 35-7 ▲ Radiograph of a grid for seizure monitoring. (Courtesy of Frederick Lenz, MD, and Ira Garonzik, MD, The Johns Hopkins University, Baltimore, MD.)

other drugs.[43,45] Table 35-10 describes current AEDs with general indications, doses, side effects, and nursing considerations.

Status epilepticus is an emergency situation and requires rapid pharmacological management. Parenteral drugs are given to provide fast absorption. Drugs are given intravenously or intramuscularly. In emergent circumstances benzodiazepines such as diazepam (Valium) may be administered rectally in an emergency in the absence of intravenous access.[48] Many fast-acting drugs are lipid soluble and have a tendency to redistribute from the plasma to the fat and muscle, thereby leading to an initial drop in blood and brain concentrations. This may lead to seizure recurrences. Repeat boluses or a continuous infusion must be administered judiciously because the drugs saturate fat, and muscle and plasma levels will increase. This may result in prolonged decreased mental status, obtundation, and even death. In-hospital mortality of status epilepticus overall is low but may increase significantly in patients with controlled ventilation and other comorbidities such as increased age and hypoxic/ischemic brain injury.[49] Emergency management of status epilepticus is summarized in Box 35-6.

Surgical Management

There are situations in which AEDs are unable to control epilepsy. Attempts at monotherapy and adjunctive therapy have been exhausted and multiple drug regimens have failed; seizure activity has compromised a patient's quality of life. In these cases, seizures are intractable, and surgery is considered to obtain seizure control. It may also be considered in situations in which the side effects of therapy are so debilitating that the patient is unable to function at an acceptable capacity.[43,45]

In many cases, the patient is monitored before surgery in an epilepsy monitoring unit using a video EEG. Surgery for grid or strip placement to localize seizure focus and identify functional areas is often performed. After surgery, the nurse

Table 35-10 Drug Therapy: Antiepileptic Drugs

Drug Name (Brand)	Indications	Daily Adult Dose	Side Effects	Nursing Considerations
Carbamazepine (Tegretol, Tegretol XR, Carbatrol)	Mono/adjunctive therapy Partial and generalized seizures	400–2,000 mg; serum levels of 4–12 mcg/mL	Drowsiness, fatigue, dizziness, blurred vision, rash, hyponatremia, bone marrow dyscrasia	Interactions with other AEDs; rare aplastic anemia, hepatic failure; obtain blood levels
Gabapentin (Neurontin)	Adjunctive Partial and secondary generalized seizures	900–4,800 mg	Sedation, dizziness, weight gain	No significant drug interactions; minimal side effects
Levetiracetam (Keppra)	Adjunctive therapy for partial seizures	1,000–3,000 mg	Somnolence, asthenia, irritability, dizziness, infection	Can be administered IV
Phenobarbital	Mono/adjunctive therapy Partial or generalized (myoclonus/absence)	90–180 mg	Sedation, depression, ataxia, rash, impotence, hyperactivity	Potential CNS toxicity, especially in children; taper slowly
Phenytoin (Dilantin)	Mono/adjunctive therapy Partial and generalized (not absence/myoclonus)	300–600 mg; serum levels of 10–20 mg/L	Ataxia, dizziness, sedation, rash, gingival hyperplasia	Interactions with multiple other drugs; dose guided by blood levels; less expensive
Fosphenytoin	Replacing IV phenytoin	Loading dose 15–20 mg IV or IM; administered 100–150 mg/min		
Valproate (Depakote)	Mono/adjunctive therapy Generalized seizures; childhood epilepsy; febrile convulsions	1,000–3,000 mg	Nausea, weight gain, endocrine, thrombocytopenia, hair loss	Interactions with other AEDs; can be administered IV

BOX 35-6 Emergency Treatment of Status Epilepticus

- **Goals:** maintain airway, breathing, circulation; stop seizures; stabilize patient; identify and treat cause
- **Treatment:** airway; O_2; intubation, if necessary; EEG monitoring; monitor ECG and BP; catheter for incontinence; CT scan; lumbar puncture if CNS infection is suspected; CPR if needed
- **Blood work:** electrolytes; magnesium; calcium; anticonvulsant levels; blood gases; complete blood count; renal and liver function studies; coagulation studies; toxicology studies may be needed
- **Medications:**
  - Benzodiazepines (lorazepam, 1 to 2 mg/min as a starting dose over 8 minutes or diazepam to total of 20 mg). These are short-acting drugs, and simultaneous loading with phenytoin at 50 mg/min or fosphenytoin at equivalent of 150 mg phenytoin/min is necessary; may total 20 mg/kg
  - For persistent seizures, add 5 to 10 mg/kg phenytoin *or* phenobarbital at 50 to 100 mg/min to total of 20 mg/kg
  - If ineffective, barbiturate anesthesia: pentobarbital and intubation. Benzodiazepines (eg, midazolam) may be attempted prior to barbiturate anesthesia. Continuous EEG monitoring in ICU environment.

performs regular, intermittent neurological examinations and has the patient attempt to perform certain tasks. Language deficits and motor weakness are observed. The goals of this procedure are to localize epileptiform discharges in relation to speech, memory, and sensory or motor function. It is also useful in identifying the relationship between seizure discharges and a focal lesion, such as a tumor, when present. This enhances the safety and accuracy of tumor surgery.[45]

The decision to proceed with surgery depends on a thorough discussion among the multidisciplinary team members. The neurologist, neurosurgeon, patient, and family review the medical treatment used to date and establish that therapy has been maximized. They evaluate the likelihood of seizure control with surgery. Preoperative neuropsychological testing is obtained, and other appropriate testing is established and discussed. Some or all of the diagnostic studies previously outlined may be indicated.

The goal of seizure surgery is to remove or disrupt the seizure focus. Patients are often maintained on AEDs for 2 years postoperatively because seizure recurrence is most likely during this period. Table 35-11 summarizes the most commonly performed surgical procedures, along with expected outcomes, possible complications, and nursing considerations.

Nursing Management

ASSESSMENT. Careful history taking is a central component of the accurate diagnosis and management of epilepsy. Family history, age at onset, frequency, and a description of symptoms and their duration all aid in the development of a plan of care tailored to the patient's particular situation. AEDs are prescribed based on all these data. Changes in severity or frequency of symptoms require modification of the treatment regimen. Drug therapy may last indefinitely; therefore, treatment should address both efficacy and tolerability. Side effects can compromise quality of life, necessitating the use of different, and possibly less effective, medications or multiple AEDs.[45]

PLAN. Inpatient nursing care includes monitoring the patient during the seizure (the patient is never left alone) and providing support and protection without attempting to restrain the individual. Turning the patient to his or her side during a generalized seizure, if possible, helps maintain a patent airway.

Patient Education and Discharge Planning

Patient education should provide instructions for independent functioning. The following patient education points are critical parts of discharge planning:

1. Make the home environment safe, particularly in the case of tonic-clonic epilepsy.
2. Assess for injury after each seizure.
3. Keep a log to record a description of the seizure and postictal period, duration, time of day, severity, and any new characteristics.
4. Know the specifics of a ketogenic (high-fat, low-carbohydrate) diet, when appropriate, for children with intractable seizures.
5. Be aware of state laws on driving restrictions related to epilepsy.
6. Wear a medical identification bracelet.
7. Monitor serum AED levels when appropriate.
8. Be aware of circumstances when emergency treatment is required.
9. Consult seizure experts for intractable epilepsy.

Guillain-Barré Syndrome

Guillain-Barré syndrome, also known as acute inflammatory demyelinating polyneuropathy, is a rapidly evolving illness that commonly presents as symmetrical weakness, sensory loss, and areflexia. This condition is an inflammatory peripheral neuropathy in which lymphocytes and macrophages strip myelin from axons. The diffuse inflammatory reaction may be seen in the peripheral nervous system, cranial nerves, and spinal nerve roots. It is referred to as a syndrome, as opposed to a disease, because of the combination of signs and symptoms seen in the patient.[50,51]

Etiology

Guillain-Barré syndrome is an immune-mediated neuropathy that is associated with a broad range of symptoms, severity, and length of progression. This disorder follows an antecedent infection in some patients.[50–53] Approximately half of the people in whom Guillain-Barré syndrome develops have a mild febrile illness 2 to 3 weeks before the onset of symptoms. The febrile infection is usually respiratory or GI. *Campylobacter jejuni* and cytomegalovirus are causes of the most frequent antecedent infections, which usually occur 1 to 4 weeks before the onset of symptoms of Guillain-Barré syndrome.[50–53] Some studies show a potential association between vaccinations and increased risk of Guillain-Barré syndrome.[51,52] Although previously administered vaccinations, including H1N1 and rabies vaccines, have been linked to the development of Guillain-Barré syndrome,[51,52] a strong causal relationship has not been demonstrated. Currently, no recent vaccine has been conclusively demonstrated to induce the disease.

The attack on the immune system is extensive and occurs proximally at the nerve roots and distally at the motor axon terminal. Both cellular and humoral immune mechanisms appear to be implicated. Lymphocytes and macrophages are the effector

Table 35-11 Clinical Applications of Seizure Surgery

Procedure	Indications	Expected Outcomes	Possible Complications	Nursing Considerations
Temporal lobectomy: removal of 6 cm of temporal lobe in the nondominant hemisphere and 4–5 cm in the dominant hemisphere	Intractable anterior temporal lobe seizures >5 y' duration Significant quality-of-life compromise	60%–70% seizure free 20% greatly improved seizure control	Visual (superior quadrantanopsia) field defects Dysphasia (usually temporary) Mild memory problems Depression Transient psychiatric disturbance Infection and/or bleeding	At 1 y postoperatively, it is expected that seizure status will not change Medication management continues for 2–3 y postoperatively
Hemispherectomy: surgical removal (or disconnection) of a hemisphere in children and adolescents	Severe seizures Often multiple seizure types and daily seizures	90%–95% improvement in seizure activity 70%–85% resolution of seizures	Contralateral neurological deficits Late hydrocephalus Chronic bleeding into surgical cavity would produce increased morbidity re: hematoma formation, infection risk, ICP elevation. → neurological disability and death	Careful patient selection required May see improved behavior and social development
Corpus callosectomy: transection of the corpus callosum (or anterior two thirds)	Cases of severe secondarily generalized epilepsy; drop attacks	Reduces number of generalized seizures Seizure-free periods usually temporary and occur in only 5%–10% of patients	Hemiparesis Transient syndrome of mutism, urinary incontinence, and bilateral leg weakness	Used when other options have failed Many patients have learning disabilities Wada test recommended for left-handed patients
Vagal nerve stimulator: implanted programmable signal generator in the chest with stimulating electrodes to the left vagus nerve	Refractory to medication Often partial seizures	Reduction in seizure frequency: high stimulation 25%; low stimulation 15%	Changes in voice Dyspnea Tingling in neck during stimulation Rare cases of bradycardia or asystole	Does not generally resolve seizures Used when resective surgery is not an option
Deep brain stimulator: Electrodes placed in deep brain structures (thalamus, hippocampus, internal capsule) and programmed to activate when seizure activity is recorded	Uncontrolled epilepsy	Reduction in seizures	Bleeding Infection Neurological deficits	Has been used for tremor in Parkinson's disease Relatively new use in refractory seizures with unknown long-term outcomes

cells that result in damage to myelin and adjacent axons. Motor, sensory, and autonomic nerves are involved. Weakness and sensory disturbances result from blockage of nerve fiber action potentials (secondary to demyelination or axon damage).[50–53]

In affected patients, the immune system most likely is first primed as it responds to a virus or bacteria. Then, the immune system inappropriately attacks host tissue that shares an epitope (surface portion of an antigen capable of triggering an immune response). This process is referred to as molecular mimicry.[51,52]

Epidemiology

Guillain-Barré syndrome occurs with equal frequency in both sexes and all races. It can develop at any age. The annual incidence is approximately 1 to 3 cases per 100,000 population in the United States.[50]

Pathophysiology

In Guillain-Barré syndrome, the myelin sheath surrounding the axon is lost. The myelin sheath is quite susceptible to injury by many agents and conditions, including physical trauma, hypoxemia, toxic chemicals, vascular insufficiency, and immunological reactions. Demyelination is a common response of neural tissue to any of these adverse conditions.[50–53]

Myelinated axons conduct nerve impulses more rapidly than nonmyelinated axons. Along the course of a myelinated fiber are interruptions in the sheath (nodes of Ranvier), where there is direct contact between the cell membrane of the axon and the extracellular fluid. The membrane is highly permeable at these nodes, resulting in especially good conduction. The movement of ions into and out of the axon

can occur rapidly only at the nodes of Ranvier; therefore, a nerve impulse along a myelinated fiber may jump from node to node (known as saltatory conduction) quite rapidly. Loss of the myelin sheath makes saltatory conduction impossible, and nerve impulse transmission is aborted.[51–53]

A current theory regarding the disease process of Guillain-Barré syndrome speculates that a primary lymphocytic T-cell mechanism is the cause of the inflammation. Cells migrate through the vessel walls to the peripheral nerve. The result is edema and perivascular inflammation. Macrophages then break down the myelin.[51] A potential secondary process is that demyelination is initiated by an antibody attack on the myelin early in the course of the disease. Demyelination causes axon atrophy, which results in slowed or blocked nerve conduction.[52]

Clinical Manifestations

Guillain-Barré syndrome may develop rapidly over the course of hours or days, or it may take up to 3 to 4 weeks to develop. Most patients demonstrate the greatest weakness in the first weeks of the disorder and are weakest by the third week of the illness.[50–53]

In the beginning, a flaccid, ascending paralysis develops quickly. The patient is most commonly affected in a symmetrical pattern. The patient may first notice weakness in the lower extremities that may quickly extend to include weakness and abnormal sensations in the arms. Deep tendon reflexes are usually lost, even in the earliest stages. The trunk and cranial nerves may become involved. Respiratory muscles can become affected, resulting in respiratory compromise.[50–53] Autonomic disturbances such as urinary retention and orthostatic hypotension may also occur. Some patients experience tenderness and pain on deep pressure or movement of some muscles.[50–53]

Sensory symptoms of paresthesias, including numbness and tingling, may occur. Pain is a complaint in many patients. It is aching in nature and often compared with the feeling of muscles that have been overexerted. If there is cranial nerve involvement, cranial nerve VII, the facial nerve, is most often affected. Guillain-Barré syndrome does not affect LOC, pupillary function, or cognitive functioning.[50–53]

Symptoms may progress for several weeks. The level of paralysis may stop at any point. Progression usually occurs in three stages: acute, plateau, and recovery. The acute stage starts at the onset of symptoms and rapidly progresses until no additional deterioration occurs. The plateau stage, during which patients are symptomatic, lasts for a few days up to a few weeks. The recovery phase can take up to 2 years. It is thought that the recovery phase coincides with remyelination and axonal regeneration. Although demyelination occurs rapidly, the rate of remyelination is approximately 1 to 2 mm/d. Motor function returns in a descending fashion.[50–53]

Diagnosis

The diagnosis of Guillain-Barré syndrome depends greatly on the patient's history and clinical progression of symptoms. As noted, onset is usually sudden, and the history often reveals an upper respiratory or GI disorder occurring 1 to 4 weeks before onset of the neurological manifestations. The history of the onset of symptoms can be revealing because symptoms of Guillain-Barré syndrome usually begin with weakness or paresthesias of the lower extremities and ascend in a symmetrical pattern.[50–53]

A lumbar puncture may be performed and reveals increased protein. However, negative results from this test should be interpreted cautiously because only 50% of patients in the first week of illness have increased protein. By 3 weeks, this percentage increases to greater than 90%. Also, nerve conduction studies record impulse transmission along the nerve fiber. In the patient with Guillain-Barré syndrome, the velocity of conduction is reduced.[50–53]

Pulmonary function tests are performed when Guillain-Barré syndrome is suspected to establish a baseline for comparison as the disease progresses. Declining pulmonary function capacity may indicate the need for mechanical ventilation and management in an ICU.[50–53] Severe respiratory failure consequent to Guillian-Barré syndrome requiring mechanical ventilation is a predictor of mortality in the hospitalized patient. Additional predictors of mortality include comorbid states, cardiovascular complications, sepsis, and advanced age.[54]

Clinical Management

Because of the risks associated with respiratory failure, bulbar symptoms, and autonomic dysfunction, all patients with Guillain-Barré syndrome, except those with mild disease, should be admitted to a hospital that has specialized ICUs. Progression to mechanical ventilation is expected in patients with rapid disease progression, bulbar involvement, bilateral facial weakness, or dysautonomia. ICU admission is recommended for patients with vital capacity below 20 mL/kg, vital capacity checks that are required more than every 4 hours, aspiration, autonomic instability, or rapid progression or weakness.[50–53] Patients who are older, have rapid progression or prior GI infection, or are ventilator dependent, tend to have a poor prognosis and need to be monitored closely.[54]

Certain strategies can lessen the severity of the illness and hasten recovery. A useful clinical sign of respiratory compromise is the strength of the neck flexor muscles. When the head cannot be lifted against gravity, the phrenic nerves are also affected, causing diaphragm paralysis and reduction of the forced vital capacity (FVC)—the amount of air a patient can forcefully exhale after maximal inhalation. Under these circumstances, the airway cannot be successfully managed without intubation.

Preventive measures need to be established so that DVT and subsequent PE do not develop. DVT prophylaxis includes subcutaneous heparin, 5,000 units twice daily, along with antiembolism stockings and sequential compression devices. Also, autonomic nervous system fluctuations need to be evaluated by checking BP and monitoring for cardiac dysrhythmias.[51–53]

Plasmapheresis was the first therapy proved to benefit patients with Guillain-Barré syndrome. It is the only therapy that has been proved superior to supportive treatment alone. This procedure mechanically removes humoral factors. Currently, it is recommended that patients with Guillain-Barré syndrome receive plasmapheresis. A dual-lumen central vascular access device and a specially skilled team are needed to perform the plasmapheresis treatments. The physician may order plasmapheresis when the patient's

condition is worsening in an attempt to reduce the severity of the disease.[50–53] Of note, two prominent risks associated with plasmapheresis are catheter-related infections and hemorrhage during catheter placement.

Intravenous immunoglobulin (IVIG) is also useful in managing Guillain-Barré syndrome. A blood product that has been derived from large pools of plasma donors, IVIG has the potential to bind many common pathogens and modulate a wide range of effectors in autoimmune disease, such as Guillain-Barré syndrome. The major component is immunoglobulin G, with a trace amount of immunoglobulin A. Immunoglobulins can be infused easily, even in the home setting, without expensive equipment. The optimal dosages and frequency of administration are individualized. Immunoglobulin, which binds to receptors on T cells or receptors on nerves, induces only a temporary improvement because of the turnover of T cells or the loss of antibodies from the receptors. Daily treatments with IVIG may be helpful in acute Guillain-Barré syndrome when the patient is rapidly deteriorating.[51–53]

The dose of IVIG is set at 2 g/kg, and usually the total dose is divided into five daily infusions of 400 mg/kg each. Neurologists who use IVIG for Guillain-Barré syndrome are familiar with the side effects, which include low-grade fever, chills, myalgia, diaphoresis, fluid overload, hypertension, nausea, vomiting, rash, headaches, aseptic meningitis, and neutropenia.[52,53] The most serious adverse effect is acute tubular necrosis, which occurs with any concomitant disease that compromises renal glomerular filtration.

Currently, there are no efficacy data that favor IVIG rather than plasmapheresis in managing Guillain-Barré syndrome. IVIG and plasma exchange have a similar ability to speed a patient's recovery. The individual patient's circumstances, such as availability of resources to perform plasmapheresis and underlying medical conditions, dictate the specific treatment for each patient. IVIG is an attractive treatment because it can be easily administered in the critical care setting.[52,53]

Nursing Management

ASSESSMENT. For the patient with Guillain-Barré syndrome, careful assessment and the resultant plan help minimize the complications of immobility and move the patient toward rehabilitation without deficits. Although patients are critically ill, their chances of returning to a productive life are good if they survive the acute stages and avoid the complications of immobility. Most deaths are due to preventable respiratory complications or autonomic dysfunction.

Once Guillain-Barré syndrome is suspected, the patient is hospitalized so that frequent assessments can be performed to monitor the patient for deterioration. Because of the progressive nature of the disease, assessment should focus on the neurological examination (ie, cranial nerve involvement, motor weakness, and sensory changes). Cranial nerve deficits identify if the patient is at risk for aspiration. The patient's level of numbness, tingling, and pain should be assessed.[50]

Cardiovascular assessment is done to monitor BP and heart rate. The autonomic nervous system is frequently involved in Guillain-Barré syndrome. Dysautonomia manifests itself as sinus tachycardia but may result in other cardiac dysrhythmias or labile BPs that require close monitoring because they may be life threatening. The patient's respiratory status should be monitored, and FVC should be assessed at least once every shift. GI and urinary function should come under surveillance as well. The patient is at risk for constipation and urinary tract infections resulting from urinary retention. Other complications of immobility are the potential for pressure ulcers and DVT.[50–53]

PLAN. When caring for a patient with Guillain-Barré syndrome, the major goals are to prevent infections and complications of immobility, provide functional maintenance of the body systems, treat life-threatening crises promptly, and provide psychological support for the patient and family. In terms of the patient's neurological status, weakness results in impaired mobility. Range-of-motion exercises should be performed at least once per shift to minimize the patient's risk for contractures.[50] Families are encouraged to help with this beside therapy.

Also, steps are taken to maintain proper body alignment. Measures such as splint placement are implemented to prevent wrist hyperflexion and footdrop.[50,53] Physical therapy is initiated early in the course of hospitalization and continued throughout the recovery period.

Cranial nerve involvement places the patient at risk for aspiration, and adequate nutrition must be maintained. If the patient is unable to take oral feedings, enteral tube feedings are initiated. A nutritional consultation with a registered dietitian should occur to provide adequate calories for remyelination as well as rehabilitation activities. Enteral nutritional support that supplies at least 1,500 to 2,000 kcal/d should be instituted if the patient is unable to receive food by mouth.[50–53]

Additionally, because of intubation or impaired verbal communication, alternate methods to facilitate communication are important. Alternate ways to communicate are established using communication boards as well as nonverbal means such as gestures and eye blinking. Inability to communicate can be frustrating for the patient and cause undue anxiety.

Respiratory failure is the most severe complication of Guillain-Barré syndrome. Weakened respiratory muscles put the patient at great risk for hypoventilation and repeated pulmonary infections. Fifty percent of patients with Guillain-Barré syndrome have some respiratory compromise, resulting in reduced tidal volume and vital capacity or perhaps complete respiratory arrest. A tracheostomy may be indicated if the patient requires long-term mechanical ventilation.[50]

If there is autonomic nervous system involvement, drastic changes in BP (hypotension or hypertension), heart rate, or both can occur. Labile hypertension and dysrhythmias occur frequently, prompting admission and management in the ICU. Cardiac monitoring allows quick identification and treatment of dysrhythmias. Because Valsalva maneuver, coughing, and suctioning may trigger an autonomic nervous system disturbance, the patient needs to be monitored closely.[50–53]

Comfort measures, such as frequent position changes, may be helpful. When remyelination occurs, it is often uncomfortable, and the patient may complain of numbness and pain. This can be an encouraging sign to the patient because the disease process is reversing.

Although the patient is incapacitated physically, he or she is fully aware of the surroundings. The patient may experience

a sense of fear, loss of control, as well as helplessness and hopelessness. Frequent explanations of the interventions and of progress are useful.[50] The patient should be allowed to participate in care as much as functionally possible. It is essential that the nurse in the critical care setting provide empathy, compassion, sensitivity, and active listening to the patient with Guillain-Barré syndrome so that his or her emotional concerns can be addressed. In addition, the nurse can provide positive reinforcement for achievement of physical gains.[50]

Patient Education and Discharge Planning

Education of the patient and family about all issues of care is important. Knowledge is power and is helpful in a situation in which the patient is powerless. The nurse can provide information about the disease process, course, and recovery.[50] Patients need to know that the disease may progress to the point at which mechanical ventilation is required. In addition, they should understand that they may be discharged to a rehabilitation facility where recovery can continue. Many months of rehabilitation may be required for them to regain strength and previous level of functioning. Patients may continue to show improvement for up to 2 years. The nurse can tell patients and their families about the Guillain-Barré Syndrome International Foundation, which provides information and resources. Before discharge to home, the patient can be referred to a support group so that he or she can interact with other people who have had Guillain-Barré syndrome.[50]

Myasthenia Gravis

Myasthenia gravis is an autoimmune disorder of the neuromuscular junction transmission that presents with fatigue and muscle weakness in the ocular, bulbar, diaphragm, or limb muscles. Myasthenia is derived from the Greek words for "muscle" and "weakness," whereas gravis means "grave" in Latin. Because of the high mortality related to diaphragmatic muscle weakness, the disorder was called "grave muscle weakness."[1] However, myasthenia gravis is not a grave disease today because of advancements with immunomodulatory treatments and the ability to manage respiratory failure.

Etiology

Myasthenia gravis is an autoimmune disorder characterized by weakness and fatigability of skeletal muscles. The disease involves a reduction in the number of acetylcholine receptors (AChRs) at the neuromuscular junction caused by antibodies against the AChR.[55–57] The factors that trigger the autoimmune process are not known, but the thymus gland plays an important role. The thymus gland lies behind the sternum and may extend down to the diaphragm and up to the neck. This gland plays a role in the responsiveness of T cells to foreign antigens. The thymus gland is large in children and small in adults. By adulthood, the gland has shrunken and has nearly been replaced by fat. Abnormalities in the thymus gland occur in approximately 75% of patients with myasthenia gravis. Eighty-five percent of patients with myasthenia gravis have thymic hyperplasia, and approximately 15% have tumor (thymoma) of the thymus gland.[55]

Epidemiology

Myasthenia gravis is seen more often in women than in men at a ratio of 3:2. It is primarily a disease of young women and older men. Symptoms most commonly appear in the third decade of life, although any age group may be affected.[55] Myasthenia gravis is uncommon with an estimated annual incidence of 2 cases per 100,000 population.[56] During the past 40 years, prevalence has increased because of improved recognition of the disorder, medical management, and survival. Myasthenia gravis is not hereditary in the Mendelian sense; however, there may be a history of autoimmune disorders in the family, including thyroid disorders or lupus. It should be remembered that approximately 10% of women with myasthenia gravis may transmit a transient type of neonatal myasthenia to the infant that resolves within days after birth.

Pathophysiology

Myasthenia gravis is a result of circulating antibodies directed toward the AChRs in the skeletal muscle.[55–57] The AChR is a protein composed of five subunits situated in a specialized surface of the muscle membrane termed the end plate. When acetylcholine is released from the nerve after depolarization, it binds to the AChR and causes the ion channel to open. This passage of ions moving through the channel leads to depolarization of the end plate, action potential generation, and subsequent muscle fiber contraction. With this process, the depolarization of the end plate is three to four times greater than what is required for action potential generation. Therefore, fluctuations in end plate depolarization do not affect action potential generation or the overall strength of muscle contraction.[56]

The AChR antibodies in myasthenia gravis lead to the loss of AChRs by increased internalization of the receptor and complement-based lysis of the muscle membrane. Compromise of ion flow through AChR occurs, leading to a decrease in end plate depolarization, which may be insufficient to generate an action potential. This results in a failure of the muscle to contract. Often, at rest, the compromise of neuromuscular transmission is mild, and action potentials are still generated. However, with exercise or with repetitive nerve stimulation, the end plate potential is further reduced, the action potential is not generated, and muscle weakness occurs. In summary, antibodies attack the AChRs of the neuromuscular junction, thereby blocking the transmission of nerve impulses to muscle.[55–57]

Clinical Manifestations

Myasthenia gravis can be characterized as ocular myasthenia or generalized myasthenia, depending on whether the symptoms are limited to the eye muscles or are spread elsewhere. In ocular myasthenia, patients may present with problems such as droopy eyelids or double vision. More than 90% of patients have ocular symptoms. In only 16% of the patients, the symptoms remain confined to the eye muscles. In the rest, the symptoms spread to other

muscles (bulbar, limb, diaphragm) within a year of the onset of eye symptoms.[55,56]

In generalized myasthenia gravis, patients may demonstrate ocular manifestations as well as bulbar symptoms, presenting as difficulty with chewing, swallowing, talking, and handling secretions, and neck weakness. The voice may have a nasal quality. Prolonged talking brings on slurred speech. Weakness of jaw closure (masseter muscle) may lead to the jaw hanging open. Muscle weakness in the limbs is also apparent and can vary among patients. Patients often demonstrate more proximal than distal weakness. They often report increased weakness with sustained activity and improvement with rest. Patients may also demonstrate respiratory compromise due to weakness of the respiratory muscles. The most serious potential complication of myasthenia gravis is respiratory failure (myasthenic crisis) secondary to intercostal and diaphragmatic muscle weakness.[55,56] Respiratory failure may result in death. With earlier diagnosis and intervention for patients with myasthenia gravis, in-hospital mortality is low. Respiratory failure consequent to myasthenia gravis as well as advanced age are predictors of increased mortality.[58]

Diagnosis

Similar to other neurological disorders, the patient's history along with other diagnostic tests aids in an accurate diagnosis. Patients may present with complaints of double vision or drooping eyelids. Also, myasthenia gravis causes weakness of the shoulder girdle muscles. Therefore, the patient may complain of the inability to perform a variety of self-care activities, such as drying the hair with a blow dryer.

The neurological examination is also valuable in making the diagnosis. The cranial nerve examination may reveal ptosis and diplopia as well as other cranial nerve involvement. Motor weakness may be exhibited.[55,56] In addition, laboratory studies are obtained. Blood is drawn for AChR antibodies, which are present in 74% of patients.[56]

Repetitive nerve conduction study during electromyography (EMG) is helpful in the diagnosis of myasthenia gravis. In EMG, a needle electrode is inserted into a skeletal muscle, and a recording of the electrical activity at rest, during voluntary activity, and with electrical stimulation is displayed on an oscilloscope. The patient should be informed that the needle causes some discomfort. In myasthenia gravis, the loss of functional AChRs results in a decrease in action potential size with repeated stimulation. Repetitive muscle stimulation produces a rapid decline in muscle action potential because of the deficient numbers of AChRs. Single-fiber EMG is a very sensitive test to assess the functioning of neuromuscular junction transmission.[55,56,59]

The Tensilon, or edrophonium, test is a classic diagnostic tool for confirming a diagnosis of myasthenia gravis. A positive test result lends strong support for the diagnosis of myasthenia gravis. In this test, 10 mg of IV Tensilon, a short-acting anticholinesterase agent, is given over approximately 1 minute. When injected, it transiently inhibits the breakdown of acetylcholine at the neuromuscular junction. A response is anticipated within 2 to 3 minutes. The test is most useful if there is improvement of ptosis or strength of the extraocular muscles. Limb strength or improved bulbar function may be difficult to interpret. When Tensilon is administered, atropine should be readily available in the event that the patient develops bradycardia. Because there have been reports of ventricular tachycardia and death as well, Tensilon should be administered in a monitored setting.[55,56]

A CT scan or MRI of the chest may also be performed to rule out thymoma or thymal hyperplasia. As noted earlier, patients with myasthenia gravis may have thymic tumors and should be screened. Thyroid function tests and vitamin B_{12} levels should also be checked, along with antinuclear antibodies, parietal cell antibodies, and antimicrosomal antibodies.[55,56]

Clinical Management

The clinical management of myasthenia gravis includes the following strategies: use of medications to enhance neuromuscular transmission; long-term immunosuppression with corticosteroids, mycophenolate mofetil (CellCept), azathioprine (Imuran), or cyclosporine; cyclophosphamide (Cytoxan); short-term immunomodulation with plasmapheresis or IVIG; or thymectomy.[55,56,60]

Pharmacological Management

Pharmacological management includes the use of anticholinesterases, steroids, or other immunosuppressive drugs. Pyridostigmine (Mestinon) is available in three formulations: liquid, a 60-mg tablet, or a 180-mg time-span formula. If the patient is unable to swallow the tablet or has a nasogastric tube or percutaneous endoscopic gastrostomy, the 60-mg tablet may be crushed, or the liquid form of pyridostigmine may be used. This drug inhibits the enzymatic elimination of acetylcholine, thus prolonging its action at the postsynaptic membrane and enhancing neuromuscular transmission. As a result of this action, more acetylcholine is available at the neuromuscular junction, and the patient has improved muscle strength. Medication onset of action is 30 minutes after administration, peaks in 1 hour, and lasts for 3 to 4 hours.[55,56]

Pyridostigmine should always be administered promptly as prescribed. It should be given every 3 to 4 hours when the patient is awake. If there is difficulty with chewing and swallowing, timing the medication 30 minutes before meals is helpful. The 180-mg time-span tablet is administered at bedtime and should never be crushed. Because the medication is given at night, the patient will have the benefit of sleep. Muscarinic side effects include diarrhea, abdominal cramping, increased salivation, blurred vision, bradycardia, and increased perspiration. Nicotinic side effects include muscle twitching, weakness, and fatigue.[55,56]

If the patient cannot take oral pyridostigmine because of fasting, or NPO status, or intubation, a comparable approach is to use IV neostigmine. IV neostigmine bromide 1 mg is equivalent to pyridostigmine 60 mg. Neostigmine can be infused as a continuous infusion, and care should be taken to ensure the patency of the IV access. Cardiac monitoring is essential.[55,56]

Steroids and other immunosuppressive medications may be used with pyridostigmine in managing myasthenia gravis.[55,56] A summary of the mechanism of action, onset of immunosuppression, usual dosage, and nursing considerations can be found in Table 35-12.

The patient should not receive some medications. For example, D-penicillamine is contraindicated in patients with myasthenia gravis. Other drugs, including some antibiotics, can cause an increase in myasthenic weakness (Box 35-7).[56] Both patients and health care professionals need to be

Table 35-12 Immunosuppressive Drugs for the Treatment of Myasthenia Gravis

Drug Mechanism of Action	Onset of Immunosuppression	Dose	Adverse Effects	Considerations and Patient Teaching
Corticosteroid (Prednisone):reduces amount of antibodies produced, blocks immune mechanism, and restores chemical reaction at neuromuscular junction	3 wk	60 mg daily	Steroid-induced diabetes Osteoporosis Weight gain Fluid retention Hirsutism Moon facies Insomnia Mood changes Gastric ulcers Susceptibility to infection	Starting with high dose may increase motor weakness temporarily
Azathioprine (Imuran):reduces the level of circulating acetylcholine receptor (AChR) antibodies	3–6 mo Maximal improvement in 36 mo	2–3 mg/kg/d	Bone marrow depression Increased risk for carcinoma Hepatotoxicity	Should be taken with food Pregnancy precautions Nurse to follow safe handling of hazardous drugs
Mycophenolate mofetil (CellCept):inhibition of purine synthesis by the *de novo* pathway	3–6 mo	1 g twice daily	Neutropenia; anemia; thrombocytopenia; leukopenia GI hemorrhage/perforation	Cautious use in digestive system disorders
Cyclosporine: suppresses T-cell function that decreases circulating AChR antibodies	4 wk	2–3 mg/kg/d; after morning and evening meals	Hypertension Nephrotoxicity	Traditionally used to provide immunosuppression after organ transplantation More costly than azathioprine
Cyclophosphamide (Cytoxan):chemotherapeutic agent; potent immunosuppressant	4 wk	2–3 mg/kg/d	Hair loss Hemorrhagic cystitis Bone marrow suppression Jaundice Kidney failure	Chemotherapy administration protocol to be followed Nurses to follow safe handling principles Education about reproductive risks

cognizant of these medications. Although physicians and nurses working in the neuroscience arena are often familiar with these drugs, patients may face potential difficulties in settings, such as the emergency department or surgery, where health care professionals do not encounter patients with myasthenia gravis on a regular basis and may not be familiar with these medications.[56]

Plasmapheresis

Plasmapheresis may be indicated for patients in crisis or who are otherwise refractory to treatment. Plasmapheresis is initiated to remove circulating anti-AChR antibodies from the plasma, which results in clinical improvement. This procedure is performed through a dual-lumen central vascular access device, which is similar to a dialysis catheter. A specialized team of nurses, trained in plasmapheresis, performs the treatment, usually three times a week, while the patient is an inpatient.[55,56] Treatments may continue on an outpatient basis. The patient's circulating blood volume is removed through one of the lumens, filtered, and then returned through the second lumen. The patient's plasma is removed and albumin is returned, along with the solid components of the patient's blood. The procedure takes several hours, and

BOX 35.7 PATIENT SAFETY

Medications to Avoid in Myasthenia Gravis

Antibiotics
Aminoglycosides, "mycins," tetracycline, polymyxin B and E, colistin

Antiepileptic Drugs
Phenytoin, mephenytoin, trimethadione

Cardiovascular Medications
Quinidine, procainamide, beta-blockers

Psychotropic Drugs
Lithium carbonate, phenothiazines

Muscle Relaxants
Curare, succinylcholine

Others
Magnesium preparations, quinine, D-penicillamine, chloroquine

Data from Shah AK: Myasthenia gravis. Updated January 15, 2009; accessed June 15, 2010 from: http://emedicine.medscape.com/article/1171206-print; Arbour, R. Mastering neuromuscular blockade. Dimens Crit Care Nurs 19(5):4–18, 2000.

the patient is monitored for hypotension. Electrolytes and clotting factors are evaluated after each treatment.

The catheters must be managed appropriately because they are a potential source of infection. They can pose a special challenge because the patient with myasthenia gravis may be receiving steroids or other immunosuppressive therapy. The nurse must also be aware that plasmapheresis removes medications, including pyridostigmine, which the patient has taken. The nurse must obtain an order from the physician to withhold the medication.

Intravenous Immunoglobulin

Another treatment in place of plasma exchange is IVIG. It is used either for acute disease management or as a long-term treatment for patients with myasthenia gravis who do not respond to other types of treatments. It is often used before thymectomy to stabilize the patient. The patient's dose is individualized. Patients may exhibit clinical improvement in 2 to 4 days, and it may last for varying intervals. The mechanism of action of IVIG is unknown. The patient needs to be monitored for fever and chills, leukopenia, headache, fluid overload, and renal failure.[55,56]

Thymectomy

Thymectomy is a standard treatment for patients younger than 52 years with generalized myasthenia gravis. This surgical procedure promotes sustained remission and improvement, although no controlled studies have been performed. However, the fall in antibody titers after surgery supports the use of thymectomy. Patients must be aware that this procedure is performed for its long-term benefit so that they do not expect a dramatic improvement immediately after surgery. Clinical improvement may not be realized for 6 to 12 months after thymectomy. In some cases, benefit may not be seen for several years.[55,56] Patients may demonstrate enough improvement so that their medications may be reduced, thereby reducing adverse effects.

Postthymectomy care involves a short stay in the ICU. Epidural analgesia is used to manage pain after the procedure. The patient is usually extubated immediately after the surgery. Intermittent positive-pressure breathing may be used to minimize postoperative respiratory complications. After the critical care stay, patients are transferred to an inpatient setting, where they are monitored for complications. Over the long term, thymectomy is considered more effective than conservative management as measured by rates of remission, overall survival, and clinical improvement.[60]

Management of Myasthenic Versus Cholinergic Crisis

Factors such as stress, respiratory infection, too rapid a steroid taper, or medication affecting the neuromuscular junction may predispose the patient to a crisis. A myasthenic crisis needs to be differentiated from a cholinergic crisis because the management of each is different.[55]

A myasthenic crisis is characterized by respiratory failure along with sudden exacerbation of weakness in other muscle groups. It is usually caused by lack of medication or lack of responsiveness at the neuromuscular junction to cholinergic treatment, as well as a worsening of the disease process. The patient is unresponsive to an increase in anticholinesterase medications and can experience severe weakness, dysphagia, and respiratory compromise. Frequent FVC checks should be performed, and when FVC falls below 15 mL/kg, the patient should be intubated. Any patient with myasthenia gravis with uncertain respiratory status should be admitted to an ICU to permit close monitoring of FVC, negative inspiratory force, and anxiety, as well as to facilitate a physical examination.[55,56]

The hallmarks of cholinergic crisis are muscarinic or nicotinic side effects, namely, increased perspiration, abdominal cramping, and diarrhea. Cholinergic crisis results from too much medication that causes neuromuscular blockage (which prevents muscle depolarization because of excess acetylcholine). The patient may also experience respiratory failure. Indeed, respiratory failure may be seen in both types of crises.[55]

Patients should be managed intensively. Admission to the ICU includes DVT prophylaxis and ulcer prevention. Providers need to be aware that patients in myasthenic crisis are not intubated due to lung or systemic problems, but rather to difficulties with muscle strength.[55,56,58] Given the hazards of intubation and controlled ventilation, including airway injury as well as lung injury and ventilator-associated pneumonia, measures to avoid intubation should be considered as clinically appropriate. One option is noninvasive positive pressure ventilation (NIPPV). Vigilant assessment and early recognition of impending respiratory failure may identify appropriate candidates for NIPPV. Using NIPPV in patients with myasthenic crisis before hypercapnia develops may prevent intubation and prolonged mechanical ventilation as well as decrease hospital length of stay and reduce risk of pulmonary complications.[61]

In the past, the Tensilon test was used to determine whether the patient was in a myasthenic or cholinergic crisis. If there was an improvement in muscle strength when Tensilon was administered, it indicated a myasthenic crisis.[53,54] If there was no improvement or further deterioration of muscle strength, the patient was most likely experiencing a cholinergic crisis. This test is no longer required because the withdrawal of cholinesterase drugs is necessary for improvement in both crises. Respiratory and nutritional support is provided in both situations.

Nursing Management

ASSESSMENT. The nurse must focus the neurological assessment on cranial nerve involvement, motor strength, and the extent of respiratory involvement. The patient should be monitored for ptosis and double vision. The patient's motor strength should be evaluated by the use of arm abduction times up to 5 minutes. One valuable tool for monitoring respiratory function a handheld spirometer to measure FVC. The nurse must be vigilant in monitoring the patient's respiratory status because the patient's diaphragm and intercostal muscles may become weak. If the patient's FVC falls below 1 liter, it usually indicates respiratory failure, and intubation and mechanical ventilation are necessary. An easy bedside test involves counting out numbers in one breath. Most patients should be able to count out up to 50 in

one breath. In summary, useful clinical assessment involves arm abduction times, FVC, range of eye movements, and time to the development of ptosis on upward gaze. Muscle strength testing is also valuable.

PLAN. The patient with myasthenia gravis may need assistance with activities of daily living. Adaptive equipment can help him or her perform self-care activities. Short rest periods are planned throughout the day, to help reduce patient fatigue and conserve energy.

Nutrition also needs to be addressed. Meals should be planned when pyridostigmine is at its peak. Aspiration precautions should be established. Thin liquids should be given only if the patient can tolerate them well. If the patient chokes when swallowing water, oral intake should cease. If the patient has a wet, gurgling voice or respiratory sounds or develops stridor, intubation may be necessary for airway protection, and nutrition may need to be offered through the enteral route. Caloric intake must be sustained to prevent a negative energy balance that interferes with weaning from the ventilator.

Skin care also needs to be incorporated into the care routine, and measures should be taken to avoid pressure ulcers. Pressure relief devices can be used in bed or on chairs.

An effective method of communication should also be developed, particularly if it becomes difficult to understand the patient because of the nasal quality of the voice. A communication board may be helpful as an alternate communication device.

Patient Education and Discharge Planning

Support and education about myasthenia gravis are crucial for the successful management of the disease. The patient needs to learn about the purpose of medications, medication schedules, and side effects. Patients should be instructed to adhere to their medication schedules and to contact the physician for modification if needed. Doses of medication should be kept at home and work so that they are readily available. During travel, the patient should always carry his or her medication (eg, in a purse or camera case) so that it does not become lost with luggage.

It is also helpful for the patient to obtain a medical identification bracelet and card so that rapid identification can occur in the event of a medical emergency. If the patient is unable to communicate, successful management may hinge on health care professionals recognizing that the person has myasthenia gravis.

The patient and family are taught to recognize signs and symptoms of a crisis. In addition, the importance of avoiding potential triggers for a crisis, such as respiratory infection or undue stress, is emphasized. During the winter months, when colds and flu are prevalent, the patient should be instructed to stay away from places where large groups of people gather, such as movies or concerts.

The patient should also be educated about community support groups. The Myasthenia Gravis Foundation can provide valuable resources. Box 35-8 lists patient teaching guidelines for myasthenia gravis. Long-term outcomes in myasthenia gravis have improved markedly because of the availability of immune-modulating therapies, and patients need to be educated on how to live with their disease.

BOX 35-8 TEACHING GUIDE *Living With Myasthenia Gravis*

Safety
- Obtain a medical identification (MedicAlert) bracelet and wear it at all times. Always carry personal identification in your wallet or purse.
- Avoid respiratory infections. Stay away from concert halls and movie theaters in the winter months or flu season.
- Avoid stress and stressful situations.
- Consider grinding or pureeing foods to make them easier to eat. Choose calorie-rich, nutritious snacks. Request a consultation with a dietitian if you have difficulty maintaining your weight.

Activity
- Plan frequent rest periods during the day.
- Conserve energy as much as possible.
- Avoid shopping at peak times or substitute "online" shopping so items can be delivered directly to your door.

Medications
- Mestinon (pyridostigmine) should be taken as directed by your physician. Adjustments should be made only under the direction of your physician. Remember not to crush Mestinon Timespan product.
- Mestinon should be stored at home and at work. Do not store medication in your car where it could be exposed to extreme heat and cold. When traveling, keep medication with you; do not store it in your luggage.
- Avoid medications (certain antibiotics, such as aminoglycosides, anticonvulsants, and muscle relaxants) that could exacerbate myasthenia gravis.
- Do not take any over-the-counter preparations or complementary medications without first contacting your physician.
- If you are taking Imuran (azathioprine) or cyclophosphamide, you need to follow precautions about hazardous drugs. Be certain to discuss family planning issues with your physician.

When to Call the Physician
- Call your physician if you are experiencing increased weakness, swallowing problems, or respiratory difficulties.
- Contact your physician immediately if you are hospitalized for any reason.

The Myasthenia Gravis Foundation of America is a valuable resource. Contact the Foundation for additional information and to find local support groups.

▲ Clinical Applicability Challenges

CASE STUDY

Ms. E.B. a 44-year-old marketing executive, was working in her office when she experienced severe dizziness and what she described as the "worst headache of her life." She called for help, and within moments, a colleague entered her office. E.B. was unable to stand or open her eyes. She was experiencing severe headache as described. Acting quickly, E.B.'s colleague activated the EMS, and E.B. was transported to the emergency department of a nearby tertiary care facility. Upon arrival, she had a neurological assessment performed revealing severe headache, nuchal rigidity, and deficit in the oculomotor nerve. The patient reported diplopia; dysconjugate gaze was noted on examination, and ptosis was evident. IV accesses were initiated, and the family was notified. Given the physical assessment findings of hard neurological signs, E.B. was taken immediately for CT of the brain. Head CT revealed slight effacement of the sulci and concurrent layering of blood in the subarachnoid space of 1 mm. Based on radiological and neurological assessment findings, the diagnosis of SAH was made. E.B. remained responsive, able to communicate and answer questions as well as maintain her own airway and lung ventilation. As such, she was transferred to an available monitored bed in the medical intensive care unit (MICU). While in the MICU, E.B.'s neurological status was monitored and documented hourly, stimulation was minimized, and she received analgesics for pain. Appropriate care was taken to balance effective analgesia with avoiding the side effect of sedation as a side effect of opioid analgesia. The plan of care for the next day included four-vessel cerebral angiography to identify location and distribution of vascular lesions such as cerebral aneurysms.

At 9:00 AM on day 2 of hospital admission, E.B. was transported to the angiography suite for urgent four-vessel cerebral angiography. The procedure was performed using a right femoral artery approach with a procedural duration of 94 minutes. E.B. tolerated the procedure well with no additional neurological deficits. Effective hemostasis was obtained following removal of the femoral arterial sheath. Upon return to the MICU, E.B. experienced diuresis consequent to crystalloid administration during the procedure as well as the osmotic diuretic effect of contrast media. Cerebral angiography showed aneurysms in the MCA, tip of the basilar artery, and the PCA, for a total of three.

The next step was deciding on a plan of treatment. Given location of the lesions and higher risk of morbidity from open craniotomy for clipping or wrapping the aneurysms, E.B. and her family elected a neurointerventional embolization procedure to treat the aneurysms. The procedure was planned in stages, with one aneurysm embolized at a time. The MCA aneurysm was identified as the lesion that had bled. As such, it was to be embolized first. Stage 1 of the embolization procedure was scheduled for hospital day 5. During the intervening time, E.B. was to remain in the monitored setting. Due to blood within the subarachnoid space, cerebral vasospasm was a risk. To maintain vigilance, neurological assessments were frequent and included arousal, consciousness, sensory and motor function, as well as communication.

During the night of ICU day 3, E.B. became lethargic and her pupils became dilated and sluggish. Her trachea was intubated orally, and she was placed on controlled ventilation. After stabilization, she was transported to the radiology department for emergent CT of the head. Head CT showed dilation of the ventricles as compared with the CT study on admission. She was transported immediately back to the MICU. An emergent ventriculostomy was performed. Opening CSF pressures were 25 to 30 mm Hg, and 25 mL of slightly blood-tinged CSF were drained, decreasing her ICP to 13 to 17 mm Hg. Monitoring parameters included placing the catheter to monitor continuously, with orders to drain 5 mL CSF for ICP readings sustained over 20 mm Hg. Within 7 hours following ventricular catheter placement, E.B.'s neurological assessment returned to baseline. She followed commands and was more responsive and interactive with care givers and family members. The embolization procedure was delayed pending stabilization of these clinical issues. Multiple IV accesses were maintained for administration of fluids and medications. E.B. was at risk for seizure activity from altered neuronal physiology, which can decrease electrical stability in the brain and increase the risk of seizure activity consequent to neurological injury. Administration of anticonvulsant drugs was potentially necessary.

Over the ensuing 5 ICU days, E.B.'s ICP remained stable, requiring CSF drainage on only two occasions. On ICU day 9, the ventricular catheter was clamped. ICP measurements remained stable (15 mm Hg or less), and neurological assessment remained stable as well, reflecting an interactive patient, able to protect her own airway. Lung ventilation and pulmonary status remained stable on continuous positive airway pressure and pressure support settings, giving her control over rate, depth, and pattern of her respiratory cycle. Follow-up head CT revealed resolution of ventricular dilation and no effacement of the sulci. The ventricular catheter was removed, and E.B. was extubated and received supplemental oxygen delivered by nasal cannula at 4 L/min.

Following stabilization, the embolization procedure for the MCA aneurysm was rescheduled for the following week. E.B. was transported to the interventional radiology suite and monitored for oxygenation and cardiovascular status by the anesthesiology provider. EEG monitoring was initiated to closely monitor neurologic status intraprocedure while anesthesia was being administered. The neurointerventional radiologist successfully placed detachable coils within the aneurysm, effectively eliminating it from the circulation. Extreme care was taken to avoid coil placement outside of the aneurysm, which may

(continued on page 803)

▲ Clinical Applicability Challenges

CASE STUDY *(Continued)*

cause ischemic stroke. Following coil placement within the aneurysm, follow-up angiography was done, demonstrating coil placement within the aneurysm and absence of any blood flow within the aneurysm. No EEG findings of neurologic injury were evident intraprocedure, and E.B. recovered from anesthesia in stable condition.

Neurologic assessment findings were intact, were interactive, and demonstrated no neurologic sequelae from the aneurysm and subsequent hydrocephalus. Long-term follow-up was planned and reviewed with E.B. and her family for two reasons. First, two additional aneurysms needed coil embolization. Second, coils may become compressed with long-term exposure to hemodynamic stress within the native blood vessel, increasing the risk for rebleeding and possibly requiring subsequent coil embolization. E.B. was discharged ultimately to home with anticipated embolization of the remaining aneurysms in a staged procedure. She ultimately returned to work following hospital discharge.

1. Increased ICP is one means by which a patient with ischemic stroke, hemorrhagic stroke, or SAH may experience increased mortality or morbidity. What are the mechanisms by which ICP elevations occur in these populations?

2. For a patient with a long-standing seizure disorder or a patient experiencing seizures in an acute-care setting, what are the means by which emergent administration of anticonvulsant medications is accomplished?

3. Pain may be managed with opioid analgesics and anxiety managed with sedative-hypnotic drugs, such as benzodiazepines. What are the significant nursing considerations in administering these drugs to a patient with a neurological disorder?

References

1. Hickey JV: The Clinical Practice of Neurological and Neurosurgical Nursing. Philadelphia, PA: Wolters Kluwer/Lippincott Williams & Wilkins, 2009
2. Paolino A, Ruppert J, Anderson M, et al: Guide to the care of the patient with craniotomy post-brain tumor resection: AANN Reference Series for Clinical Practice. Am Assoc Neurosci Nurses 2006. Revised 2009. Accessed June 4, 2010 from http://www.aann.org/pdf/cpg/aanncraniotomy.pdf
3. Adult Brain Tumors Treatment: Health Professional Version. Accessed June 9, 2010 from http://www.cancer.gov/cancertopics/pdq/treatment/adultbrain/HealthProfessional. Updated October 13, 2009
4. Wen PY, Kesari S: Malignant gliomas in adults. N Engl J Med 359(5):492–507, 2008
5. Stummer W: Mechanisms of tumor-related brain edema. Neurosurg Focus 2007. Accessed June 9, 2010 from http://www.medscape.com/viewarticle/559000
6. Clarke J, Butowski N, Chang S: Recent advances in therapy for glioblastoma. Arch Neurol 67(3):279–283, 2010
7. Giubilei C, Ingrosso G, D'Andrea M, et al: Hypofractionated stereotactic radiotherapy in combination with whole brain radiotherapy for brain metastases. J Neurooncol 91(2):207–212, 2009
8. Serizawa T, Yamamoto M, Nagano O, et al: Gamma knife surgery for metastatic brain tumors. J Neurosurg 109(Suppl12):118–121, 2008
9. Suh JH: Stereotactic radiosurgery for the management of brain metastases. N Engl J Med 362:1119–1127, 2010
10. Van Meir EG, Hadjipanayis CG, Norden AD, et al: Exciting new advances in neuro-oncology: The avenue to a cure for malignant glioma. CA Cancer J Clin 60(3):166–193, 2010
11. Treibel KL, Martin RC, Nabors LB, et al: Medical decision-making capacity in patients with malignant glioma. Neurology 17(24):2086–2092, 2009
12. Brisman JL, Soliman E. Cerebral aneurysm. Updated May 22, 2009; accessed May 16, 2010 from http://www.emedicine.medscape.com/article/252142-print
13. Leibskind DS. Cerebral aneurysms. Updated May 10, 2009; accessed from http://emedicine.medscape.com/article/1161518-print
14. Alexander S, Gallek M, Prescuitti M, et al: Care of the patient with subarachnoid hemorrhage: AANN Reference Series for Clinical Practice. Am Assoc Neurosci Nurses 2007. Revised December, 2011. Accessed December 20, 2011 from http://www.aann.org/pdf/cpg/aanncraniotomy.pdf
15. Diringer MN: Management of aneurismal subarachnoid hemorrhage. Crit Care Med 37(2):432–440, 2009
16. Norton CK, Linenfelser P: Patient with intracranial subarachnoid hemorrhage requiring an endovascular coiling procedure. Adv Emerg Nurs J 31(1):12–18, 2009
17. Schmidt U, Bittner E, Pivi S, et al: Hemodynamic management and outcome of patients treated for cerebral vasospasm with intraarterial nicardipine and/or milrinone. Anesth Alang 110(3):895–902, 2010
18. Westermaier T, Stetter C, Vince GH, et al: Prophylactic intravenous magnesium sulfate for treatment of aneurismal subarachnoid hemorrhage: A randomized, placebo-controlled, clinical study. Crit Care Med 38(5):1284–1290, 2010
19. Friedlander RM: Arteriovenous malformations of the brain. N Engl J Med 356(26):2704–2712, 2007
20. Sen S, Webb SW, Selph J: Arteriovenous malformations. Updated May 18, 2010; accessed June 1, 2010 from http://emedicine.medscape.com/article/1160167-print
21. Altschul D, Smith M, Sinson GP: Intracranial arteriovenous malformation. Updated May 26, 2009; accessed June 10, 2010 from http://emedicine.medscape.com/article/252426-print
22. Guedin P, Gaillard S, Boulin A, et al: Therapeutic management of intracranial dural arteriovenous shunts with leptomeningeal venous drainage: Report of 53 consecutive patients with emphasis on transarterial embolization with acrylic glue. J Neurosurg 112(3):603–610, 2010
23. Andreou A, Loannidis I, Lalloo S, et al: Endovascular treatment of intracranial microarteriovenous malformations. J Neurosurg 109(6):1091–1097, 2008
24. Chung WY, Shiau CY, Wu HM, et al: Staged radiosurgery for extra-large cerebral arteriovenous malformations: Method, implementation and results. J Neurosurg 109(Suppl 6):65–72, 2008
25. Geibprasert S, Peirera V, Krings T, et al: Hydrocephalus in unruptured brain arteriovenous malformations: Pathomechanical considerations, therapeutic implications and clinical course. J Neurosurg 110(3):500–507, 2009

26. Air EL, Leach JL, Warnick RE, et al: Comparing the risks of frameless stereotactic biopsy in eloquent and noneloquent regions of the brain: A retrospective review of 284 cases. J Neurosurg 111(4), 2009. DOI: 10.3171/2009.3.JNS081695
27. Feiden W, Bise K, Steude U, et al: The stereotactic biopsy diagnosis of focal intracerebral lesions in AIDS patients. Acta Neurol Scand 87(3):228–233, 2010
28. Kim SS, McCutcheon IE, Suki D, et al: Awake craniotomy for brain tumors near eloquent cortex: Correlation of intraoperative cortical mapping with neurological outcomes in 309 consecutive patients. Neurosurg 64(5):836–846, 2009.
29. Sciarretta V, Mazzatenata D, Ciarpaglini R, et al: Surgical repair of persisting CSF leaks following standard or extended endoscopic transsphenoidal surgery for pituitary tumor. Minim Invasive Neurosurg 53(2):55–59, 2010
30. Trembly A: Stroke care in the 21st century. Nurs Manag 41(6):30–36, 2010
31. Saver JL, Kalafut M: Thrombolytic therapy in stroke. Updated April 15, 2010; accessed June 5, 2010 from http://emedicine.medscape.com/article/1160840-print
32. Becker JU, Wira CR, Arnold JL: Stroke, ischemic. Updated May 12, 2010, accessed June 15, 2010 from http://emedicine.medscape.com/article/793904-print
33. Minino AM, Murphy SL, Xu J, et al: Deaths: Final data for 2008. National Vital Statistics Reports. December 7, 2011; 59(10):1–157, 2011.
34. Pugh S, Mathiesen C, Meighan D, et al: Care of the Patient With Ischemic Stroke: AANN Reference Series for Clinical Practice. Am Assoc Neurosci Nurses, 2009
35. Summers D, Leonard A, Wentworth D, et al: Comprehensive overview of nursing and interdisciplinary care of the acute ischemic stroke patient. Stroke 40:2911–2944, 2009
36. del Zoppo GJ, Saver JL, Jauch EC, et al: Expansion of the time window for treatment of acute ischemic stroke with intravenous tissue plasminogen activator: A science advisory from the American Heart Association/American Stroke Association. Stroke 40:2945–2948, 2009
37. Smith WS, Sung G, Saver J, et al: Mechanical thrombectomy for acute ischemic stroke: Final results of the Multi MERCI trial. Stroke 39:1205–1212, 2008
38. Nussbaum ES, Janjua TM, Defillo A, et al: Emergency extracranial-intracranial bypass surgery for acute ischemic strok. J Neurosurg 112(3): 666–673, 2010
39. Brott TG, Hobson RW, Howard G, et al: Stenting versus Endarterectomy for treatment of carotid artery stenosis. N Engl J Med 363(1): 11–23, 2010
40. Nassisi D: Stroke, hemorrhagic. Updated April 7, 2010; accessed June 6, 2010 from http://emedicine.medscape.com/article/793821-print
41. Qureshi AI, Mendelow AD, Hanley DF: Intracerebral hemorrhage. Lancet 373:1632–1644, 2009
42. Elliott J, Smith M: The acute management of intracerebral hemorrhage: A clinical review. Anesth Analg 110(5):1419–1427, 2010
43. Pillow MT, Howes DS: Seizures. Updated January 22, 2010; accessed June 15, 2010 from http://emedicine.medscape.com/article/1609294-print
44. Cavazos JE, Spitz M: Seizures and epilepsy: Overview and classification. Updated November 10, 2009; accessed June 5, 2010 from http://emedicine.medscape.com/article/1184846-print
45. Fisher RE, Long L: Care of the patient with seizures: AANN Reference Series for Clinical Practice. Am Assoc Neurosci Nurses 2009
46. Haut SR, Hall CB, Masur J, et al: Seizure occurrence: Precipitants and prediction. Neurology 69(20):1905–1910, 2007
47. Oddo M, Carrera E, Claassen J, et al: Continuous electroencephalography monitoring in the medical intensive care unit. Crit Care Med 37(6):2051–2056, 2009
48. De Haan GJ, van der Geest P, Doelman G, et al: A comparison of midazolam nasal spray and diazepam rectal solution for the residential treatment of seizure exacerbations. Epilepsia 51(3):478–482, 2010
49. Koubeissi M, Alshekhlee A: In-hospital mortality of generalized convulsive status epilepticus: A large US sample. Neurology 69(9):886–893, 2007
50. Simmons S: Guillian-Barre' syndrome: A nursing nightmare that usually ends well. Nursing 40(1):24–29, 2010
51. Miller AC, Rashid RM, Sinert RH: Guillain-Barre' sundrome. Updated April 23, 2010; accessed June 3, 2010 from http://emedicine.medscape.com/article/792008-print
52. Ramachandran TS, Sater RA: Acute inflammatory demyelinating polyradiculoneuropathy. Updated January 15, 2009; accessed June 5, 2010 from http://emedicine.medscape.com/article/1169959-print
53. Davids HR, Oleszek JL: Guillain-Barre' syndrome. Updated March 29, 2010; accessed June 15, 2010 from http://emedicine.medscape.com/article/315632-print
54. Alshekhlee A, Hussain Z, Sultan B, et al: Guillain-Barre' syndrome: Incidence and mortality rates in US hospitals. Neurology 70(18):1608–1613, 2008
55. Goldenberg WD, Sinert RH: Myasthenia gravis. Updated May 21, 2010; accessed June 15, 2010 from http://emedicine.medscape.com/article/793136-print
56. Shah AK: Myasthenia gravis. Updated January 15, 2009; accessed June 15, 2010 from http://emedicine.medscape.com/article/1171206-print
57. Meriggioli MN: Myasthenia gravis with anti-acetylcholine receptor antibodies. Front Neurol Neurosci 26:94–108, 2009
58. Alshekhlee A, Miles JD, Katirji B, et al: Incidence and mortality rates of myasthenia gravis and myasthenic crisis in US hospitals. Neurology 72(18):1548–1554, 2009
59. Guardia C, Berman SA: Assessment of neuromuscular transmission. Updated April 7, 2010; accessed June 3, 2010 from http://emedicine.medscape.com/article/1140870-print
60. Bachmann K, Burkhardt D, Schreiter I, et al: Thymectomy is more effective than conservative treatment for myasthenia gravis regarding outcome and clinical improvement. Surgery 145(4):392–398, 2009
61. Seneviratne J, Mandrekar J, Wijdicks EF, et al: Noninvasive ventilation in myasthenic crisis. Arch Neurol 65(1):54–58, 2008

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

36 Traumatic Brain Injury

Elizabeth Zink and Elizabeth Kozub

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Explain the significance of the mechanism of injury when assessing the patient with traumatic brain injury.
2. Compare and contrast various types of head injuries and typical patient presentation.
3. Differentiate between primary and secondary brain injury.
4. Explain the importance of and technique for serial neurological assessment in the patient with traumatic brain injury.
5. Discuss the rationale for medical and nursing management in the care of the patient with a traumatic brain injury.
6. Describe the roles of multidisciplinary health care team members in caring for the patient with traumatic brain injury.

Traumatic brain injury (TBI) affects as many as 1.7 million Americans each year and has devastating effects on patients and their families. About 1.4 million people, or 80% of those with TBI, are evaluated and discharged from emergency departments (EDs) each year; 275,000 are admitted to hospitals and 52,000 die. It is suspected that a large number of people who sustain TBI do not seek health care. Falls are the leading cause of TBI, constituting 35% of all cases, followed by unknown causes (21%) and motor vehicle crashes, which account for 17% of all TBI.[1] The incidence of TBI is greater in males than females, and TBI occurs most frequently in children younger than age 4 years and in adolescents between ages 15 and 19 years.[1] TBI in adults older than age 65 most often result from falls (61%) (Box 36-1). Critical care nurses play an important role in reducing the incidence of head injury through patient and family teaching as well as participation in primary prevention efforts (eg, helmet safety, violence prevention, fall prevention, and drug and alcohol awareness).

TBI can have a profound and lasting effect on the patient and family. Neurologic deficits may affect the patient's ability to resume his or her chosen career or to return to work at all. Emotional and behavioral changes may affect interpersonal relationships and family roles. A thorough understanding of the pathophysiology of TBI enables the critical care nurse to individualize nursing care and positively affect patient and family outcomes. Critical care nurses play a key role in planning and implementing the multidisciplinary care of these complex patients and their families.

▲ Mechanisms of Traumatic Brain Injury

Typical mechanisms of injury include acceleration, acceleration–deceleration, coup–contrecoup, rotational injury, and penetrating injury (Fig. 36-1):

- Acceleration injuries occur when a moving object strikes the stationary head (eg, a bat striking the head or a missile fired into the head).
- Acceleration–deceleration injuries occur when the head in motion strikes a stationary object. For example, a motor vehicle crash in which the head strikes the windshield produces an acceleration–deceleration injury. Acceleration–deceleration injuries can also occur with falls or physical assaults.
- Coup–contrecoup injuries occur when the brain "bounces" back and forth within the skull, striking both poles of the brain (ie, front and back or right side and left side). Coup refers to the area of brain tissue initially making forceful contact with the inside of the skull, and contrecoup refers to the second impact of brain tissue with the inside of the skull, usually on the opposite side. When assessing a patient struck in the back of the head, the clinician evaluates for injury to posterior structures (ie, the occipital lobes and cerebellum) as well as anterior brain structures (ie, the frontal lobes).
- Rotational forces cause the brain to twist within the meninges and the skull, resulting in stretching and tearing of blood vessels and shearing of neurons. Physical assaults

BOX 36-1 CONSIDERATIONS FOR THE OLDER PATIENT

Preventing Falls in the Older Adult

Screen patients 75 years old and older for history of falling or at age 70 if they are deemed to be at a higher risk for falling

Consider the following factors, which may need to be evaluated and/or modified in patients found to be at a greater risk for future falls:

- Impaired vision
- Medications
- Blood pressure (postural hypotension)
- Balance and gait
- Hazards in living environment

Tinchi ME: Preventing falls in elderly persons. N Engl J Med 384(1):42–49, 2003.

and motor vehicle crashes are examples of situations in which rotation and torsion may be a mechanism of injury.

- Penetration injuries may be caused by a bullet, shrapnel, or another sharp object traveling at a velocity substantial enough to disrupt the integrity of the skull. Depending on the speed and trajectory of the object, underlying brain structures may or may not be injured.

Cervical spine injury must be automatically assumed and systematically excluded with all types of TBI before immobilization devices are removed. TBI is categorized by severity based on radiographic injury and the Glasgow Coma Scale (GCS) (Table 36-1).

▲ Primary and Secondary Brain Injury

The term *primary brain injury* describes injury occurring at the time of trauma. Injury occurring after the traumatic event is referred to as *secondary brain injury*. The initial injury causes immediate disruption of the skull, brain structures (ie, meninges, blood vessels, brain tissues, neurons), and functions (blood flow, oxygenation, cellular metabolism). Secondary injury describes the physiological response to brain injury, including cerebral edema, cerebral ischemia, and biochemical changes. Current research and existing therapies are aimed at preventing and mitigating secondary brain injury to maximize chances for positive functional outcomes.

Primary Brain Injury

Scalp Laceration

A scalp laceration frequently causes significant bleeding because of the vascularity of the scalp and may be associated with other underlying injuries to the skull and brain. The scalp should be carefully palpated assessing for deformation. Skull fractures may be present even if deformities are not palpable; therefore, care must be exercised in applying pressure to scalp wounds. Scalp lacerations can be sutured at the bedside or may require surgical repair, depending on the size and extent of injury. Avulsion of areas of the scalp may require surgical reimplantation to address injured vascular structures.

Skull Fracture

The skull protects the brain by distributing forces outward, lessening direct impact to the brain. Skull fractures are categorized by location; the fractured bones may be located in the anterior, middle, or posterior fossae, or the base of the skull. Skull fractures may be compound (ie, occurring with an open wound), displaced (closed wound in which the edges of the fracture no longer meet), or linear. Depressed skull fractures are fractures in which bone fragments are driven into the underlying meninges or brain tissue; this often presents as a depression or dip on palpation. Patients with depressed skull fractures may require surgical management to débride bone fragments, repair the skull or dura, evacuate a hematoma, or repair other adjacent structures, such as sinuses or blood vessels.[2] Blood vessels travel along bony grooves on the inside surface of the skull; as a result, they are vulnerable to injury during a direct blow to the skull. Injury to the dura places the patient at risk for meningitis; therefore, careful monitoring for signs and symptoms of infection is important.

Basilar skull fractures occur at the base or floor, of the skull, typically in the areas of the anterior and middle fossae. Basilar skull fractures are linear or displaced. Assessment of extraocular movements is important in detecting impingement of cranial nerves. Nasogastric and nasotracheal intubation are avoided to reduce the risk for passing the tube through fractured areas into the brain.

Drainage of cerebrospinal fluid (CSF) from the ear or nose indicates injury to the dura. Drainage from the ear, otorrhea, typically signifies a fracture in the middle fossa. Ecchymosis (bruising) behind the ear (Battle's sign) is a delayed sign of a basilar skull fracture in the middle fossa. Rhinorrhea, CSF drainage from the nose, occurs with a fracture in the anterior fossa. "Raccoon eyes," a ring-like pattern of bruising around the eyes, is a late sign of this type of fracture.

Drainage from the ear or nose may be mixed with blood, making identification of CSF difficult. A layering of fluids, with blood on the inside and CSF in a yellowish ring on the outside (the "halo sign"), will appear when the area is wiped with gauze. Patients may also report a sweet or salty taste if CSF is draining into the pharynx. Clear fluid may be tested for a substance called beta-2 transferrin to distinguish between CSF and other body fluids.

CSF leaks typically heal on their own with rest; however, when a CSF leak persists, diversion of CSF into an external drainage device may be necessary to reduce pressure on the dural tear and allow time for healing. In some cases, surgical repair of the damaged region of dura must be performed. A loose gauze dressing can be applied to the ear or nose to quantify the amount and character of drainage while allowing unobstructed drainage of the fluid. The skin around the site of drainage is kept clean, and the patient is instructed not to blow his or her nose.

Concussion

A concussion is defined as any alteration in mental status resulting from trauma. The patient may or may not lose consciousness. Often patients are unable to recall events leading up to the traumatic event, and occasionally short-term memory is affected. Concussions are not associated with structural abnormalities on radiographic imaging, however, a growing body of research suggests that neuronal injury does

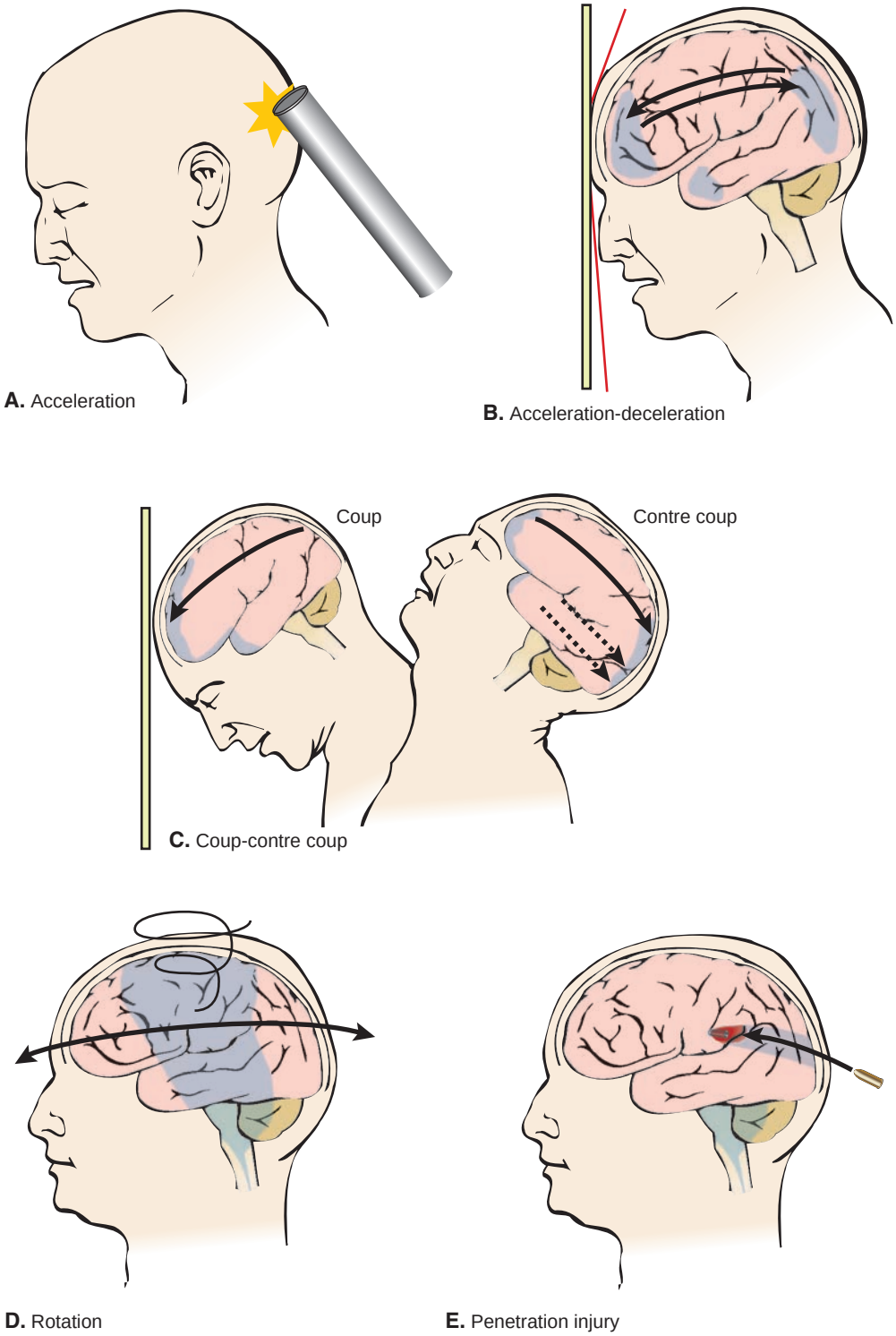


FIGURE 36-1 ▲ Typical mechanisms of head injury. **A:** Acceleration. **B:** Acceleration–deceleration. **C:** Coup-contrecoup. **D:** Rotation. **E:** Penetration.

occur with concussions and is associated with a metabolic crisis at the cellular level. This cellular metabolic crisis may cause the symptoms attributed to post-concussive syndrome.[3] Recovery after a concussion is usually quick and complete; however, some patients exhibit symptoms of postconcussive syndrome, which include headaches, decreased attention span, short-term memory impairment, dizziness, irritability, emotional lability, fatigue, visual disturbances, noise and light sensitivity, and difficulties with executive functions.[4] These symptoms may last for months to 1 year and can be alarming to the patient and the family. Discharge teaching must include a review of these signs and symptoms as well as criteria for obtaining medical follow-up.

Contusion

Contusions in the brain are the result of laceration of the microvasculature. They are focal and superficial, occasionally

Table 36-1 Defining the Severity of Head Injury

Severity	Description
Mild	GCS score 13–15 Loss of consciousness or amnesia for 5–60 min No abnormality on CT scan and length of hospital stay <48 h
Moderate	GCS score 9–12 Loss of consciousness or amnesia for 1–24 h May have abnormality on CT scan
Severe	GCS score 3–8 Loss of consciousness or amnesia for >24 h May have a cerebral contusion, laceration, or intracranial hematoma

GCS, Glasgow Coma Scale.

spreading to deeper layers of the brain. Cerebral contusions can range from mild to severe depending on the location, size, and extent of brain tissue injury. The diagnosis of cerebral contusion is made using computed tomography (CT). Small lesions may result in focal neurological deficits, whereas multiple or large contusions may result in a depressed level of consciousness and coma. Complications of a cerebral contusion include development of a hematoma and cerebral edema. Cerebral edema peaks 24 to 72 hours after injury, causing increased intracranial pressure (ICP). The patient's clinical condition may progressively deteriorate over the first 72 hours; therefore, the patient requires intensive anticipatory monitoring (serial neurological assessments) to identify signs and symptoms of increased ICP quickly and prevent further brain injury.

Epidural Hematoma

An epidural hematoma is a collection of blood between the dura and inside surface of the skull, often caused by laceration of the middle meningeal artery (Fig. 36-2). Although this type of hemorrhage is often associated with injury to an artery, injury to an extradural vein or venous sinus may also produce an epidural hematoma. Prompt recognition and expeditious surgical intervention to evacuate the hematoma result in improved outcomes. Patients may present in a coma or fully conscious.

Subdural Hematoma

A subdural hematoma is an accumulation of blood below the dura and above the arachnoid layer covering the brain (see Fig. 36-2). Tearing of the surface veins or disruption of venous sinuses can cause a subdural hematoma. The risk for subdural hematoma is increased in the elderly and in people with alcoholism. Cortical atrophy in these two populations causes tension on bridging veins leading from the surface of the brain to the inner surface of the dura. An increased incidence of falls also compounds the risk for subdural hematomas in these populations. Subdural hematomas can be separated into three categories based on the time from injury to the onset of symptoms: acute, subacute, and chronic.

Patients with acute subdural hematomas manifest symptoms within 24 to 48 hours after injury depending on the rate and amount of blood accumulation. Symptoms include headache, focal neurological deficit, unilateral pupillary abnormalities, and a decreasing level of consciousness. Size and location of the hematoma and the degree of neurological dysfunction are considered in making the decision to surgically evacuate the hematoma.

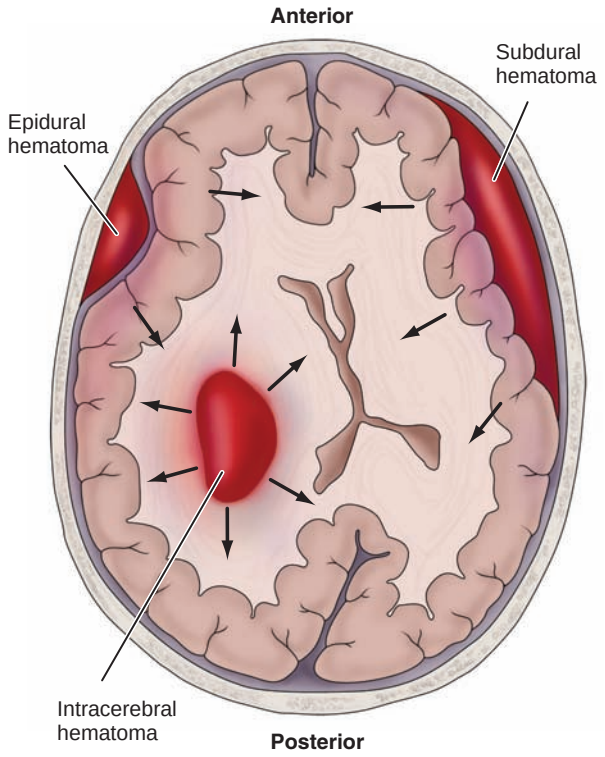


FIGURE 36-2 ▲ Cerebral hematomas. **A:** An epidural hematoma. **B:** A subdural hematoma. **C:** An intracerebral hematoma. (From Smeltzer: Brunner & Suddarth's Textbook of Medical-Surgical Nursing, 12th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 1922.)

Patients with subacute subdural hematomas have a delayed onset of symptoms, 2 days to 2 weeks after injury. The delay in symptom onset may be explained by a slower accumulation of blood caused by disruption of smaller blood vessels. In some cases, cerebral atrophy may allow for a greater amount of fluid to collect before symptoms of increased ICP manifest. Surgical evacuation of the clot may be performed on an elective basis depending on the degree of neurologic dysfunction.

Patients with chronic subdural hematomas may initially experience a small bleed that does not cause symptoms. Over time, slow capillary leaking of proteinaceous fluid causes expansion of the mass and produces symptoms of increased ICP. Chronic subdural hematomas are often seen in elderly patients with a history of falling. The slow accumulation of fluid accounts for delayed presentation of signs and symptoms of increased ICP.[5] Common symptoms include headache, lethargy, confusion, and seizures. Surgical intervention may include drilling burr holes into the skull or craniotomy to remove the hematoma. A drain may be placed intraoperatively to prevent reaccumulation of fluid. It is necessary to keep the head of the patient's bed flat to decrease tension placed on bridging veins. When the head is elevated, the brain settles downward. Therefore, the head of the patient's bed must be raised slowly to prevent rebleeding.

Intracerebral Hematoma

An intracerebral hematoma is a collection of blood within brain tissue caused by disruption of blood vessels (see Fig. 36-2). Traumatic causes of intracerebral hematoma include depressed skull fractures and penetrating injuries. Surgical management of intraparenchymal hematomas is indicated in patients with deteriorating neurological status referable to the injured region of brain tissue or patients with increased ICP that is uncontrolled with maximal medical therapies (eg, osmotic therapy, hyperventilation, and sedation). Medical therapy aims to manage cerebral edema and promote adequate cerebral perfusion.

Traumatic Subarachnoid Hemorrhage

Traumatic subarachnoid hemorrhage occurs with tearing or shearing of microvessels in the arachnoid layer where CSF flows around the brain. A traumatic subarachnoid hemorrhage often accompanies other severe brain injuries and appears to be associated with poor neurologic outcome and increased mortality.[6] Additional complications, such as hydrocephalus and cerebral vasospasm, add to the complexity of the injury.

Diffuse Axonal Injury

Diffuse axonal injury (DAI) is characterized by direct tearing or shearing of axons, and during the first 12 to 24 hours edema develops. DAI prolongs or disables signal conduction from the white matter to gray matter in the brain and is thought to occur with rotational and acceleration–deceleration forces. DAI can be classified as mild, moderate, or severe based on length of coma and degree of neurological dysfunction. Mild DAI is associated with a coma lasting no longer than 24 hours. Moderate DAI is characterized by a coma lasting longer than 24 hours with transient flexor or extensor posturing. Severe DAI is characterized by prolonged coma, fever, diaphoresis, and severe extensor posturing. DAI is not easily identified through radiographic imaging in the first 24 hours; however, small punctate hemorrhages may be visualized deep in the white matter, a finding that increases suspicion that DAI has occurred. Magnetic resonance imaging (MRI) may be helpful in identifying neuronal damage after 24 hours.

Cerebrovascular Injury

Carotid or vertebral artery dissection must be considered in situations in which a patient presents with neurologic deficits unexplained by other brain injuries. Arterial dissection is caused by shearing of the innermost or middle vessel layers, the intima and media. Damage to the intima can result in clot formation or an intimal flap, either of which can occlude the vessel, resulting in stroke. The key to preventing stroke in these patients is early identification of the injury, exclusion of concomitant hemorrhage, and possibly initiation of anticoagulation therapy. To detect this type of injury, cerebral angiography may be performed in patients who have sustained injury to the neck or have unexplained focal neurological deficits. Damage to the intima or media allows blood to leak between the blood vessel layers, resulting in a ballooning of the outermost vessel layers and creating an aneurysm. This type of aneurysm is referred to as a traumatic intracerebral aneurysm and may also be called a pseudoaneurysm.

Secondary Brain Injury

Secondary brain injury events occur after the initiating traumatic event and cause additional brain injury. Examples of conditions causing or exacerbating secondary brain injury are uncontrolled ICP, cerebral ischemia, hypotension, hypoxemia, and local or systemic infection. Secondary brain injury occurs as a function of the inflammatory response, reduced cerebral blood flow, and dysfunctional cerebral autoregulation causing damage to neurons. These secondary processes can result in cerebral infarction, coma, and increased cerebral edema. Prevention of hypotension, hypercarbia, hypoxemia, and seizures is extremely important in attempting to prevent further injury.[7]

Understanding intracranial dynamics and the nature of cerebral blood flow is essential to preventing and treating secondary brain injury (see Chapter 34 for a complete discussion of intracranial dynamics and the Monro-Kellie doctrine).

Compensation for increased volume in the cranium occurs when CSF is channeled through the foramen magnum into the spinal canal, CSF production is decreased, and venous blood is channeled out of the cranium into the jugular veins. The compliance curve (Fig. 36-3) illustrates the body's ability to compensate for the addition of water, CSF, or blood into the cranial vault and the point at which intracranial compliance is maximized. Decreased intracranial compliance results in a small addition of volume causing disproportionate increases in ICP. Examples of conditions causing decreased intracranial compliance are cerebral edema (an increase in brain water), expansion of a hematoma (an increase in blood), and hydrocephalus (an increase in CSF). An understanding of pressure–volume relationships allows the nurse to anticipate deterioration of the patient's clinical condition, tailor nursing interventions, and anticipate potential medical or surgical treatments.

Cerebral autoregulation is a protective mechanism that enables the brain to receive a constant blood flow over a range of systemic blood pressures (see Chapter 34 for a complete discussion). Several studies have suggested that cerebral blood flow may decrease up to 50% during the first 24 to 48 hours after TBI.[8]

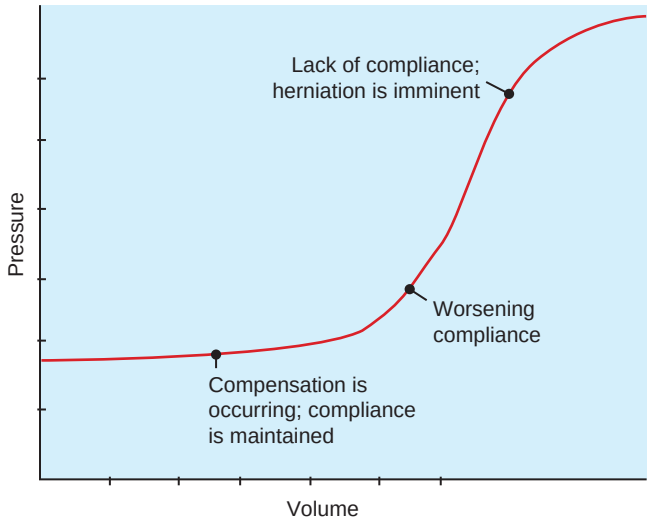


FIGURE 36-3 ▲ The compliance curve. The body is able to compensate for the addition of water, blood, or CSF to the cranial vault until a critical point is reached where compensation has been maximized. At this point, the addition of a small amount of volume will cause a disproportionate increase in ICP.

Biochemical mechanisms also play a significant role in causing secondary brain injury. The inflammatory response has been implicated as a potential cause or exacerbating factor in secondary brain injury.

Cerebral Edema

Cerebral edema commonly occurs in patients with TBI 24 to 48 hours after the primary insult and typically peaks at 72 hours.[9] Patients require increased observation during this period because of the increased risk for neurological deterioration. If cerebral edema is not quickly and aggressively treated, herniation syndrome may ensue. The treatment of cerebral edema is discussed in Chapter 34.

The two most common types of edema occurring after TBI are cytotoxic and vasogenic edema. Cytotoxic edema occurs as a result of intracellular sodium–potassium pump failure, allowing an influx of sodium and water into the cell. Vasogenic edema occurs as a result of a disruption to the blood–brain barrier.[9]

Ischemia

Cerebral ischemia, a major cause of morbidity and mortality, may be a result of direct vascular injury or cerebral edema that causes compression or occlusion of blood vessels within the brain. Ischemia in the brain may occur at the time of injury or during the period subsequent to the injury. Cerebral ischemia occurs whenever blood flow is inadequate to meet metabolic demands of the brain. If the cause of cerebral ischemia is not controlled, cerebral infarction (stroke) may result (see Chapter 35 for further information on cerebral ischemia and stroke).

Herniation Syndrome

Herniation syndrome occurs when pressure builds within the cranium, exceeding the brain's ability to compensate for the increase in pressure and causing brain tissue to be displaced. Cushing's triad refers to the three late signs of herniation: increased pulse pressure, decreased heart rate, and an irregular respiratory pattern. It is of critical importance to identify early signs of increased ICP (such as change in level of consciousness) in order to prevent herniation syndrome. The examination findings of a patient with increased ICP differ significantly from those of a patient with herniation syndrome (Table 36-2).

Cerebral herniation syndrome is classified according to the brain structures involved. The most common syndromes in the setting of critical care and trauma are uncal and central herniation (Table 36-3). Herniation of the medial temporal lobe (uncus) through the tentorium and into the brainstem is called uncal herniation. Herniation of the uncus through the tentorium results in pressure on the midbrain where the third cranial nerve exits, producing ipsilateral pupillary dilation. Contralateral hemiparesis occurs during uncal herniation. Central or tonsillar herniation describes the downward displacement of the cerebellar tonsils through the foramen magnum, causing compression of the brainstem. Clinical signs of central herniation syndrome include loss of consciousness, bilateral pupillary dilation, respiratory pattern changes or respiratory arrest, and flaccid paralysis. Central herniation may be caused by bilateral expanding lesions or a centrally located mass lesion causing downward displacement of the cerebral hemispheres and midline structures (ie, the basal ganglia, diencephalon, and the midbrain) through the tentorium (Fig. 36-4).

Critical care nurses have the opportunity to make a significant contribution to patient outcome by performing thorough serial neurological examinations, taking into account subtle changes. Once thought to be immediately fatal, herniation syndrome may be reversible in certain circumstances if it is identified early and aggressive therapies are administered.

Coma

Coma is an alteration in consciousness caused by damage to both hemispheres of the brain or the brainstem. Coma results from disruption of the reticular activating system (RAS), which is a physiologic region encompassing nuclei from the medulla to the cerebral cortex. The RAS is responsible for wakefulness, heightened arousal, and alertness. Consciousness spans a continuum from full consciousness to coma (see Chapter 33, Box 33-3). The states of coma can be subdivided into light coma, coma, and deep coma.

Persistent Vegetative State

Several terms describe a persistent vegetative state, such as irreversible coma or coma vigil. A persistent vegetative state is characterized by a period of sleep-like coma followed by a return to the awake state with an inability to respond to the environment. In a persistent vegetative state, higher cortical functions of the cerebral hemispheres have been damaged permanently, but the lower functions of the brainstem remain intact. The patient's eyes open spontaneously and may appear as if they are opening in response to verbal stimuli. Sleep–wake cycles exist, and the patient maintains normal cardiovascular and respiratory control. Also seen are involuntary lip smacking, chewing, and roving eye movements. A diagnosis of persistent vegetative state cannot be made for at least 4 weeks after onset of TBI and coma.[10]

The critical care nurse organizes resources for family and patient support such as pastoral care and social services. Support groups and assistance programs are often available for families of patients with TBI. Supporting the family in

Table 36-2 Increased Intracranial Pressure Versus Herniation Syndrome

	Increased Intracranial Pressure	Herniation Syndrome
Level of arousal	Increased stimulus required	Unarousable
Motor function	Subtle motor weakness or pronator drift	Dense motor weakness, posturing or absent response
Pupillary response	Sluggish pupillary response	Unilateral dilated and fixed pupil ("blown pupil")
Vital signs	May be stable or labile	Cushing's triad (increased systolic blood pressure, decreased heart rate, irregular respiration)

Used with permission from an unpublished lecture, Lower J, 1997.

Table 36-3 Herniation Syndromes

Name	Tissue Displaced	Common Causes	Clinical Signs
Central (transtentorial) herniation	Supratentorial	Compression and impaired blood flow to the brainstem Chronic increases in ICP Tumor in frontal, parietal, occipital lobes	Early altered alertness Respiratory sighs, yawns, pauses Roving eyes, small pupils Late sign: decorticate or decerebrate posturing
Uncal herniation	Supratentorial	Rapidly expanding lesions—hematoma	Early unilateral dilating pupil Once brainstem signs begin, deterioration is rapid
Upward cerebellar herniation	Infratentorial	Posterior fossa mass	Coma Cerebellar infarct if superior cerebellar arteries occluded Hydrocephalus with involvement of sylvian aqueduct
Tonsillar herniation	Infratentorial	Elevated ICP Expanding mass	Cranial nerve abnormalities Respiratory changes (apneustic/cluster breathing) Change in level of consciousness (rapid)

the process of gathering information and making decisions is an essential role of the multidisciplinary critical care team.

▲ Assessment

Physical Examination

Two essential tenets of neurological assessment are (1) level of consciousness is the most sensitive indicator of increased ICP and (2) maximal stimulus must be applied to achieve the maximal patient response.

Performing serial neurological examinations that include evaluation of level of consciousness and motor and cranial nerve function is necessary to identify increased ICP and prevent herniation syndrome. The GCS (see Chapter 33, Box 33-4) is useful for assessing trends of neurological function over time; however, focal motor deficits are not taken into consideration. The advantages of the GCS are ease of use and proven consistency across evaluators.

Assessment of Cognitive Function

Cognitive function is usually assessed by asking three orientation questions regarding person, place, and time. However,

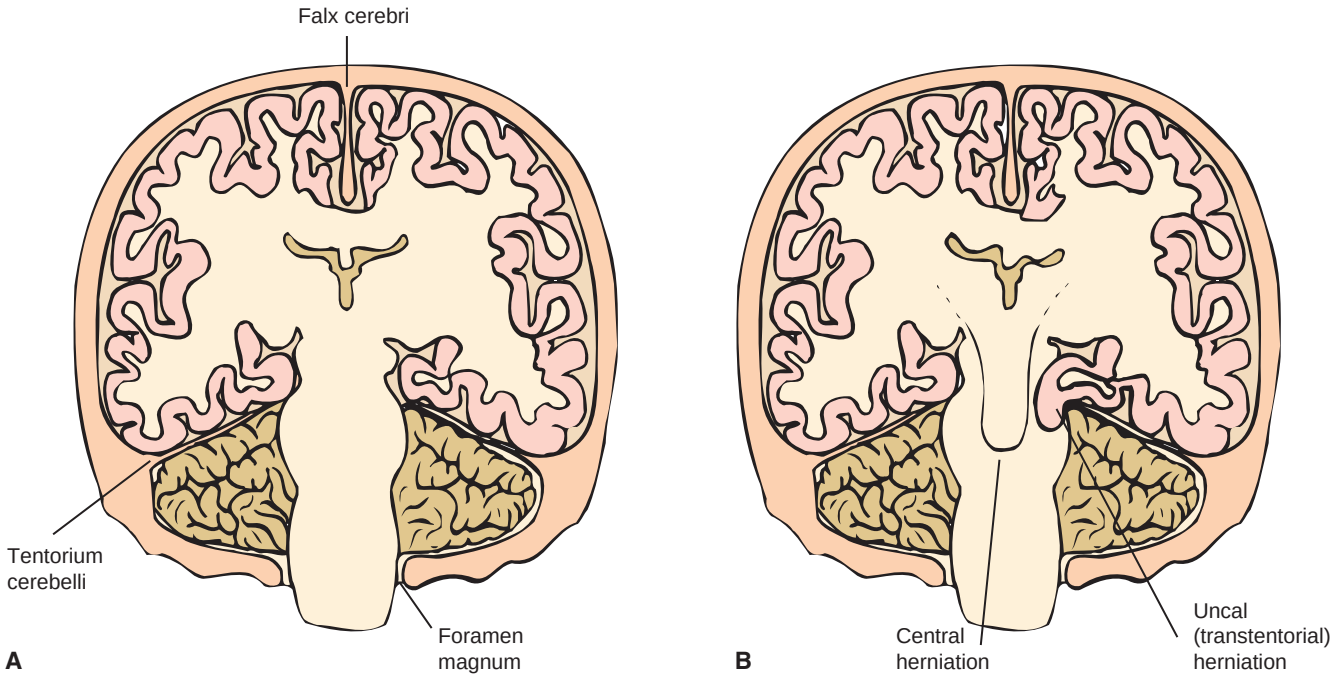


FIGURE 36-4 ▲ **A:** Normal brain. **B:** Herniated brain. Herniation associated with brainstem compression is called *central herniation*, whereas herniation associated with the supratentorial structures is called *uncal* (*transtentorial*) *herniation*.

it is necessary to elicit an "embroidered" or specific history from the patient to facilitate the detection of subtle changes over time. Patients may learn to answer the same questions correctly because of repetition but may continue to be confused when questioned further. Instead of asking the patient to state his or her location, the nurse may ask the patient to recall what type of place he or she is in or ask the name of the hospital, the city, and the state. Asking the patient to name his or her children, spouse, or close family members may also be helpful.

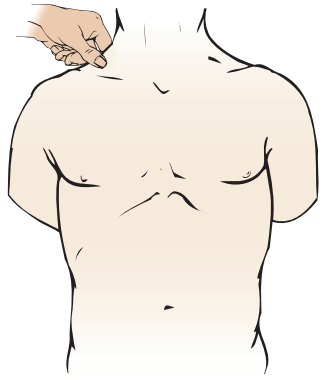
Assessment of Level of Arousal

Assessment of arousal determines a patient's capacity for wakefulness. A maximum stimulus must be applied in a systematic and escalating fashion to effectively elicit the patient's best, or maximal, response. A patient should be stimulated first by calling his or her name (in the same manner as you would try to wake a person who is sleeping), then by shouting the name (as you would to wake a "sound sleeper"), next by shaking, and finally by applying central pain. This staged approach affords the patient the opportunity to demonstrate increasing wakefulness or his or her best response. If the patient awakens readily, the ability to follow simple commands is assessed by asking the patient to move his or her extremities or "show two fingers." When asking a patient to grip or squeeze the evaluator's hand, it is important to make sure that the person can squeeze and release the grip. Patients with injury to the frontal lobe may have damaged the area of grasp inhibition, which develops in infancy. In this instance, the patient would grasp because of a reflex instead of a voluntary action.

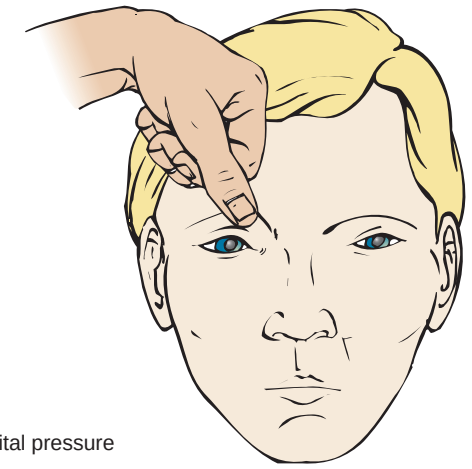
If a painful stimulus must be applied, the following techniques are useful: Squeeze the belly of the trapezius muscle with the thumb and first finger where the neck and shoulder meet, apply pressure over the supraorbital notch, or perform a sternal rub (Fig. 36-5). If a response is not elicited with these maneuvers, pressure may be applied to the nail beds of the patient's fingers or toes by placing a pencil on the nail and rolling it back and forth while applying pressure. Movement elicited by nail bed pressure is a result of activation of a spinal cord reflex. A painful stimulus should be applied for 15 to 30 seconds before the patient is considered not to have a motor response. Patients with brain injury may exhibit delayed responses to stimuli.

Assessment of the Eyes

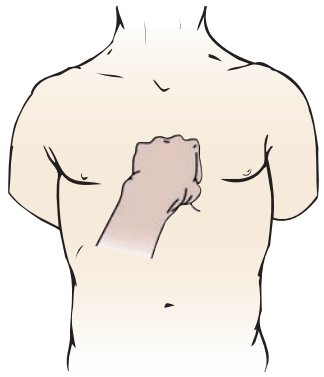
Assessment of the eyes includes evaluation of the pupils and extraocular movements, which assists in localizing cranial nerve dysfunction. Testing of cranial nerve II (the optic nerve) involves detection of gross visual field defects and visual acuity. Visual fields can be adequately assessed by the patient's ability to detect movement of the evaluator's finger in each field of vision (see Chapter 33 for technique). Visual



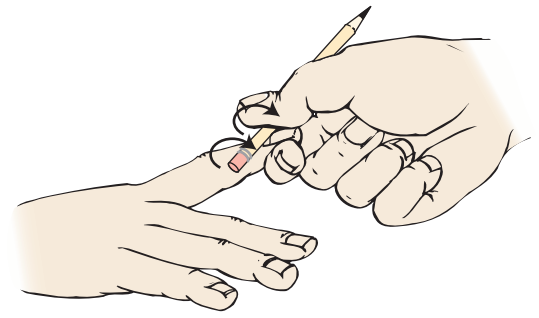
A. Trapezius squeeze



B. Supraorbital pressure



C. Sternal rub



D. Nailbed pressure

FIGURE 36-5 ▲ Methods of applying a painful stimulus. **A:** Trapezius squeeze. **B:** Application of supraorbital pressure. **C:** Sternal rub. **D:** Application of pressure to the nail bed.

acuity can be grossly assessed by asking the patient to read printed words on a page or by using a Snellen eye chart. If there is concern about optic nerve impairment, a full evaluation by an ophthalmologist is recommended.

Evaluation of cranial nerve III (the oculomotor nerve) involves inspection of the pupil, including size, shape, equality, and reaction to light. Increased ICP can cause irregularities in shape, pupillary inequality (anisocoria), and sluggish or absent reaction to light. Cranial nerves III, IV, and VI (the oculomotor, trochlear, and abducens nerves) enable movement of the eyes. Cranial nerves III and IV exit at the level of the midbrain, and cranial nerve VI exits at the level of the pons. Assessment of these nerves is accomplished by asking the patient to follow the evaluator's finger while it is moved in an "H" pattern. Double vision (diplopia) is a sign of eye muscle weakness and cranial nerve impairment.

In the comatose patient, the following tests are performed to evaluate cranial nerves III, VI, and VIII (the oculomotor and abducens nerves, and the vestibular portion of the acoustic nerve). The oculocephalic reflex (ie, the "doll's eyes" phenomenon; see Chapter 33, Fig. 33-6) is tested by moving the head from side to side in a horizontal plane (after confirming the absence of cervical spinal fracture). If the oculocephalic response is present, the eyes move together in the opposite direction of the head as it is turned from side to side. Absence of eye movement on head turning reflects brainstem dysfunction. The oculovestibular reflex (see Chapter 33, Fig. 33-7) is tested by instilling cold water into each ear and observing the eyes for movement. A normal oculovestibular response is characterized by movement of the eyes toward the stimulus with nystagmus. The absence of movement signals loss of function of the vestibular portion of the eighth cranial nerve as well as the brainstem.

Assessment of Brainstem Responses

The brainstem can be further assessed in the unconscious patient by testing corneal, cough, and gag reflexes. The corneal reflex reflects function of cranial nerves V and VII (the trigeminal and facial nerves), which exit the brain at the level of the pons. This reflex is tested by passing a wisp of cotton over the lower conjunctiva of each eye. Movement of the lower eyelid indicates the presence of the reflex. Sensation of the irritating stimulus represents gross function of one branch of the trigeminal nerve, and movement of the lower eyelid represents motor function of the facial nerve. Care must be taken in testing the corneal reflex to avoid corneal abrasions.

Cranial nerves IX and X (the glossopharyngeal and vagus nerves) exit at the level of the medulla and are responsible for the cough and gag reflexes and protection of the airway from aspiration. The cough and gag reflexes should be evaluated in the awake and unconscious patient.

Assessment of Motor Function

Motor function is evaluated by using the staged approach described earlier. Further detailed assessment of motor function is tested in the awake and cooperative patient by having the patient move his or her extremities against gravity and with passive resistance, grading the movement on a scale of 1 to 5 (see Chapter 33).

The unresponsive patient may exhibit localization, withdrawal, flexor posturing, or extensor posturing in response to noxious stimuli. Localization of a painful stimulus is observed as a purposeful response in which the patient is able to locate the source of pain and move toward it with one or both extremities crossing the midline of the body. A patient may try to remove the evaluator's hand when he or she performs a trapezius squeeze, or the patient may attempt to grab medical equipment (eg, catheters or endotracheal tubes). A withdrawal response is characterized by movement away from a painful stimulus. Flexor (decorticate) posturing is indicative of diffuse cortical injury and is characterized by the bending or flexing of the upper extremities and extension of the lower extremities and feet. Extensor (decerebrate) posturing indicates injury to the brainstem and is observed as extension and internal rotation of the upper extremities and extension of the lower extremities and feet (see Chapter 33, Fig. 33-1). It is possible that a patient may exhibit one type of movement in one extremity and another type of movement in another extremity. Presence of the Babinski reflex may also be observed in the patient with severe TBI.

Assessment of Respiratory Function

Assessment of respiratory patterns is important in detecting worsening neurologic injury and the need for airway management and mechanical ventilation. Numerous locations in both cerebral hemispheres regulate voluntary control over the muscles used in breathing. The cerebellum synchronizes and coordinates the muscles involved in respiration. The cerebrum controls the rate and rhythm of respiration. Nuclei in the pons and midbrain regulate the automaticity of respiration.

Abnormal respiratory patterns may be correlated with injured regions of the brain (Fig. 36-6). Cheyne-Stokes breathing is periodic breathing in which the depth of each breath increases to a peak and then decreases to apnea. The hyperpneic phase usually lasts longer than the apneic phase. This breathing pattern may be seen in patients with bilateral lesions located deep in the cerebral hemispheres. Compression of the midbrain can cause central neurogenic hyperventilation. Hyperventilation is sustained, regular, rapid, and deep. It is usually caused by a lesion above the midbrain. Apneustic breathing is characterized by respiration with a long pause at full inspiration or full expiration. The etiology of this pattern is loss of all cerebral and cerebellar control of breathing, with respiratory function at the brainstem level only. Cluster breathing may be seen in a patient when the lesion is high in the medulla or low in the pons. This pattern of respiration is seen as gasping breaths with irregular pauses.

The critical centers of inspiration and expiration are located in the medulla oblongata. Rapidly expanding intracranial lesions, such as cerebellar hemorrhage, can compress the medulla, resulting in ataxic breathing. This irregular breathing consists of both deep and shallow breaths with irregular pauses. This pattern of respiration signals the need for definitive airway control (ie, endotracheal intubation).

Assessment of Other Body Systems

In addition to thorough assessment of the central nervous system, comprehensive assessment of all other body systems is crucial in the early identification of complications in patients with TBI. Organ dysfunction, particularly respiratory failure, is common in patients with severe TBI.[11]

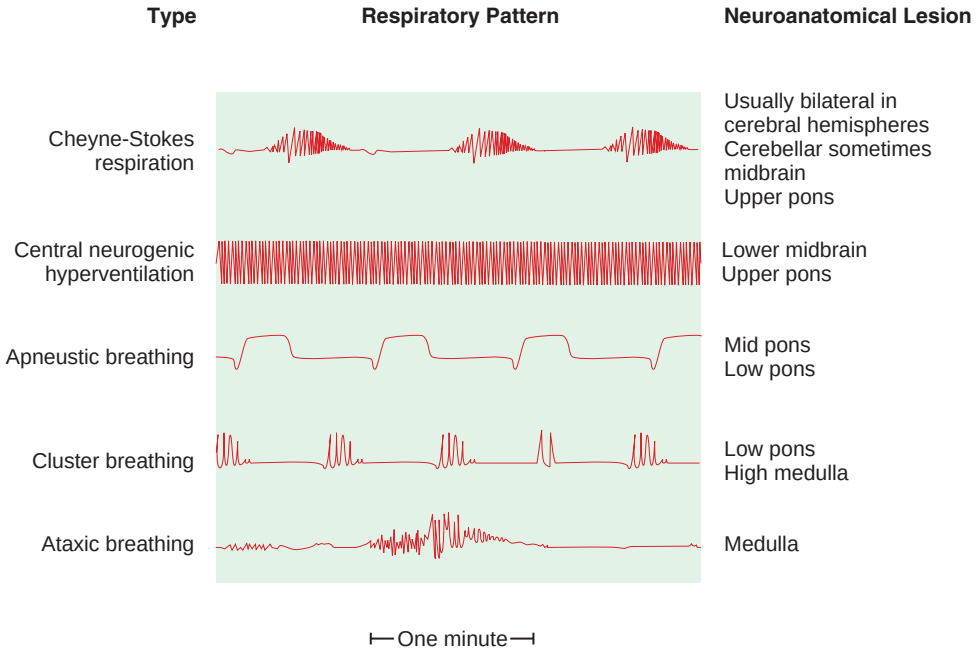


FIGURE 36-6 ▲ Injury to the brainstem can result in various abnormal respiratory patterns.

Diagnostic Testing

CT is performed as an initial diagnostic test to identify structural injuries in the brain and intracranial bleeding. A CT scan can be obtained quickly. One disadvantage of the CT scan is that it does not provide adequate views of the cerebelli and brainstem. An initial CT scan is performed without contrast. CT scans performed with intravenous contrast are used to investigate suspected masses (ie, tumors or abscesses). MRI is useful to assess structures in the posterior fossa and spinal cord. Magnetic resonance angiography may be used to evaluate cerebral vascular injuries such as carotid or vertebral dissection.

Cerebral angiography is the gold standard diagnostic test to investigate injuries to cerebral blood vessels. A cerebral angiogram may also be obtained to confirm the absence of cerebral blood flow in brain death.

Transcranial Doppler (TCD) ultrasonography indirectly evaluates cerebral blood flow and autoregulatory mechanisms by measuring the velocity at which blood travels through blood vessels. TCD may also be used to document cessation of blood flow to the brain.

Other diagnostic tests are used to assess electrical impulse transmission in the brain. These tests are often obtained to provide information for patient prognosis. Neurophysiological tests include the electroencephalogram (EEG), brainstem auditory evoked responses (BAERs), and somatosensory evoked potentials (SSEPs). The EEG measures electrical activity in all regions of the cortex and is useful in identifying seizures and correlating the abnormal neurological examination with abnormal cortical function. The EEG is necessary in ruling out subclinical or nonconvulsive seizures in the comatose patient. It may also be used as a confirmatory test in brain death to demonstrate cessation of electrical conduction to the cerebral cortex. A common finding in a patient with TBI is slowing of electrical activity in the area of injury. BAER and SSEP are useful prognostic tests in a patient with TBI. Abnormal results of either of these tests may help confirm a diagnosis of severe brainstem or cortical dysfunction.

Global cerebral oxygenation can be measured using jugular venous oxygen saturation (SjO_2), which is determined by inserting an intravenous catheter into the internal jugular vein and directing it upward toward the brain. The SjO_2 is indicative of oxygen extraction in the brain; in the healthy brain, SjO_2 is 55% to 70%.[12] If SjO_2 is less than 55%, cells are not extracting oxygen from the hemoglobin molecule efficiently (see Chapter 17 for discussion of oxygen consumption). If SjO_2 is greater than 70%, the rate of oxygen extraction is increased. An increase in cerebral oxygen extraction often occurs as a result of cerebral ischemia.

Measurement of oxygen delivery and extraction in brain tissue using an oxygen sensor placed in the deep white matter may be used as an adjunct to ICP and cerebral perfusion pressure (CPP) monitoring. Therapies aimed at keeping the partial pressure of brain tissue oxygen ($PbtO_2$) greater than 15 to 20 mm Hg in conjunction with ICP and CPP management may optimize the patient's chances for maximal functional recovery.[13,14]

In addition, microdialysis is used to measure cellular metabolites, such as glutamate, as markers of cellular dysfunction and injury. A small microdialysis catheter is inserted into the brain tissue through a hole made in the skull. Small amounts of CSF-like fluid are infused at very slow rates by a specialized infusion pump, and extracellular fluid is pumped out into a collection chamber. Currently, this monitoring modality primarily is used in the context of research.

▲ Management

Guidelines for the management of severe TBI have been developed by the Brain Trauma Foundation and the American Association of Neurological Surgeons to disseminate evidence-based recommendations.[14] The goal of these guidelines is to create a consistent standard for the care and treatment of patients with severe TBI. Several studies have suggested the benefit of evidence-based standardization of

care in improving functional outcomes after TBI.[15] Specific guidelines for managing severe TBI in infants, children, and adolescents are available, outlining the unique needs of the pediatric population.[16] The focus of the discussion in this chapter is the management of severe TBI in adults.

Initial Management

Initial assessment and treatment of the patient with TBI begins immediately after the insult, often with prehospital care providers. Specific guidelines for prehospital management of TBI were revised and published by the Brain Trauma Foundation in 2007. Prehospital treatment of the patient with a head injury focuses on rapid neurological assessment, definitive airway management, and treatment of hypotension.[17] These guidelines emphasize early correction of hypoxia and hypercarbia, which have been shown to affect morbidity and mortality in patients with TBI (Fig. 36-7).[7]

Airway management is a crucial initial step to prevent hypoxia and hypercarbia, which exacerbate secondary brain injury. Initial mechanical ventilation strategies aim to maintain normal ventilation or a partial pressure of carbon dioxide ($PaCO_2$) within normal limits (35 to 45 mm Hg). Signs

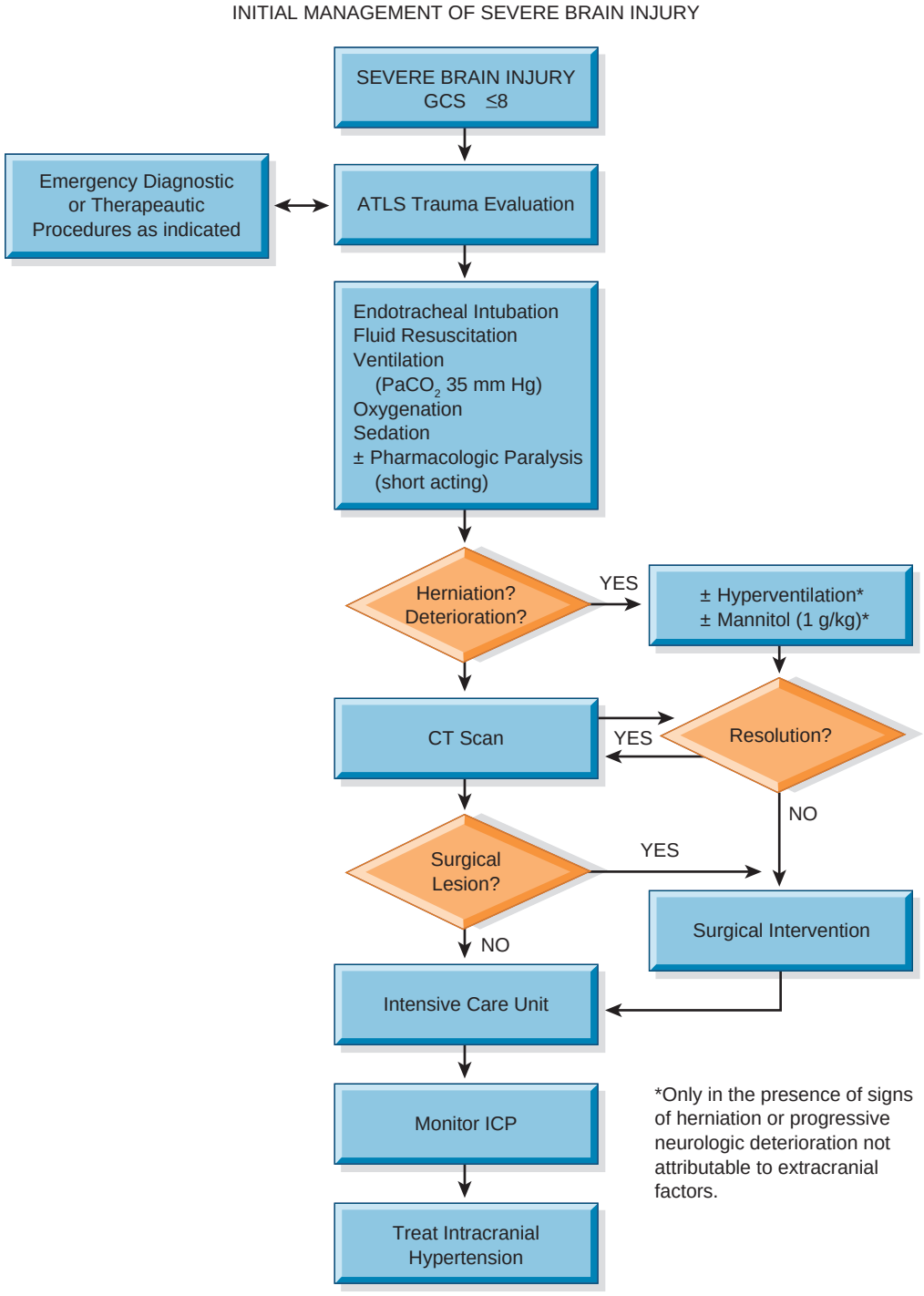


FIGURE 36-7 ▲ Flowchart for resuscitation of the patient with a severe head injury before ICP monitoring. (© 2000 Brain Trauma Foundation, Inc. Used with permission.)

of cerebral herniation may necessitate hyperventilation therapy ($PaCO_2$, 30 to 35 mm Hg). The goal of hyperventilation in TBI is to decrease $PaCO_2$, causing constriction of cerebral blood vessels and decreased cerebral blood volume. Decreased blood volume in the brain results in decreased ICP. Global cerebral vasoconstriction places healthy regions of brain tissue at risk for developing ischemia and should not be used prophylactically.[14] Continuous surveillance of end tidal carbon dioxide ($EtCO_2$) or frequent assessment of $PaCO_2$ is essential to prevent cerebral ischemia. Monitoring cerebral oxygenation (ie, SjO_2 or $PbtO_2$) is an option for identifying cerebral ischemia. Research suggests that the brain experiences decreased blood flow in the first 24 hours after injury; therefore, hyperventilation should be avoided during this period.[14]

Diagnostic testing is performed subsequent to the initial resuscitation to evaluate the need for immediate surgical intervention. Typical tests include radiographs of the cervical spine and a CT scan of the brain, which is useful in diagnosing intracranial bleeding that may require surgical intervention. Additional imaging and blood tests may be obtained to rule out systemic injuries and assist in treating complications. The mechanism of injury helps determine appropriate diagnostic testing.

Continuing management seeks to control ICP, promote cerebral perfusion, and correct the primary pathological process. Examples of nursing diagnoses for the patient with TBI are given in Box 36-2. General management of the patient with TBI requires a holistic, multisystem, multidisciplinary approach, taking into consideration the unique physiological and psychosocial characteristics of the patient (Box 36-3).

Monitoring and Controlling Intracranial Pressure

ICP monitoring, which is discussed in depth in Chapter 34, allows the health care team to make rapid treatment decisions based on pressure displays and ICP waveform analysis. ICP monitors are typically inserted by neurosurgeons at the bedside or in the operating room. ICP monitoring is recommended for patients with severe head injury (GCS score < 8) and CT scan abnormalities on admission. ICP monitoring may also be considered when the CT scan is normal but the patient meets two or more of the following criteria: older than 40 years, posturing, or a systolic blood pressure less than 90 mm Hg.[14]

Nursing interventions to manage increased ICP include maintaining body alignment as well as avoiding sharp turning of the head to one side and sharp hip flexion. Turning the head to one side causes compression of the jugular vein, preventing drainage of venous blood from the head, increasing ICP. Sharp hip flexion increases intra-abdominal pressure, decreasing venous outflow and causing an increase in ICP.

BOX 36-2 EXAMPLES OF NURSING DIAGNOSES

For the Patient With a Head Injury

- Risk for Ineffective Cerebral Tissue Perfusion
- Ineffective Airway Clearance related to diminished airway protective reflexes
- Risk for Infection related to multiple indwelling monitoring devices
- Impaired Skin Integrity related to physical immobilization
- Imbalanced Nutrition: Less Than Body Requirements related to increased energy expenditure
- Acute Pain related to injury
- Disturbed Sleep Pattern related to ICU routine care and environment
- Interrupted Family Processes related to acute crisis
- Anticipatory Grieving related to uncertain prognosis and critical illness

Maintaining Cerebral Perfusion

Management of cerebral perfusion involves control of ICP and maintenance of mean arterial pressure (MAP). Cerebral perfusion pressure (CPP) is calculated by subtracting ICP from MAP: CPP = MAP − ICP. Maintenance of CPP within the range of 50 to 70 mm Hg prevents cerebral ischemia at the lower end and mitigates the risk for acute respiratory distress syndrome (ARDS), which has been shown to occur more frequently when CPP is pushed over the upper limit of 70 mm Hg.[14]

Preventing and Treating Seizures

Seizures during the early stages of TBI can have severe negative effects on ICP and cerebral metabolic demands. Evidence-based guidelines support the use of antiseizure medication in the first 7 days after TBI.[14] Seizures that occur after this initial period are called late posttraumatic seizures and are not prevented by prophylactic administration of antiseizure medications.[14,18]

Phenytoin is one of the most common drugs used in the acute period. Phenytoin is usually given as a bolus dose intravenously, followed by a maintenance dosing schedule. The patient is monitored closely for hypotension, bradycardia, rashes, and IV infiltration during and after administration. Hypotension can be mitigated by administering the drug slowly (no greater than 50 mg/min). Truncal rashes with varying severity, including Stevens-Johnson syndrome, can occur with administration of phenytoin. The drug should be discontinued at the appearance of rash. The drug, fosphenytoin, is administered IV or IM and is metabolized in the body to phenytoin. Fosphenytoin can be administered rapidly without the infusion site reactions associated with phenytoin.

Maintaining a Normal Body Temperature

Hyperthermia (body temperature >37.5°C) in a patient with severe TBI increases cerebrometabolic demands and may compound secondary brain injury. Frequent monitoring of body temperature is necessary to maintain normothermia (35°C to 37.5°C). Infection must be ruled out as the cause of fever, and cooling methods are used as needed to maintain a normal body temperature. Inducing hypothermia in patients with TBI may be beneficial in improving functional

BOX 36-3 *COLLABORATIVE CARE GUIDE for the Patient With a Head Injury*

Outcomes	Interventions
Oxygenation/Ventilation	
Patient will maintain a patent airway. Lungs will be clear to auscultation. Arterial pH, PaO_2, and SaO_2 will be maintained within normal limits. $ETCO_2$ or PCO_2 will be maintained within prescribed range. There will be no evidence of atelectasis or pneumonia on chest x-ray.	• Auscultate breath sounds every 2–4 h and as needed. • Hyperoxygenate before and after each suction pass. • Avoid suction passes >10 s. • Monitor ICP and CPP during suctioning and chest physiotherapy. • Provide meticulous oral hygiene. • Monitor for signs of aspiration. • Encourage nonintubated patients to use incentive spirometer, cough, and deep breathe every 4 h and as needed. • Turn side-to-side every 2 h. • Move patient out of bed to chair one to two times daily when ICP has been controlled.
Circulation/Perfusion	
Patient will exhibit normal sinus rhythm without ectopy or ischemic changes. Patient will not experience thromboembolic complications.	• Monitor for myocardial ischemia and dysrhythmias due to sympathetic activation and catecholamine surges. • Prevent DVT with the use of pneumatic compression devices, antiembolism stockings, and subcutaneous heparin. • Implement early mobilization. Facilitate moving to a chair one to two times daily. • Monitor blood pressure continuously by arterial line or frequently by noninvasive cuff. • Monitor oxygen delivery (hemoglobin, SaO_2, cardiac output). • Administer red blood cells, inotropes, intravenous fluids as indicated.
Cerebral Perfusion/Intracranial Pressure	
CPP will be >60 mm Hg. ICP will be <20 mm Hg. Patient will not experience seizure activity.	• Monitor ICP and CPP every hour. • Perform neurological checks every 1–2 h. • Elevate the head of bed to 30 degrees unless contraindicated. • Maintain proper body alignment, keeping the head in a neutral position, and avoiding sharp hip flexion. • Maintain normothermia. • Maintain a quiet environment, cluster care, and provide rest periods. • Provide sedation as necessary and as prescribed. • Administer prophylactic antiepileptic agents as prescribed to prevent seizure activity.
Fluids/Electrolytes	
Serum electrolytes will be within normal limits. Serum osmolality will remain within prescribed range.	• Strict documentation of input/output; consider insensible losses from intubation, fever, and the like. • Monitor serum electrolytes, glucose, and osmolality as ordered. • Consider need for electrolyte replacement therapy and administer per physician order or protocol.
Mobility/Safety	
There will be minimal and transient changes in ICP/CPP during treatments or patient care activities. ICP/CPP will return to baseline within 5 min. Patient will not experience complications related to prolonged immobilization (eg, DVT, pneumonia, ankylosis). Patient will not harm self by dislodging medical equipment or falling.	• Provide range of motion and functional splinting for paralyzed limbs or patients in a coma. • Position patient off of pressure points at least every 2 h. • Consider use of specialty mattresses based on skin and risk factor assessments. • Keep bed rails in the upright position. • Provide restraints if necessary to prevent dislodgment of medical devices as policies permit.

outcome, although further research is required before this practice is recognized as a standard.[14]

Identifying and Managing Sympathetic Storming

Patients with severe TBI may experience a condition known as sympathetic storming. This condition is characterized by diaphoresis; agitation, restlessness, or posturing; hyperventilation; tachycardia; and fever. Sympathetic storming occurs as a result of an imbalance of the sympathetic and parasympathetic nervous systems. The precise cause of this imbalance is poorly understood; however, theories have suggested that TBI causes disruption in autonomic relay pathways and injury to the cerebral cortex, which limits control of autonomic function.[19] Triggers of a storming episode may include any stressful event, such as endotracheal suctioning, turning, development of fever, or alarm sounds in the patient's room.[19] The diagnosis of sympathetic storming is typically based on the appearance of suggestive signs and symptoms. Treatment focuses on finding a medication regimen that suppresses the sympathetic nervous system while avoiding adverse effects such as hypotension and bradycardia. Medication regimens may include one or more of the following drug classes: alpha-adrenergic blockers, beta blockers, opiates, sedatives, gamma-aminobutyric acid agonists, and dopamine agonists. Nursing management of the patient with sympathetic storming includes monitoring and assessing the patient to determine the effectiveness of the medication regimen, reducing environmental stimuli to reduce triggers of storming episodes, and preventing complications such as skin breakdown or injury from restlessness or agitation. Patients experiencing sympathetic storming may appear uncomfortable and evoke concern in family members; therefore, family education is important.

Monitoring Fluid and Electrolyte Status

Administration of osmotic diuretics, insensible fluid loss, and pituitary gland dysfunction may be responsible for fluid and electrolyte disturbances in patients with TBI. Strict monitoring of intake and output, as well as hemodynamic monitoring, guides the health care team in prescribing adequate fluid replacement. Routine monitoring of serum osmolality is helpful in preventing excessive systemic dehydration when administering osmotic diuretics or hypertonic saline. Surveillance of serum electrolytes allows for early identification and treatment of electrolyte abnormalities.

Disorders of sodium imbalance are common in the patient with TBI (Table 36-4). Hyponatremia most commonly occurs as a result of the syndrome of inappropriate antidiuretic hormone secretion (SIADH), in which antidiuretic hormone (ADH) is released in excessive amounts, resulting in hemodilution.[20] Hemodilution leads to a lower concentration of sodium in the blood. SIADH often is a transient phenomenon that can be treated with fluid restriction.

Cerebral salt-wasting syndrome may also cause hyponatremia. The precise physiologic mechanism of cerebral salt-wasting syndrome is poorly understood but involves a primary loss of sodium and free water through the kidneys. Treatment of this disorder requires fluid and sodium replacement in amounts that equal losses.[20,21]

Diabetes insipidus (DI) is a cause of hypernatremia and hypovolemia that occurs commonly in patients with injury or ischemia in the pituitary gland. Herniation syndrome often causes direct compression of the pituitary gland or compression to the supplying blood vessels. Damage to the pituitary gland prevents or decreases the secretion of ADH. DI is diagnosed by increasing serum sodium level, low urine specific gravity, and increased urine output. Treatment of DI includes aggressive fluid replacement that matches hourly fluid losses and the administration of exogenous ADH (vasopressin). Vasopressin may be given intravenously, subcutaneously, or intranasally, depending on the severity of the disorder.

Managing Cardiovascular Complications

Myocardial stunning and a transient decrease in cardiac function may occur in severe TBI. Inversion of T waves and ST-segment elevation or depression may be noted. Serum cardiac enzyme levels, electrocardiography, and echocardiography may be used to evaluate myocardial function. Hemodynamic monitoring devices, such as arterial and central lines and pulmonary artery catheters, may be used to guide medical therapies during the critical phases of TBI.

Disorders of coagulation are a significant concern in patients with TBI causing the release of large amounts of thromboplastin in response to brain injury. Disseminated intravascular coagulation may result. If hypothermia is

Table 36-4 Disorders of Sodium Imbalance: Comparison of Diabetes Insipidus, the Syndrome of Inappropriate Antidiuretic Hormone Secretion, and Cerebral Salt-Wasting Syndrome

	Diabetes Insipidus	Syndrome of Inappropriate Antidiuretic Hormone Secretion	Cerebral Salt-Wasting Syndrome
Urinary output	Increased	Decreased	Increased
Specific gravity	Decreased	Increased	Decreased
Volume status	Decreased	Mildly increased	Decreased
Serum sodium	Increased	Decreased	Decreased
Treatment	Administration of exogenous vasopressin, fluid replacement	Fluid restriction, judicious sodium replacement	Fluid and sodium replacement

employed, it is important to recognize that coagulopathies may be exacerbated as the body temperature decreases.

Prophylaxis of deep venous thrombosis (DVT) is an essential component in the care of patients with head injuries, who are often immobile for extended periods. Sequential compression devices provide intermittent pulsatile pressure to the lower extremities, increasing venous return and promoting systemic fibrinolysis. Antiembolic stockings, anticoagulant administration, and early mobilization are recommended to prevent DVT and pulmonary emboli.[14]

Managing Pulmonary Complications

Pulmonary complications in the patient with TBI include pneumonia, acute respiratory distress syndrome (ARDS), neurogenic pulmonary edema, and pulmonary embolus. Pulmonary toilet, vigilant oral hygiene, and monitoring of endotracheal tube cuff pressure are necessary to prevent nosocomial pneumonia and mitigate pulmonary complications in patients with head injuries who require prolonged mechanical ventilation (see Chapter 25 for a discussion of the causes and prevention of ventilator-associated pneumonia).

Early mobility is critical in facilitating pulmonary toilet, preventing atelectasis, and preventing pulmonary emboli due to DVT. Early consideration of extubation to reduce the number of days on mechanical ventilation as well as early planning for tracheostomy in patients unable to protect their airway may prevent additional pulmonary complications.[14]

ARDS is a hypoxic lung disease resulting from the activation of the inflammatory cascade, causing leakage of protein-rich fluid from the pulmonary capillaries into the interstitium of the lungs as well as destruction of alveolar cells. There are many causes of ARDS in patients with head injuries, including concomitant pulmonary contusion, aspiration pneumonia, sepsis, and massive blood transfusion. Medical management of ARDS may involve the use of pressure modes of mechanical ventilation to decrease the volume needed to deliver each breath (high volumes have been implicated in furthering alveolar injury); see Chapter 27 for a complete discussion of ARDS management.

Neurogenic pulmonary edema may result from injury to the brainstem, increased ICP, or an increase in sympathetic tone that causes a catecholamine surge at the time of trauma. Neurogenic pulmonary edema often presents as "flash pulmonary edema" because it has a sudden onset. This type of pulmonary edema is thought to be caused by massive vasoconstriction due to an acute increase in ICP causing activation of the sympathetic nervous system. Consequently, there is marked increase in systemic afterload resulting in left ventricular failure. Pulmonary edema resulting from left ventricular failure is exacerbated by an increase in pulmonary capillary permeability, causing further edema.[22] Treatment includes judicious use of low-dose diuretics. The condition is typically self-limiting in patients without cardiac disease.

Multidisciplinary care of the patient with TBI with respect to pulmonary complications requires the involvement of the nursing and the physician teams; the respiratory therapist; the occupational therapist; the physical therapist (for early mobilization); and the speech–language pathologist (to address issues with aspiration).

Managing Nutrition and Maintaining Glycemic Control

Head injury is thought to cause hypermetabolic and hypercatabolic states as well as a decrease in immunocompetency.[23] Morbidity and mortality may significantly increase if nutritional requirements are not met. Indirect calorimetry is useful in determining resting energy expenditure (REE).[23] Indirect calorimetry is performed using a machine (metabolic cart) that interfaces with the ventilator to measure oxygen consumption and carbon dioxide production.

Nutrition in the form of enteral or parenteral nutrition is administered in amounts that meet metabolic needs within 5 to 7 days of the injury.[14,23] Enteral feeding may prevent the translocation of bacteria from the intestines to the bloodstream as well as prevent gastrointestinal ulceration and bleeding.[24] Early nutrition starting within 5 days of injury may reduce mortality and be one of the few treatment options that can directly affect mortality in TBI patients.[23] Current recommendations suggest replacement of 140% of REE in patients who are not paralyzed and 100% of REE in patients who are paralyzed.[14] Recognition of the importance of nutrition and multidisciplinary collaboration with a nutrition support team are essential to optimize patient outcome. Research suggests a detrimental effect of hyperglycemia on morbidity and mortality of patients with TBI; however, specific treatment thresholds have not been established. Hyperglycemia with a blood glucose exceeding 200 mg/dL and hypoglycemia should be avoided in patients with TBI.[14,25,26]

Managing Musculoskeletal and Integumentary Complications

Comprehensive assessment of the musculoskeletal and integumentary systems is necessary to prevent skin breakdown and other complications, such as contractures. Collaboration with other disciplines, such as occupational and physical therapy, is also essential in developing a plan of care to prevent or mitigate the effects of immobility on the skin and musculoskeletal systems. Splinting of the hands and feet in an unresponsive patient is necessary to preserve musculoskeletal function and ensure the best conditions for future rehabilitation. Functional splinting and range-of-motion exercises also help reduce dependent edema in immobile extremities. Frequent turning of patients, even in the critical phase of the illness, is integral in maintaining skin integrity and facilitating pulmonary drainage.

Caring for the Family

Caring for families in crisis, as well as coordinating available services (such as social work and pastoral care), is an important function of the critical care nurse. Bond et al[27] surveyed the needs of family members of patients with severe TBI and found the following four needs:

- The need for specific truthful information
- The need for information to be consistent
- The need to be actively involved in care
- The need to make sense of the entire experience.

BOX 36-4 NURSING INTERVENTIONS

For Sensory Stimulation

Sound

- Explain to the patient what you are going to do.
- Play the patient's favorite television or radio program for 10 to 15 minutes. Alternatively, play a recording of a familiar voice of a friend or family member.
- During the program, do not converse with others in the room or perform other activities of patient care. The goal is to minimize distractions so the patient may learn to attend to the stimulus selectively.
- Another approach is to clap your hands or ring a bell. Do this for 5 to 10 seconds at a time, moving the sound to different locations around the bed.

Sight

- Place a brightly colored object in the patient's view. Present only one object at a time.
- Alternatively, use an object that is familiar, such as a family photo or favorite poster.

Touch

- Stroke the patient's arm or leg with fabrics of various textures. Alternatively, the back of a spoon can simulate smooth texture and a towel rough texture.
- Rubbing lotion over the patient's skin will also stimulate this sense. For some, firm pressure may be better tolerated than very light touch.

Smell

- Hold a container of a pleasing fragrance under the patient's nose. Use a familiar scent, such as perfume, aftershave, cinnamon, or coffee.
- Present this stimulation for very short periods (1 to 3 minutes maximum).
- If a cuffed tracheostomy or endotracheal tube is in place, the patient will not be able to appreciate this stimulation fully.

Critical care nurses have an opportunity to meet all of these specific needs and to change unit culture to meet these needs. Encouraging family members to touch the patient or allowing family members to assist in providing sensory stimulation (Box 36-4) may be helpful and comforting to some family members. Finding opportunities to involve family members in the patient's plan of care may also be therapeutic for the patient and the family. The Ranchos Los Amigos Scale can be used by the critical care nurse to describe the stages of coma as they relate to rehabilitative methods and interventions (Table 36-5). Attention is given to including both spiritual and cultural needs in the plan of care.

A patient with TBI may be discharged to home, a rehabilitation program, or a nursing facility depending on the severity of his or her neurologic deficits. Families must be informed and educated about the expected course of events and potential scenarios for continued care after the acute hospitalization, especially when the patient has severe TBI. Family resources, as well as other support systems and services available to the patient, should be assessed early in all patients with TBI to facilitate a smooth transition into the next stage of care. Social workers and case managers play an integral role in obtaining information and communicating with the patient, the family, and the multidisciplinary team.

▲ Brain Death

A patient's condition may be so severe that brain death is the final outcome. The critical care nurse continues to provide nursing care to the patient as treatment is continued or life support measures are withdrawn.

In the past, the declaration of brain death was controversial with regard to the standardization of tests needed to make the decision and ethical considerations. The Uniform Determination of Death Act was developed in 1981 by the President's Commission for the Study of Ethical Problems in Medicine and Biomedical Behavior Research and adopted by all 50 states. This act states: "An individual, who has sustained either (1) irreversible cessation of circulatory and respiratory functions, or (2) irreversible cessation of all functions of the entire brain, including the brainstem, is dead. A determination of death must be made in accordance with accepted medical standards."[28]

The brain death examination seeks to confirm the following three cardinal findings: coma or unresponsiveness, absence of brainstem reflexes, and apnea.[29] Tests specific for brain death include, but are not limited to, motor testing; evaluation of pupillary responses; evaluation of the oculocephalic reflex ("doll's eyes" phenomenon); evaluation of the oculovestibular reflex (caloric ice-water test); evaluation of the corneal, cough, and gag reflexes; and apnea testing. Electrolyte abnormalities, hypothermia or hyperthermia, severe hypotension, or the presence of medications in amounts that could cause coma must be resolved before brain death testing can be performed. Apnea testing is performed by removing the patient from the ventilator, inspecting the chest for spontaneous respiratory effort while providing supplemental oxygen, and monitoring for an increase in $PaCO_2$. Baseline acid–base balance is established with an arterial blood gas (ABG) measurement prior to removal from the ventilator, and then serial ABG measures are obtained. A $PaCO_2$ greater than 60 mm Hg or an increase in the $PaCO_2$ of 20 mm Hg or more above the patient's baseline $PaCO_2$ is regarded as a positive test, supporting the diagnosis of brain death.[29] The patient is simultaneously observed for spontaneous respiration and hemodynamic instability, which may cause the test to be aborted. An increased $PaCO_2$ is the single strongest stimulus for the initiation of breathing; therefore, the absence of respiratory effort in the presence of severe hypercarbia constitutes strong evidence of brain death. Confirmatory tests for brain death, such as cerebral angiography (to test for the absence of cerebral blood flow), TCD ultrasonography, EEG, BAER, and SSEP, can be used if any doubt exists after a full clinical examination has been completed.

The American Academy of Neurology recommends repeating the clinical evaluation for brain death after 6 hours.[29] Time of death is recorded at the time that brain death is declared. Different institutions specify requirements based on state laws and statutes for physicians declaring brain death. Brain death determination in pediatric patients differs from that in adults because of the increased viability of

Table 36-5 Ranchos Los Amigos Scale

Level	Guidelines for Interacting With Patient
1. **No response** to any stimuli occurs.	• Assume that the patient can understand all that is said. Converse with, not about, the patient.
2. **Generalized response.** Stimulus response is incoherent, limited, and nonpurposeful with random movements or incomprehensible sounds.	• Do not overwhelm the patient with talking. Leave some moments of silence between verbal stimuli.
3. **Localized response.** Stimulus response is specific but inconsistent; patient may withdraw or push away, may make sounds, may follow some simple commands, or may respond to certain family members.	• Manage the environment to provide only one source of stimulation at a time. If talking is taking place, the radio or television should be turned off. • Provide short, random periods of sensory input that are meaningful to the patient. A favorite television program or recording or 30 min of music from the patient's favorite radio station will provide more meaningful stimulation than constant radio accompaniment, which becomes as meaningless as the continual bleep of the cardiac monitor.
4. **Confused–agitated.** Stimulus response is primarily to internal confusion with increased state of activity; behavior may be bizarre or aggressive; patient may attempt to remove tubes or restraints or crawl out of bed; verbalization is incoherent or inappropriate; patient shows minimal awareness of environment and absent short-term memory.	• Be calm and soothing when handling the patient. Approach with gentle touch to decrease the occurrence of defensive emotional and motor reflexes. • Watch for early signs that the patient is becoming agitated (eg, increased movement, vocal loudness, resistance to activity). • When the patient becomes upset, do not try to reason with him or her or "talk him or her out of it." Talking will be an additional external stimulus that the patient cannot handle. • If the patient remains upset, either remove him or her from the situation or remove the situation from him or her.
5. **Confused, inappropriate–nonagitated.** Patient is alert and responds consistently to simple commands; however, patient has a short attention span and is easily distracted; memory is impaired and patient exhibits confusion of past and present events; patient can perform previously learned tasks with maximal structure but is unable to learn new information; may wander off with vague intention of "going home."	• Present the patient with only one task at a time. Allow time to complete it before giving further instructions. • Make sure that you have the patient's attention by placing yourself in view and touching the patient before talking. • If the patient becomes confused or resistant, stop talking. Wait until he or she appears relaxed before continuing with instruction or activity.
6. **Confused–appropriate.** Patient shows goal-directed behavior but still needs external direction; can understand simple directions and reasoning; follows simple directions consistently and requires less supervision for previously learned tasks; has improved past memory depth and detail and basic awareness of self and surroundings.	• Use gestures, demonstrations, and only the most necessary words when giving instructions. • Maintain the same sequence in routine activities and tasks. Describe these routines to the patient and relate them to time of day.
7. **Automatic–appropriate.** Patient is able to complete daily routines in structured environment; has increased awareness of self and surroundings but lacks insight, judgment, and problem-solving ability.	• Supervision is still necessary for continued learning and safety. • Reinforce the patient's memory of routines and schedules with clocks, calendars, and a written log of activities.
8. **Purposeful–appropriate.** Patient is alert, oriented, and able to recall and integrate past and recent events; responds appropriately to environment; still has decreased ability in abstract reasoning, stress tolerance, and judgment in emergencies or unusual situations.	• The patient should be able to function without supervision. • Consideration should be given to job retraining or a return to school.

the immature brain.[30] Specific guidelines for the determination of brain death in children were developed by a federal task force in 1987 and updated in 2011. These guidelines delineate physical examination features unique to pediatric patients as well as specific time frames for observation when brain death is suspected, depending on the age of the child.[30]

The concept of brain death is often confusing for families because death is typically associated with cardiopulmonary death. Therefore, the language used in discussions is very important. Some family members may interpret the term "brain dead" to mean that the rest of the body can continue to live, so care must be taken to assess the understanding and coping behaviors of family members.[31]

The discussion of brain death should be separated in time from conversations regarding the opportunities for organ donation. It is essential to work closely with an organ procurement organization to provide the most complete and accurate information regarding organ donation.

▲ Clinical Applicability Challenges

CASE STUDY

Mr. M. is a 21-year-old male who was transported to the emergency department (ED) after being assaulted. Emergency medical service personnel placed a rigid collar to immobilize his cervical spine. On arrival in the ED, Mr. M. was unresponsive. His vital signs were blood pressure of 116/76; heart rate, 61; respiratory rate, 22 breaths/min; and temperature, 35.8°C orally. Neurological examination showed no movement to noxious stimulation of his upper and lower extremities; pupils were small, sluggishly reactive to light (cranial nerve III); corneal reflex intact in each eye (cranial nerves V and VII); and intact cough and gag reflexes (cranial nerves IX and X). The Glasgow Coma Scale (GCS) score was 5.

Because of Mr. M's depressed level of consciousness and GCS score, he was intubated using rapid-sequence intubation technique and mechanical ventilation was begun. Secondary survey was performed to detect other obvious traumatic injuries and did not reveal additional deformities. An emergency head CT scan was obtained and revealed a left fronto-temporal subdural hematoma, bilateral frontal contusions with shifting of the midline brain structures, and right uncal herniation. Radiographs of the patient's cervical spine were obtained to rule out vertebral fracture. No fractures were revealed nor was there dislocation of the vertebrae.

Based on the patient's clinical neurological examination and CT findings, neurosurgeons took him emergently to the operating room to evacuate the subdural hematoma and performed a left frontotemporal decompressive craniectomy with durotomy and a ventriculostomy (external ventricular drain) was placed to monitor ICP and allow for drainage of cerebrospinal fluid if necessary.

Mr. M. was admitted to the intensive care unit (ICU) postoperatively. His GCS was now 6T (eye opening = 2, best motor response = 3, flexion to pain, best verbal response = 1T). Both pupils were now large and sluggishly reactive to light. An infusion of propofol was initiated at 20 mcg/kg/hour for sedation. ICP readings remained stable in the range of 10–15 mm Hg.

On ICU day 3, Mr. M continued to be comatose and his intracranial pressure rose to 35 mm Hg. Drainage of CSF from the ventriculostomy was performed until the ICP was sustained in the range of 15–20 mm Hg. A head CT revealed an increase in cerebral edema. A dose of mannitol, 0.5 grams/kg was administered and maintenance IV fluids were increased from 100 mL/h to 125 mL/hour in an attempt to prevent systemic dehydration which could cause a decrease in blood pressure leading to vasodilation of cerebral blood vessels and increased ICP. On day 7, the propofol infusion was weaned to off and the patient was no longer requiring frequent drainage of CSF to maintain normal ICP. On day 8, the ventriculostomy was discontinued and on day 10 tracheostomy and gastrostomy tubes were inserted to facilitate prolonged mechanical ventilation and nutrition support. Physical and occupational therapy were consulted to begin the rehabilitation process.

On ICU day 18, his neurologic examination revealed localization to pain, spontaneous eye opening with tracking of his caregivers from side to side.

On ICU day 20, Mr. M. was transferred to a rehabilitation facility specializing in the care of patients with traumatic brain injury. Mr. M. began to follow simple commands, such as showing two fingers, and he was alert and oriented to person and place. Eventually, he was able to have his tracheostomy removed and he was able to continue outpatient physical and occupational therapy to improve motor and cognitive deficits.

1. Based on the radiological findings and your knowledge of the pathophysiology of head injury, explain why Mr. M. has a decreased level of consciousness.

2. Name at least three multisystem complications from having a severe TBI that Mr. M. may experience.

3. Using the description of the patient's neurologic status at the beginning and end of the case study, place the patient into the appropriate level on the Ranchos Los Amigos Scale (see Table 36-5). Name two interventions that could be used when interacting with patients in these categories.

References

1. Faul M, Xu L, Wald MM, et al: Traumatic Brain Injury in the United States: Emergency Department Visits, Hospitalizations, and Deaths. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control, 2010
2. Manson PN, Stanwix MG, Yaremchuk MJ, et al: Frontobasal fractures: Anatomical classification and clinical significance. Plast Reconstr Surg 124(6):2096–2106, 2009
3. Barkhoudarian G, Hovda DA, Giza CC: The molecular pathophysiology of concussive brain injury. Clin Sports Med 30(1):33–48, 2011
4. Sigurdardottir S, Andelic N, Roe C, et al: Post-concussion symptoms after traumatic brain injury at 3 and 12 months post-injury: A prospective study. Brain Inj 23(6):489–497, 2009
5. Tsai TH, Lieu AS, Hwang SL, et al: A comparative study of patients with bilateral or unilateral chronic subdural hematoma: Precipitating factors and post-operative outcomes. J Trauma 68(3):571–575, 2010

6. Tong WS, Zheng P, Xu JF, et al: Early CT signs of progressive hemorrhagic injury following acute traumatic brain injury. Neuroradiology 53(5):305–309, 2011
7. Jeremitsky E, Omert L, Dunham CM, et al: Harbingers of poor outcome the day after severe brain injury: Hypothermia, hypoxia, and hypoperfusion. J Trauma 54(2):312–319, 2003
8. Ng SC, Poon WS, Chan MT: Cerebral hemisphere asymmetry in cerebrovascular regulation in ventilated traumatic brain injury. Acta Neurochir Suppl 96:21–23, 2006
9. Donkin JJ, Vink R: Mechanisms of cerebral edema in TBI: therapeutic developments. Curr Opin Neurol 23(3):293–299, 2010
10. Quality Standards Subcommittee of the American Academy of Neurology: Practice parameters: Assessment and management of patients in the persistent vegetative state [summary statement]. Report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology 45:1015–1018, 1995
11. Kemp CD, Johnson, JC, Riordan WP, et al: How we die: the impact of non-neurologic organ dysfunction after severe TBI. Am J Surg 74(9):866–872, 2008
12. Kidd KC, Criddle L: Using jugular venous catheters in patients with traumatic brain injury. Crit Care Nurse 21(6):17–22, 2001
13. Martini RP, Deem S, Yanez ND, et al: Management guided by brain tissue oxygen monitoring and outcome following severe traumatic brain injury. J Neurosurg 111(4):644–649, 2009
14. Brain Trauma Foundation: Guidelines for the management of severe traumatic brain injury, 3rd ed. J Neurotrauma 24(Suppl 1):s1–s106, 2007
15. Arabi YM, Haddad S, Tamim HM, et al: Mortality reduction after implementing a clinical practice guidelines-based management protocol for severe traumatic brain injury. J Crit Care 25(2):190–195, 2010
16. Kochanek PM, Carney N, Adelson PD, et al: Guidelines for the acute medical management of severe traumatic brain injury in infants, children, and adolescents, 2nd edition. Crit Care Med 13(1 Suppl):S1–S82, 2012
17. Badjatia N, Carney N, Crocco TJ, et al: Guidelines for prehospital management of traumatic brain injury 2nd edition. Prehosp Emerg Care 12:S1–S52, 2008
18. Chang BS, Lowenstein DH: Practice parameter: Antiepileptic drug prophylaxis in severe traumatic brain injury. Report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology 60(1):10–16, 2003
19. Baguley IJ, Heriseanu RE, Cameron ID, et al: A critical review of the pathophysiology of dysautonomia following traumatic brain injury. Neurocrit Care 8:293–300, 2008
20. Powner DJ, Boccalandro C, Alp MS, et al: Endocrine failure after traumatic brain injury in adults. Neurocrit Care 5:61–70, 2006
21. Agha A, Thornton, E, O'Kelly P, et al: Posterior pituitary dysfunction after traumatic brain injury. J Clin Endocrinol Metab 89:5987–5992, 2004
22. Mascia L: Acute lung injury in patients with severe brain injury: A double hit model. Neurocrit Care 11:417–426, 2009
23. Härtl R, Gerber LM, Ni Q, et al: Effect of early nutrition on deaths due to severe traumatic brain injury. J Neurosurg 109: 50–56, 2008
24. Krakau K, Omne-Ponten M, Karlsson T, et al: Metabolism and nutrition in patients with moderate and severe traumatic brain injury: A systematic review. Brain Injury 20:345–367, 2006
25. Griesdale DE, Tremblay MH, McEwen J, et al: Glucose Control and Mortality in Patients with Severe Traumatic Brain Injury. Neurocrit Care 11:311–316, 2009
26. Liu-DeRyke X, Collingridge DS, Orme J, et al: Clinical impact of early hyperglycemia during acute phase of traumatic brain injury. Neurocrit Care 11:151–157, 2009
27. Bond AE, Draeger CRL, Mandleco B, et al: Needs of family members of patients with severe traumatic brain injury: Implications for evidenced-based practice. Crit Care Nurse 23(4):63–71, 2003
28. Uniform Determination of Death Act. Presented and approved at the 89th Annual Conference of Commissioners on Uniform State Laws, July 26–August 1, 1980, Kauai, Hawaii. Chicago, IL: National Conference of Commissioners on Uniform State Laws, 1980
29. Wijdicks EF, Varelas PN, Gronseth GS, et al: American Academy of Neurology. Evidenced-based guideline update: determining brain death in adults: report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology 74(23):1911–1918, 2010
30. Nakagawa TA, Ashwal S, Mathur M, et al: Society of Critical Care Medicine, Section on Critical Care and Section on Neurology of American Academy of Pediatrics; Child Neurology Society. Clinical report-Guidelines for the determination of brain death in infants and children: an update of the 1987 taskforce recommendations. Pediatrics 128(3):e720–e740, 2011
31. Manuel A, Solberg S, MacDonald S. Organ donation experiences of family members. Nephrol Nurs J 37(3):229–236, 2010

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

37 Spinal Cord Injury

Janice J. Hoffman and Kathy A. Hausman

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Describe the mechanism of spinal cord injury (SCI).
2. Discuss the various classification systems for spinal cord injuries.
3. Differentiate between the following syndromes: central cord syndrome, Brown-Séquard syndrome, anterior cord syndrome, and posterior cord syndrome.
4. Differentiate between spinal shock, neurogenic shock, and orthostatic hypotension.
5. Perform an assessment of a patient with an SCI.
6. Develop a collaborative plan of care for a patient with an acute SCI.
7. Describe immediate nursing actions when the patient develops autonomic dysreflexia.
8. Explain other typical complications that occur after an SCI.

Spinal cord trauma is often a devastating injury resulting in permanent paralysis and disability. The estimated annual incidence of spinal cord injury (SCI) in the United States is 40 cases per million or approximately 12,000 new cases, and these numbers do not include those individuals who die at the accident scene.[1] According to the National Spinal Cord Injury Database (2009), there are about 262,000 people living with SCI in the United States.

SCI continues to most often affect young adults, but as the age of the general population has increased, so has the average age at injury of these people. From 1973 to 1979, the average was 28.7 years, while since 2005, the average age at injury increased to 40.7 years.[1] Males are overwhelmingly most often affected, composing 80.8% of those with injuries in the national database. An analysis of race/ethnicity reveals that since 2005, 66.2% were Caucasians, 27% were African American, 7.9% were Hispanic, and 2% were Asian.

Motor vehicle accidents are the most common etiology of SCI (41.3%), followed by falls (27.3%), acts of violence (15%), and sports (7.9%).[1] While the life expectancy of persons with SCI has increased over the years, it is still below those without SCI. Leading causes of death in these people include pneumonia, septicemia, pulmonary emboli, and renal failure. The mortality rates are highest during the first year after injury and greater in those with higher level spinal cord injuries.[1]

The average length of stay in acute care settings for patients after SCI is 12 days. They are then usually transferred to a rehabilitation unit/facility, where the average length of stay is 38 days. Most people (87.7%) discharged from rehabilitation settings reside in noninstitutional settings (most often their homes), with only 5% discharged to long-term care facilities. The cost of care differs significantly based upon the level of injury. Estimated first costs for patients with high tetraplegia (quadriplegia) (C1–C4) is $829,843, low tetraplegia (C5–C8) is $535,877, and paraplegia is $303,220; for succeeding years, the estimated annual cost is $148,645, $60,887, and $30,855, respectively.[1]

▲ Classification of Injury

Understanding the anatomy and physiology of the spinal cord is important to correlating the cord damage to the clinical presentation. The spinal cord extends from the base of the brain to approximately the level of the first or second lumbar vertebra. Blood is supplied to the cord by the anterior and posterior spinal arteries. Extending off of the spinal cord are the spinal nerve roots. The spinal cord is enclosed in the vertebral canal, which consists of 33 vertebrae: 7 cervical, 12 thoracic, 5 lumbar, 5 sacral (fused), and 4 coccygeal (fused). The vertebrae are held in place by ligaments, muscles, and other supporting structures.

Spinal cord injuries can be classified by mechanism, type of vertebral injury, level of injury, or cause. Spinal cord injuries occur as a result of penetrating injury or mechanical forces. Penetrating injuries, which are most often caused by gunshot or stab wounds, damage the spinal cord and cause loss of neurological functioning.

Mechanism of Injury

Mechanical forces that can result in SCI include hyperflexion, hyperextension, axial loading (compression), and rotational forces (Fig. 37-1):

- Hyperflexion, depicted in Figure 37-1A, is caused by a sudden deceleration of the head and neck and is often seen in patients who have sustained trauma from a head-on motor vehicle crash (MVC) or diving accident. The cervical region is most often involved, especially at the C5–C6 level.
- Hyperextension (see Fig. 37-1B) is the most common type of injury and can be caused by a fall, a rear-end MVC, or getting hit in the head (eg, during a boxing match). Hyperextension of the head and neck may cause contusion and ischemia of the spinal cord without vertebral column damage. Whiplash injuries are an example of a hyperextension injury.
- Axial loading, also known as compression (see Fig. 37-1C), typically occurs when a person lands on the feet or buttocks after falling or jumping from a height, or when there is a direct blow to the head. Injury results from vertebral column compression leading to a fracture that causes damage to the spinal cord.
- Rotational injuries result from forces that cause extreme twisting or lateral flexion of the head and neck (see Fig. 37-1D). Fracture or dislocation of vertebrae may also occur.

Type of Vertebral Injury

Mechanical forces can result in fracture or dislocation of vertebrae, or both. If vertebral injury occurs, the type of vertebral injury can be used to describe the person's SCI.

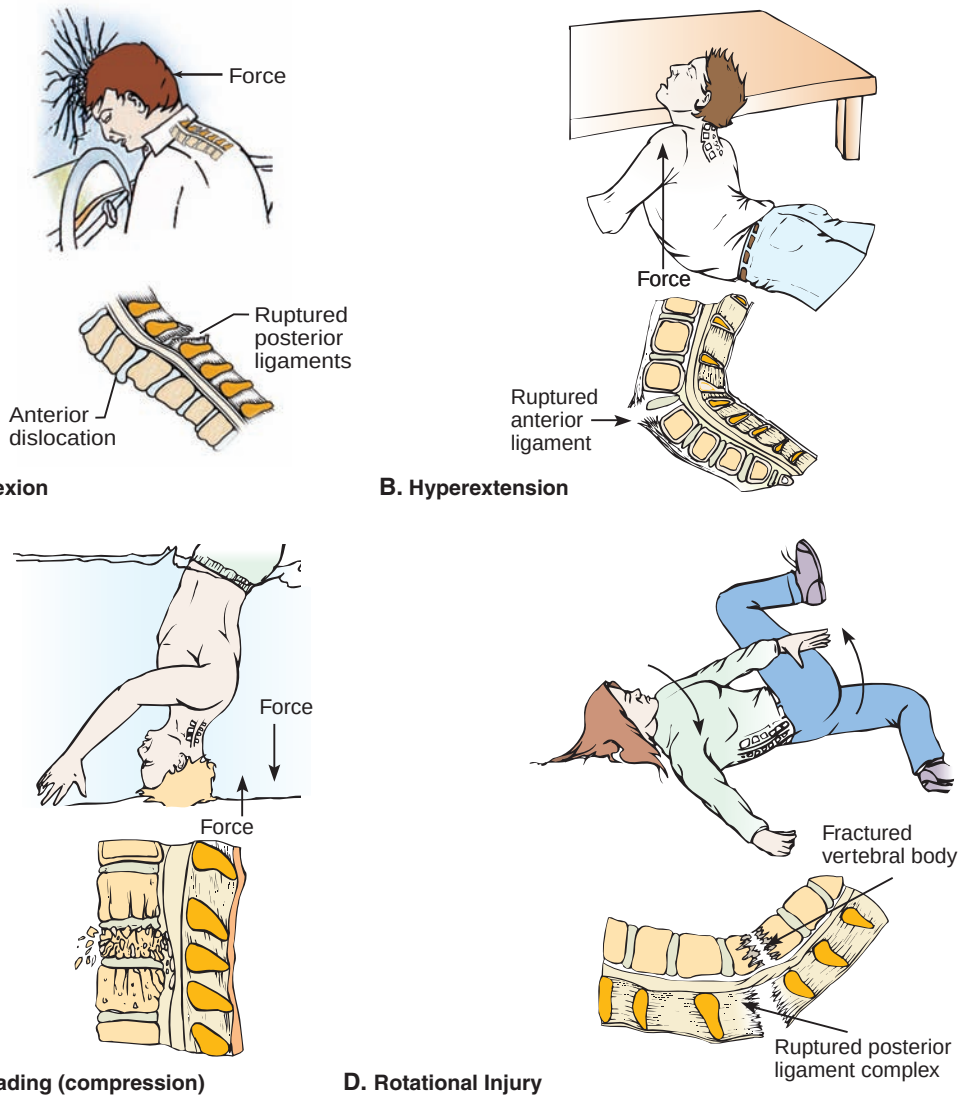


FIGURE 37-1 ▲ Spinal cord injuries can be classified according to the mechanism of injury. **A:** With hyperflexion to the cervical spine, there may be tearing of the posterior ligamentous complex, resulting in anterior dislocation. **B:** Hyperextension injury can result in rupture of the anterior ligament. **C:** Axial loading (compression) of the spine results in fracture and subsequent spinal cord damage. **D:** When rotational force occurs, there is concurrent fracture and tearing of the posterior ligamentous complex. (Adapted from Hickey JV: Clinical Practice of Neurological and Neurosurgical Nursing, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, pp 412–415.)

BOX 37-1 Types of Vertebral Fractures and Dislocations

Fractures

Simple fracture: single fracture; alignment of the vertebrae is intact, and neurological deficits do not occur
Compression fracture: fracture caused by axial loading and hyperflexion
Wedge compression fracture: a stable fracture that involves compression of the vertebral body in the cervical area
Teardrop fracture: an unstable fracture that involves a piece of bone breaking off the vertebra; seen in wedge fractures
Comminuted fracture: the vertebra is shattered into several pieces; bone fragments may be driven into spinal cord

Dislocations

Dislocation: one vertebra overrides another
Subluxation: partial or incomplete dislocation
Fracture–dislocation: fracture and dislocation

Box 37-1 presents definitions of types of fractures and dislocations. A fracture may be considered unstable if the posterior ligaments are torn.

Level of Injury

Spinal cord injuries can also be classified according to the segment of the spinal cord that is affected:

1. Upper cervical (C1–C2) injuries (atlas fractures, atlantoaxial subluxation, odontoid fractures, and hangman's fractures)
2. Lower cervical (C3–C8) injuries
3. Thoracic (T1–T12) injuries
4. Lumbar (L1–L5) injuries
5. Sacral (S1–S5) injuries

The degree of functional recovery depends on the location and extent of the injury. The level of SCI is determined by the effect of the injury on sensory and motor function (Table 37-1). Total loss of voluntary muscle control and sensation below the level of injury suggests that the lesion is complete. Complete lesions involving spinal cord regions C1 to T1 result in tetraplegia (Fig. 37-2). Complete lesions involving spinal cord regions T2 to L1 result in paraplegia (Fig. 37-2). In incomplete injuries, there is some motor or sensory function below the level of injury. The level of sensory loss seen in a person with

Table 37-1 Functional Loss From Spinal Cord Injury (Based on Complete Lesions)

Level of Spinal Injury	Motor Function	Deep Tendon Reflexes	Sensory Function	Respiratory Function	Voluntary Bowel and Bladder Function	Rehabilitative Potential
C1–C4	Tetraplegia: loss of all motor function from the neck down	All lost	Loss of all sensory function in the neck and below (C4 supplies the clavicles)	Loss of involuntary (phrenic) and voluntary (intercostals) respiratory function; ventilatory support and a tracheostomy needed	No bowel or bladder control	May be discharged home on a ventilator with home care
C5	Tetraplegia: loss of all function below the upper shoulders **Intact:** sternomastoids, cervical paraspinal muscles, and the trapezius; can control head	C5, C6 biceps	Loss of sensation below the clavicle and most portions of arms, hands, chest, abdomen, and lower extremities **Intact:** head, shoulders, deltoid, clavicle, portion of forearms (C5 supplies the lateral aspect of the arm)	Phrenic nerve intact, but not intercostal muscles	No bowel or bladder control	Use of extremity-powered devices to achieve some upper limb control Head control facilitates wheelchair (W/C) balance Adaptive tools, held in mouth, for typing and writing Some adaptive tools and use of special computer technology
C6	Tetraplegia: loss of all function below the shoulders and upper arms; lacks elbow, forearm, and hand control **Intact:** deltoid, biceps, and external rotator muscles of shoulders	C5, C6 brachioradialis	Loss of everything listed for a C5 lesion, but greater arm and thumb sensation **Intact:** head, shoulders, arms, palms of hands, and thumbs (C6 supplies the forearm and thumb)	Phrenic nerve intact, but not intercostal muscles	No bowel or bladder control	Needs assistive devices to use arms (may be able to help feed, groom, and dress self) Needs a motorized W/C Dependent for all transfers

(Continued on page 827)

Table 37-1 Functional Loss From Spinal Cord Injury (Based on Complete Lesions) (continued)

Level of Spinal Injury	Motor Function	Deep Tendon Reflexes	Sensory Function	Respiratory Function	Voluntary Bowel and Bladder Function	Rehabilitative Potential
C7	Tetraplegia: loss of motor control to portions of the arms and hands **Intact:** voluntary strength in shoulder depressors, shoulder abductors, internal rotators, and radial wrist extensors	C7, C8 triceps	Loss of sensation below the clavicle and portions of arms and hands **Intact:** head, shoulders, most of arms and hands (C7 supplies the middle finger)	Phrenic nerve intact, but not intercostal muscles	No bowel or bladder function	Can perform some activities of daily living (ADLs) Can use wrist extensor with a special splint to induce finger flexion Can push a W/C with special hand grasps May be able to drive a specially equipped car
C8	Tetraplegia: loss of motor control to portions of the arms and hands **Intact:** some voluntary control of elbow extensors, wrist, finger extension, and finger flexors		Loss of sensation below the chest and in portions of hands **Intact:** sensation to face, shoulders, arms, hands, and part of chest (C8 supplies the little finger)	Phrenic nerve intact, but not intercostal muscles	No bowel or bladder function	Able to push up in the W/C Improved sitting tolerance Can grasp and release hands voluntarily Independent in most ADLs from W/C Independent in use of W/C Can use hands for catheterization and rectal stimulation for bowel movements
T1–T6	Paraplegia: loss of everything below the midchest region, including the trunk muscles **Intact:** control of function to the shoulders, upper chest, arms, and hands		Loss of sensation below the midchest area **Intact:** everything to the midchest region, including the arms and hands (T1 and T2 supply the inner aspect of the arm; T4 supplies the nipple area)	Phrenic nerve functions independently Some impairment of intercostal muscles	No bowel or bladder function	Full control of upper extremities and completely independent in W/C Full-time employment possible Independent in managing urinary drainage and inserting suppositories Able to live in a dwelling without major architectural changes
T6–T12	Paraplegia: loss of motor control below the waist **Intact:** shoulders, arms, hands, and long trunk muscles		Loss of everything below the waist **Intact:** shoulders, chest, arms, and hands (T10 supplies the umbilicus; T12 supplies the groin area)	No interference with respiratory function	No bowel or bladder control	In addition to the previously described capabilities, there is complete abdominal and upper back control. Good sitting balance (allows for greater ease of W/C operation and athletics)
L1–L3	Paraplegia: loss of most control of legs and pelvis **Intact:** shoulders, arms, hands, torso, hip rotation and flexion, and some leg flexion	L2–L4 (knee jerk)	Loss of sensation to the lower abdomen and legs **Intact:** all of the above plus some sensation to the inner and anterior thigh (L3 supplies the knee)	No interference with respiratory function	No bowel or bladder control	Independent for most activities from W/C
L3–L4	Paraplegia: loss of control of portions of lower legs, ankles, and feet **Intact:** all of the above, plus increased knee extension		Loss of sensation to portions of the lower legs, feet, and ankles **Intact:** all of the above, plus sensation to the upper legs	No interference with respiratory function	No bowel or bladder control	Voluntary control of hip extensors; weak abductors Walking with braces possible

(Continued on page 828)

Table 37-1 Functional Loss From Spinal Cord Injury (Based on Complete Lesions) (continued)

Level of Spinal Injury	Motor Function	Deep Tendon Reflexes	Sensory Function	Respiratory Function	Voluntary Bowel and Bladder Function	Rehabilitative Potential
L4 to S5	Paraplegia: incomplete Segmental motor control L4 to S1: abduction and internal rotation of hip, ankle dorsiflexion, and foot inversion L5 to S1: foot eversion L4 to S2: knee flexion S1–S2: plantar flexion (ankle jerk) S2–S5: bowel/bladder control	S1–S2 (ankle jerk)	Lumbar sensory nerves innervate the upper legs and portions of the lower legs L5: medial aspect of foot S1: lateral aspect of foot S2: posterior aspect of calf/thigh Sacral sensory nerves innervate the lower legs, feet, and perineum	No interference with respiratory function	Bowel and bladder control possibly impaired S2–S4 segments control urinary continence S3–S5 segments control bowel continence (perianal muscles)	Can walk with braces or may use W/C Can be relatively independent

From Hickey JV: The Clinical Practice of Neurological and Neurosurgical Nursing, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, pp 428–429, with permission.

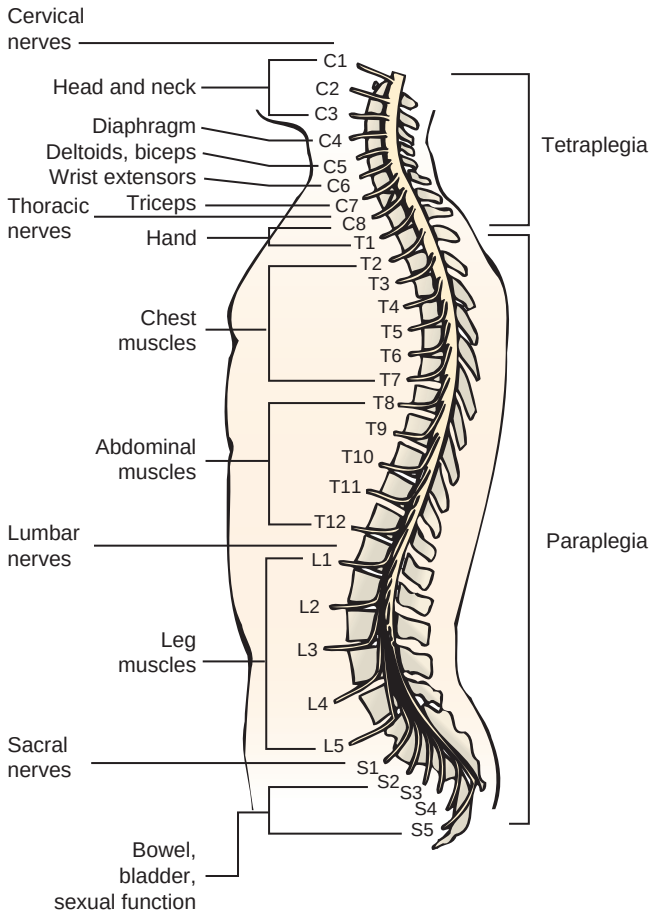


FIGURE 37-2 ▲ The level of spinal cord injury (SCI) relates to functional loss. The higher the SCI, the more motor, sensory, and autonomic functional losses are incurred. (Adapted from Hickey JV: Clinical Practice of Neurological and Neurosurgical Nursing, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 411.)

a complete cord injury follows the corresponding dermatome pathway as shown in Figure 37-3.

Cause of Injury

Spinal cord injuries are also classified according to the cause of injury. Causes of SCI include concussion or jarring injuries, compression of the neural elements by bony fragments or hemorrhage, contusion (bruising) of the spinal cord, and laceration, transection, or blockage of the blood vessels that supply the cord.

Functional Outcome

Spinal cord injuries may be classified as complete or incomplete injuries based upon functional outcome. Injuries that result in total loss of all sensory and motor function below the level of the injury are classified as complete injuries. The clinical presentation depends greatly upon the level of injury; patients with injuries at or above C4 are particularly vulnerable to complete respiratory failure resulting from loss of innervations to both the diaphragm (C2–C4) and the intercostal muscles (T1–T4). Incomplete cord injuries often cause recognizable neurological syndromes that are classified according to the area damaged (Fig. 37-4).

▲ Spinal Cord Syndromes

Central Cord Syndrome

Damage to the spinal cord in central cord syndrome is centrally located. Hyperextension of the cervical spine often is the mechanism of injury, and the damage is greatest to the

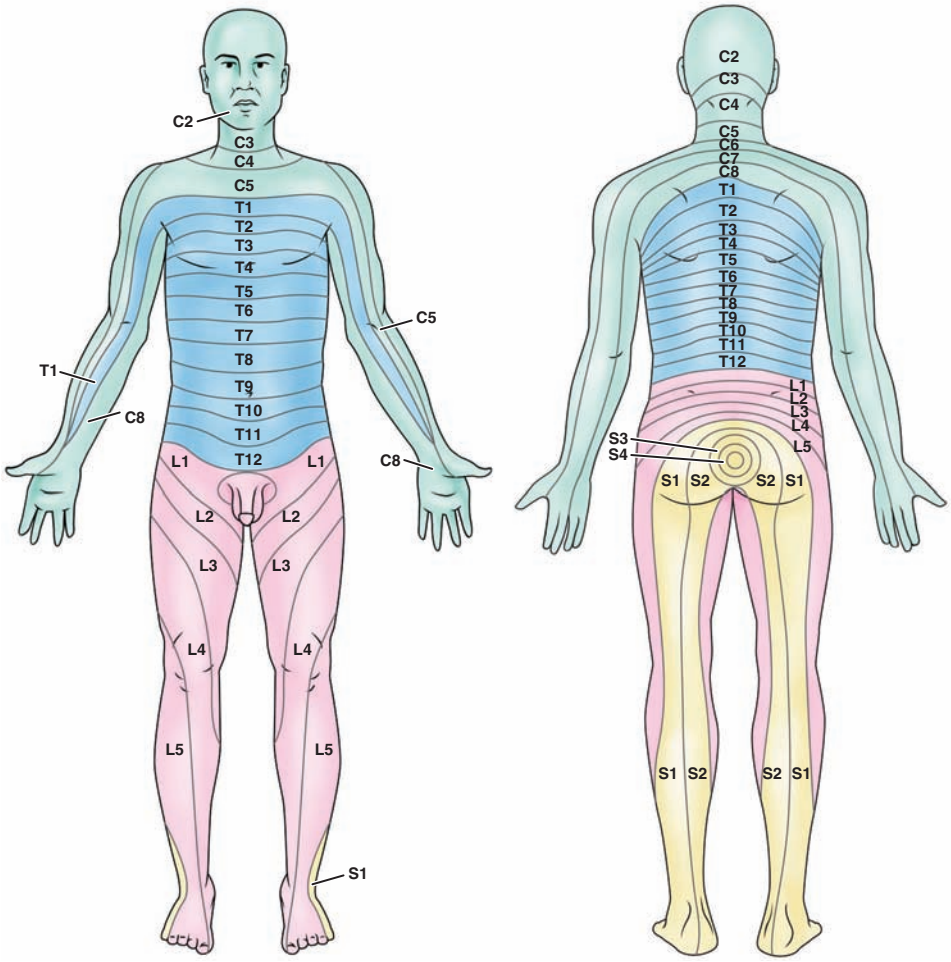


FIGURE 37-3 ▲ A person with complete SCI follows the dermatome pathways for the level of sensory loss. (From Smeltzer SC, Bare BG: Brunner & Suddarth's Textbook of Medical-Surgical Nursing, 12th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 1837.)

cervical tracts supplying the arms.[2] Clinically, the patient may present with paralyzed arms but with no deficit in the legs or bladder (see Fig. 37-4A).

Brown-Séquard Syndrome

The damage in Brown-Séquard syndrome is located on one side of the spinal cord. The clinical presentation is one in which the patient has either increased or decreased cutaneous sensation of pain, temperature, and touch on the same side of the spinal cord at the level of the lesion. Below the level of the lesion on the same side, there is complete motor paralysis. On the patient's opposite side, below the level of the lesion, there is loss of pain, temperature, and touch because the spinothalamic tracts cross soon after entering the cord. The posterior columns are interrupted ipsilaterally (on the same side), but this does not cause a major deficit because some fibers cross instead of running ipsilaterally. Clinically, the patient's limb with the best motor strength has the poorest sensation. Conversely, the limb with the best sensation has the poorest motor strength (see Fig. 37-4B).

Anterior Cord Syndrome

The area of damage in anterior cord syndrome is, as the name suggests, the anterior aspect of the spinal cord. Clinically, the patient usually has complete motor paralysis below the level of injury (corticospinal tracts) and loss of pain, temperature, and touch sensation (spinothalamic tracts), with preservation of light touch, proprioception, and position sense (see Fig. 37-4C).

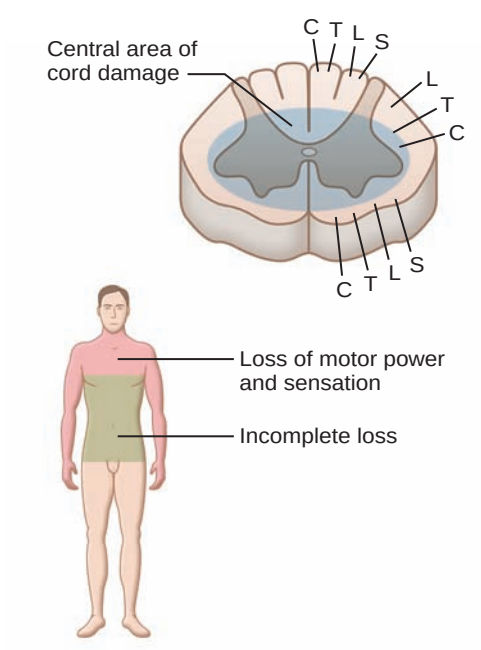
Posterior Cord Syndrome

Posterior cord syndrome is usually the result of a hyperextension injury at the cervical level and is not commonly seen. Position sense, light touch, and vibratory sense are lost below the level of the injury, while motor function and pain and temperature sensation remain intact (Fig. 37-4D).

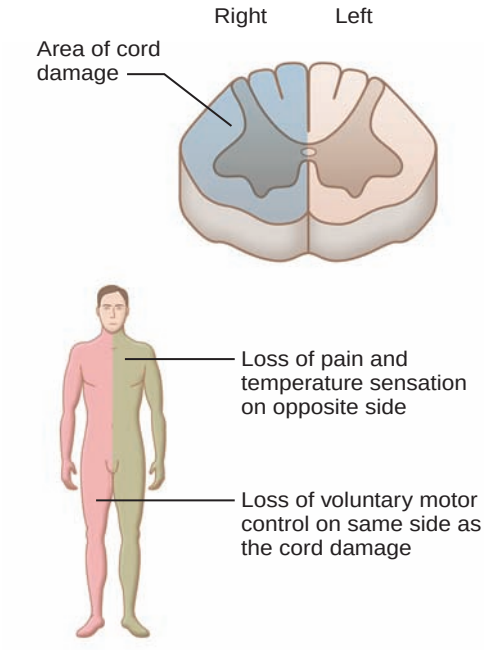
▲ Pathophysiology

Primary Injury

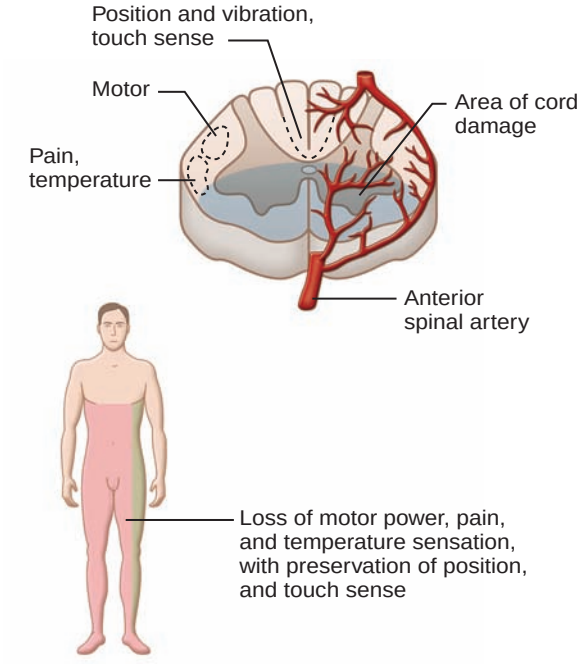
Injury to the spinal cord that occurs at impact is referred to as the primary injury and is most often associated with damage to the vertebral column. The vertebrae may be fractured, dislocated, or compressed leading to concussion, contusion, compression, laceration, or transaction of the spinal cord. The more mobile areas of the vertebral column (such as the cervical area) are most frequently involved.



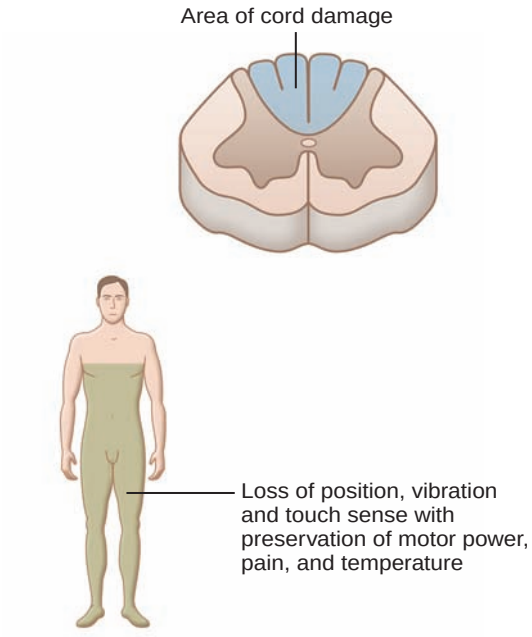
A. Central cord syndrome



B. Brown-Sèquard syndrome



C. Anterior cord syndrome



D. Posterior cord syndrome

FIGURE 37-4 ▲ Selected syndromes related to SCI. C, cervical; L, lumbar; S, sacral; T, thoracic. (Adapted from Hickey JV: Clinical Practice of Neurological and Neurosurgical Nursing, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, pp 424–425.)

Secondary Injury

Equally destructive is the secondary injury or damage to the spinal cord that continues for hours to days after the initial trauma. Complex vascular, inflammatory, and chemical processes result in additional axonal damage and further neurological deficit. Mechanisms of secondary injury include:

- Immune cells, which normally do not enter the spinal cord, engulf the area after an SCI. These immune cells respond as they normally do with inflammation in other parts of the body, resulting in release of regulatory chemicals, some of which are harmful to the spinal cord. Highly reactive oxidizing agents (free radicals) are produced, damaging the cell membrane and disrupting the sodium–potassium pump. Intracellular calcium increases secondary to disruption of sodium–potassium pump leading to release of vasoactive substances (catecholamines, histamine, and prostaglandins). This series of events ultimately leads to decreased blood flow to the spinal cord further exacerbating cord ischemia.

- Hypoperfusion of the spinal cord from microscopic hemorrhage and edema leads to cord ischemia. Ischemic areas develop at the injury site as well as one or two segments above and below the level of injury.
- The release of catecholamines and vasoactive substances (norepinephrine, serotonin, dopamine, and histamine) contributes to decreased circulation and impaired cellular perfusion of the spinal cord.
- The release of excess neurotransmitters leads to overexcitation of the nerve cells. Excitotoxicity allows high levels of calcium to enter the cells, causing further oxidative damage and damage to mitochondria. Excitotoxicity is thought to damage oligodendrocytes (the cells that produce myelin), leading to demyelinated axons that are unable to conduct impulses.

BOX 37-2 Clinical Manifestations of Spinal Shock

- Flaccid paralysis below the level of injury
- Absence of cutaneous and proprioceptive sensation
- Hypotension and bradycardia
- Absence of reflex activity below the level of injury; may cause urinary retention, bowel paralysis, and ileus
- Loss of temperature control; vasodilation and inability to shiver make it difficult for the patient to conserve heat in a cool environment, and the inability to perspire prevents normal cooling in a hot environment

▲ Autonomic Nervous System Dysfunction

Spinal Shock

Spinal shock is a condition that occurs immediately or within several hours of an SCI and is caused by the sudden cessation of impulses from the higher brain centers (Fig. 37-5). Due to a decrease in sympathetic innervation to the vascular system, massive vasodilation occurs that initiates a series of events including decreases in preload and stroke volume. The loss of sympathetic nervous system function, accompanied by unopposed parasympathetic nervous system stimulation to the heart leads to a decrease in heart rate, and also further decreases stroke volume. The vasodilation also leads to a decrease in afterload. Due to the changes in innervation, the patient develops hypotension and bradycardia. Spinal shock is a unique shock state as there is not the reflex tachycardia that usually accompanies the decrease in blood pressure. Characteristics include the loss of motor, sensory, reflex, and autonomic function below the level of the injury, with resultant flaccid paralysis (Box 37-2), and loss of bowel and bladder function also. Additionally, the body's ability to control temperature is lost, and the patient's temperature tends to equilibrate with that of the external environment (poikilothermia).

If the SCI produces an incomplete transection, the suppression of function below the level of injury is temporary, lasting a few days to weeks or months. The duration of spinal shock is variable, depending on the severity of the insult and the presence of other complications. The return of perianal reflex activity signals the end of the period of spinal shock. Reflexes associated with the area surrounding the injured cord return last. The skeletal muscles become spastic, and there is increased muscle tone and exaggerated flexor muscle movement.

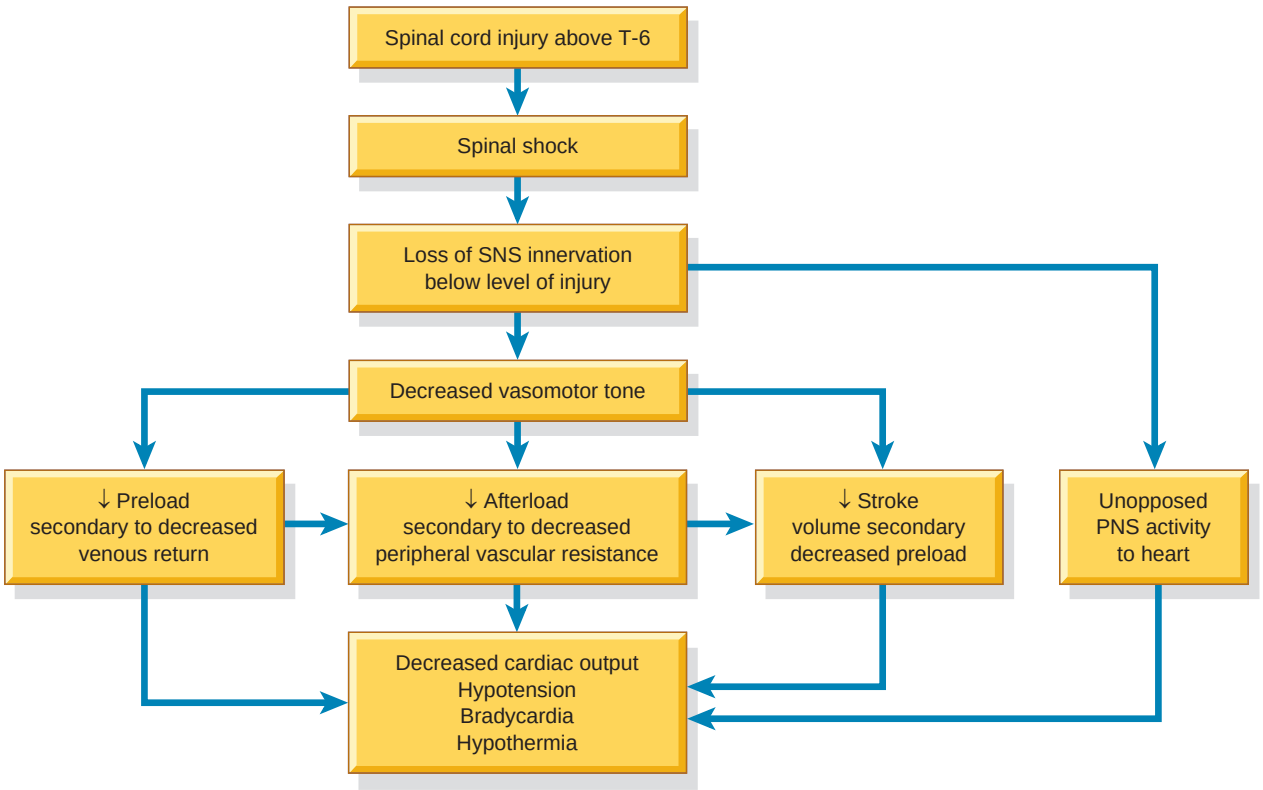


FIGURE 37-5 ▲ Mechanisms involved in spinal shock. PNS, parasympathetic nervous system; SNS, sympathetic nervous system.

Neurogenic Shock

Neurogenic shock, a form of distributive shock, is a condition seen in patients with severe cervical and upper thoracic injuries. It is caused by the loss of sympathetic input to the systemic vasculature of the heart and subsequent decreased peripheral vascular resistance. Signs and symptoms include hypotension, severe bradycardia, and loss of the ability to sweat below the level of injury. The same clinical findings pertaining to disruption of the sympathetic transmissions in spinal shock occur in neurogenic shock.

Orthostatic Hypotension

Orthostatic hypotension may occur in a patient with an SCI because the patient is unable to compensate for changes in position. The vasoconstriction message from the medulla cannot reach the blood vessels because of the cord injury.

▲ Initial Assessment and Management

Prehospital Management

An SCI should be suspected at the scene of an accident when the patient has decreased or absent movement or sensation. An unconscious patient or one with a head injury is treated as though an SCI has occurred until cord injury is ruled out. Because elapsed time from injury significantly affects prognosis, the patient with an SCI is transported as safely and rapidly as possible to a specialized trauma center or a hospital with adequate diagnostic and treatment facilities to handle such trauma. A primary survey performed at the scene of an accident includes a rapid assessment of airway, breathing, and circulation (ABCs). Airway patency is assessed, and the cervical spine is immobilized and stabilized. It is important to remember that a cervical collar increases the level of stability but does not provide complete immobilization, especially in the case of complete ligamentous disruption, in which the collar has a minimal immobilization effect on spinal stability.[3]

In-Hospital Management

After the patient has been admitted to the emergency department, assessment of the patient's airway is a priority. Facial, mandibular, or laryngeal injuries, as well as broken teeth or a swollen tongue, may be a cause of airway obstruction. Based on assessment findings, the emergency department staff promptly initiates appropriate ventilatory support, which may include elective intubation and mechanical ventilation followed by a chest radiograph. Breathing is assessed after an airway is established. Absence of breath sounds or hyperresonance on chest percussion may indicate an open or tension pneumothorax, flail chest, or hemothorax. These injuries require prompt treatment to prevent deterioration in the patient's respiratory status.

Once airway and breathing are stabilized, the circulatory status is assessed. Hypotension generally occurs secondary to volume loss from hemorrhage. Fluid resuscitation is accomplished by infusing intravenous fluids, crystalloids, or blood. Early administration of blood enhances oxygenation and may minimize the secondary ischemic injury to the spinal cord.[4] The clinical staff completes a thorough neurological and orthopedic assessment and assesses the patient for additional injuries, and then the patient is removed from the backboard to minimize the development of pressure injuries. Last, the emergency department team stabilizes the patient before transfer to the intensive care unit (ICU) or a specialized trauma center.

The administration of high-dose methylprednisolone (Solu-Medrol) in the emergency department remains controversial. The drug reduces swelling and helps minimize secondary injury by reversing the intracellular accumulation of calcium, reducing the risk for cord degeneration and ischemia. However, steroid use has been associated with severe pneumonia and sepsis. The physician determines whether to prescribe methylprednisolone based on assessment of the patient, past medical history, and diagnostic testing.[5]

Physical Examination

RESPIRATORY ASSESSMENT. The nurse assesses and records the patient's respiratory rate and arterial oxygen saturation (by pulse oximetry). Clinical manifestations other than those associated with concomitant injuries may include hypoventilation or respiratory failure. The risk for hypoventilation results from loss of innervation to both the diaphragm (C2–C4) and the intercostal muscles (T1–T4) and is a priority assessment in patients with high cervical injuries. Spinal cord edema can act like an ascending lesion and may compromise function of the diaphragm with injuries to C5 or C6. Assessment of tidal volume and vital capacity and auscultation of breath sounds are vital to determining early clinical manifestations of respiratory compromise.

The patient with an SCI may have additional respiratory compromise because of preexisting pulmonary disease or coexistent chest, laryngeal, tracheal, or esophageal injuries. Major cranial nerves and surrounding arteries and veins may also be injured. Pulmonary collapse or consolidation from retained secretions or aspiration of vomitus may directly affect alveolar ventilation. Pulmonary edema may also result from incorrect management of intravenous fluids. Because paralytic ileus and gastric dilation may increase the pressure on the diaphragm and cause further respiratory compromise, insertion of a nasogastric tube may be indicated.

CARDIOVASCULAR ASSESSMENT. The trauma team immediately places the patient on a cardiac monitor, obtains vital signs, and completes a cardiovascular assessment. Hypotension and bradycardia may be due to neurogenic shock or hemorrhagic shock. Causes of hemorrhagic shock (manifested by hypotension, tachycardia, and cold, clammy skin) include intrathoracic, intra-abdominal, or retroperitoneal injury or pelvic or long bone fractures. Examination of the patient determines whether other injuries are present. The rate of intravenous infusion is adjusted based on the patient's presenting signs and symptoms and past medical history. Insertion of an indwelling Foley catheter allows for accurate monitoring of the fluid status. Chest injury often accompanies thoracic spinal cord trauma, and it is important to examine the chest, head, and abdomen for evidence of concomitant injuries. If neurogenic shock is suspected, the patient will demonstrate bradycardia, in contrast to the tachycardia associated with hypovolemia, due to loss of autonomic innervation.

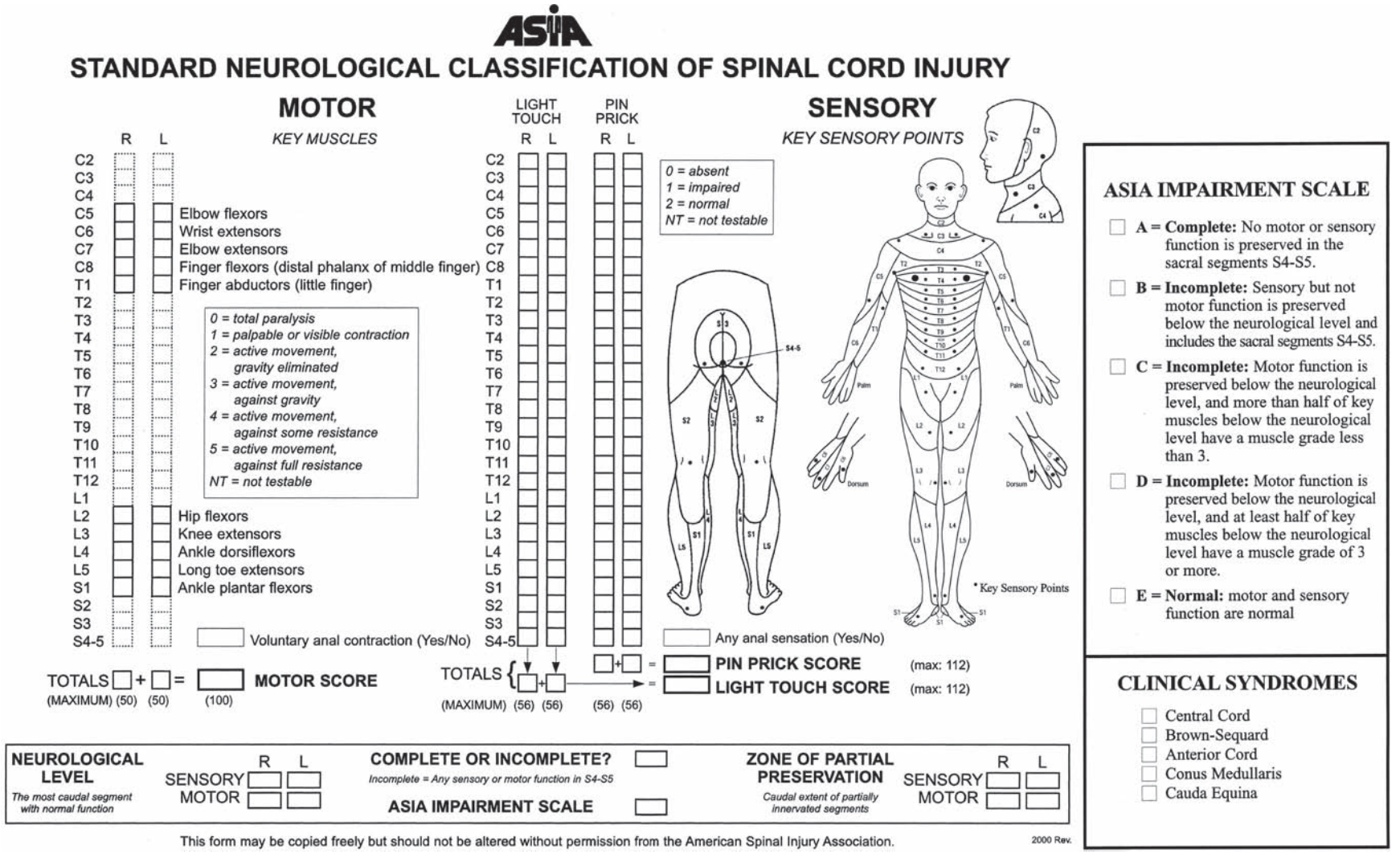


FIGURE 37-6 ▲ This Standard Neurological Classification of SCI flow sheet defines key motor and sensory impairments and clinical syndromes and also includes a functional assessment scale. Motor movement of the major muscle groups is scored on a scale of 0 to 5, with 0 indicating total paralysis and 5 indicating active movement against full resistance or normal movement. Sensation testing is done starting at the area of absent or decreased sensation and proceeding to the area of normal sensation, following the sensory distribution of the skin dermatomes. Each spinal cord segment is tested for sensation and is scored as 0 for absent, 1 for impaired, or 2 for normal. (Courtesy of the American Spinal Injury Association.)

NEUROLOGICAL ASSESSMENT. Frequent assessment of neurological status determines the extent of the SCI and allows for early recognition of changes in level of consciousness that may occur secondary to traumatic brain injury. The trauma team uses the Glasgow Coma Scale (GCS) or other standardized tools to determine patient's level of consciousness. Most SCI centers use a specialized flow sheet, such as the Standard Neurological Classification of Spinal Cord Injury flow sheet, to assess and document the patient's level of functioning (Fig. 37-6). Cranial nerve testing is necessary, particularly if the cause of the injury was penetrating trauma or involved a head injury. For a further discussion of the care of patients with a head injury, see Chapter 36.

During the early assessment of the patient, a digital rectal examination is important to determine whether the injury is incomplete or complete. The lesion is incomplete if the patient can feel the palpating finger or can contract the perianal muscles around the finger voluntarily. Sensation may be present in the absence of voluntary motor activity. Sensation seldom is absent when voluntary perianal muscle contraction is present. In either case, the prognosis for further motor and sensory return is good. Preservation of sacral function might be the only finding that indicates an incomplete lesion, and significant neurological recovery may occur in the patient with an incomplete cord injury. Rectal tone by itself, without the presence of voluntary perianal muscle contraction or rectal sensation, is not evidence of an incomplete cord injury.

BOWEL AND BLADDER ASSESSMENT. Incontinence of urine and possibly feces may have occurred at the scene of the accident. To prevent the bladder from becoming distended secondary to an atonic bladder, insertion of an indwelling urinary catheter is indicated. There may be an imbalance between parasympathetic and sympathetic innervation to the bowel and therefore a loss of voluntary control.

DIAGNOSTIC STUDIES. Once the patient is stabilized, definitive diagnostic tests may be completed safely. The diagnostic workup consists of radiographs of the spine, chest, and other structures as clinically indicated. A computed tomography (CT) scan provides additional information concerning bony structures and fractures. Soft tissue injury is more easily diagnosed with magnetic resonance imaging (MRI). Typical laboratory tests ordered include a complete blood count, electrolytes, glucose, blood urea nitrogen, creatinine, blood type and crossmatch, arterial blood gases, and coagulation studies.

Although rare, the incidence of missed spinal fractures is usually less than 2% with CT.[6] Consequences of a missed injury include chronic pain, deformity, and a delayed injury to the spinal cord or adjacent nerve root. The factors most associated with a missed injury are high-energy trauma, older age, closed head injury, and insufficient imaging. Ideally, flexion–extension views or an MRI is necessary in patients with altered mental status and in those who complain of pain, even when plain radiographs and CT are negative.

When the patient is in the critical care or acute care environment, a physician often orders a somatosensory evoked potential test (see Chapter 33). This test measures the ability of the spinal cord to transmit impulses along the neural pathways to the higher centers in the brain and is used in determining treatment. In this test, a peripheral nerve in the arm or leg below the level of injury is stimulated, and the neurological response (evoked potential) is recorded. If the injury is complete, there is no response. In incomplete injuries, varying responses occur.

▲ Ongoing Assessment and Management

Box 37-3 presents examples of nursing diagnoses and collaborative problems for the patient with SCI. Based on the assessment data, the interdisciplinary team develops an individual treatment plan (Box 37-4), and management is based on the type and severity of injury. Collaboration with all health care disciplines is necessary to enable the patient to achieve the fullest potential after injury. The initial goals are to realign or stabilize the spine to prevent further neurological deterioration, to prevent complications, and to initiate prompt interventions to treat any complications that do occur.

Realignment and Stabilization of the Spine

The trauma team carefully evaluates the patient to determine the most effective treatment based on the type and cause of injury. The surgeon must balance the risks of surgery against the possible benefits associated with eventual patient outcome.[7]

Medical Management

Regardless of the treatment approach, medical management of the patient is an important cornerstone of initial treatment.[8] Closed reduction of a cervical fracture often involves skeletal traction. Cervical traction is used when the fracture is unstable or if subluxation has occurred. Gardner-Wells, Vinke, or Crutchfield tongs are common forms of cervical traction; however, because of the complications that accompany prolonged immobility, long-term traction with tongs

BOX 37-3 EXAMPLES OF NURSING DIAGNOSES

For the Patient With Spinal Cord Injury

- Ineffective Airway Clearance
- Impaired Gas Exchange
- Ineffective Breathing Pattern
- Decreased Cardiac Output
- Risk for Ineffective Peripheral Tissue Perfusion
- Risk for Deficient Fluid Volume
- Acute Pain
- Imbalanced Nutrition: Less Than Body Requirements
- Risk for Impaired Skin Integrity
- Risk for Autonomic Dysreflexia
- Ineffective Thermoregulation
- Impaired Physical Mobility
- Impaired Urinary Elimination
- Bowel Incontinence
- Anxiety
- Disturbed Body Image
- Ineffective Coping
- Compromised Family Coping
- Risk for Infection
- Bathing Self-Care Deficit
- Toileting Self-Care Deficit
- Feeding Self-Care Deficit
- Sexual Dysfunction, Ineffective Sexuality Pattern

BOX 37–4 COLLABORATIVE CARE GUIDE for the Patient With Spinal Cord Injury

Outcomes	Interventions
Oxygenation/Ventilation	
Arterial blood gas values will be within normal limits. No evidence of atelectasis is demonstrated.	• Assess need for mechanical ventilation. • Provide routine pulmonary toilet, including: Airway suctioning Chest percussion, couth and deep breathing Incentive spirometer, nebulizer treatment • Turn frequently. • Mobilize out of bed to chair. • Apply abdominal binder when out of bed. • Consult pulmonologist as needed. • Obtain pulmonary function tests.
Circulation/Perfusion	
There will be no evidence of neurogenic (spinal) shock (T10 injuries and higher). Blood pressure (BP) will be adequate to maintain vital organ function. There will be no development of deep venous thrombosis (DVT) or pulmonary embolism.	• Monitor for bradycardia, vasodilation, and hypotension. • Assess for dysrhythmias. • Prepare to administer intravascular volume, vasopressors, and positive chronotropic agents. • Begin DVT prophylaxis on admission (eg, external compression device, low-dose heparin). • Measure calf and thigh circumference daily and at same location; report increase. • Apply elastic bandage/wraps to lower extremities before mobilizing out of bed.
There will be no evidence of orthostatic hypotension.	• Monitor for orthostatic hypotension when raising head of bed and getting out of bed. • Consult cardiology department as needed.
Neurological	
There will be no evidence of deterioration in neurological status.	• Perform neurological check and spinal cord function checks every 2–4 h. • Monitor for deterioration in neurological status and report to the physician or nurse practitioner. • Monitor for and prevent complications. • Provide patient and family education concerning injury, effects of injury, and rehabilitation.
Fluids/Electrolytes	
Serum electrolytes will be within normal limits. Fluid balance will be maintained as evidenced by stable weight, absence of edema, normal skin turgor.	• Monitor laboratory studies as indicated by patient condition. • Assess for dehydration. • Administer mineral/electrolyte replacement as ordered. • Monitor gastrointestinal and insensible fluid loss. • Make accurate daily fluid intake and output measurements. • Weigh weekly. • Monitor results of laboratory studies, particularly albumin and electrolyte levels.
Mobility/Safety	
Joint range of motion will be maintained and contractures prevented. Skin integrity will be maintained under or around stabilization devices (eg, cervical collar, Yale brace, halo vest).	• Position in correct alignment. • Consult with wound care specialist to determine correct type of bed. • Begin range-of-motion exercises early after admission. • Use high-top tennis shoes, moon boots, extremity splints routinely. • Consult with physical and occupational therapists. • Maintain splint, brace, and adaptive device schedule; check for pressure ulcers every 4 h or more often if indicated. • Monitor skin or pin sites of stabilization devices. • Use meticulous skin care/pin care under or around stabilization devices.

(Continued on page 836)

BOX 37–4 *COLLABORATIVE CARE GUIDE for the Patient With Spinal Cord Injury (continued)*

Outcomes	Interventions
Skin Integrity	
Skin will remain intact.	• Consult with wound care specialist to determine correct type of bed. • Reposition at least every 2 h while patient is in bed. • Position patient to prevent pressure on bony prominences. • Use upright, straight-backed chair when patient is out of bed (not a reclining chair). Use felt pad on chair seat. • Reposition/shift weight every hour when patient is sitting upright. • Use Braden scale to monitor risk for skin breakdown.
Nutrition	
Protein, carbohydrate, fat, and calorie intake will meet minimal daily requirements.	• Consult dietitian. • Encourage fluids, high-fiber diet. • Monitor fluid intake and output, calorie count. • Administer parenteral and enteral nutrition as appropriate. • Assist with feeding/feed as needed.
Comfort/Pain	
Pain will be <"4" on visual analog scale.	• Assess and differentiate pain from anxiety or stress response. • Administer appropriate analgesic or sedative to relieve pain and monitor patient response. • Use nonpharmacological pain relief techniques (eg, distraction, music, relaxation therapies).
Psychosocial	
Patient will adapt to loss of motor and sensory function.	• Provide emotional support by: Encouraging ventilation of sadness, fears, and the like. Arranging for social services, clergy, neuropsychologist, or support groups to see patient.
Therapeutic strategies will be used to cope with anxiety and chronic pain syndrome.	• Provide information and counseling regarding: Personal resources Nonpharmacological pain management techniques Stress management strategies Appropriate use of prescribed pharmacological agents
Integration will be made into prior social role.	• Provide patient/family counseling regarding: Stages of grief Sexual function and management techniques Social services and community resources
Teaching/Discharge Planning	
Patient will adapt to loss of bowel/bladder control. Patient will participate in bowel and bladder program.	• Teach patient/family: Bowel program and training Dietary habits to maintain bowel function Bladder training/intermittent catheterization Prevention of and signs/symptoms of autonomic dysreflexia
Complications of immobility will be prevented.	• Teach patient/family: Positioning to prevent skin breakdown Physical therapy exercises Pulmonary toilet
Patient will be placed in appropriate postacute setting.	• Consult rehabilitation/discharge planner/social services early after admission to initiate placement arrangements.

is seldom used, especially since the advent of the halo vest (Fig. 37-7). Boxes 37-5 and 37-6 present collaborative care guides for the patient in cervical traction and for the patient in a halo vest, respectively.

A halo device, Miami J, or Aspen collar is used for cervical immobilization. Immobilization devices for cervical and thoracic injuries may involve the use of a metal and plastic (Minerva) brace. Thoracolumbar–sacral orthosis may be accomplished using a fiberglass and plastic canvas corset or Jewett brace. Each of these devices is fitted to the patient to provide support and stabilization of the spine. Surgical stabilization may also be necessary. Bed rest is the recommended treatment for sacral and coccygeal injuries.

Surgical Management

The goal of surgical management is to stabilize and support the spine. Emergency surgery may be necessary to remove bone fragments, a hematoma, or a penetrating object, such as a bullet. If the patient's motor status continues to decline, a laminectomy (removal of a portion of the vertebral column) may be performed to allow for swelling of the spinal cord secondary to edema. Rod placement, laminectomy and fusion,

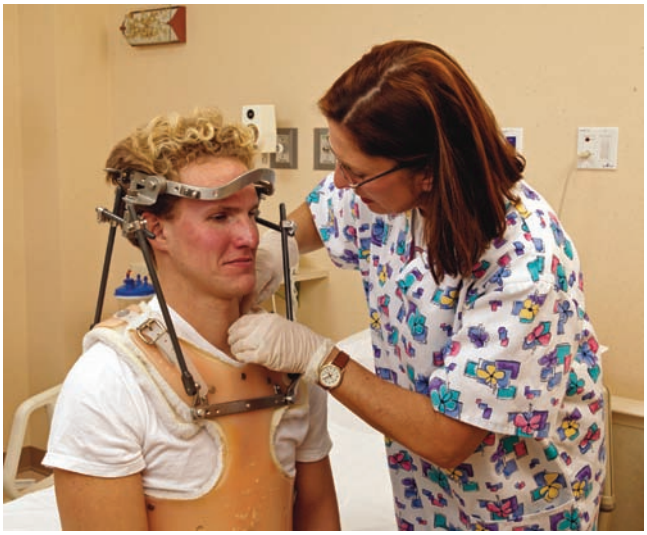


FIGURE 37-7 ▲ A lightweight fleece-lined vest with a halo may be used to stabilize the cervical vertebrae. Note that the vest comes in various sizes and does not need to be removed for magnetic resonance imaging studies. (Courtesy of Bremer Medical, Inc., Dawin Road, Jacksonville, FL).

BOX 37–5 *COLLABORATIVE CARE GUIDE for the Patient in Cervical Traction*

Outcomes	Interventions
Equipment Management	
The orthopedic frame will remain intact. Tongs will not slip. Traction weights will hang freely. There will not be any extension of cord injury secondary to slippage of the traction apparatus.	• Check the orthopedic frame and traction daily to ensure that nuts and bolts are secure. • Check tongs daily to be sure that they are secure. • Be sure that traction weights are hanging freely and not resting on the floor or frame. (Releasing the traction is dangerous because cord injury could be extended.)
Oxygenation/Ventilation	
Airway patency will be maintained. The patient will not aspirate. The patient will not develop a respiratory infection.	• Have suction available to maintain a patent airway. • Provide respiratory care. • Provide for deep breathing, assistive coughing, and incentive spirometer exercises every 1–2 h
Circulation/Perfusion	
Air boots and thigh-high elastic hose (antiembolism stockings) will be worn at all times. Vital signs will be maintained within normal limits. The patient will be observed for DVT and pulmonary emboli.	• Maintain antiembolism stockings and sequential compression boots. • If the patient is receiving heparin, observe for signs and symptoms of bleeding. • Monitor for DVT and pulmonary emboli. (May be receiving minidoses of subcutaneous heparin every 12 h, if not contraindicated.)
Mobility/Safety	
The patient will be free from pain. Contractures will not develop. Strategies will be used to manage spasticity if it occurs.	• Provide comfort measures. • Provide range-of-motion exercises four times per day. • Position the patient in proper body alignment. • Reposition the patient frequently. • Stretch the patient's heel cord with exercises.

(Continued on page 838)

BOX 37-5 COLLABORATIVE CARE GUIDE for the Patient in Cervical Traction (continued)

Outcomes	Interventions
Skin Integrity	
The pin site will remain free of infection. Skin integrity will be maintained. The vertebral column will be maintained in a neutral position and in proper alignment.	• Inspect tong sites, and clean and dress daily as ordered (may be referred to as "pin care"). • Turn the patient every 2 h from side to back to other side using a triple log-roll technique as described below if a patient is on a regular hospital bed: Nurse #1 stands behind the head of the bed and places hands firmly on the patient's head and neck, maintaining the head and neck in a neutral position; the head and neck are turned as a unit. Nurse #2 stands at the patient's side and moves the patient's shoulders. Nurse #3 stands at the patient's side and moves the patient's hips and legs. Plan ahead, identifying desired position and pillow placement *before* moving the patient. When all three nurses are ready, turn the patient as a log on the count of three. Leave the patient positioned in the middle of the bed (if not, he or she will be uncomfortable); use pillows to support the patient's body in alignment. Nurse #1 should hold the head and neck until the patient is supported adequately (if traction slips, manual traction can be supplied by Nurse #1).
Nutrition	
A diet high in protein and carbohydrates, which includes a fluid intake of up to 3,000 mL, will be provided. Aspiration will be prevented.	• Encourage an adequate diet. • Ask the dietitian to see the patient. • Provide for adequate fluid intake up to 3,000 mL daily. • Encourage the patient to take small portions of food into the mouth and chew well to prevent aspiration. • Keep suction equipment handy.
Elimination	
Pattern of bowel evacuation every 1 to 2 days will be established. Intake will be 3,000 mL unless contraindicated. Postvoid residuals will be <100 mL. Strict aseptic technique will be used for catheter protocols.	• Institute a bowel retraining program. • Auscultate the abdomen for bowel sounds. • Record the frequency and consistency of stool. • Monitor intake and output. • Force fluids. • If an intermittent catheterization protocol is initiated, use aseptic technique. • If the patient is voiding on his or her own, monitor postvoid residuals.
Psychosocial	
The patient's mental health will be supported. Social interaction and diversion will be provided based on the patient's ability to participate. A positive body image will be supported. Necessary information will be provided. Sexual function and SCI are discussed with the patient when he or she is ready.	• Provide for social interaction and diversion based on the patient's functional level. • Reinforce a positive self-image. • Allow the patient to participate in decision making as much as possible. • Provide for patient teaching. • Provide information about sexual function.

BOX 37-6 COLLABORATIVE CARE GUIDE for the Patient in a Halo Vest

Outcomes	Interventions
Equipment Management	
The patient will be comfortable and without signs of skin irritation. Proper body alignment will be maintained.	• Check the pins on the halo ring to be sure they are secure and tight. • Check the edges of the fiberglass vest for comfort and fit by inserting the small finger or index finger between the vest and the patient's skin. If the vest is too tight, skin breakdown, edema, and possible nerve injury can occur. • The vest should be supported while the patient is in bed. • Place a rubber cork over the tips of the halo device to diminish magnification of sound if the pin is bumped.
Oxygenation/Ventilation	
The patient will not develop a respiratory infection.	• Provide for deep-breathing exercises at least four times daily.
Circulation/Perfusion	
Risk for thrombus or embolus formation will be decreased.	• Apply thigh-high elastic hose to the legs to improve blood return to the heart. • Observe the legs for development of thrombophlebitis or DVT.
Mobility/Safety	
The patient will maintain muscle tone. The patient will ambulate in a safe manner to the best of his or her ability.	• If the patient's neurological function is intact, he or she will be able to ambulate in the halo vest. • Start to assess the patient's tolerance of the upright position by having him or her sit on the edge of the bed ("dangle"). Check vital signs. (Orthostatic hypotension may be a problem to overcome in the early stages.) • Teach the patient to compensate for lost head and neck movement by making increased use of eye movement to scan the area. • Accompany patients when ambulating because they are more accident prone owing to a displaced center of gravity, a tendency for loss of balance, and decreased peripheral vision. • Consider the patient's use of a walker for ambulation as a means of support and greater safety.
Skin Integrity	
The patient will maintain skin integrity. The patient will maintain proper body alignment without injuries. Early signs of skin irritation or breakdown will be detected.	• Inspect and cleanse the pin site once or twice daily, as prescribed, to prevent infection. • Turn the patient in bed every 2 h by means of the triple log-roll technique to prevent the development of hypostatic pneumonia, atelectasis, and skin breakdown. • Provide sponge pads to prevent pressure on prominent body areas, such as the forehead and shoulder, while the patient is in bed. • Inspect under the vest, and keep all areas of skin dry.
Elimination	
Pattern of bowel elimination every 1 to 2 days will be established. Intake will be 3,000 mL unless contraindicated; postvoid residuals will be <100 mL.	• Institute a bowel retraining program. • Monitor intake and output.
Comfort/Pain Control	
The patient will be comfortable, with pain controlled.	• Administer mild analgesics to control headache and discomfort, which are common, around the pin site. • Provide a soft diet because many patients have jaw pain if they attempt to chew.
Psychosocial	
The patient's emotional well-being will be supported.	• Help the patient adjust to the distorted body image that the halo device can create. • Encourage self-care as much as possible.
Teaching/Discharge Planning	
The patient will be provided the necessary information to ensure competent home care. The patient will use equipment in a safe manner.	• If the patient is to go home with the halo vest, begin a patient and family teaching plan using a booklet or other printed material. Review any written material prepared by the manufacturer for accuracy before giving it to the patient or family.

EVIDENCE-BASED PRACTICE HIGHLIGHT 37-1

Timing Decompressive Surgery for Acute Spinal Cord Injury

A systematic review of literature with a prospective survey was conducted to seek expert opinion regarding the timing of decompressive surgery for patients with acute spinal cord injury (SCI). The lack of consistent practices related to timing of this surgery in this patient population results in wide variability in clinical practice. The methods of this study included a systematic review of the literature related to the optimal timing of decompressive surgery, and the development of a 20-question survey. Data were collected from neurosurgical and orthopedic spine surgeons across the world, with a total of 971 responses. More than 80% of the respondents reported a preference for decompression within 24 hours. Additionally, in complete cervical spinal cord injuries, 46.2% of the surgeons would decompress within 6 hours, and 72.9% would decompress within 6 hours in patients with incomplete injury. Findings of this study found that the majority of spine surgeons recommend decompression of the spine within 24 hours of acute injury, and early surgical decompression should be considered in patients with most spinal cord injuries.

From Fehlings MG, Rabin D, Sears W, et al: Current practice in the timing of surgical intervention in spinal cord injury. Spine 35(21Suppl):A166–A173, 2010.

and anterior fusion are types of surgical stabilization. Bone for fusion usually comes from the iliac crest, tibia, or ribs, or may be retrieved through a tissue bank. See Evidence-Based Practice Highlight 37-1 for information about the timing of decompression surgery.

Postoperatively, the nurse monitors the patient's neurological status at least every hour for the first 24 hours and then every 4 hours. Any indication of deterioration in neurological status is reported immediately to the physician. A major complication of surgery is postoperative infection. This is especially true in older patients and in those with preexisting comorbidities or with open wounds, injuries to the thoracolumbar spine, or complete injuries.[9] The causative organisms are typically Gram-positive organisms, although other concurrent organisms may include *Enterococcus faecalis*, *Enterobacter cloacae*, *Pseudomonas* species, *Klebsiella* species, and *Escherichia coli*.

Prevention of Respiratory Problems

Patients with an SCI, especially injuries above T6, are at risk for respiratory problems, such as ineffective airway clearance, ineffective breathing patterns, and impaired gas exchange. The degree of respiratory compromise is determined primarily by the level of the injury, although not entirely. For example, a 28-year-old patient with C5 tetraplegia with no lung disease may have better ventilation than a 65-year-old patient with C8 tetraplegia with a long history of smoking and chronic obstructive pulmonary disease.

Normally, ventilation is accomplished through a complex interaction between muscles of the chest, the abdominal wall, and the diaphragm. An SCI above T6 results in paralysis of the inspiratory and expiratory muscles. Dysfunction of the intercostal and accessory muscles decreases ventilation and predisposes the patient to atelectasis. Dysfunction of the abdominal muscles and expiratory intercostal muscles diminishes the patient's ability to generate a cough to clear secretions. The intercostal muscles also normally provide support to the lateral chest wall. When the intercostals are impaired, this part of the chest wall collapses during inspiration as the abdomen expands. This breathing pattern is easily discernible and results in ineffective ventilation.

Respiratory complications are the leading cause of death in the acute and chronic phases of SCI, especially among tetraplegic patients. The nurse and respiratory therapist auscultate breath sounds and measure respiratory parameters (eg, tidal volume and vital capacity) frequently. Respiratory failure is anticipated if the patient's vital capacity is less than 15 to 20 mL/kg and the respiratory rate is greater than 30 breaths/min. Other interventions include measuring oxygenation via pulse oximetry. If the value is less than 85 mm Hg or if the arterial carbon dioxide tension ($PaCO_2$) is above 45 mm Hg, intubation may be required. Other interventions include providing oxygen per nasal cannula and ensuring that the patient is well hydrated.

Kinetic therapy involves placing the patient on a special bed that rotates a minimum of 40 degrees on a continuous basis. This stabilizes the spine and the continuous slow rotation prevents pulmonary complications.

The nurse encourages the patient to take deep breaths and to use an incentive spirometer every 2 hours, or more frequently if tolerated. For example, if the patient has a TV or radio in the room, every time there is a commercial, the patient can take four to five deep breaths or use the incentive spirometer independently or with the assistance of the nurse or a family member. Assisting the patient with the quad coughing technique may help clear airways more effectively despite weakness or loss of the respiratory muscles that produce the automatic cough reflex. The quad coughing technique involves compressing the sides of the patient's chest (if patient is on his or her side or abdomen) or the diaphragm (if the patient is supine) during exhalation. This technique often is most helpful after postural drainage or vibration of the chest.

Suctioning may be necessary if the patient's airway cannot be cleared effectively with other techniques. Nurses should remember that suctioning (or nasogastric tube insertion) might trigger an abnormal vasovagal response, resulting in bradycardia.

When turning a patient to the prone position on a Stryker frame, the nurse needs to remain at the bedside for the first few turns to evaluate the patient's respiratory tolerance of the turn. Patients with high-level tetraplegia can experience respiratory arrest in the prone position because movement of the diaphragm is compromised. When the patient is in the prone position, bradycardia is also common.

Restoration of Hemodynamic Stability

The management of arterial oxygenation and blood pressure (BP) support are critical in optimizing the potential for neurological recovery. Continuous hemodynamic monitoring is essential to measure cardiac output and systemic perfusion. Insertion of a pulmonary artery catheter and central venous line may be necessary if this was not done in the emergency department. It is important to maintain mean arterial pressure between 85 and 90 mm Hg for the first week after injury. The systolic BP should be above 90 mm Hg. The patient is at risk for cardiovascular compromise because of disruption in the autonomic nervous system. Bradycardia, hypotension, and dysrhythmias may occur. Hypotension and tachycardia may indicate hemorrhage from intra-abdominal bleeding or bleeding around fracture sites. Sequential compression devices, antiembolism stockings, or an abdominal binder may be used to promote venous return.

Left ventricular dysfunction may occur secondary to release of β-endorphins. Cardiac enzymes should be obtained if there are electrocardiographic changes. Dysrhythmias and heart block may occur. It is necessary to address adequate tissue perfusion to the spinal cord and other vital organs, such as the kidneys. Careful intravenous fluid replacement provides hydration without fluid overload. Vasopressors may not be necessary to maintain BP during spinal shock, but when the BP is not high enough to sustain vital organ perfusion, usually low-dose dopamine is useful. Bradycardia also may not need treatment, but if necessary, atropine may be used to speed up the heart rate. A transcutaneous or transvenous pacemaker may be essential if the patient remains bradycardic despite atropine.

Neurological Management

While in the ICU, the patient requires neurological assessment every hour until stable, then every 4 hours. The patient's motor and sensory states warrant particular attention. If there is any deterioration in the patient's condition, the frequency of assessment increases, and the nurse notifies the physician or nurse practitioner.

Depending on the patient's motor function, an adapted nurse call system may be necessary. Useful devices include low-pressure, voice-controlled, and sip-and-puff straw-like call systems. Other communication systems can be developed in conjunction with the speech language pathologist for the patient on a mechanical ventilator.

Pain Management

It is not unusual for the patient to complain of pain, frequently severe pain. The source of the pain may be neuropathic, musculoskeletal, central, or visceral. Abnormal sensation may occur at the level of the lesion in injuries causing diverse nerve root damage, such as occurs with gunshot or knife wounds. Pain resulting from either the SCI, surgery, or both is treated aggressively following institutional protocol and within the pain management standards of the institution.

Medication Administration

Medications used in treating the patient with SCI are listed in Table 37-2. Nurses administering medications to patients with spinal cord injuries must take into account several special considerations. Subcutaneous and intramuscular injections are not absorbed well because of the lack of muscle tone. Sterile abscesses may result, causing autonomic dysreflexia or an increase in spasms. Injection sites are the deltoid area, the anterior thigh, and the abdominal area. Rotation of injection sites is necessary, and the volume injected should not exceed 1 mL at any one site.

Nurses often start peripheral intravenous lines, but the intravenous site of choice is the subclavian vein. In this area of high blood flow, there is less chance of thrombosis secondary to vasomotor paralysis, especially during spinal shock. For this reason, the veins of the lower extremities should never be used for intravenous administration.

Thermoregulation

Ineffective thermoregulation is a common problem seen in patients with spinal cord injuries above the thoracolumbar area. Interruption of the sympathetic nervous system prevents the thalamic thermoregulatory mechanisms. As a result, the patient fails to sweat to get rid of body heat, and there is an absence of vasoconstriction, resulting in an inability to shiver to increase body heat. The degree of thermal control and dysfunction is directly proportional to the extent of body area with loss of thermal regulation. Hence, a tetraplegic patient has more difficulty with thermoregulation than a paraplegic patient.

Hypothermia is usually managed by using warmed blankets. The room temperature is adjusted to maintain patient comfort. Electric heating blankets or hot water bottles may present a danger for body parts with no sensation. An attempt is made to stabilize the patient's temperature above 96.5°F (35.8°C). Ideally, the patient is placed in a private room so that the room temperature does not adversely affect the other patient in the room. Over the long term, thermal control can be facilitated by use of clothing appropriate for the weather conditions.

Nutrition

The possibility of inadequate nutrition is of significant concern during the acute phase of injury and must not go unnoticed while the focus is on hemodynamic stability. Negative nitrogen balance contributes to skin breakdown, poor wound healing, and lack of energy for rehabilitative efforts. Along with other required blood work, a serum albumin test is necessary. A value less than 3.5 g/dL and/or total lymphocyte count less than 1,500 to 2,000/mm^2 is indicative of clinical malnutrition. A dietary consult should be obtained as early as possible. Caloric requirements are calculated to ensure adequate, but not excessive, nutritional support. If the patient must remain nothing by mouth (NPO) for more than a few days, total parenteral nutrition should be initiated.

Patients with spinal cord injuries often have increased energy needs secondary to metabolic stress response. This

Table 37-2 Drugs Used in the Treatment of the Patient With a Spinal Cord Injury

Drug	Description	Administration
Drugs to Minimize Injury		
Methylprednisolone	Synthetic adrenal corticosteroid used as an anti-inflammatory agent	Loading dose: 30 mg/kg IV over 15 min Pause for 45 min; infuse normal saline solution or other IV fluid Maintenance dose: 5.4 mg/kg/h IV for 23 h if loading dose given within 3 h of injury Loading dose must be given within 8 h of injury. Maintenance dose must begin within 1 h of loading dose.
Drugs for Cardiovascular Stabilization		
Atropine	Anticholinergic used to treat symptomatic bradycardia	Dosage: 0.4–1.0 mg IV slowly; maximum dose 2 mg
Dobutamine (Dobutrex)	β-Adrenergic agent that enhances myocardial contractility, stroke volume, and cardiac output; improves perfusion to the spinal cord	Dosage 2.5–10 mcg/kg/min up to maximum dosage of 30 mcg/kg/min; usually given for a total of 72 h if needed
Dopamine (Intropin)	α- and β-Adrenergic agent used to treat hypotension related to neurogenic shock	Dosage: 3–5 mcg/kg/min; increase gradually at 10- to 30-min intervals up to 20–50 mcg/kg/min until optimum blood pressure (BP) achieved Start the older adult on lower doses; use with caution if the patient is on a monoamine oxidase inhibitor
Phenylephrine (Neosynephrine)	α-Adrenergic agent used to treat hypotension related to neurogenic shock	Dosage: 0.1–0.5 mg; may administer every 10–15 min as needed. Do not exceed initial dose.
Drugs for Paralytic Ileus and Stress Ulcers		
Proton pump inhibitors (lansoprazole, omeprazole, pantoprazole)	Suppress gastric acid secretion; used to prevent or treat gastric ulcers	Dosage: dependent on medication prescribed
Histamine blockers (cimetidine, famotidine, nizantine, ranitidine hydrochloride)	Inhibit histamine action at H_2 receptor sites; used to treat and prevent ulcers and gastric reflux	Dosage: dependent on medication prescribed
Drugs for Autonomic Hyperreflexia		
Nitroglycerin	Organic nitrate used if systolic BP rises above 140 mm Hg	Dosage: 1 inch of nitroglycerin paste placed above the level of injury
Nifedipine (Procardia)	Calcium channel blocker used to decrease systemic vascular resistance and BP	Dosage: 10–20 mg orally every 20–30 min if necessary
Hydralazine, trimethaphan, diazoxide	Antihypertensive agents	Dosage: dependent on medication prescribed Trimethaphan: dilute per hospital protocol (usually 500 mg in 500 mL D_5W); usual dose 0.5–1 mg/min Hydralazine: 25 mg orally four times daily or 10–20 mg IV every 4–6 h Diazoxide: 1–3 mg/kg, up to 150 mg, IV; repeat at 5- to 15-min intervals if needed
Drugs for Skeletal Muscle Spasm		
Dantrolene (Dantrium)	Skeletal muscle relaxant used to treat spasticity	Dosage: initial dose 25 mg/d; may increase to 25–100 mg twice or thrice a day. Max dose is 400 mg/d
Baclofen (Lioresal)	Skeletal muscle relaxant used to treat spasticity	Dosage: initially 5 mg three times a day for 3 d, then 10 mg three times a day for 3 d, then 20 mg three times a day for 3 d; then titrated to response Max daily dose is 80 mg/d.
Tizanidine hydrochloride (Zanaflex)	Central-acting skeletal muscle relaxant used to treat acute and intermittent increased muscle tone associated with spasticity	Dosage: initially 4 mg; gradually increase to 8 mg every 6–8 h for a maximum dose of 36 mg/24 h
Investigational Drugs		
Fampridine-SR ("4-AP," "4-aminopyridine")	This drug has been approved by FDA for patients with multiple sclerosis; however, evidence for use in patients with acute SCI is inconclusive.	

can lead to a severe catabolic state and malnutrition. It is not unusual to see a significant weight loss within the first few days of injury. Total parental nutrition or enteral feedings are instituted until the patient is able to start on an oral diet. The patient's intake and output are strictly monitored.

Ensuring adequate nutrition is a collaborative problem involving the patient, family, dietitian, occupational therapist (OT), and nurse. The dietitian meets with the patient and family to identify the foods the patient enjoys and develops a menu that incorporates patient preference into the required dietary plan. Family members learn what foods they can bring from home to include in the patient's prescribed diet. The OT assesses the patient's need for assistive devices to use during mealtimes and teaches the patient and family how to use the adapted silverware. The nurse reinforces the information provided by the dietitian and OT. The nurse may teach family and friends how to assist the patient with meals and about the importance of allowing the patient as much independence as possible at mealtime. It is necessary to encourage fluid and a high-roughage diet, unless contraindicated. Before meals, the nurse assists the patient with mouth care and ensures that the patient has not been incontinent.

Mobilization and Skin Care

Rehabilitation begins in the critical care unit and is a collaborative effort involving the patient, physician, physical therapist (PT), OT, nurse, and the patient's family. Initially, the nurse assists the patient with range-of-motion exercises. When the patient is stable, family members may be taught to assist the patient with these exercises. Based on their assessment findings, the PT and OT develop an individualized treatment plan to begin mobilization of the patient.

Positioning is not simply based on the usual every-2-hour turn schedule. Development of proper positioning protocols occurs in conjunction with the physical and OT to maximize range of motion and prevent joint contractures. When turning the patient, the nurse places a pillow under the patient's head but not under the forearms. When the patient is on his or her side, most of the upper body weight should be over the scapula that rests on the bed. Flexing of the hips and knees allows the patient to remain on the side with minimal support. The nurse places pillows and foam cushions between the knees and against the back.

Pressure is a common cause of structural damage to a muscle and its peripheral nerve supply. Preventing skin breakdown is a priority for the nurse in the critical care unit. There is a definite time–pressure relationship in the development of pressure ulcers. Microscopic tissue changes secondary to local ischemia occur in less than 30 minutes. Pressure interferes with arteriolar and capillary blood flow. When the pressure is prolonged, there is definite damage to superficial circulation and tissue. The damage may be associated with congestion and induration of the area or blistering and loss of the superficial epidermal layers of the skin. As the pressure continues, the deeper skin layers are lost, leading to necrosis and ulceration. Serous drainage from such ulceration can constitute a continuous protein loss of as much as 50 g/d. Prolongation of the pressure results in deep penetrating necrosis of the skin, subcutaneous tissue, fascia, and muscle. The destruction may progress to gangrene of the underlying bony structure. Pressure necrosis can begin from within the tissue over a bony prominence, where the body weight is greatest per square inch.

A turn schedule for the patient is important, even if the patient has not had stabilizing surgery. It may take three staff members to accomplish this safely, particularly in the patient with a cervical injury. One person stabilizes the neck, and the other two flex the hips, knees, and ankles and hold the feet flat on the bed surface while turning the patient's trunk. To maintain alignment, the nurse uses foam wedges, pillows, or air-filled rolls. Turning occurs a minimum of every 2 hours. Use of an air or egg-crate mattress does not preclude the need to turn. The nurse checks the condition of the skin before and after the position change, paying particular attention to the patient's earlobes, back of head, elbows, inner aspects of the knees, heels, and sacral area. The posterior thigh and ischial tuberosities are prone to skin breakdown from prolonged lying on the back with the head of bed elevated or when sitting in a chair. The nurse documents any change in skin integrity, notifies the wound care specialist, and implements a plan of care. Numerous kinetic beds and air beds are available for patient comfort, preventing skin breakdown and treating complications of immobility.

Together with the PT and the OT, the nurse develops a plan to prevent foot drop. Initially, the heels are placed in "bunny boots." Frequently, high-top sneakers/basketball shoes are worn. It is important to ensure that the boots or shoes are the correct size, to check for signs of skin breakdown, and to ensure that the patient's feet are dry. It is necessary to develop an "on and off" schedule with the PT. When the boots or shoes are off, the nurse pays careful attention to foot positioning and assesses for pressure ulcers.

The OT determines the need for splints or braces for the patient's wrists and hands. The nurse must assess the patient's skin frequently to identify pressure areas early. If necessary, the splints are modified to prevent skin breakdown.

Urinary Management

Acute tubular necrosis may occur within 48 hours of injury as a result of hypotension. An indwelling urinary catheter is necessary to allow for hourly measurement of urinary output during this phase, with the goal of keeping it at least 30 mL/h. The nurse closely monitors fluid and electrolyte balance. Removal of the indwelling catheter as soon as spinal shock has resolved reduces the risk for infection.

The long-range objective of bladder management, regardless of the level of the injury, is to achieve a means whereby the bladder consistently empties, the urine is sterile, and the patient remains continent. The ultimate goal is to have the patient catheter free, with consistent low residual urine checks, no urinary tract infection, and no evidence of damage to the upper urinary tract structures.

One method of bladder management involves intermittent catheterization, and it may begin in the early recovery phase after spinal shock is resolved. The purpose of this program is to exercise the detrusor muscle, again with the goal of keeping the patient catheter free. The advantage of this method is that no irritant remains in the bladder; consequently, the risk for urinary tract infection, periurethral abscess, and epididymitis is reduced.

Bowel Management

Before the initiation of a bowel program, it is necessary to perform a systematic, comprehensive evaluation of the type of cord injury, bowel function, impairment, and possible problems. This includes an abdominal assessment, a rectal examination, and evaluation of anal sphincter tone. In addition, the anocutaneous reflex (contraction of the anal sphincter secondary to cutaneous stimulation) and bulbocavernosus reflex (also referred to as the penile reflex; compression or tapping on the dorsum of the glans penis, which leads to contraction of the bulbocavernosus muscle at the tip of the penis) warrant assessment to determine whether the patient has upper motor neuron or lower motor neuron dysfunction. Equally important, a bowel program must consider the ability of the patient and care givers to carry out the planned interventions at discharge.

Simple steps can prevent constipation and begin progress toward bowel continence. It is necessary to maintain appropriate intake, either through intravenous or oral fluids and diet. The nurse records bowel movements in an area that is easily accessible for review and administers stool softeners daily. Development of a consistent schedule for the bowel program is warranted. The timing of the program is usually after meals to coincide with peristalsis that occurs after meals to move food through the gastrointestinal tract. Rectal stimulation may be necessary to trigger defecation.

Psychological Support

As soon as the patient is medically stable, the nurse begins to focus on the psychosocial issues that are of concern to the patient and family. Questions often asked of the nurse include the following: Am I going to die? Will I walk or use my arms again? What is going to happen to me? There are no easy answers to these questions. It can be difficult for patients and family members to accept this uncertainty. Most patients are accustomed to receiving treatment for illnesses or conditions that have a predictable course of treatment and outcome. For example, antibiotics are prescribed for 10 days and the infection clears up, or surgery is performed and we are discharged home and able to return to our normal activities within an expected timeframe.

Answer questions to the best of your knowledge. Never predict the future, tell stories about patients who made a complete recovery, or ignore a question or a concern. Listen to the patient and the family. Let the patient and family talk about their fears and anxieties. Detailed patient education in a critical care unit is not generally appropriate. However, providing information the patient and family need to know while they are being cared for in the critical care unit is important. Focus on the patient care issues that present each day and on the patient's abilities. Do not minimize the patient's disabilities. Refer questions to other members of the health care team as appropriate. If indicated, ask for an order for a psychiatric consult.

Incorporate the use of technology to help the patient stay connected with family and friends. For example, place a "hands free" telephone in the room. Use a laptop computer and web cam if wireless internet is available. Explore other enhancements that may be on the patient's personal laptop that will enable continued support with others and provide a means for helping pass the long days in the critical care unit.

Psychological transition from the loss of previous physical abilities to the current state is unique to each person. Feelings of grief, loss, anger, and frustration are common. Certain emotions are characteristic after an SCI, whatever names are given to the stages of grief (Box 37-7). The rate at which a person works through this process varies, and no stage is static. A person can move back and forth between stages. The emotions felt and displayed by someone with an SCI are no different from the emotions felt by everyone at one time or another, and recognition of that fact may help promote empathy with the patient.

All staff members should have an understanding of the types of feelings and reactions the patient with an SCI may exhibit. They can share this process of recovery with family members in helping them to support the injured person and participate in recovery. It is necessary to provide psychological support for family members, who no doubt have many concerns, such as finances, role changes, and long-term prognosis. It is important to be supportive of them and help them and the patient with coping strategies.

Addressing Concerns About Sexuality

After an SCI, patients have concerns about their ability to function sexually, although they may not verbalize this issue immediately. Critical care nurses probably will not deal with this problem specifically, but it is important to have some knowledge of the functional potential of the patient to begin to manage the patient's fears and concerns in this area. By avoiding discussion of this important issue, professionals validate the patient's fear that there can be no sex after an SCI, which is certainly not true.

Male Sexuality

Many men with an SCI believe that their total sexuality is tied to erection and ejaculation. There are three general types of erection in men: psychogenic, reflexogenic, and spontaneous. A psychogenic erection can result from sexual thoughts. The area of the cord responsible for this type of erection is between T11 and L2. Therefore, if the lesion is above this level, the message from the brain cannot get through the damaged area.

Reflexogenic erections are a direct result of stimulation to the penis. Some patients may get this type of erection when changing their catheter or pulling the pubic hairs. The length of time the erection can be maintained is variable; therefore, its usefulness for sexual activity is variable. Reflexogenic erections are better with higher cervical and thoracic lesions. Damage to lumbar and sacral regions may destroy the reflex arc.

The third type of erection is spontaneous. This may occur when the bladder is full, and it comes from some internal stimulation. How long the spontaneous erection lasts will determine its usefulness for sexual activity. The ability to achieve a reflexogenic or spontaneous erection comes from nerves in the S2, S3, and S4 segments of the spinal cord.

BOX 37-7 Stages of Grief in a Patient With a Spinal Cord Injury

Stage	Description	Implications for the Nurse
1. Shock and disbelief	During this phase, the patient does not request an explanation of what has happened. The patient is overwhelmed by the injury. There may be more concern with whether he or she will live than with whether he or she will walk again. This period may result in extreme dependence on the staff members.	The nurse may feel that the patient does not understand the ramifications of the injury. The nurse may identify with the feelings of being overwhelmed because he or she is often overwhelmed with the acute medical management of this catastrophic illness.
2. Denial	The process of denial is an escape mechanism. Usually, the whole disability is not denied, but particular aspects of it are. For instance, the patient may say he or she cannot walk now but will be able to in 6 months. Bargaining, instead of being a separate stage, can be considered a form of denial. Bargains with God may be in the form of offering Him the legs if He will just return function of the arms.	The nurse often finds it difficult to deal with patients in this stage. A helpful approach is to focus on the present problems. This is not the stage to discuss long-term changes, such as ordering a wheelchair or making modifications to the home. More appropriate matters to deal with would be skin care and range-of-motion exercises.
3. Reaction	During this stage, instead of denying the impact of the injury, the patient expresses this impact. There may be severe depression and loss of motivation and involvement. Previous hobbies or interests lose their meaning. There is great helplessness during this period, and there may be suicidal statements.	The nurse can help at this stage by listening to the patient as feelings are verbalized. The nurse should avoid setting up failure situations, which could happen if he or she pushes the patient too fast. It is important to note that both the sudden absence of muscular activity and sensations in the patient with an SCI and the mental state of helplessness appear to alter central nervous system metabolism. Depression coincides with a fall in a brain metabolite excreted in the urine as tryptamine. Thus, it is important for the nurse to understand that depression in some patients with SCI might have a metabolic basis and that a trial of pharmacological therapy might be beneficial.
4. Mobilization	Problem-solving behavior is seen during this stage. The patient is looking toward the future and wants to learn about self-care. In fact, the patient may become very possessive of the therapist or nurse and resent the time spent with other patients. This is a time of sharing and planning between patient and staff.	
5. Coping	Some authorities think that patients do not accept the disability *per se* but instead learn to cope with it. Disability still is an inconvenience, but it is no longer the center of the patient's life. Life is again meaningful to the patient, and the patient is again involved with others.	

Female Sexuality

In 50% of women with an SCI, the menstrual pattern is interrupted for approximately 6 months after injury but then is reestablished. Women are able to become pregnant and seem to have no increase in rate of miscarriage. There are potential complications for the pregnant woman, such as urinary tract infection, pressure sores, and anemia, but with careful medical attention, complications usually can be avoided or minimized.

Labor may be painless, or the woman may experience other signs that indicate labor is occurring (eg, abdominal or leg spasms, back pain, difficulty breathing). Autonomic dysreflexia is a complication of labor in women with injuries above T4 to T6 and should be anticipated so that it can be controlled. Women may breastfeed if they wish.

▲ Complications

Autonomic Dysreflexia

Autonomic dysreflexia, or hyperreflexia, is a syndrome that sometimes occurs after the acute phase in patients with a spinal cord lesion at T7 or above. Autonomic dysreflexia constitutes a medical emergency. The syndrome presents quickly and can precipitate a seizure or stroke. Death can occur if the cause is not relieved.

Triggering conditions include bladder or intestinal distention, spasticity, pressure ulcers, or stimulation of the skin below the level of the injury. In men, ejaculation can initiate the reflex, and in pregnant women, strong

BOX 37-8 Precipitating Factors in Autonomic Dysreflexia

- Bladder distention or urinary tract infection
- Bladder or kidney stones
- Distended bowel
- Pressure areas or decubitus ulcers
- Thrombophlebitis
- Acute abdominal problems (eg, ulcers, gastritis)
- Pulmonary emboli
- Menstruation
- Second stage of labor
- Constrictive clothing
- Heterotopic bone
- Pain
- Sexual activity; ejaculation by a man
- Manipulation or instrumentation of bladder or bowel
- Spasticity
- Exposure to hot or cold stimuli

uterine contractions can elicit it. Box 37-8 lists potential precipitating factors.

These stimuli produce a sympathetic discharge that causes a reflex vasoconstriction of the blood vessels in the skin and splanchnic bed below the level of the injury. The vasoconstriction produces extreme hypertension and a throbbing headache. Vasoconstriction of the splanchnic bed distends the baroreceptors in the carotid sinus and aortic arch. These baroreceptors in turn stimulate the vagus nerve, producing a bradycardia, in an attempt to lower the BP. The body also attempts to reduce the hypertension by superficial vasodilation of vessels above the SCI. As a result, there is flushing, blurred vision, and nasal congestion. Because the SCI interrupts transmission of the vasodilation message below the level of the injury, the vasoconstriction continues below the level of the injury until the stimulus is identified and interrupted. The vasoconstriction results in pallor below the injury, whereas flushing occurs above the injury. Box 37-9 summarizes the signs and symptoms of autonomic dysreflexia.

When autonomic dysreflexia is recognized, there are several things the nurse can do quickly to relieve the patient's condition. The nurse can elevate the head of the bed and make frequent checks of BP. In addition, he or she quickly checks the bladder drainage system for kinks in the tubing. The urine collection bag should not be overly full. Some protocols for checking the patency of the urinary drainage system include irrigating the catheter with 10 to 30 mL of irrigating solution.

BOX 37-9 *PATIENT SAFETY*

Signs and Symptoms of Autonomic Dysreflexia

- Paroxysmal hypertension
- Pounding headache
- Blurred vision
- Bradycardia
- Profuse sweating above the level of the injury
- Flushing or splotching of the face and neck
- Piloerection
- Nasal congestion
- Nausea
- Pupil dilation

Absolutely no more than that amount is used because the addition of the fluid may aggravate the massive sympathetic outflow already present. If the symptoms persist, the catheter is changed so that the bladder can empty. Foley catheter placement may be necessary for patients on a bladder management program and those who have not voided in the past 4 to 6 hours.

If the urinary system does not appear to be the cause of the stimulus, it is necessary to check the patient for bowel impaction. Removal of the impaction should not occur until the symptoms subside. Rectal application of dibucaine or lidocaine ointment anesthetizes the area until symptoms subside. If the patient's BP does not return to normal, sublingual nifedipine (Procardia) may be very effective. A sympathetic ganglionic blocking agent, such as atropine sulfate, guanethidine sulfate (Ismelin), reserpine, or methyldopa (Aldomet), may be useful. Hydralazine (Apresoline) and diazoxide (Hyperstat) also may help. Box 37-10 presents nursing intervention guidelines for managing autonomic dysreflexia.

Pulmonary Complications

Pulmonary complications are the most common cause of death in people with SCI, both in the acute and chronic phases. These pulmonary complications are especially prevalent in people injured above T10. If there is concomitant chest trauma or preexisting pulmonary disease, a history of smoking, or older age, there is higher risk for these complications.

Atelectasis and Pneumonia

Atelectasis is possible in any immobilized patient. Early mobilization, ensuring the airways are clear of secretions, and bronchial hygiene may be useful in minimizing or preventing atelectasis. Pneumonia may also result from hypoventilation and an inability to keep the airways clear. Adequate hydration helps keep secretions liquefied for ease of removal, and bronchoscopy may be necessary to remove mucous plugs. Supplemental oxygen administration is used to treat hypoxia. Ventilator-dependent patients need exquisite pulmonary care (see Chapter 25).

Deep Venous Thrombosis and Pulmonary Embolus

The Virchow triad for venous thrombosis—venous stasis, vein injury, and hypercoagulability—is found in the patient with an SCI. As a result, the patient is at increased risk for deep venous thrombosis (DVT) and pulmonary embolus. In addition to swelling and pain, obstruction to venous return can lead to compartment syndrome and limb ischemia. Although it is infrequent, hypovolemic shock can occur if enough blood and interstitial fluid pool in the extremity.[10] (If the thrombus breaks off, pulmonary emboli can obstruct venous return and lead to cardiovascular collapse and death.) Patients particularly at risk for a fat embolus are those with long bone fractures. Signs of chest or neck petechiae and low-grade fever may be early indications.

Leg veins should not be used as sites from which to draw blood, lest the trauma to the vessel wall enhance platelet aggregation and clot formation. It is important to encourage smokers to quit because nicotine causes vasoconstriction, thereby slowing blood flow through the periphery.

There is some controversy about the effectiveness of serial leg measurements in monitoring for DVT. A standard measurement protocol is necessary, and all staff should follow

BOX 37-10 NURSING INTERVENTIONS

For Managing Autonomic Dysreflexia

1. Elevate the head of bed.
2. Apply BP cuff, and check BP every 1 to 2 minutes.
   - If BP is above 180/90 mm Hg, proceed to step 5.
   - If BP is below 180/90 mm Hg, proceed as follows.
3. Quickly insert bladder catheter or check bladder drainage system in place to detect possible obstruction.
   - Check to make sure plug or clamp is not in catheter or on tubing.
   - Check for kinks in catheter or drainage tubing.
   - Check inlet to leg bag to make sure it is not corroded.
   - Check to make sure leg bag is not overfull.
   - If none of these are evident, proceed to step 4.
4. Determine whether catheter is plugged by irrigating the bladder slowly with no more than 30 mL of irrigation solution. Use of more solution may increase the massive sympathetic outflow already present. If symptoms have not subsided, proceed to step 5.
5. Change the catheter and empty the bladder.
6. When you are sure the bladder is empty and if BP is:
   - Above 180/90 mm Hg, call physician immediately.
   - Below 180/90 mm Hg, proceed as follows: Give sublingual nifedipine (Procardia) if protocol calls for it. Give atropine according to physician's order. If BP rises or fails to subside, call physician immediately. Guanethidine monosulfate (Ismelin), hydralazine (Apresoline), or inhaled amyl nitrate may then be ordered by the physician. Dibenzylene may be used for chronic dysreflexia.
7. Ideally, this procedure requires three people: one to check the BP, one to check the drainage system, and one to notify the physician.

If bladder overdistention does not seem to be the cause of the dysreflexia,

- Check for bowel impaction. Do not attempt to remove it, if present. Apply Nupercainal ointment or Xylocaine jelly to the rectum and anal area. As the area is anesthetized, the BP should fall. After the BP is again stable, using a generous amount of anesthetizing ointment or jelly, manually remove impaction.
- Change the patient's position. Pressure areas may be the source of dysreflexia.

it. For example, use a special measuring tape rather than a sewing tape, mark the area where the tape is placed, and use running averages.

Treatment of DVT and pulmonary embolus includes heparin infusion, insertion of an intravenous vena cava filter, or dissolving the clot with thrombolytic agents. Measures to prevent DVT may include the administration of low–molecular-weight heparin and the use of antiembolism stockings. Other modalities include sequential compression devices, passive range-of-motion exercises, and early mobilization. In addition, a kinetic bed can be useful; this device works by keeping the patient in continuous motion.

Paralytic Ileus and Stress Ulcers

Early medical management for paralytic ileus and stress ulcers includes NPO, particularly for cervical spinal cord–injured patients. Nasogastric tube placement with intermittent suction is useful in treating the paralytic ileus that frequently accompanies SCI. Nasogastric tube placement also decreases the risk for aspiration and reduces abdominal distention. As soon as bowel sounds are present, safe stimulation of peristalsis may be facilitated with stool softeners, mild laxatives, or suppositories. It is important to avoid enemas, other than the oil-retention type, because the risk for intestinal perforation is high.

Patients with cervical injuries are more likely to experience gastrointestinal bleeding as a result of stress ulcers. Medical treatment includes histamine-2 (H_2) receptor blockers, proton pump inhibitors, antacids, or a combination of the three.

Heterotropic Ossification

Calcification around a joint, especially the hip joint, may occur within 12 weeks of injury. Clinical manifestations include limited range of motion, swelling of the affected joint, and an elevated alkaline phosphatase level. Pain may or may not be present. The treatment goal is to prevent further damage and progression. Additional treatment includes irradiation, nonsteroidal anti-inflammatory drugs, and disodium etidronate.

Spasticity

Spasticity develops after recovery from the period of spinal shock and affects the flexor muscles of the arms and the extensor muscles of the legs. An interdisciplinary approach is the hallmark of treatment. A physical therapy consult is warranted to develop an exercise, stretching, and positioning program for the patient. A variety of medications such as baclofen, dantrolene sodium, diazepam, and clonidine may be useful.

▲ Research in the Treatment of Spinal Cord Injuries

Progress in clinical investigations may contribute to the treatment of SCI.

- Stem cell research holds promise for a number of neurological disorders, including spinal cord regeneration. Some studies have advanced to phase I clinical trials.
- Medications are under investigation to identify drugs that may be useful in the treatment of the primary and secondary effects of the cord injury itself. Other studies are focusing on the development of medications to treat complications such as spasticity or neuropathic pain. Other research is focusing on designing DNA vaccines for the treatment of axonal degeneration and demyelination. The goal is to improve motor function.
- Medical devices and assistive devices continue to be developed and older tools improved to enable the person with an SCI to function at the maximal level possible. With the increased numbers of military personnel suffering spinal cord injuries, this area will continue to expand.
- The use of systemic hypothermia is being investigated as an intervention during the acute phases of SCI.

▲ Patient Teaching and Discharge Planning

The nurse plays an integral role in working with the patient and family in finding a rehabilitation program that has a program specific for spinal cord–injured patients. Usually, the patient is discharged to a rehabilitation setting to learn the skills needed for activities of daily living and, when possible, independent living. The nurse helps the family find a rehabilitation program specifically for patients with SCI. When searching for an appropriate rehabilitation program, the family should obtain answers to the following questions:

1. How many patients with SCI are treated in the program each year?
2. What is the average age of the patients in the program?
3. Does the treatment plan identify both long-term and short-term goals?
4. Will the patient be assigned an experienced case manager to coordinate the transition between the rehabilitation center and home?
5. How much time is spent teaching the patient and family about sexuality, bowel and bladder care, and other activities of daily living?

The family should also ask if the staff has specialized training in SCI. Rehabilitation therapies should be available for a minimum of 3 hours per day. There should be activities or programs for the patients on weekends and in the evenings. Most importantly, the facility should have 24-hour staffing with registered nurses and respiratory therapists. Box 37-11 offers a teaching guide for a person living with an SCI.

BOX 37–11 TEACHING GUIDE *Living With a Spinal Cord Injury*

Respiratory Management
- Cough and deep-breathe routinely.
- Drink plenty of fluids, unless contraindicated.
- Use postural drainage or chest physiotherapy.
- Because you are at risk for pneumonia, be careful around anyone with a cold and get an annual influenza vaccination.

Nutritional Management
- Eat a well-balanced diet that includes protein (lean meat, dairy foods, legumes), fresh fruits, vegetables, and liquids.
- Maintain ideal body weight.

Skin Management
- You and your helper should check your skin twice a day. Look for redness, bruises or scrapes, blisters, and rashes. Pay particular attention to bony areas. Check your groin for rashes and reddened areas.
- Keep your skin clean and dry, especially in areas where skin touches skin (eg, between the toes, underneath the breasts). Do not use antimicrobial or harsh soaps. Apply moisturizing lotion. Avoid lotions or creams that dry the skin.
- Check your feet whenever you wear new shoes. Check for ingrown toenails. Keep your nails trimmed and filed smooth and have calluses treated by a podiatrist.
- Use the wheelchair and cushion prescribed by your PT.
- Change positions frequently to relieve pressure on bony areas.
- Be sure you are not sitting or lying on anything. Avoid putting objects in your pockets.
- Check to be sure braces, leg bags, and other adaptive equipment are not too tight.
- When in bed, use padding over bony areas. Use a firm mattress. If possible, sleep on your stomach.
- Notify your health care provider of any skin breakdown.

Urinary Tract Management
- Follow the bladder program developed by the rehabilitation team.
- Drink plenty of fluids unless contraindicated.
- If you have a Foley catheter, keep it free of kinks and change it as directed by your health care provider.
- Watch for signs and symptoms of urinary tract infection (eg, cloudy urine with a foul odor, sediment in the urine).

Bowel Management
- Follow the bowel program developed by your rehabilitation team. Avoid the regular use of laxatives. Schedule sufficient time to complete the required activities. Notify your health care provider if you have not had a bowel movement in 3 or 4 days.
- Drink plenty of fluids, unless contraindicated.
- Monitor your diet to see what foods cause constipation and diarrhea.
- Prevent constipation through diet and fluid intake and medications as needed.
- Avoid foods that cause gas, such as beans, corn, and apples.
- Be aware of the potential for the development of autonomic dysreflexia during your bowel program.

Home Environment Management
- Arrange for representatives from physical and occupational therapy to evaluate the patient's home for the following:
  Wheelchair accessibility
  Clearance for maneuvering a wheelchair within the home
  Necessary adaptations to the bedroom, bathroom, and kitchen
  Smoke and fire alarms
- Obtain needed equipment for home care, depending on patient's level of injury. Be sure the equipment is delivered before the patient leaves the rehabilitation facility. Also be sure that the patient and his or her family members know how to operate the equipment.
- Make arrangements for home health care, physical therapy, occupational therapy, and job training or vocational rehabilitation, as necessary.
- Notify the electric company if there will be lifesaving equipment in the home, such as a respirator.
- Carry out patient and family education
- Locate community support groups.

Complications

Autonomic Dysreflexia
- This complication occurs in patients with injury at T5 or above.
- The most common cause is overfilling of the bladder. Other causes include constipation or gas, skin irritations, pressure sores, wounds, and ingrown toenails.
- Autonomic dysreflexia can be a life-threatening emergency. You or a helper must take immediate action to correct this problem.
- Signs and symptoms include a severe headache, nasal congestion, goose pimples, and restlessness.
- Be sure your head is up. If you are sitting in a chair, stay there; if you are in bed, get your head elevated.
- If you have an indwelling catheter:
  Check for kinks along the tubing.
  Empty the Foley bag; if there is no drainage, the Foley may be obstructed—change the Foley.
  Check the catheter and drainage bag for deposits.
  Check urine for color.

(Continued on page 849)

BOX 37–11 TEACHING GUIDE *Living With a Spinal Cord Injury* (continued)

- If you are on intermittent catheterization, catheterize yourself.
- If the problem is related to the bowel, perform a digital stimulation and empty the bowel.
- If it is not related to a bladder or bowel problem, check for a pressure sore, ingrown toenail, or possible bone fracture.
- If none of the above actions relieve the signs and symptoms, get emergency medical treatment.

Deep Venous Thrombosis

- Prevention is important.
- Signs and symptoms include leg swelling, chest pain, and cough.
- Call your health care provider immediately if signs or symptoms develop.

Hypothermia and Hyperthermia

- To prevent hyperthermia:
  Drink lots of fluids.
  Dress according to the temperature you will be in.
  Watch for signs and symptoms of hyperthermia.
  Use sun block.
- To prevent hypothermia:
- Dress appropriately for the weather.
- Watch for signs and symptoms of hypothermia and frostbite.

Heterotopic Ossification

- Check for development of abnormal bone in soft tissue, usually around the hip or knee.
- Signs and symptoms include a change in range of motion, decreased ability to perform activities of daily living, swelling, warmth, redness over the hip or knee, spasticity, and fever.
- Notify your health care provider immediately if any of these signs or symptoms develop.

Medication-Related Complications

- Select a pharmacy that will keep your medication profile on record, including any allergies you may have.
- Be sure the pharmacy has a system to check for and identify drug and food interactions.
- Have all of your prescriptions filled at the same pharmacy.
- Follow the directions on the label.
- Take all the medication that is ordered.
- Keep a current list of all medications.
- Know the important side effects of the medication; if any occur, notify your health care provider.

Pain

- Prevention of other problems such as pressure ulcers, stress ulcers, and infections is important.
- Maintain your activity program, range-of-motion exercises, and a healthy diet.
- Notify your health care provider of the type of pain you are experiencing.
- Medications and stress reduction techniques may be used.

Orthostatic Hypotension

- Know that this is a drop in BP when you first sit up.
- Wear elastic hose or an abdominal support.
- Sit up slowly.
- If you experience orthostatic hypotension while you are sitting up, ask someone to tilt your wheelchair back until your head is parallel to the floor.
- Be sure to drink plenty of fluids.

Spasticity

- Prevention is crucial. Watch for and immediately treat skin problems such as an ingrown toenail. Prevent pressure ulcers. Maintain your bowel and bladder program.
- Notify your health care provider if spasticity develops.

▲ Clinical Applicability Challenges

CASE STUDY

D.C. is a 24-year-old college student who was admitted to the Neuroscience Critical Care Unit 2 days ago secondary to injuries suffered when the bicycle he was riding was hit by an automobile. When paramedics arrived at the scene of the accident, D.C. had minimal spontaneous breathing and was unconscious. After stabilizing his neck and removing his helmet, the paramedics intubated him in the field and placed him on a spinal board. Two peripheral IV lines were started with normal saline solution. His pulse rate was 80 beats/min and BP was 90/60 mm Hg. Other than moderate abrasions on his lower arms, D.C. had no other obvious areas of injury or overt bleeding.

Upon arrival to the emergency department, D.C. began to open his eyes and attempted to talk around his endotracheal tube. The trauma staff explained where he was and began a more comprehensive neurological examination. D.C. had no voluntary movement of his arms or legs. He was able to shrug his shoulders, and he opened his eyes spontaneously. He had some sensation intact in the neck area and on the top of his shoulders. When tested, he had no perianal reflex activity, suggesting that he was in spinal shock. Radiographic evaluation and computed tomography (CT) scan demonstrated fracture at the C4–C5 level, with moderate spinal cord edema. There were no findings of head trauma on the CT scan. A loading dose of methylprednisolone (Solu-Medrol) was administered, followed by a continuous IV infusion for 23 hours.

(continued on page 850)

CASE STUDY *(Continued)*

D.C. was admitted to the critical care unit, where he was placed in a halo fixation device to realign the cervical vertebrae and stabilize the fracture. During the first 24 hours after admission, BP control was problematic as D.C. demonstrated signs of neurogenic shock, including decreased BP with bradycardia. In addition to IV fluids, he was also started on a continuous infusion of dopamine at 3 mcg/kg/min to maintain a mean arterial pressure of 85 to 90 mm Hg. Atropine was to be administered if the bradycardia became symptomatic.

Currently, 2 days after the injury, D.C. is alert and oriented to person and place but unable to talk because of oral intubation with a 7.5 endotracheal tube. He is on a mechanical ventilator, as he is still unable to initiate sufficient respiratory effort. Physical assessment reveals flaccid paralysis below the level of the injury. He is able to shrug his shoulders and discriminate between sharp and dull sensations at the corresponding level of his injury. Two peripheral IV lines are in place with normal saline solution infusing at 75 mL/h. He also has a nasogastric tube connected to low intermittent suction; a Foley catheter draining clear, yellow urine; and sequential pressure devices on both legs.

Nursing interventions include monitoring vital signs every 1 to 2 hours with complete motor and sensory evaluations every 4 hours. The nurses who are caring for D.C. continue vigilant monitoring of his respiratory status. He is repositioned (turned) every 2 hours, and the outputs of his Foley catheter and nasogastric tube are measured and recorded every 4 hours. Every effort is made to prevent complications of immobility, both in the critical care and acute care units. After consulting physical and occupational therapy, D.C.'s health care team initiates a treatment plan. Once he is past the acute phase of his injury, plans will be made to transfer him to a rehabilitation facility for further recovery and adaptation to his injury.

1. Correlate D.C.'s clinical presentation to his C4–C5 level of injury.
2. When D.C. questions (via word board or some other method of communication) whether he will require mechanical ventilation for the rest of his life, what is the nurse's best response?
3. What is the significance of loss of perianal reflex activity immediately after the spinal cord injury?

References

1. The National Spinal Cord Injury Statistical Center: Spinal Cord Injury Facts and Figures at a Glance, 2010. Available at: https://www.nscisc.uab.edu
2. Yadla S, Klimo P, Harrop JS: Traumatic central cord syndrome: Etiology, management, and outcomes. Top Spinal Cord Inj Rehabil 15(3): 73–84, 2010
3. Rechtine GR: Nonoperative management and treatment of spinal injuries. Spine 31(11 Suppl):S22–S27, 2006
4. Frederickson MD: Acute spinal cord injury management. J Trauma 62 (6 Suppl):S9, 2007
5. Ito Y, Sugimoto Y, Tomioka M, et al: Does high does methylprednisolone sodium succinate really improve neurological status in patient with acute cervical injury? Spine 34(20):2121–2124, 2009
6. Hashem R, Evans CC, Farrokhyam F et al: Plain radiography does not add any clinically significant advantage to multidetector row computed tomography in diagnosing cervical spine injuries in blunt trauma patients. J Trauma 66(2):423–428, 2009
7. Singal B, Mohammed A, Samuel J, et al: Neurological outcome in surgically treated patients with closed traumatic cervical spinal cord injury. Spinal Cord 46:603–607, 2008
8. Schinker C, Anastasiadis AP: The timing of spinal stabilization in polytrauma and in patient with spinal cord injury. Curr Opin Crit Care 14:685–689, 2008
9. Lim MR, Lee JY, Vaccaro AR: Surgical infections in the traumatized spine. Clin Orthop 444(3):114–119, 2006
10. Agarwl NK, Mathur N: Deep vein thrombosis in acute spinal cord injury. Spinal Cord 47:769–772, 2009

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

PART 9

GASTROINTESTINAL SYSTEM

38

Anatomy and Physiology of the Gastrointestinal System

Allison G. Steele and Valerie K. Sabol

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Describe the processes of ingestion, digestion, absorption, and elimination.
2. Define the functions of the major structures of the gastrointestinal system.
3. Explain digestion and absorption of carbohydrates, proteins, fats, vitamins, and minerals.
4. Describe bile production, secretion, and excretion.
5. Discuss the processes involved in emesis and defecation.

The gastrointestinal system consists of the gastrointestinal tract and the accessory glandular organs that empty their contents into the gastrointestinal tract. The major structures of the gastrointestinal tract are the mouth, pharynx, esophagus, stomach, small intestine (duodenum, jejunum, ileum), and large intestine (colon, rectum, anus). The accessory glandular organs include the salivary glands, liver, gallbladder, and pancreas.

The primary physiological functions of the gastrointestinal system are to take in nutrients for cell maintenance and growth and to eliminate waste. Cell maintenance and growth are accomplished through the processes of ingestion (taking in food), motility (mixing and propelling food through the gastrointestinal tract), digestion (breaking down food), and absorption (movement of food particles into the bloodstream). Elimination is the process by which waste is eliminated from the body.

Gastrointestinal function is regulated and coordinated by the autonomic nervous system (ANS) and a variety of peptides, which are further classified as endocrines (hormones), paracrines, and neurocrines. Endocrines are released in the general circulation and reach all tissues. Endocrine cells release paracrines, which target specific tissues. Neurocrines, or neurotransmitters, diffuse across a synaptic gap and can stimulate or inhibit the release of endocrines and paracrines.

▲ Structure of the Gastrointestinal System

Macroscopic Anatomy of the Gastrointestinal System

The gastrointestinal system is composed of the gastrointestinal tract (also called the alimentary canal), a hollow tube about 8 m (25 ft) long that begins at the mouth and ends at the anus (Fig. 38-1). The accessory glands (eg, salivary glands) and organs (eg, liver and pancreas) release secretory products into the gastrointestinal tract.

The oral cavity opens into the pharynx, a structure that allows the passage of nutrients and air. The anterior pharynx, divided into the oropharynx and nasopharynx, connects the oral and nasal cavities. The posteroinferior end of the pharynx (at about the level of the sixth cervical vertebra) connects to the esophagus and larynx. The epiglottis, a thin

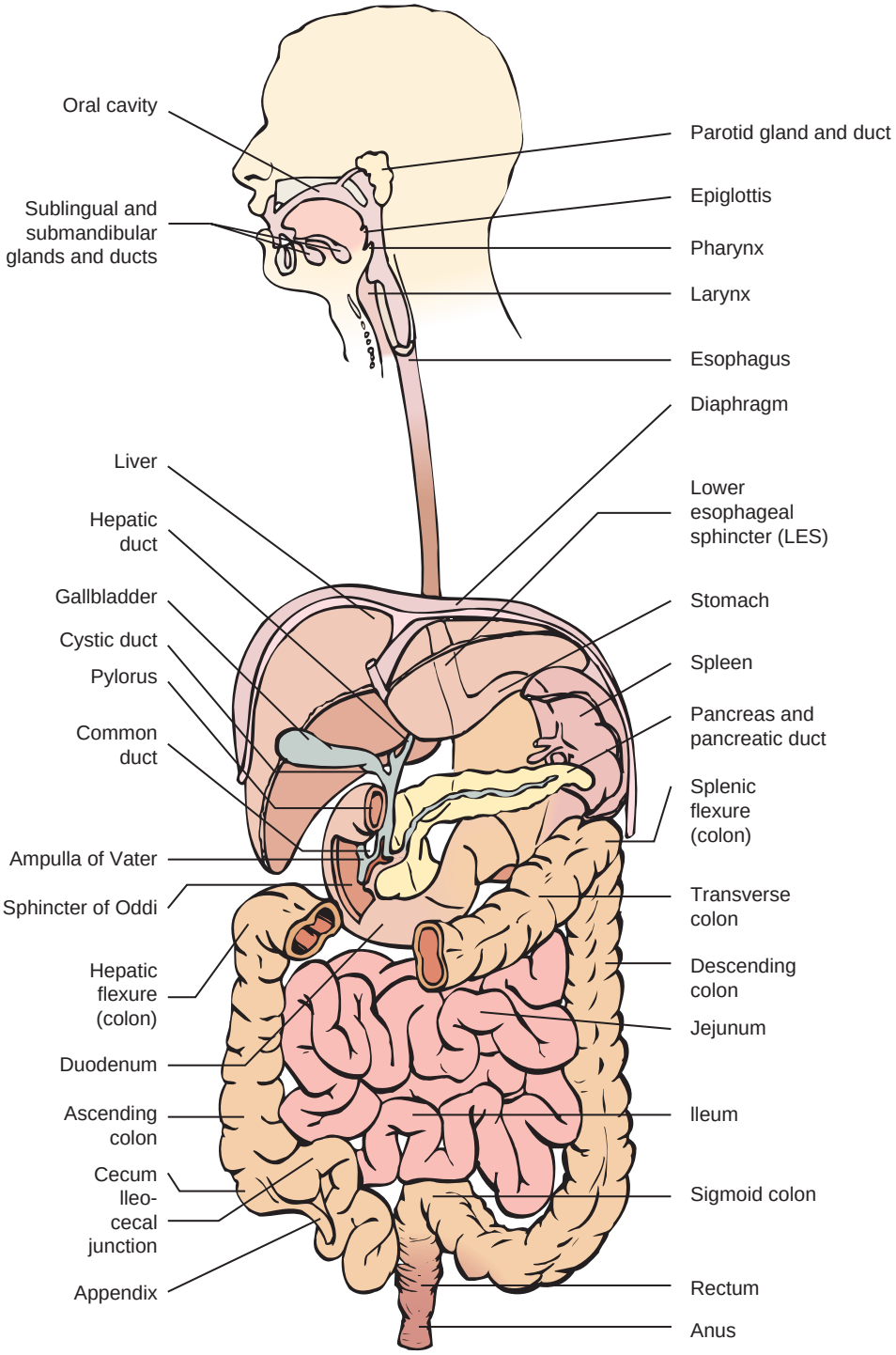


FIGURE 38-1 ▲ The gastrointestinal tract.

cartilaginous flap covered by soft tissue, reflexively covers the larynx during swallowing and prevents the passage of food and water into the trachea.

The esophagus, a 25-cm (10-inch) collapsible tube, connects the pharynx to the stomach at the cardiac orifice (Fig. 38-2). The esophagus is posterior to the trachea, in the posterior mediastinum, and crosses through the diaphragm. Its main function is to deliver food to the stomach. Two muscular rings, the upper and lower esophageal sphincters, border the esophagus. The upper esophageal sphincter (UES) prevents aspiration and swallowing of excessive air. The lower esophageal sphincter (LES), a muscular ring at the gastroesophageal junction, prevents reflux of gastric contents into the esophagus. The esophageal lumen, a central hollow tube through which food passes, is surrounded by four layers of tissue (see Microscopic Anatomy of the Gastrointestinal System section for more details). From the lumen outward, these layers are the mucosa, submucosa, muscularis propria, and serosa (Fig. 38-3).

The stomach is a flask-shaped organ that lies in the upper abdomen below the diaphragm (Fig. 38-4). The main

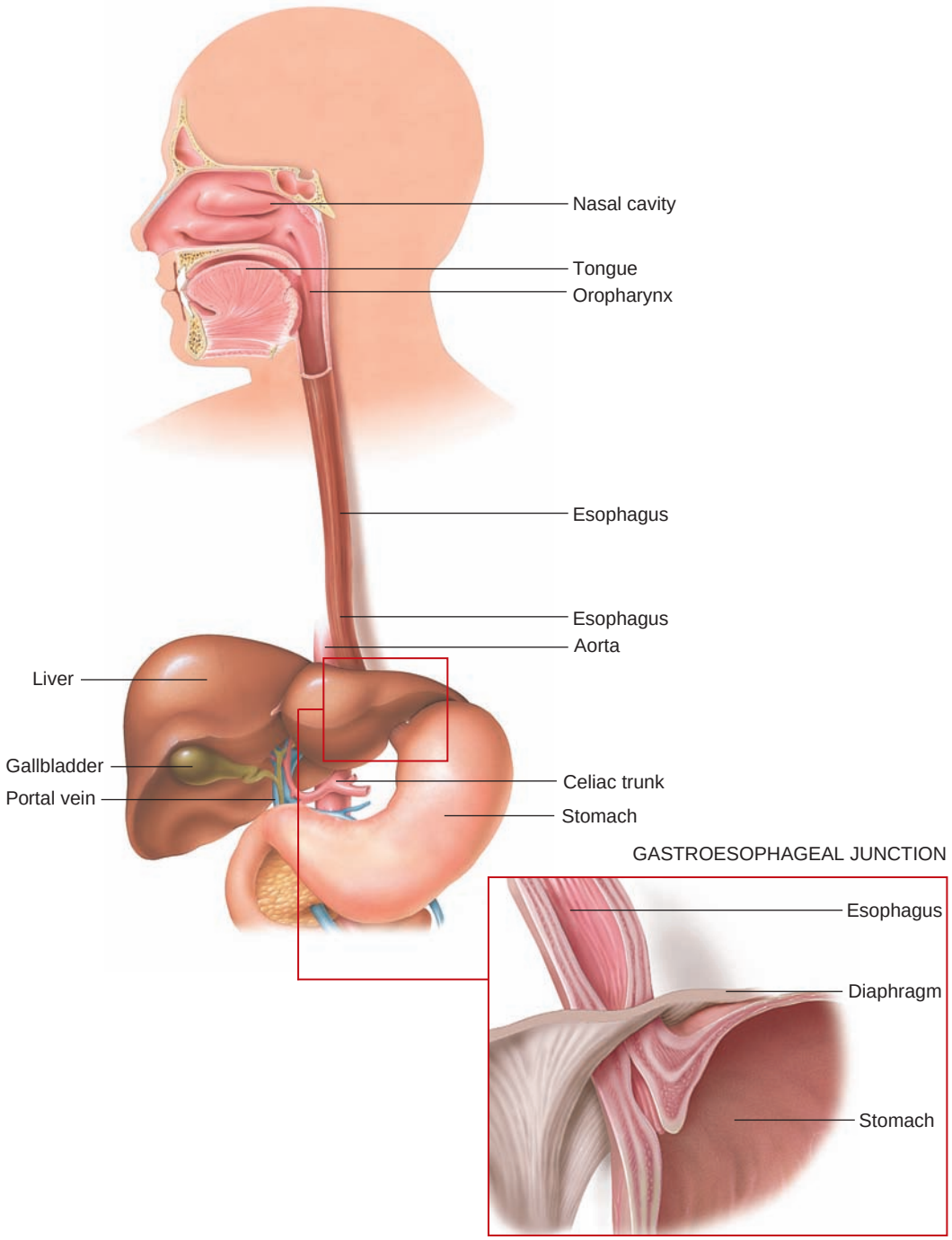


FIGURE 38-2 ▲ Gastroesophageal junction. (From Anatomical Chart Company: Atlas of Human Anatomy. Springhouse, PA: Springhouse, 2001, p 203.)

function of the stomach is storage; it acts as a reservoir for chewed food. The stomach also mixes ingested food with gastric secretions to form a semisolid liquid called chyme and regulates the release of chyme into the duodenum at a controlled rate. The esophagus joins the stomach at the cardia of the stomach. The cells of the cardia secrete mucus that helps protect the esophagus from the acidic secretions of the stomach. The dome-shaped fundus, located to the left of the cardia, acts as a reservoir. The body and the fundus have coarse folds called rugae that allow for expansion of the stomach. Gastric pits, which contain the acid-secreting cells of the stomach, are located mainly in the body of the stomach. The antrum, the most distal area of the stomach, is the site of G cells, which secrete gastrin. The antrum narrows into the pyloric channel, or pylorus, ending in the gastroduodenal junction at the pyloric sphincter. The pyloric sphincter, a muscular structure between the stomach and the duodenum, minimizes intestinal reflux.

Most digestion and absorption take place in the small intestine. The duodenum, the first 25 to 30 cm (10 to 12 inch) of the small intestine, begins at the pylorus. The common bile duct opens into the duodenum at the duodenal papilla through the

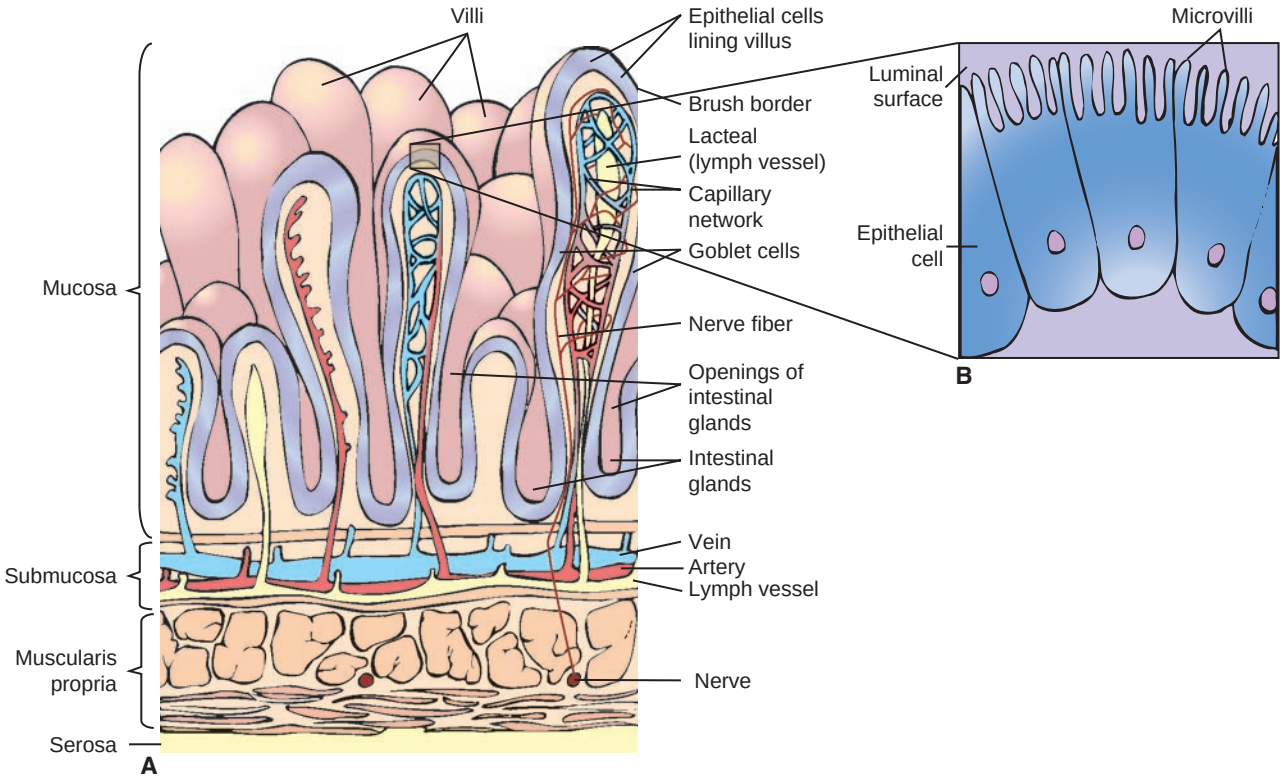


FIGURE 38-3 ▲ **A:** Layers of tissue in the gastrointestinal tract. **B:** Microvilli on the luminal surface of intestinal epithelial cells.

ampulla of Vater. The next 2.6 m (8.5 ft) of the small intestine is the jejunum. The ileum, the last 1.1 m (3.6 ft) of the small intestine, connects to the colon (cecum) at the ileocecal valve. The ileocecal valve prevents reflux of colonic contents into the ileum.

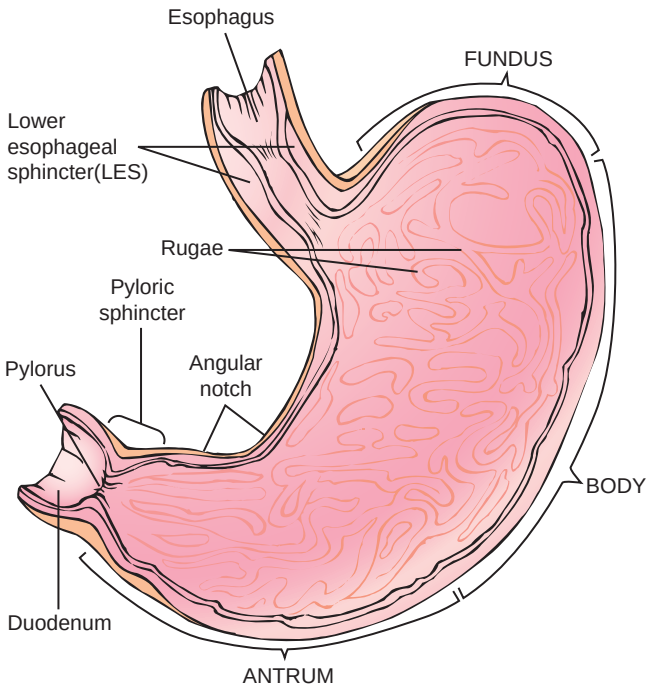


FIGURE 38-4 ▲ Anatomy of the stomach.

Traditionally, the colon is considered to have six sections. The *cecum* is the most proximal section and is the location of the ileocecal valve. The *vermiform appendix*, a blind-ended 2.5- to 20-cm (1- to 8-inch) tube, protrudes posteriorly from the cecum. The *ascending colon* extends superiorly from the cecum to the hepatic flexure. The *transverse colon* lies between the hepatic and splenic flexures. The *descending* colon extends from the splenic flexure to the level of the iliac crest. At the iliac crest, the colon becomes the sigmoid colon. The *sigmoid colon* continues downward to the pelvic floor as the rectum. The last 2.5 cm (1 inch) or so of the rectum, the anal canal, passes between the levator ani muscles of the pelvic floor and opens to the exterior body surface as the anal orifice. Two sphincters, which work to provide fecal continence, guard this orifice: an internal sphincter composed of smooth muscle and an external sphincter composed of skeletal muscle. The colon, although not necessary for life, is responsible for the reabsorption of electrolytes and fluid, thus allowing the body to maintain fluid and electrolyte balance with less fluid intake.

Microscopic Anatomy of the Gastrointestinal System

The microscopic structure of the gastrointestinal tract varies depending on location but possesses common features that are independent of the location.

Mucosa

The mucosa is composed of three layers: the epithelium, the lamina propria, and the muscularis mucosae. A single

layer of epithelial cells lines the mucosa. The tight junctions between the epithelial cells act as a barrier to bacteria and other large molecules. In the small intestine, this layer is more convoluted and possesses finger-like projections called villi (see Fig. 38-3). Such structural modifications dramatically increase the surface area of the small intestine, thereby facilitating absorption. The lamina propria, a layer of connective tissue, contains capillaries and lymph vessels. The muscularis mucosae, the innermost layer, is composed of two layers of smooth muscle. The mucosa contains cells that produce gastrointestinal secretions and cells that are sensitive to chemical and mechanical stimuli.

Submucosa

The submucosa contains blood vessels, nerve networks, and connective tissue. The submucosa of the small intestine contains aggregates of lymphatic tissue (Peyer's patches), which are especially numerous in the ileum. Specialized mucosal cells that lie superiorly to the patches of lymphatic tissue in the small intestine absorb viral and bacterial antigens. These specialized cells sensitize the lymphatic cells to antigens and manufacture and secrete antibodies of immunoglobulin class A (IgA). The antibodies protect the body from the antigen the next time (or times) it enters the small intestine.

Muscularis Propria

The muscularis propria consists of two layers of smooth muscle, an inner circular muscle layer and an outer longitudinal layer. The two smooth muscle layers function in the two major types of gastrointestinal motility: propulsive motion and mixing movements. The stomach has an additional layer of smooth muscle to facilitate its food-mixing movements.

Serosa

The serosa, or adventitia, is the outermost layer of the gastrointestinal tract. The serosa is continuous with the mesentery and forms part of the visceral peritoneum.

Innervation

The gastrointestinal tract is innervated by the ANS. The ANS can be divided into the extrinsic nervous system and the intrinsic (enteric) nervous system.

Extrinsic Nervous System

The extrinsic nervous system is further divided into parasympathetic and sympathetic branches. Parasympathetic stimulation increases gastrointestinal activity through sensory and motor fibers to promote motility, relax sphincters, and promote secretion. Activation of the sympathetic nerves usually inhibits the motor and secretory activities of the gastrointestinal system.

Parasympathetic Branch

The parasympathetic innervation of the gastrointestinal tract is primarily through the vagus and pelvic nerves. The vagal nerve (cranial nerve X) innervates the esophagus, stomach, pancreas, gallbladder, small intestine, cecum, and proximal colon. Vagal efferents synapse onto neurons in the myenteric plexus, or Auerbach plexus, which lies between the circular and longitudinal layers of smooth muscle cells in the muscularis propria. Postganglionic fibers then synapse with secretory and smooth muscle cells.

Vagal afferent (sensory) fibers originate in the esophagus, stomach, small intestine, and possibly the large intestine. The cell bodies are located in the nodose ganglion (in the neck) and join the vagus high in the neck. Afferent fibers relay information about pain and distention to the brain and spinal cord.

The pelvic nerve, issuing from spinal routes S2 to S4, carries parasympathetic afferent and efferent fibers to innervate the rectum and descending colon. For sacral efferents, the cell bodies are located in the spinal cord. Afferent cell bodies are in the corresponding dorsal root ganglia.

Sympathetic Branch

Sympathetic efferent fibers exit the spinal cord and synapse on ganglia near the spinal cord. Then, long postganglionic fibers travel to the gut and synapse on blood vessels, myenteric plexus ganglia, and secretory cells. The esophagus receives dense sympathetic innervation. Sympathetic fibers to the stomach and duodenum exit T6 to T9, synapse in the celiac ganglion, and then travel along the celiac artery. Sympathetic fibers exiting at T9 and T10 synapse in the superior mesenteric ganglion and then travel with the celiac artery to the large and small intestine. Fibers terminate on enteric neurons and blood vessels; a few fibers innervate the muscle layers.

Intrinsic (Enteric) Nervous System

The intrinsic, more commonly known enteric nervous system (ENS), coordinates gastrointestinal motility and secretion. The ENS is grouped into several nerve plexuses, with the myenteric and submucosal plexuses being the most prominent. The nerves in these plexuses receive input from receptors in the gastrointestinal tract and from the ENS. When integrated into the intrinsic system, this input helps coordinate function. Peripheral fibers innervate the voluntary muscles responsible for chewing, swallowing, and defecating.

The ENS is a complex network embedded in the wall of the gastrointestinal tract from the pharynx to the anus. It includes enteric neurons and the processes of afferent and efferent extrinsic neurons. There are two main ganglionic plexuses containing the cell bodies of enteric neurons. The outer plexus, the myenteric, or Auberbach's plexus lies between the longitudinal and circular muscle layers. The inner plexus, the submucosal plexus, or Meissner plexus lies between the circular muscle and the mucosa. The myenteric plexus mainly controls gastrointestinal movement, and the submusocal plexus mainly controls gastrointestinal secretion and blood flow.

The ENS can function on its own, independent of the extrinsic nerves, although stimulation by the parasympathetic or sympathetic nerves can further activate or stimulate its function.

Nerves in the ENS are characterized by both their function and by the neurotransmitters they contain. These include acetylcholine, norepinephrine, serotonin, and dopamine. In addition, many GI hormones have been identified in the nerves of the ENS where they act as neurotransmitters, and in the brain where they influence autonomic outflow. These include substance P, vasoactive intestinal polypeptide, gastric inhibitory peptide (GIP), and opioid peptides. There is evidence

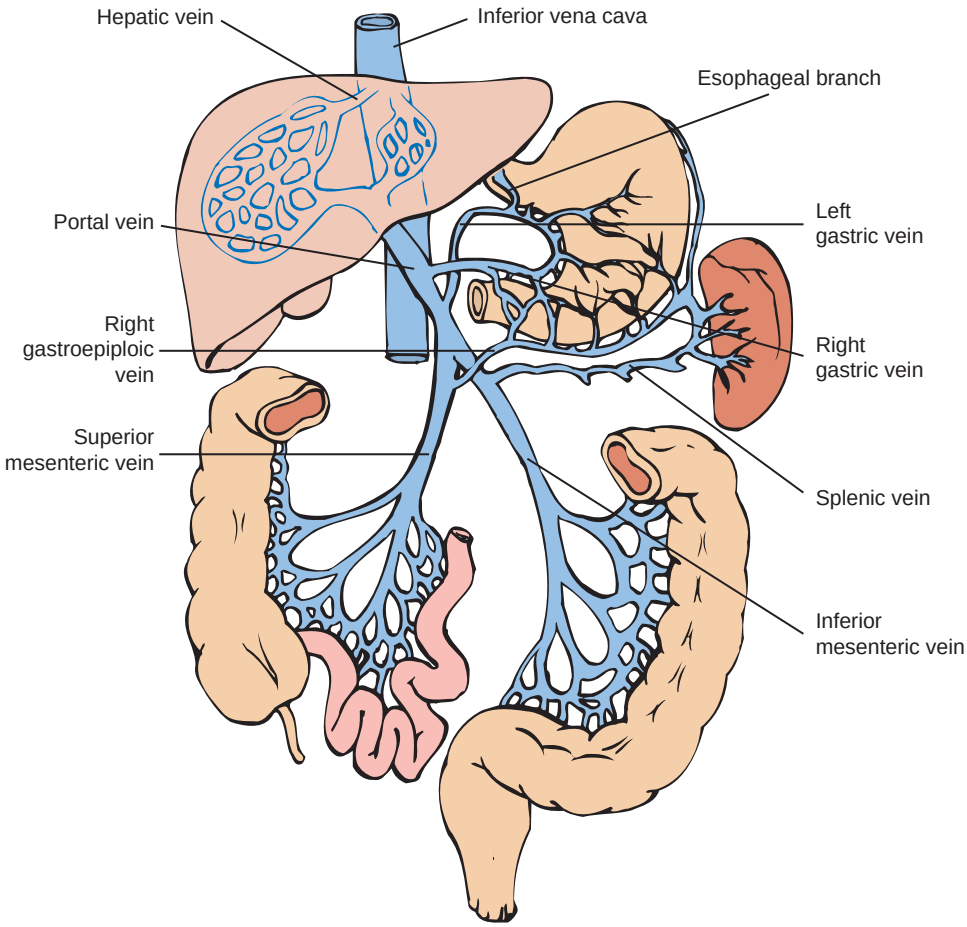


FIGURE 38-5 ▲ Portal circulation. Blood from the gastrointestinal tract, spleen, and pancreas travels to the liver by way of the portal vein before moving into the inferior vena cava for return to the heart.

that these neuropeptides participate in the control of all gastrointestinal functions (secretion, motility, and absorption).

Circulation

Blood supply to the gastrointestinal tract and spleen is called the splanchnic circulation. The gastrointestinal system receives about one fourth of the resting cardiac output, more than any other organ system. When circulation is impaired (as in shock), perfusion to the splanchnic bed is shunted to the systemic circulation. Because splanchnic organs normally extract only about 20% of the oxygen from the perfusing blood, splanchnic perfusion can be reduced without compromising the organs. However, a severe reduction in splanchnic perfusion can damage the mucosal lining of the gut.

The esophageal artery branches from the thoracic aorta and perfuses the esophagus. Three branches of the abdominal aortic artery perfuse the gastrointestinal organs:

- The celiac axis (consisting of the left gastric artery, the common hepatic artery, and the splenic artery) perfuses the lower esophagus, stomach, duodenum, gallbladder, and liver.
- The superior mesenteric artery perfuses the small intestine to transverse colon.
- The inferior mesenteric artery perfuses the descending colon, sigmoid colon, and rectum.

The areas of perfusion overlap, providing some protection against ischemia.

Venous drainage of the stomach and small and large intestines is primarily through the portal vein to the liver. The blood supply from the lower rectum and the lower esophagus bypasses the portal system. Blood from the rectum drains into the inferior vena cava through the rectal veins, which empty into the external iliac vein. Blood from the esophagus drains through the hemiazygos and azygos veins into the inferior vena cava.

The blood supply of the liver is unique. The liver receives its blood supply from both venous and arterial sources. The venous blood is supplied by the portal vein, which drains most of the blood from the gastrointestinal tract (Fig. 38-5). The portal vein forms behind the spleen at the confluence of the superior mesenteric and splenic veins and leads to the liver. The arterial supply is by the common hepatic artery, which branches from the celiac trunk near the aorta and then perfuses the liver. Both sets of vessels form capillaries and then drain into the hepatic vein, which in turn feeds into the inferior vena cava.

▲ Function of the Gastrointestinal System

The main function of the gastrointestinal tract is to break down nutrients into a form of usable energy. Food is ingested in the form of macromolecules that cannot be absorbed. These macromolecules are converted into usable forms of energy by mixing with digestive enzymes and secretions as

they move through the gastrointestinal tract. These processes will be discussed in relationship to the various parts of the gastrointestinal tract and the secretions and motility that make digestion possible.

Oropharynx

Secretions

The salivary glands of the oropharynx produce saliva. There are three pairs of salivary glands: the submaxillary glands, the sublingual glands, and the parotid glands. These glands are drained by ducts into the mouth. Saliva is composed of mucus (a lubricant that facilitates swallowing), lingual lipase (a fat-digesting enzyme secreted by tongue glands), salivary amylase (an enzyme that breaks down starch), and class A (IgA) antibodies (which provide a first line of defense against bacteria, viruses, and bacteriostatic and carcinogenic chemicals; Table 38-1). A moist oral cavity also facilitates speech. The pH of saliva is 7; saliva contains bicarbonate, which allows it to neutralize acid substances that enter the oral cavity, including regurgitated gastric acid. Lingual lipase

Table 38-1 Major Gastrointestinal Secretions

Location	Daily Volume	Composition (and Action)
Mouth	1,000–2,000 mL	Amylase (carbohydrate digestion) Lipase (fat digestion) Immunoglobulins Mucus Water, electrolytes
Esophagus	300–800 mL	Mucus
Stomach	2,000 mL	Intrinsic factor (vitamin B_{12} absorption) Hydrochloric acid (activates pepsinogen) Pepsinogen (protein digestion) Mucus Water, electrolytes Gastrin (stimulates hydrochloric acid release; trophic effects on mucosa, especially in stomach)
Pancreas	1,200–1,800 mL	Enzymes • Amylase (carbohydrate digestion) • Trypsinogen (protein digestion) • Chymotrypsin (protein digestion) • Elastase (protein digestion) • Carboxypeptidase (protein digestion) • Lipase (fat digestion) • Colipase (fat digestion) • Esterase (cholesterol digestion) • Phospholipase (phospholipid digestion) • Nucleases (RNA and DNA digestion) Bicarbonate (protects luminal wall by neutralizing acid) Water, electrolytes
Liver	500–1,000 mL	Bile salts (emulsify fats) Bilirubin (excretory end product of hemoglobin breakdown) Water, electrolytes
Small intestine	3,000–4,000 mL	Enzymes • Enterokinase (activates trypsinogen) • Lipase (fat digestion) • Enteropeptidase (protein digestion) • Peptidase (protein digestion) • Nucleases (RNA and DNA digestion) • Maltase (carbohydrate digestion) • Lactase (carbohydrate digestion) • Sucrase (carbohydrate digestion) Mucus Bicarbonate Water, electrolytes CCK into blood (stimulates pancreatic secretion and gallbladder contraction) Glucose-dependent insulinotropic peptide into blood (stimulates insulin release and gastric motility, secretion) Gastrin (stimulates gastric acid secretion)
Large intestine	Variable	Mucus

digests about 30% of the dietary fat in the stomach. The salivary glands secrete about one half of the digestive amylase used in digestion; the rest is secreted by the pancreas.

Saliva production is elicited by multiple stimuli, including the sight, smell, or thought of food, and by the pleasant taste or smooth texture of food in the mouth. Rough, bad-tasting, unpleasant-smelling foods reduce salivary gland secretions. Stimuli are received by the two salivary centers in the medulla of the brainstem, which then send impulses to the salivary glands through the seventh and ninth cranial nerves (parasympathetic fibers) and the first and second thoracic nerves (sympathetic fibers). Parasympathetic stimulation, or the administration of drugs that mimic stimulation (cholinergics) or enhance it (neostigmine), promotes copious secretion of watery saliva. Sympathetic stimulation or sympathomimetic drug administration produces a scanty output of thick saliva. Cholinergic blockers (eg, atropine) also inhibit salivation.

Motility

In the mouth, chewing mechanically breaks down food into smaller particles. This produces a bolus of food held together and lubricated by saliva that can then be propelled into the stomach by the process of swallowing. Swallowing is a complex process that has several phases (Fig. 38-6). During the oral phase, the tongue propels the food or fluid bolus to the posterior pharynx. This is a voluntary process. During the involuntary pharyngeal phase, the presence of food or fluid in the pharynx stimulates pharyngeal sensory receptors that initiate impulses through cranial nerve V (the trigeminal nerve) to the swallowing center in the medulla. Sensory impulses reflexively trigger the outflow of impulses down motor fibers in cranial nerve IX (the glossopharyngeal nerve) and cranial nerve X (the vagus nerve) to pharyngeal and laryngeal structures. This causes the following coordinated events, which propel the solid or fluid substance into the esophagus:

1. The soft palate elevates and retracts, sealing off the nasopharynx to prevent regurgitation.
2. The vocal cords close, and the epiglottis closes over the larynx to prevent aspiration.
3. The UES relaxes.
4. The larynx pulls up and increases the opening of the esophagus and UES.
5. The pharyngeal muscles contract, propelling food or fluid into the opened esophagus.

During this phase, respiration is reflexively inhibited. Damage to sensory or motor fibers (in cranial nerves V, IX, or X) or to the swallowing center in the brainstem weakens or eliminates the ability to swallow or causes poorly coordinated swallowing, wherein food or fluid enters the nasopharynx or larynx, or both.

Esophagus

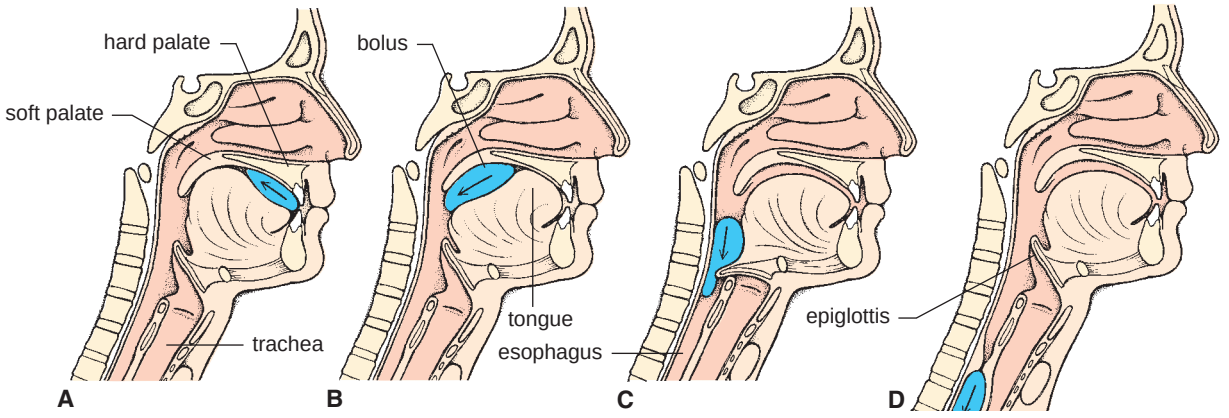
Secretions

Esophageal mucosal cells secrete mucus (see Table 38-1, p. 857). The mucus protects the esophageal lining from damage by gastric secretions or food and acts as a lubricant to facilitate the passage of food.

Motility

The esophageal phase of swallowing begins once food or fluid enters the esophagus (Fig. 38-7). Swallowing-induced contractions of the esophagus are called primary peristalsis. The wave of peristalsis causes the LES to relax, thereby allowing food to enter the stomach. If primary peristalsis cannot clear the esophagus, food or fluid distends the esophagus. This distention stimulates stretch receptors that reflexively promote relaxation of the esophageal muscles ahead of the area of distention as well as contraction of the esophageal muscles in and behind it. This propels the food or fluid ahead into the newly relaxed area, which then becomes distended. This is called secondary peristalsis. The peristalsis reflex repeatedly recurs until the food or fluid arrives at the LES.

The tone of the LES can be altered by a variety of agents (Table 38-2). Some people suffer from a hypertrophic LES, which impedes esophageal emptying (and can lead to overdistention of the lower esophagus), whereas others have an incompetent LES, which results in repeated episodes of gastric reflux (which can lead to lower esophageal strictures).



swallowing: (A) bolus is pushed back; (B) nasopharynx closes; (C) epiglottis closes the trachea; (D) bolus is moved down the esophagus

FIGURE 38-6 ▲ Swallowing. Passage of bolus of food from the mouth through the pharynx.

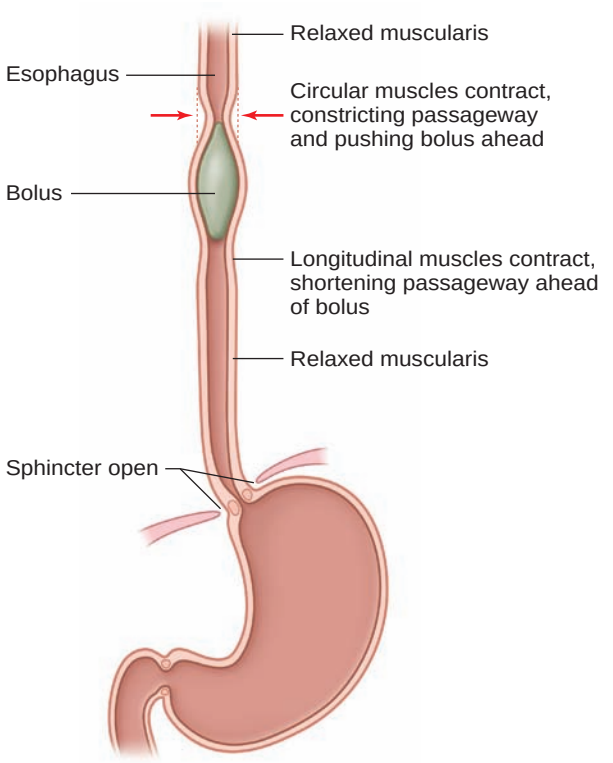


FIGURE 38-7 ▲ Movement of a bolus of food through the esophagus by a peristaltic contraction.

Stomach

Secretions

The major secretions of the stomach are hydrochloric acid, intrinsic factor, pepsinogen, gastrin, and mucus (see Table 38-1, p. 857). The oxyntic glands contain parietal cells that secrete hydrochloric acid and intrinsic factor. Hydrochloric acid converts pepsinogen, which is secreted by the chief cells of the stomach, to pepsin, a proteolytic enzyme. The hydrochloric acid provides an ideal pH for the activity of pepsin; together, hydrochloric acid and pepsin begin the digestion of protein. The chemical action of hydrochloric acid also breaks down food molecules and helps protect the gastrointestinal tract from bacterial invasion. Intrinsic factor is necessary for the absorption of vitamin B_{12} in the small intestine.

G cells, located in the gastric antrum, secrete the hormone gastrin, which promotes the secretion by the chief and parietal cells and promotes the growth of the gastric mucosa (Table 38-3). Overproduction of gastrin, a condition known as Zollinger-Ellison syndrome, results in gastric hypersecretion and peptic ulceration.

Gastric mucosal cells continuously secrete a thin coat of mucus. Mucus, a lubricant, works together with bicarbonate to neutralize acid, protecting the stomach wall from damage. This barrier can be disrupted by a variety of agents, including bile salts, alcohol, aspirin, nonsteroidal anti-inflammatory drugs, and infection with *Helicobacter pylori*.

Table 38-2 Factors Influencing Lower Esophageal Sphincter Tone

Increased Tone	Decreased Tone
Food substances:	Food substances:
Protein	Fats
Drugs:	Coffee
Metoclopramide	Chocolate
Some prostaglandins (F_2)	Alcohol
	Peppermint
	Tomato products
	Citrus juices
	Carbonated beverages
	CCK
	Progesterone (as in pregnancy)
	Somatostatin
	Dopamine
	Some prostaglandins (E_2, A_2)
	Cigarette smoking

Factors Affecting Gastric Secretions

The gastric parietal cells contain receptors for acetylcholine, histamine, and gastrin. Stimulation of these receptors prompts the parietal cells to secrete hydrochloric acid. Hydrochloric acid secretion is inhibited by chemicals that block the histamine receptors (eg, H_2-receptor antagonists) or the acetylcholine receptors (eg, atropine). Proton pump inhibitors inhibit the H^+/K^+-adenosine triphosphatase (ATPase) enzyme pathway, the final common step in the acid secretory pathway. Some prostaglandins also inhibit hydrochloric acid secretion.

Factors that stimulate gastric secretions include alcohol, caffeine, and hypoglycemia. The first two factors act directly by way of gastric chemoreceptors and the intramural nerve plexuses in the stomach wall. Hypoglycemia acts by way of the brainstem and vagal fibers.

Control of Gastric Secretions

Gastric secretions are regulated in three phases: the cephalic phase, the gastric phase, and the intestinal phase (Table 38-4). These phases are controlled by neural and hormonal mechanisms.

In the cephalic phase, the sight, smell, taste, or thought of food stimulates brainstem centers, reflexively prompting parasympathetic (vagal) stimulation of salivation, pancreatic secretion, bile release, and gastric secretions of pepsinogen and hydrochloric acid by the chief and parietal cells, respectively. Sympathetic stimulation can alter the cephalic phase response. This is the mechanism by which emotions can influence gastrointestinal secretions. Fear, anger, and depression decrease secretions.

During the gastric phase, distention of the stomach by food stimulates stretch receptors in the stomach wall. Chemicals, mainly proteins, stimulate chemoreceptors in the mucosa. The stretch receptors and chemoreceptors in turn activate neurons in the submucosal plexus, which then stimulate neurons in the myenteric plexus, which in turn stimulate secretion by the parietal and chief cells. Proteins in the chyme also directly promote gastrin secretion by G cells; the gastrin provides an additional stimulus for parietal and chief cell secretion.

Table 38-3 Hormones Controlling Secretion and Motility

Hormone	Source	Stimulation of Release	Major Function
Gastrin	Stomach, small intestine	Gastric distention, presence of partially digested protein near pylorus	Stimulates • Gastric acid secretion • Gastric intrinsic factor secretion • Gastric motility • Intestinal motility • Mucosal growth • Pancreatic growth • Pancreatic insulin release • Lower esophageal tone
Secretin	Small intestine	Acid entering small intestine	Stimulates • Pancreatic bicarbonate secretion • Pancreatic enzyme secretion • Pancreatic growth • Gastric pepsin secretion • Bile bicarbonate secretion • Gallbladder contraction Inhibits • Gastric emptying • Gastric motility • Intestinal motility
CCK	Small intestine	Fatty acids and amino acids in small intestine	Stimulates • Gastric acid secretion • Gastric motility • Intestinal motility • Colonic motility • Gallbladder contraction and sphincter of Oddi relaxation (thus increasing bile flow into small intestine) • Pancreatic bicarbonate secretion • Pancreatic enzyme release • Pancreatic growth Inhibits • Lower esophageal tone • Gastric emptying
GIP	Small intestine	Fatty acids and lipids in small intestine	Stimulates • Insulin release • Intestinal motility Inhibits • Gastric acid secretion • Gastric emptying • Gastric motility
Motilin	Small intestine	Acid and fat in small intestine	Stimulates • Gastric motility • Intestinal motility

A combination of events eventually brings the gastric phase to a halt: the stretch receptors and chemoreceptors in the wall of the stomach become refractory to stimulation, the acidity of the chyme inhibits further gastrin secretion, and GIP decreases hydrochloric acid secretion and gastric motility.

The intestinal phase begins after chyme reaches the duodenum. The acidity of the chyme stimulates duodenal mucosal cells to release secretin into the bloodstream; proteins and fat trigger the release of cholecystokinin (CCK) into the blood from similar cells, and glucose and fat stimulate the secretion of GIP. Secretin and CCK cause pancreatic secretion and release of gallbladder contents into the duodenum. GIP stimulates the release of insulin from the islets of Langerhans and decreases gastric motility and secretions (see Table 38-3). Stretch receptors in the duodenum trigger peristalsis so that chyme is degraded, mixed with enzymes and diluents, and moved past the highly absorbent small intestinal lumen. If the chyme is less acidic, gastrin is released. Under neural control, motilin is another hormone that is cyclically released during fasting. Motilin stimulates stomach and small intestine motility.

Motility

The passage of food from the esophagus into the stomach reflexively initiates receptive relaxation. After the stomach has filled with food, peristaltic contractions mix the food and propel gastric contents toward the pylorus, where small

Table 38-4 Phases of Gastric Secretion

Phase	Stimulus to Secretion	Effect
Cephalic (neuronal)	Sight, smell, taste of food initiates central nervous system impulse mediated by vagus nerve	Gastric effects: Hydrochloric acid (from parietal cells) Pepsinogen (from chief cells) Mucus secretion Other effects: Salivation Pancreatic secretion Bile release
Gastric (neuronal and hormonal)	Food in antrum initiates central nervous system impulse mediated by vagus nerve	Gastrin release Hydrochloric acid release Pepsinogen release
Intestinal (hormonal)	Chyme in small intestine	pH of chyme <2: release of secretin, gastric inhibitory polypeptide, CCK (decreases gastric acid secretion) pH of chyme >3: release of gastrin (increases gastric acid secretion)

amounts enter the duodenum. The pyloric sphincter plays a minor role in gastric emptying; its main function is to prevent duodenal reflux. The bile acids in the chyme that reenters the stomach through duodenal reflux damage the chemical barrier that coats the surfaces of gastric mucosal cells. Mild peristaltic contractions that persist after the stomach has completely emptied are called hunger contractions; however, they play no role in appetite regulation. Gastric emptying can be retarded by vagotomy; by the presence of fats, proteins, or hydrochloric acid in the duodenal chyme; by duodenal distention; and by intestinal hormones.

Emesis, or vomiting, is the regurgitation of food from the stomach through the mouth. During vomiting, the abdominal muscles and diaphragm contract, and the LES relaxes, allowing reflux of gastric content into the esophagus and propulsion of gastric contents out of the mouth. The reflex elevation of the palate prevents expulsion through the nasopharynx. Respiratory inhibition and closure of the glottis prevent pulmonary aspiration. In addition, irritation of the small intestine (by materials in the chyme, by inflammation, or by a disease process) can cause reverse peristalsis. These movements move chyme toward the pyloric valve. If strong enough to force open the pylorus, intestinal contents may be vomited. When yellow bile from the duodenum is exposed to acid in the stomach, the interaction turns the vomitus green. Occasionally, vomiting of intestinal contents can be so rapid that the vomitus contains yellow bile. When blood is exposed to acid in the stomach, the exposure results in a brownish-black "coffee-ground" emesis. If the rapidity of vomiting does not allow sufficient time for this interaction between acid and blood to occur, blood in the vomitus has its normal red color (hematemesis).

Pancreas

The pancreas is composed of both exocrine and endocrine tissue. The islets of Langerhans, endocrine tissue scattered throughout the pancreas, secrete insulin, glucagon, and pancreatic polypeptide hormones, which aid in the digestive process. The exocrine pancreas is composed of acinar cells, which are arranged in lobules. The acinar cells empty secretions into an internal pancreatic ductal system (Fig. 38-8). These internal ducts drain into progressively larger ducts that terminate in the duct of Wirsung, the main pancreatic duct. This pancreatic duct then joins the common bile duct to form a shared short duct called the ampulla of Vater. This ampulla, carrying bile and pancreatic secretions, opens into the duodenum. A smooth muscle ring, the sphincter of Oddi, encircles the ampulla. Because of the anatomical arrangements between the common bile duct and the duct of Wirsung, a gallstone that obstructs the ampulla of Vater can obstruct the normal flow of bile and pancreatic secretions. (Such obstruction, although rare, can lead to a stasis of pancreatic secretion, resulting in acute pancreatitis.) Some people have a second external pancreatic duct (duct of Santorini) that opens into the duodenum near the pylorus.

The exocrine acinar cells secrete both a watery alkaline bicarbonate solution and enzymes (see Table 38-1, p. 857). The large amount of water secreted by the pancreas is instrumental in diluting chyme before absorption. In addition, the bicarbonate neutralizes the highly acidic chyme from the stomach. The pancreatic enzymes digest proteins (trypsin, chymotrypsin, elastase, and carboxypeptidase), fats (lipase, colipase, and esterase), phospholipase and nucleic acids (nucleases), and starch (amylase). Although pancreatic enzymes require a pH close to neutrality for optimal activity, they are capable of nearly completing the digestion of food in the absence of all other digestive secretions.

Pancreatic enzymes are secreted from the pancreas in their inactive forms. Trypsin inhibitor prevents the premature activation of trypsinogen into its active form, trypsin. Once the pancreatic secretions arrive in the duodenum, trypsinogen is activated by an intestinal mucosal enzyme, enterokinase, into its active form, trypsin. Trypsin then activates the other pancreatic enzymes.

Regulation of pancreatic secretion occurs by neural and hormonal means. Vagal stimulation results in the secretion of pancreatic enzymes. Hormonal regulation occurs as a result of duodenal mucosal responses to chyme and is discussed later.

Gallbladder

In the duodenum, chyme mixed with pancreatic secretions is watery. The fat in chyme is not water soluble and

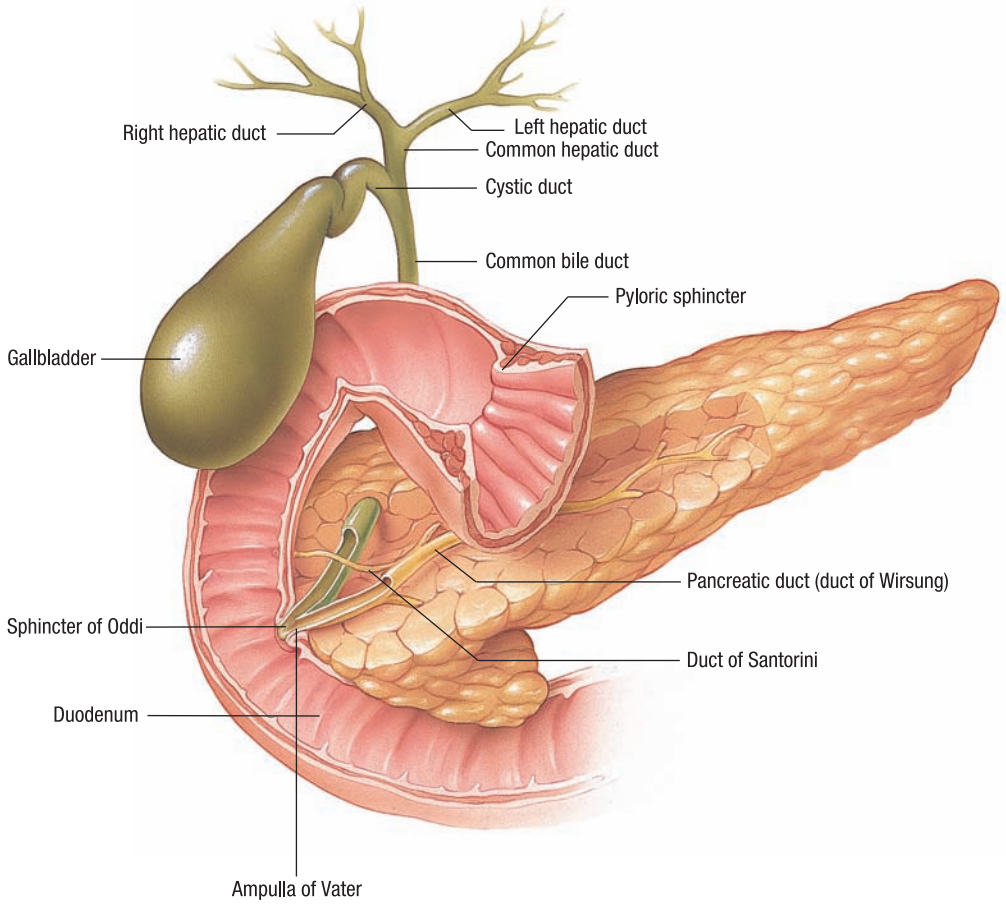


FIGURE 38-8 ▲ Biliary system. (From Anatomical Chart Company: Atlas of Human Anatomy. Springhouse, PA: Springhouse, 2001, p 217.)

requires a solvent enzyme mixture from the liver to render it absorbable by intestinal cells. Hepatocytes, among many other metabolic functions, make bile. Bile is a mixture of bile salts, cholesterol, bilirubin, and acids suspended in water. This solution emulsifies the fat in chyme, breaking the fat into small globules that can be absorbed across the intestinal lumen. The action of bile ionizes fat-soluble vitamins into absorbable forms. Bile also suspends cholesterol, triglycerides, and multiple-density lipoproteins in the bloodstream, thus preventing precipitation and deposition of these molecules in the vasculature until they can be catabolized.

Bile is stored and concentrated in the gallbladder. Gallbladder secretion is greatest during the intestinal phase of digestion. This activity is stimulated by CCK, which is secreted by the intestinal mucosa when fatty acids or amino acids are present. CCK causes gallbladder contraction and relaxation of the sphincter of Oddi, allowing the release of bile into the duodenum to mix with chyme.

Small Intestine

Secretions

In the duodenum, chyme mixes with pancreatic digestive enzymes, alkaline substances, water, mucus, and bile. When the mucosa of the small intestine is exposed to acid in the chyme, secretin is released. Secretin stimulates bicarbonate release by cells that line the bile ducts. The intestinal enzymes secretin, CCK, and enterokinase are added to this mixture, along with mucus, bicarbonate, and water. The small intestine secretes 3 to 4 L a day of alkaline secretions. These intestinal secretions help maintain the liquidity of chyme and can dilute noxious agents.

Motility

The small intestine has two types of characteristic movements, propulsive and mixing. The intramural plexuses initiate and coordinate these movements, but the movements can be enhanced or retarded by extrinsic autonomic stimulation, as discussed previously. Propulsive movements propel food forward, allowing for digestion and absorption. This peristalsis is stimulated by distention. During mixing movement, localized concentric contractions of the intestinal wall called segmentation promote mixing of food particles. These segments have the appearance of linked sausages. Repetition of this process continually kneads the chyme, which increases the exposure of the molecules to the absorptive surfaces of the intestinal mucosa.

Emptying of the small intestine into the colon occurs in the same way as gastric emptying. Peristaltic waves build pressure in the ileum behind the ileocecal valve and push the

chyme through the valve into the colon. The ileocecal valve then prevents backflow. Ileal emptying can be retarded by intramural reflexes, which are initiated by a full (distended) colon.

Absorption

The major functions of the small intestine are absorption and digestion, which are facilitated by secretions from the pancreas, liver, and gallbladder. The mucosal layer of the small intestine has many folds (valvulae conniventes) covered with numerous finger-like projections (villi) and microvilli, which dramatically increase the absorptive surface area of the small intestine.

CARBOHYDRATES. The three major sources of carbohydrates in the human diet are sucrose, lactose, and starch. The breakdown of carbohydrates begins in the mouth when food mixes with salivary amylase during chewing. The digestion continues in the duodenum. Conversion to simple sugars continues in the small intestine by intestinal enzymes. Both active and passive transport are used to absorb sugars across the intestinal lumen into the bloodstream.

PROTEINS. Protein degradation is initiated in the stomach through the actions of hydrochloric acid and pepsin. However, in the absence of pepsin and hydrochloric acid, the small intestine is capable of fully digesting all available protein. Most digestion occurs in the duodenum and jejunum by proteolytic pancreatic enzymes. Polypeptides in the small intestine are degraded into peptide fragments and amino acids by trypsin, chymotrypsin, and carboxypeptidase. Amino acids are absorbed into the blood by active and passive diffusion.

FATS. Triglycerides, lipids, and phospholipids are first degraded in the small intestine. Bile salts, in a process called emulsification, facilitate the creation of small droplets of fats from larger globules. Pancreatic enzymes then degrade the fats into fatty acid chains and monoglycerides. These smaller molecules form into even smaller globules, called micelles. Fatty acids and monosaccharides are transported across the intestinal mucosa from a micelle passively, leaving bile behind.

In the submucosa, free fatty acids are passed into the blood directly, if small enough. If too large for direct passive diffusion, the free fatty acid is reorganized into a triglyceride, coupled with lipoproteins and cholesterol, and passed into the lymph fluid as chylomicron.

The bile left behind in the intestine after absorption of fats from a micelle is reabsorbed in the ileum. If bile salts enter the colon, they decrease the reabsorption of sodium and water, thereby increasing the liquidity of the undigested food residues in the colon. Most fat is absorbed by the time chyme reaches the middle of the jejunum.

VITAMINS, MINERALS, AND WATER. Most vitamins, whether fat or water soluble, diffuse across the intestinal mucosa and submucosa into the blood. Fat-soluble vitamin B_{12} couples with intrinsic factor, forming a larger molecule. In this form, vitamin B_{12} is absorbed in the ileum.

Minerals and electrolytes vary in their absorption. Sodium and iron require active transport, whereas other minerals and electrolytes diffuse passively. Iron is primarily absorbed in the duodenum.

Water is absorbed passively throughout the stomach and small and large intestines. The gastrointestinal tract is highly permeable, in both directions, to water. If a hypertonic solution enters the duodenum, osmosis occurs within the lumen. The converse is also true: a hypotonic chyme in the stomach and duodenum causes extremely rapid movement of water into the bloodstream.

Large Intestine

Secretion

The goblet cells of the colonic mucosa secrete mucus, which lubricates the passage of chyme (see Table 38-1, p. 857). The production of mucus is stimulated by irritation and by cholinergic activation.

Motility

Colonic movements include mixing and peristaltic movements. These operate as described for the small intestine. A third movement, unique to the colon, is mass movement. This consists of simultaneous contractions of colonic smooth muscle over large portions of the descending and sigmoid portions of the colon. Mass movement rapidly moves the undigested food residue (feces) from these areas into the rectum.

Humans cannot digest the cellulose, hemicellulose, or lignin in plant tissues. These plant materials form a large portion of the undigested food residue. They are usually termed vegetable fiber or dietary bulk. These fibers attract and hold water, creating a larger, softer stool. Low quantities of bulk result in a relatively inactive colon, leading to bowel movements that are relatively infrequent and feces that are relatively small, dry, and difficult to pass. Epidemiological reports suggest that high-fiber diets are associated with a decreased incidence of diverticulitis and colon cancer.

Filling of the rectum triggers the defecation reflex by stimulating stretch receptors in the rectal wall. Stimulation of the stretch receptors causes sensory (afferent) nerve fibers to transmit impulses to the lower spinal cord. Because of anatomical arrangements of neurons in this part of the cord, these afferent impulses reflexively cause nerve impulses to travel out of the cord along parasympathetic motor fibers that innervate the smooth muscles of the descending and sigmoid colon, the rectum, and the internal anal sphincter. The afferent impulses also reflexively cause nerve impulses to be sent out of the cord along somatic motor neurons that innervate the skeletal muscle of the external anal sphincter. The total effect of these events is to produce coordinated expulsive contractions of the colon and rectum, relaxation (opening) of the sphincters, and expulsion of feces from the anus.

The urge to defecate begins after the pressure within the rectum reaches 18 mm Hg. After intrarectal pressure reaches 55 mm Hg, reflex bowel evacuation occurs. This defecation reflex is inhibited in a continent person by descending neuronal impulses from higher brain centers that inhibit the actions of the somatic motor neurons that innervate the external sphincter. Such inhibition keeps the

external anal sphincter closed, thereby averting inappropriate defecation. After a few minutes, the defecation reflex subsides, but it usually becomes active again a few hours later. Defecation is a spinal cord reflex that does not require intact pathways between the sacral cord and the brain. In the early posttraumatic phase of spinal shock, the reflex does not work. After cord shock is ended, reflex defecation occurs once again, but voluntary inhibition is not possible (neurogenic bowel).

Absorption

In the large intestine, most of the water and potassium are absorbed from the chyme. This produces a semisolid residue of undigested food (feces) that can be eliminated from the body. Diarrhea can reduce the transit time for chyme, thereby limiting such potassium and water reabsorption. This can result in hypokalemia and dehydration. Diarrhea can be caused by materials that hold water in the chyme (eg, magnesium sulfate), resulting in semiliquid stool.

At birth the colon is sterile, but large colonic bacterial populations become established soon afterward. Some of these organisms produce vitamin K and a number of B vitamins. Other bacteria produce ammonia, which is absorbed. Normally, ammonia is removed from the blood once it reaches the liver. However, in people with seriously impaired liver function or with collateral circulatory routes that bypass the liver (usually the result of portal hypertension), ammonia can remain in the circulation and lead to hepatic encephalopathy.

Liver

The liver lies in the right upper quadrant of the abdomen. It has two lobes (right and left) and lies just below the diaphragm, with its greatest portion located on the right side of the body. Its superior (rounded) surface fits into the curve of the diaphragm and is in contact with the anterior wall of the abdominal cavity. The inferior surface is molded over the stomach, the duodenum, the pancreas, the hepatic flexure of the colon, the right kidney, and the right adrenal gland.

The liver is covered with a thin layer of peritoneum over a thin fibrous coat called Glisson's capsule. This fibrous capsule encases and partitions the liver, sending inward fibrous sheets that divide the liver into functional units called lobules. Each lobule consists of sheets of hepatocytes organized around a core cluster of vessels called the portal triad (Fig. 38-9). The portal triad includes the two sets of afferent vessels (portal vein and hepatic artery) and a small bile duct. The afferent vessels lead to the liver sinusoids, which drain into the efferent hepatic vein, lying at the periphery of each lobule.

The lobule measures approximately 1.5 mm in diameter and 8 mm in length. Each lobe of the liver contains between 50,000 and 100,000 lobules. Rows of hepatocytes radiate from a central venule like spokes of a wheel. Branches of the hepatic artery and the hepatic portal vein lie at the periphery of the wheel. Blood from these branches is poured into open channels (hepatic sinuses) that run between alternate rows of hepatocytes. Kupffer's cells, specialized white cells of the reticuloendothelial system, phagocytize bacteria, debris, and

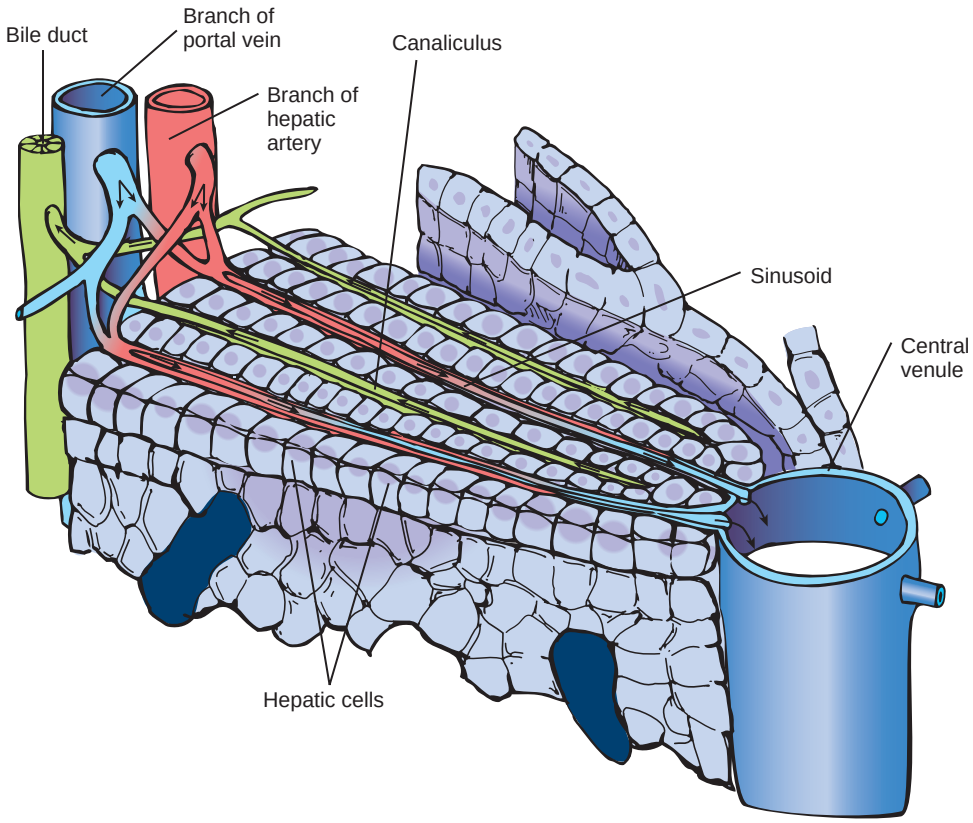


FIGURE 38-9 ▲ A section of the liver lobule showing the location of the hepatic veins, hepatic cells, liver sinusoids, and branches of the portal vein and hepatic artery.

other foreign matter in the sinus blood. The sinuses drain into the central venule, which in turn carries blood to the hepatic vein.

Blind-ended bile canaliculi arise between the other rows of hepatocytes. They carry newly secreted bile to larger ducts located at the periphery. These smaller ducts eventually drain into the common bile duct. Bile that is leaving the liver is concentrated and stored in the gallbladder. Fluid and electrolyte reabsorption in the gallbladder can increase the concentration of bile salts, cholesterol, and bilirubin 12-fold.

The gallbladder has a maximum capacity of 50 mL and can hold a 24-hour output of bile (600 mL) from the liver. The intestinal hormone CCK (secreted by the duodenal mucosa) and vagus nerve activity stimulate gallbladder contraction as a part of food digestion, particularly lipids. CCK and local reflexes initiated by duodenal peristalsis open the sphincter of Oddi. These events permit an outflow of bile down the common bile duct into the duodenum.

The common bile duct and the main duct from the pancreas usually unite just before the duct enters the lumen of the duodenum. There is often a dilation of the tube after this junction (the ampulla of Vater). The opening of the common bile duct in the duodenum is about 8 to 10 cm from the pylorus.

The liver cells perform many vital functions, as described in the sections that follow and summarized in Table 38-5.

Carbohydrate Metabolism

The liver participates in carbohydrate metabolism. The liver and skeletal muscle are the two primary sites of glycogen storage. Serum glucose levels are maintained by hepatic glycostatic function, involving two mechanisms. When plasma glucose levels are high, hepatocytes remove glucose from the plasma. Some of this glucose is then stored in the liver as glycogen. If plasma glucose levels decline, hepatocytes convert the glycogen back into glucose through a process called glycogenolysis, and the glucose is released into the bloodstream. Although many body tissues have the requisite cellular enzymes for glycogenolysis, hepatocytes are one of the few cell types that can release this intracellular glucose into the bloodstream. Hepatocytes do not simply respond directly to plasma glucose. These glycostatic functions are mediated by several hormones; some (eg, insulin) promote hepatic glucose uptake, and others (eg, glucagon, growth hormone, and epinephrine) stimulate glycogenolysis and the release of glucose from liver cells.

The liver does not contain enough glycogen reserves to be able to buffer plasma glucose during prolonged fasting or severe exercise. During these times, low plasma glucose levels stimulate the secretion of one or more hormones (glucagon, glucocorticoids, or thyroxine) that trigger the biochemical conversion of intracellular fatty and amino acids into glucose (gluconeogenesis), which the liver cell can then release into the bloodstream or store as glycogen. Only hepatocytes

Table 38-5 Hepatic Function

General Category	Specific Description
Carbohydrate metabolism	Glycogenesis (conversion of glucose to glycogen) Glycogenolysis (breakdown of glycogen to glucose) Gluconeogenesis (formation of glucose from amino acids or fatty acids)
Protein metabolism	Synthesis of nonessential amino acids Synthesis of plasma proteins (albumin, prealbumin, transferrin, clotting factors, complement factors; not γ-globulin or immunoglobulins) Urea formation from NH_3 (NH_3 formed by deamination of amino acids in liver and by action of colonic bacteria on proteins)
Lipid and lipoprotein metabolism	Synthesis of lipoproteins Breakdown of triglycerides into fatty acids and glycerol Formation of ketone bodies Synthesis of fatty acids from amino acids and glucose Synthesis and breakdown of cholesterol
Bile acid synthesis and excretion	Bile formation (containing bile salts, bile pigments [bilirubin, biliverdin]), cholesterol Bile excretion
Storage	Glucose (as glycogen) Vitamins (A, D, E, K, B_1, B_2, B_{12}, folic acid) Fatty acids Minerals (Fe, Cu) Amino acids (as albumin, β-globulins)
Biotransformation, detoxification, excretion of endogenous and exogenous compounds	Inactivation of drugs and excretion of the breakdown products Clearance of procoagulants, activated clotting factors, byproducts of coagulation
Removal of pathogens	Clearance of microorganisms by macrophages
Steroid catabolism	Conjugation and excretion of gonadal steroids Conjugation and excretion of adrenal steroids (cortisol, aldosterone)

possess the enzyme that is critical for gluconeogenesis. Glycogen storage is important for other functions of liver cells. A glycogen-rich hepatocyte conjugates bilirubin at a faster rate and is more resistant to toxins and infectious agents.

Protein Metabolism

The liver plays an essential role in the metabolism of proteins. The amino acids that result from the breakdown of proteins are deaminated to form ammonia by the liver and then converted to urea. The liver also synthesizes plasma proteins, including albumins, globulins, fibrinogens, plasma lipoproteins, and other proteins involved in clotting. The albumins maintain normal plasma oncotic pressure. A fall in this pressure leads to edema (both systemic and pulmonary) and contributes to ascites. The globulins bind thyroid and adrenal hormones. Bound, the hormones are inactive. Decreased hepatic protein levels can lead to a clinical excess of these hormones.

Lipid and Lipoprotein Metabolism

The liver contributes to adipose stores through the metabolism of triglycerides, fatty acids, and cholesterol. During fasting, triglycerides from adipose tissue are catabolized by the liver into fatty acids and glycerols. The free fatty acids in prolonged fasting are further catabolized into acetyl coenzyme A and then into ketone bodies. Ketone bodies provide an energy source for some (nonneuronal) tissues.

Bile Acid Synthesis and Excretion

Hepatocytes make bile, which contains water, bile salts, cholesterol, bilirubin, gluconate, and inorganic acids. Bile salts aid digestion by emulsifying dietary fats and fostering their absorption and the absorption of fat-soluble vitamins through the intestinal mucosa. They also prevent the cholesterol in the bile from precipitating out of solution and forming calculi. More than 90% of the daily output of bile is reabsorbed for recycling by an active transport process of the ileal mucosa.

Another hepatic function is elimination of bilirubin from the body. Old or defective erythrocytes are phagocytosed by large reticuloendothelial cells that line the large veins and the sinuses of the liver and spleen. These phagocytes degrade the hemoglobin of these cells into biliverdin, iron, and globulin molecules. The last two components are recycled by the body and used for future erythropoiesis. The biliverdin is almost immediately converted to free bilirubin. Because free bilirubin is an insoluble compound, it is transported bound to plasma albumin molecules. The hepatocytes convert this insoluble bilirubin into a soluble (and thus excretable) form by conjugating it with glucuronic acid to form bilirubin gluconate. This soluble form of bilirubin is then added to the bile and is eliminated from the body by the feces. Bilirubin gluconate gives the bile its normal golden yellow color. Organisms in the intestine convert most of the bilirubin gluconate into a darker brown compound, urobilinogen, which gives the feces its natural brown color. Because it is soluble in water, urobilinogen can also be absorbed from the colon back into the bloodstream and can be excreted by the kidneys. Excess plasma levels of either conjugated (direct) or unconjugated (indirect) bilirubin produce jaundice. Excess unconjugated bilirubin can cross the immature or damaged blood–brain barrier and bind with the basal ganglia, resulting in kernicterus.

Storage

Fat-soluble vitamins and many minerals are stored in the liver. These vitamins and minerals are released under the influence of hormones and serum concentrations of inorganic elements.

Biotransformation

Hepatocytes possess a mixed-function oxidase (MFO) system of enzymes that degrade certain drugs, including alcohol, benzodiazepines, tranquilizers, phenobarbital, phenytoin, and sodium warfarin, among others. This system operates in addition to other intracellular systems that also degrade some of these drugs. Its clinical significance lies in the nature of the drugs that this system catabolizes and in the fact that MFO system activity can be either inhibited or augmented (induced) by these same drugs, depending on when they are taken.

Administration of two MFO system–catabolized drugs within a few hours of one another or together causes each agent to act competitively, slowing down the degradation of the other. For example, simultaneous ingestion of diazepam (Valium) and alcohol can result in slower degradation of each drug. The outcome is higher blood levels of both chemicals for a longer time after administration.

The repeated administration of one MFO system–catabolized drug for several days causes the MFO system to enlarge physically and to possess more enzymes. This is called induction. Once induced, the MFO system degrades drugs more rapidly (including the drug that initiated the induction). If administration of a second MFO system–catabolized drug is begun after MFO system induction, a larger dose of this drug is required to produce a given effect. For example, induction of the MFO system by diazepam increases the dosage of warfarin needed to produce a given therapeutic effect. Other drugs are degraded by various hepatic systems.

Steroid Catabolism

The liver cells degrade steroid hormones, thereby preventing excess serum levels of estrogen, testosterone, progesterone, aldosterone, and glucocorticosteroids.

▲ Clinical Applicability Challenges

SHORT ANSWER QUESTIONS

1. Mr. K. is a 59-year-old male with a history of oropharyngeal cancer treated with radiation therapy which resulted in xerostomia. Discuss the role of saliva. Xerostomia may put Mr. K. at risk for what potential problems?

2. Mrs. C. complains of diarrhea after a cholecystectomy. Discuss how this surgery could contribute to her diarrhea.

3. Ms. Z. complains of an unintentional weight loss of 25 pounds over the past 2 months. She was subsequently diagnosed with a malabsorption deficiency. Which nutrient deficiencies would you expect and why?

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

39 Patient Assessment: Gastrointestinal System

JoAnn Coleman

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Explain the nursing role in assessing the critical care patient with gastrointestinal (GI) compromise.
2. Discuss important health history components that provide information about GI system status.
3. Describe a systematic approach for conducting a complete GI physical examination.
4. Discuss the importance of referred pain patterns in an abdominal assessment.
5. Differentiate between normal and abnormal findings detected on physical assessment of the GI system.
6. Identify the data used to make judgments about nutrition and metabolism in a critical care patient.
7. Discuss appropriate studies and procedures used to diagnose GI disorders.

The gastrointestinal (GI) system is a long tube with glands and accessory organs (salivary glands, liver, gallbladder, and pancreas). The GI tract begins at the mouth; extends through the pharynx, esophagus, stomach, small intestine, colon, and rectum; and ends at the anus. It is an unsterile system filled with bacteria and other flora. These organisms can cause superinfection from antibiotic therapy, and they can infect other systems when an organ of the GI tract ruptures. A malfunction along the GI tract can produce a variety of metabolic effects, which eventually may be life threatening.

Assessment of the GI system in a critically ill patient provides essential information. Early identification and treatment of GI disorders is necessary and serves as a foundation for developing a holistic plan of care for the patient. Ongoing assessment of the GI system in the critical care patient may help identify new complications. The ability to complete a comprehensive assessment of the GI system depends on the status of the patient. In an intensive care environment, the dynamic nature of the patient may dictate a more focused assessment of the patient. The nurse must be perceptive in obtaining information and timely in soliciting critical information. For example, a ventilated patient cannot converse in an intensive care area, and neither can a comprehensive assessment of the GI system be obtained nor would it be necessary at that time.

When a patient is critically ill, assessment of the GI system helps determine whether assessment findings relate to the current clinical problem or herald a new complication. The nurse correlates and integrates presenting GI signs and symptoms, whether they are isolated entities or related to another underlying problem: Is the bright red blood in the stool a result of GI bleeding or from external bleeding hemorrhoids? Is the abdominal pain due to recent bowel surgery or to a distended stomach? The nurse must be aware of the patient's changing metabolic state and nutritional status because this information may directly affect other health outcomes, such as length of stay, morbidity, and even mortality.[1]

▲ History

Unless emergency conditions require immediate action to preserve life, an assessment of the GI system begins with the history. A thorough and accurate history greatly enhances the assessment process. The patient's history provides information that can lay the foundation and set the direction for the rest of the assessment.

The history is the major subjective data source about a patient's health status and provides insight into actual or potential health problems. It guides the physical assessment. The history organizes pertinent physiological, psychological, cultural, and psychosocial information as it relates to the patient's current health status and accounts for factors such as lifestyle, family relationships, and cultural influences.[2] Box 39-1 lists elements for a comprehensive GI health history.

The initial presentation of the patient in the intensive care area determines how quickly the history will be obtained

BOX 39-1 HEALTH HISTORY for Gastrointestinal Assessment

Chief Complaint
- Patient's description of the problem

History of the Present Illness
- Complete analysis of the following signs and symptoms (using the NOPQRST format; see Box 17-1, p. 207):
- Abdominal pain
- Anorexia
- Indigestion (heartburn)
- Dysphagia
- Eructation
- Nausea
- Vomiting
- Hematemesis
- Fever and chills
- Jaundice
- Pruritus
- Diarrhea
- Constipation
- Flatulence
- Bleeding
- Hemorrhoids
- Melena
- Change in appetite
- Recent weight gain or weight loss
- Mouth lesions
- Anal discomfort
- Fecal incontinence
- Change in abdominal girth

Past Health History
- Relevant childhood illnesses and immunizations: hepatitis, influenza, pneumococcal, meningococcal
- Past acute and chronic medical problems: treatments and hospitalizations—diabetes, cancer, inflammatory bowel disease (Crohn's disease, ulcerative colitis, irritable bowel syndrome, diverticulitis), peptic ulcer, gallstones, polyps, pancreatitis, hepatitis or cirrhosis of the liver, previous GI bleeding, cancers or tumors of the GI tract, spinal cord injury; for women, episiotomy or fourth-degree laceration during delivery
- Risk factors: age, heredity, gender, race, tobacco use, physical inactivity, obesity, diabetes mellitus, tattoos, exposure to infectious diseases—hepatitis and influenza
- Past surgeries: previous GI surgeries (mouth, pharyngeal, esophageal, stomach, small intestine, colon, gallbladder, liver, gallbladder, pancreas), abdominal surgeries or trauma
- Past diagnostic tests and interventions: upper endoscopy, colonoscopy, upper GI, barium enema, rectal manometry
- Medications (prescription drugs, over-the-counter drugs, vitamins, herbs, and supplements): aspirin, steroids, anticoagulants, nonsteroidal anti-inflammatory drugs, laxatives, stool softeners
- Allergies and reactions to medications, foods, contrast dye, latex or other materials
- Transfusions, including type and date

Family History
- Health status or cause of death of parents and siblings: inflammatory bowel disease, Hirschsprung's disease, aganglionic megacolon, malabsorption syndrome, cystic fibrosis, celiac disease, gallbladder disease, Peutz-Jeghers syndrome, familial adenomatous polyposis, familial Mediterranean fever, any cancers of the GI tract

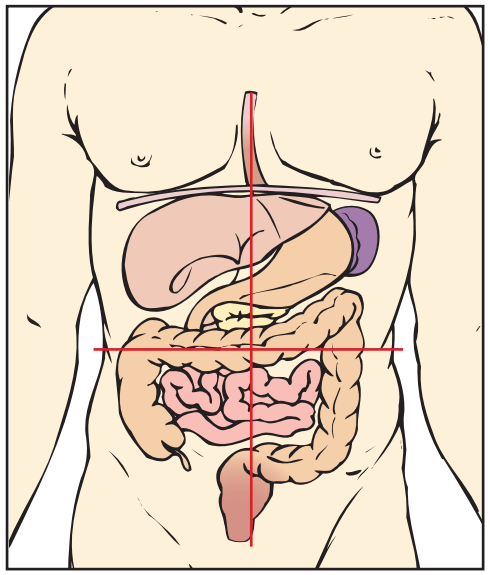
Personal and Social History
- Tobacco, alcohol, and substance use
- Family composition
- Occupation and work environment
- Living environment, water source
- Diet: food intolerances, taste sensations, coffee intake, special diet
- Oral hygiene: dental status, pattern of dental care, dentures, braces, bridges, crowns, caries
- Bowel habits
- Sleep patterns
- Exercise
- Cultural beliefs
- Spiritual/religious beliefs
- Coping patterns and social support systems
- Leisure activities
- Sexual activity
- Recent stressful events: physical or psychological
- Recent travel, overseas military duty

Review of Other Systems
- HEENT: visual changes, headaches, tinnitus, vertigo, epistaxis, sore throat, mouth lesions, swollen glands, lymphadenopathy
- Respiratory: shortness of breath, dyspnea, cough, sputum, lung disease, recurrent infections
- Cardiovascular: chest pain, palpitations, orthopnea, edema, hypertension, congestive heart failure, dysrhythmia, angina, valvular disease
- Genitourinary: incontinence, erectile dysfunction, dysuria, frequency, nocturia
- Musculoskeletal: pain, weakness, varicose veins, change in sensation
- Neurological: transient ischemic attacks, stroke, change in level of consciousness, syncope, seizures, cerebrovascular disease

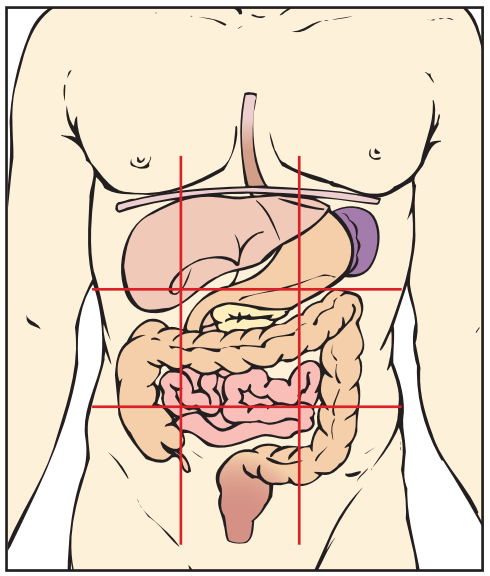
as well as the focus of the interview. If the patient is in acute distress, the history must focus on obtaining answers to pertinent questions about the patient's chief complaint and precipitating events. Information may be more readily obtained from family members or friends. The nurse can obtain a more thorough history from a patient in no obvious distress by focusing on the patient's current symptoms, medical history, and family history. Information that is fixed and needs to be obtained only once includes data about personal health, preexisting GI conditions, previous GI or abdominal surgeries or injuries, and hospitalizations. The critical care nurse must also consider the present nutritional status of the patient, the projected length of illness, and its impact on future nutritional needs or adjustments.[3]

The GI assessment may change over the course of the illness. The data gathered during the initial history may have focused on the pressing issues facing the patient at that time, but these issues can change in a short or protracted amount of time. The critical care nurse must be vigilant, and he or she must maintain data and incorporate additional information to provide individualized and holistic nursing care as the status of the patient continually evolves.

Because pain is often the chief complaint of patients presenting with abdominal disorders, it must be dealt with in detail.[4] A thorough assessment of pain must include details about the onset, progression, migration, character, localization, radiation, and duration of the pain, as well as information about factors that exacerbate or alleviate the pain.



Right upper quadrant (RUQ)	Left upper quadrant (LUQ)
Liver and gallbladder Pylorus Duodenum Head of pancreas Hepatic flexure of colon Portions of ascending and transverse colon	Left liver lobe Stomach Body and tail of pancreas Splenic flexure of colon Portions of transverse and descending colon
Right lower quadrant (RLQ)	**Left lower quadrant (LLQ)**
Cecum and appendix Portion of ascending colon	Sigmoid colon Portion of descending colon



Right hypochondriac	Epigastric	Left hypochondriac
Right liver lobe Gallbladder	Pyloric end of stomach Duodenum Pancreas Portion of liver	Stomach Tail of pancreas Splenic flexure of colon
Right lumbar	**Umbilical**	**Left lumbar**
Ascending colon Portions of duodenum and jejunum	Omentum Mesentery Lower part of duodenum Jejunum and ileum	Descending colon Portions of jejunum and ileum
Right inguinal	**Suprapubic or hypogastric**	**Left inguinal**
Cecum Appendix Lower end of ileum	Ileum	Sigmoid colon

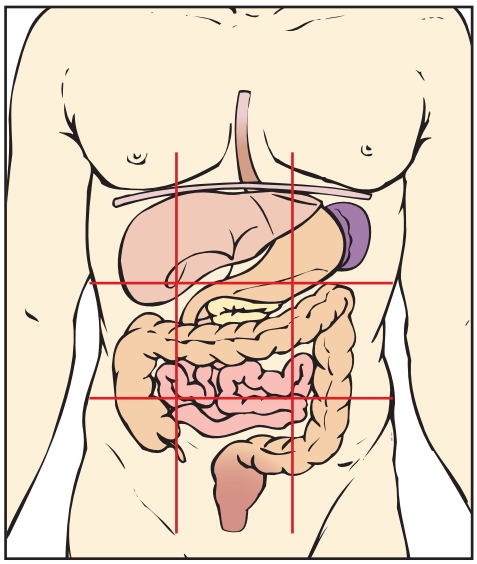
FIGURE 39-1 ▲ To aid accurate abdominal assessment and documentation of findings, the nurse can mentally divide the patient's abdomen into regions. **A:** The quadrant method. **B:** The nine regions method.

To help understand the potential origin, location, and radiation of the pain, the nurse mentally divides the abdomen into regions using either the quadrant method or the nine regions method (Fig. 39-1). Figure 39-2 summarizes common causes of pain by location.

With many GI problems, the pain is referred, which makes diagnosis especially difficult. Referred pain is pain felt at a site different from that of the involved organ. Pain results because nerves that supply an organ also supply the body surface.[4] Figure 39-3 identifies common sites of referred abdominal pain.

▲ Physical Examination

The physical examination helps establish baseline information about the physical dimensions of the patient's present situation. The physical assessment begins by observing the



Right hypochondriac	Epigastric	Left hypochondriac
Cholecystitis/ cholangitis Hepatitis Metastatic disease to the liver Pleurisy, lower lobe pneumonia, or pneumothorax Congestive hepatomegaly Pyelonephritis Renal colic Duodenal ulcer	Duodenal or gastric ulcer Duodenitis or gastritis Pancreatitis Myocardial infarction or angina Pericarditis Gastroenteritis Mesenteric embolus or thrombus Small bowel obstruction	Pleurisy, lower lobe pneumonia, or pneumothorax Myocardial infarction or angina Pericarditis Pyelonephritis Renal colic Splenic injury
Right lumbar	**Umbilical**	**Left lumbar**
Pancreatitis Pyelonephritis Renal colic Colon obstruction/ gangrene	Appendicitis Small bowel obstruction Rectus sheath hematoma Gastroenteritis Umbilical hernia Abdominal aortic aneurysm Aortic dissection Mesenteric embolus or thrombus	Pancreatitis Pyelonephritis Renal colic Sigmoid diverticulitis Colon obstruction/ gangrene
Right inguinal	**Suprapubic or hypogastric**	**Left inguinal**
Meckel's diverticulum Appendicitis Cecal perforation Groin hernia Colon obstruction/ gangrene Ectopic pregnancy Spigelian hernia Regional enteritis	Rectus sheath hematoma Salpingitis Ectopic pregnancy Tubo-ovarian torsion Mittelschmerz Regional enteritis Endometriosis Abdominal aortic aneurysm	Sigmoid diverticulitis Groin hernia Colon obstruction/ gangrene Ectopic pregnancy Spigelian hernia Regional enteritis

FIGURE 39-2 ▲ Common causes of pain by location.

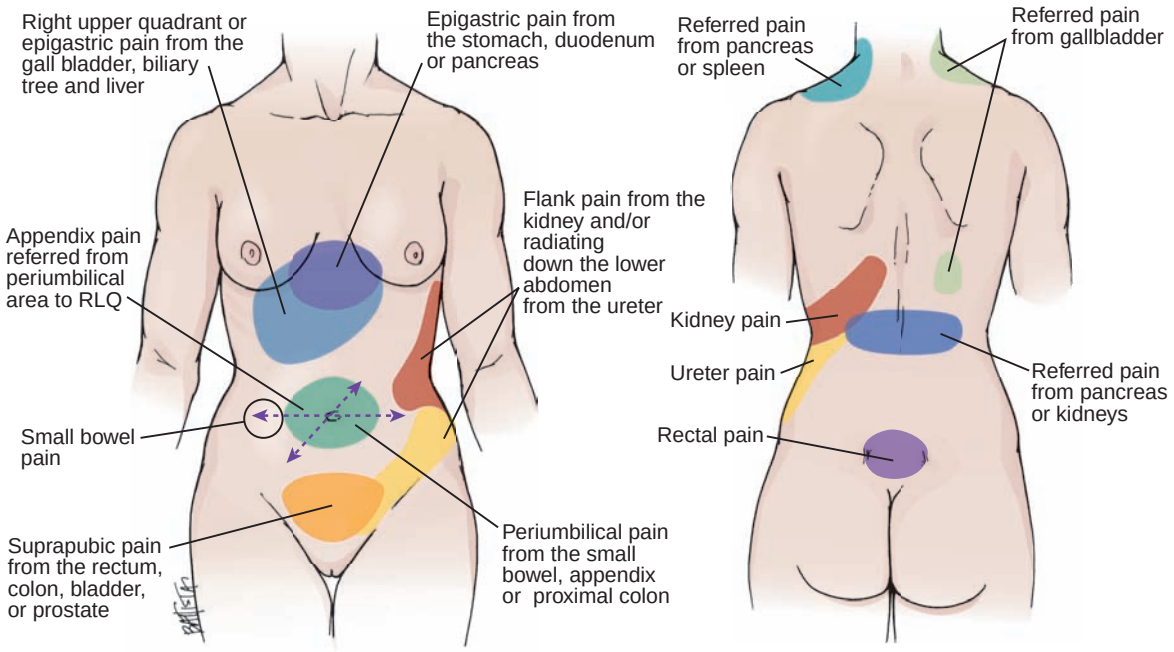


FIGURE 39-3 ▲ Mechanisms and sources of abdominal pain. Abdominal pain may be described as visceral, parietal, or referred. (From Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Wolters Kluwer Health, 2010, p 418.)

patient's overall appearance. Motor activity, body position, gait, hair (pattern, loss), skin color (jaundice, cyanosis, pallor) and quality (edema), facial expression, level of consciousness, and signs of depression, anxiety, confusion, or irritability are noted. It is important to note that changes in fluid and electrolyte balance, severe infection, drug toxicity, and hepatic disease may cause a change in behavior, such as confusion, lethargy, or agitation. Next, a focused examination of the GI system is necessary. A focused examination of the GI system includes evaluation of the oral cavity and throat, abdomen, and rectum. Assessment of the abdomen includes assessment of the liver, gallbladder, and pancreas.[3]

Oral Cavity and Throat

Adequate nutrition is related to good dental health and the general condition of the mouth. Any disorders of the upper GI tract (lips, mouth, teeth, pharynx, and esophagus) can prevent adequate nutritional intake. Changes in the oral cavity may influence the type and amount of food ingested and the extent to which food is properly mixed with salivary enzymes. Esophageal problems can also adversely affect food and fluid intake, jeopardizing a patient's health.

Examination of the oral cavity includes inspection and palpation using a good light source, a tongue depressor, an examining glove, and a mask (Fig. 39-4). The nurse should explain the procedure to the patient. The patient assumes a comfortable position that facilitates examination. Sitting upright is the best position for this part of the examination.

Inspection of the lips and jaws for abnormal color, texture, lesions, symmetry, and swellings is essential. Palpation of the temporomandibular joints for mobility, tenderness, and crepitus is also warranted. The nurse retracts the lips to allow adequate visualization. It is necessary to inspect dentures for fit and to remove them for the oral examination. The nurse should use a good light source to inspect all structures inside the mouth and the buccal mucosa (see Fig. 39-4). The nurse identifies missing, broken, loose, and decayed teeth while noting redness, pallor, white patches, plaques, ulcers, petechiae, bleeding, and masses. A pool of saliva under the tongue helps assess hydration. Palpation of the parotid and submaxillary ducts is necessary. The nurse palpates suspect areas with a gloved finger to determine tenderness or induration. The patient sticks out his or her tongue while the nurse checks for symmetry of movement, swelling, lesions, and any abnormal coating. While depressing the tongue with a tongue blade, the nurse observes the movement of the soft palate and uvula as the patient says "ah." These structures should rise symmetrically. This is a good time to inspect the hard and soft palates, uvula, tonsils, pillars, and posterior pharynx (Fig. 39-5). Note decreased or absent gag reflex, which suggests possible neurological dysfunction and increases the patient's risk for aspiration. Deviation of the uvula to one side when the patient says "ah" may indicate pathology of cranial nerve IX (the glossopharyngeal nerve) or cranial nerve X (the vagus nerve).[4] Unusual breath odors may indicate serious GI disease, such as esophageal cancer. Fecal breath odor may be caused by a bowel obstruction or hepatic failure; a fruity breath odor may be the result of diabetic or starvation ketoacidosis. Table 39-1 reviews oral assessment, normal and abnormal findings, and possible causes of abnormal findings.

Examination of the oral cavity in an intubated patient is very important, even though the tube may hinder vision during the assessment. The condition of the mouth of a critically ill patient can change rapidly, and the nurse must conduct a periodic assessment to initiate treatment and intervene to prevent complications. It is necessary to assess the presence of any secretions, oral odor, or changes in odors coming from the oral cavity promptly. Studies have indicated that microbial colonization of the oropharynx and dental plaque are associated with pneumonia in patients who are receiving mechanical ventilation.[5]

Evidence now shows that implementing the use of appropriate tools at the bedside, a comprehensive oral care protocol, and staff compliance with the protocol can reduce the rate of ventilator-associated pneumonia (VAP) and associated costs.[6–8] The economic burden from VAP is associated with increased hospital costs, longer duration of hospital stay, and a higher number of services affected.[9]

The patient with a nasogastric, orogastric, or long tube for intestinal decompression warrants close observation because these tubes prevent the lower esophageal sphincter

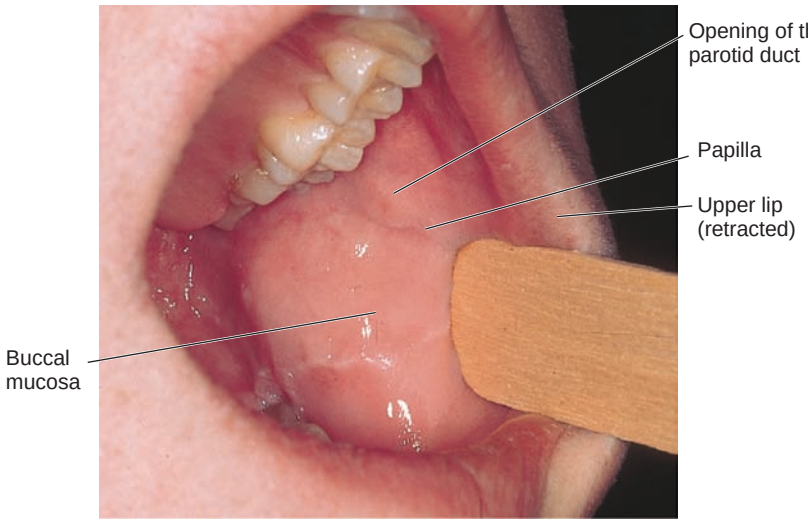


FIGURE 39-4 ▲ Examination of the oral cavity. (From Bickley L: Bates' Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 234.)

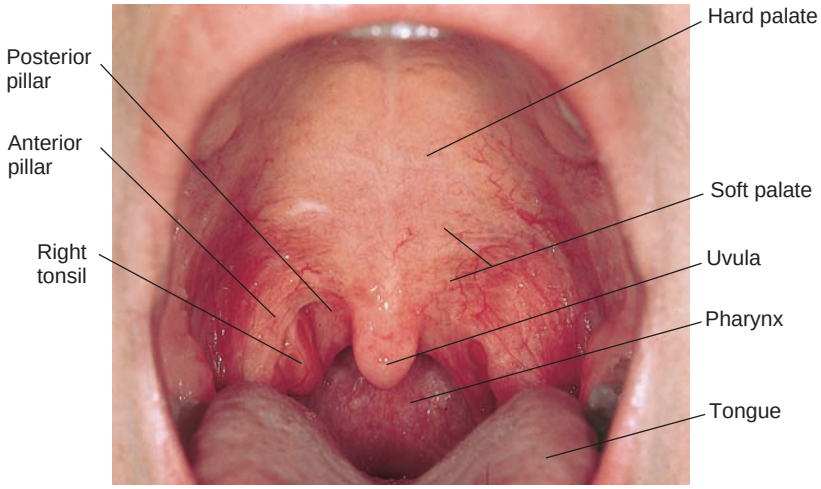


FIGURE 39-5 ▲ Structures of the mouth. (From Bickley L: Bates' Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 233.)

Table 39-1 Oral Assessment

Structure	Normal	Abnormal Findings	Possible Cause
Lips	Smooth, pink, and moist	Dry or cracked	Febrile illness
		Asymmetrical, cracked, fissured, or bleeding	Cheilitis
		Cyanotic	Cold or hypoxia
		Cracks at corner of lips	Possible vitamin B deficiency or poor hygiene
Tongue	Pink, moist with papillae present	Coated or loss of papilla and a shiny appearance (with or without redness); blistered or cracked; altered taste	Infection
		Deviation to one side	Cranial nerve XII (hypoglossal nerve) problem
		Nodules or ulcers on base of tongue	Cancerous lesion
Saliva	Watery	Thick, ropy, or absent	
Mucous membranes	Pink and moist	Reddened without ulcerations	Infection
		Ulcerations with or without bleeding	Poor nutrition
		Inflammation	Ill-fitting dentures
		Leukoplakia on buccal membrane	Precancerous lesion
		Cyanosis	Hypoxia
		Pale mucosa	Anemia
		Small areas of white scar tissue	Chronic irritation from friction of irregular tooth surfaces or biting when chewing
		Inflamed or painful Stensen's duct opening	Parotid gland infection
Gingiva	Pink, stippled, and firm	Edematous with or without redness; spontaneous bleeding or bleeding with pressure; soreness	Gingivitis
Teeth or dentures	Clean without debris	Plaque or debris in between teeth; plaque or debris along gum line or denture-bearing area	
		Toothache, tooth abscess	
		Misfit of dental appliances	
		Absent or broken teeth, cavities	
		Malocclusion, worn or flattened tooth edges	Bruxism
Voice	Normal	Deeper or raspy; difficulty talking or painful to talk	Vocal cord paralysis
Throat/swallowing	Normal	Some pain on swallowing or unable to swallow; sore throat	Cancerous lesion
Glands	Nonpalpable	Inflammation and lumps	Stones or cysts

from closing completely. Gastric reflux or even reflux into the oropharynx may occur, which can cause erosive damage to the esophagus as well as a noxious odor in the mouth. Delayed gastric emptying may also exacerbate the reflux.

Abdomen

The patient's comfort should be preserved as the nurse performs the abdominal examination (Table 39-2). The patient should empty his or her bladder before the examination, or this may be facilitated by an indwelling urinary catheter. A supine position with arms down and knees slightly bent is preferred because this position relieves tension on the abdominal wall. Draping exposes the abdomen and protects the patient's modesty.[3] This is the ideal situation for performing an abdominal examination, but it may not always be possible in a critically ill patient. The nurse must assess the circumstances and prioritize for the individual patient at that time. If the patient is experiencing any or severe pain, reevaluation of the need for an examination may be warranted. Likewise, if the procedure increases discomfort or intensity of pain, the examiner stops.

The order of the abdominal examination is inspection, auscultation, percussion, and palpation. Auscultation precedes percussion and palpation because the latter can alter the frequency and quality of bowel sounds. Likewise, if the painful area is palpated first, the patient may tense the abdominal muscles, making assessment difficult or impossible.

The abdomen is usually divided into four quadrants by imaginary lines crossing at the umbilicus: right upper, right lower, left upper, and left lower quadrants. Another way to view the abdomen is to divide it into nine sections. Figure 39-1 on page 870 shows the abdominal organs and their relationship to these two methods of identifying abdominal landmarks. Figure 39-3 on page 871 shows common causes of pain by location.

Inspection

The examination begins with the nurse standing at the foot of the patient's bed and inspecting for symmetry of the abdomen, visible masses, and pulsations. The nurse inspects the skin of the abdomen for tense, shiny skin, any areas of discoloration, rashes, striae (lines resulting from rapid or prolonged skin stretching), ecchymoses, petechiae (small red or purple spots caused by hemorrhage), lesions, scars, and prominent or dilated veins. Then the nurse moves to the patient's side and assumes a position to obtain an eye-level view across the abdomen. Size; shape; asymmetry; movements from respirations, peristalsis, and vascular pulsations; and exaggerated movement are noteworthy. It is necessary to inspect the umbilicus for position, contour, and color (Fig. 39-6). Pulsation of the aorta is normally apparent in the epigastric area. In a thin person, the femoral pulses may be visible. When ascites or abdominal bleeding is suspected, the nurse should measure the abdominal girth.[4] Measure abdominal girth at the same time of day, ideally in the morning just after voiding,

Table 39-2 Abnormal Abdominal Findings

Finding	Characteristic	Possible Cause
Abdominal contour	Concave (scaphoid)	Malnutrition
	Distention	Tumor; excessive fluid (ascites, perforation); gas accumulation; severe malnutrition
Skin abnormalities	Bulging around old scar	Incisional hernia
	Striae	Obesity; pregnancy; abdominal tumor; Cushing's syndrome (purple striae)
	Pink or blue	Recently developed striae
	White or silver	Older striae
	Tense, glistening	Ascites
	Dilated, tortuous veins	Inferior vena cava obstruction
Umbilicus	Everted	Increased intra-abdominal pressure
	Bluish ecchymosis surrounding umbilicus (Cullen's sign)	Intra-abdominal bleeding; pancreatitis; ectopic pregnancy
	Palpable nodule bulging (Sister Mary Joseph nodule)	May indicate metastasis from pelvic or gastrointestinal (GI) cancer
Peristaltic wave	Strong	Intestinal obstruction
Abdominal aortic pulsations	Obvious and pronounced	Increased intra-abdominal pressure (from tumor or ascites)
Murphy's sign	Sharp pain that stops respiration when palpating under liver border	Cholecystitis
Grey Turner's sign	Flank ecchymosis	Intra-abdominal bleeding; hemorrhagic pancreatitis
Blumberg's sign	Rebound tenderness	Peritoneal irritation; inflamed or perforated appendix
Iliopsoas muscle	Right lower quadrant pain when right leg elevated against tension	Inflamed or perforated appendix from inflamed psoas muscle
Obturator muscle	Abdominal pain when right leg rotated at hip (internal or external rotation)	Inflamed or perforated appendix

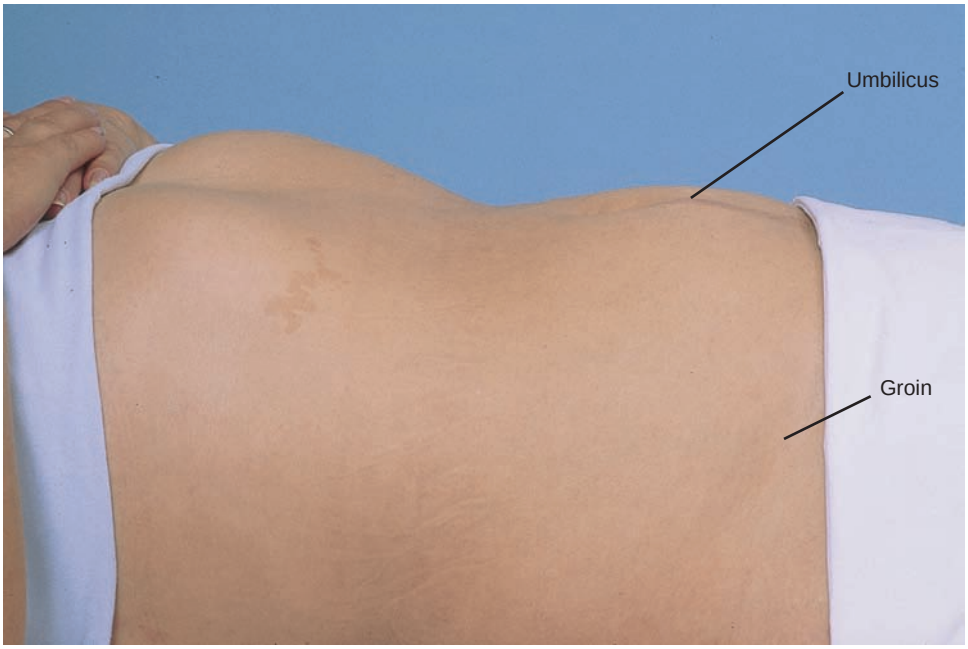


FIGURE 39-6 ▲ Inspection of the abdomen. (From Bickley L: Bates' Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 435.)

or at a designated time for bedridden patients and those with indwelling catheters. The patient will normally be supine in the intensive care unit (ICU). Place the tape measure behind the patient and measure at the umbilicus. Record the distance in designated units (inches or centimeters). Take all future measurements from the same location. The abdomen may be marked with a pen to help identify the correct site.

Auscultation

Auscultation provides information on bowel motility and the vessels and organs that lie beneath the abdominal wall. The nurse applies light pressure on the diaphragm of the stethoscope when auscultating the four quadrants of the abdomen. The nurse starts below and to the right of the umbilicus and proceeds in a methodical direction through all four quadrants. To prevent contraction of the abdominal muscles that can obscure sounds, the nurse lifts the stethoscope completely off the abdominal wall when changing its location. Normally, air and fluid moving through the bowel by peristalsis create a soft, bubbling sound with no regular pattern, often with soft clicks and gurgles interspersed, approximately every 5 to 15 seconds. Colonic sounds are low pitched with a rumbling feature. A hungry patient may exhibit a "growling stomach" resulting from hyperperistalsis, called borborygmi. High-pitched, rapid, loud, and gurgling bowel sounds are hyperactive and may occur in a hungry patient. High-pitched tinkling and rushes of high-pitched sounds with abdominal cramping usually indicate obstruction. Bowel sounds that occur once every minute or less frequently are hypoactive and usually occur after bowel surgery or when feces fill the colon.[10] Absent bowel sounds may be associated with peritonitis or paralytic ileus.

Edema of the abdominal wall can be detected when an imprint of the diaphragm remains after light auscultation. The nurse uses the bell of the stethoscope to listen for vascular sounds over the abdominal aorta and the renal and femoral arteries. Figure 39-7 illustrates auscultation sites of the abdomen for vascular sounds. If the nurse hears a bruit (a continuous purring, blowing, or humming sound), percussion and palpation do not follow. If a bruit is a new finding, it is necessary to notify the physician. Table 39-3 describes abnormal abdominal sounds.

Percussion

The nurse percusses the abdomen lightly in all four quadrants of the abdomen, listening for the location and distribution of tympany and dullness (Fig. 39-8A). The percussion may proceed clockwise or up and down over the abdomen (see Fig. 39-8B,C). Abdominal percussion helps identify air, gas, and fluid in the abdomen and helps determine the size and location of abdominal organs. The percussion sound depends on the density of the underlying structure. The sound is dull over solid organs (eg, liver), a stool-filled colon, abdominal masses, or pleural effusions. The sound is tympanic over air, such as in the gastric bubble or air-filled intestine. To determine the size of the liver, the nurse percusses along the right midclavicular line (Fig. 39-9A). One method is to begin at the iliac crest and work upward. The point at which the sound becomes dull is marked. It is necessary to perform percussion down from the clavicle. The dull sound of the rib should not be mistaken for the superior edge of the liver. After marking the superior edge, the nurse measures the distance between the marks in centimeters. The normal liver measures 6 to 12 cm in height at the midclavicular line (see Fig. 39-9B).[4]

It may be necessary to postpone performing abdominal percussion on a critically ill patient, especially if there is abdominal guarding. The nurse percusses areas where the patient is not experiencing pain before examining painful areas. Abdominal percussion or palpation is contraindicated in a patient with suspected appendicitis, abdominal aortic aneurysm, or polycystic kidneys or in a patient who has received an abdominal organ transplantation to prevent rupture of the organs or aorta.[4]

Palpation

Abdominal palpation is necessary to establish the character of the abdominal wall, including the size, condition, and

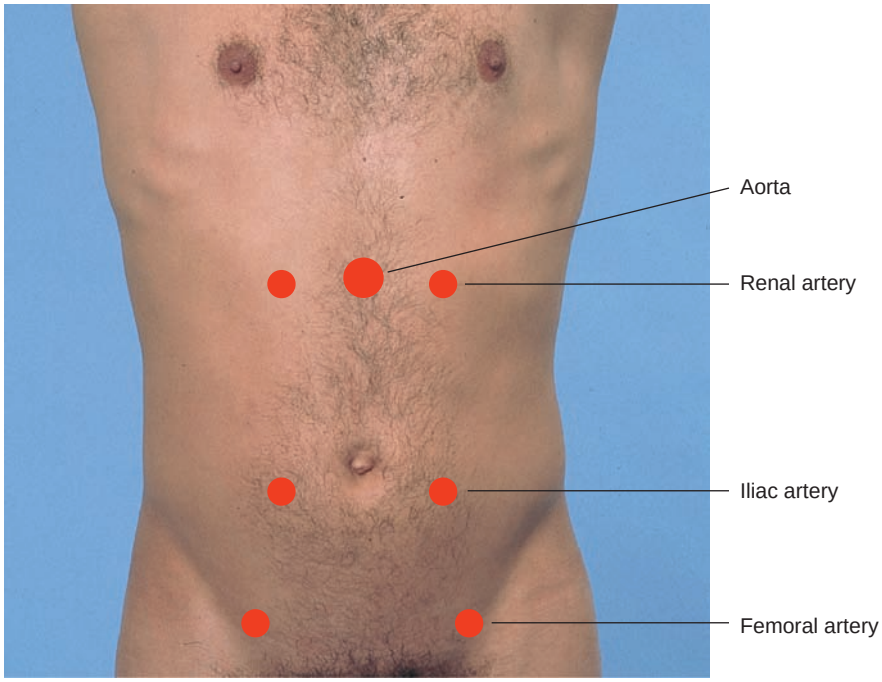


FIGURE 39-7 ▲ Auscultation sites for vascular sounds. (From Bickley L: Bates' Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 436.)

consistency of abdominal organs; the presence of abdominal masses; and the presence, location, and degree of abdominal pain. Abdominal palpation includes light palpation, deep palpation, and ballottement.

Light palpation, which is performed first, identifies muscular resistance and areas of tenderness (Fig. 39-10). Fingertips are used to depress the abdominal wall 1 cm (0.5 inch). The nurse notes skin temperature, muscle resistance, tender areas, and masses. The femoral artery is subject to bilateral palpation. To ensure patient cooperation and relaxed muscles, the nurse always palpates a symptomatic area last.

When disease is present, palpation may result in somatic or organ pain. Somatic pain is localized and reflects inflammation of the skin, fascia, or abdominal surfaces. Guarding of the abdominal muscles accompanies somatic pain. Organ pain is visceral in nature and is usually dull, diffuse, and generalized.

Deep palpation is used to locate abdominal organs (enlarged spleen, edge of the liver, pole of the right kidney; the left

Table 39-3 Abnormal Abdominal Sounds

Sound and Description	Location	Sound	Possible Cause
Bowel sounds	All four quadrants	Hypoactive sounds unrelated to hunger	Diarrhea or early intestinal obstruction
		Hypoactive, then absent, sounds	Paralytic ileus or peritonitis
		High-pitched "tinkling" sounds	Intestinal air and fluid under tension in a dilated bowel; early intestinal obstruction
		High-pitched "rushing" sounds coinciding with an abdominal cramp	Intestinal obstruction
		Hyperactive sounds, long and prolonged (borborygmi)	Hunger, gastroenteritis
		Absence of sounds more than 5 min in all four quadrants	Temporary loss of intestinal motility; occurs with ileus
Systolic bruits (vascular "blowing" sounds resembling cardiac murmurs)	Abdominal aorta	Partial arterial obstruction or turbulent blood flow	Dissecting abdominal aneurysm
	Renal artery		Renal artery stenosis
	Iliac artery		Hepatomegaly
Venous hum (continuous, medium-pitched tone created by blood flow in a large, engorged vascular organ such as the liver)	Epigastric area and umbilicus	Increased collateral circulation between portal and systemic venous systems	Hepatic cirrhosis
Friction rub (harsh, grating sound resembling two pieces of sandpaper rubbing together)	Hepatic	Inflammation of the peritoneal surface of an organ	Liver mass

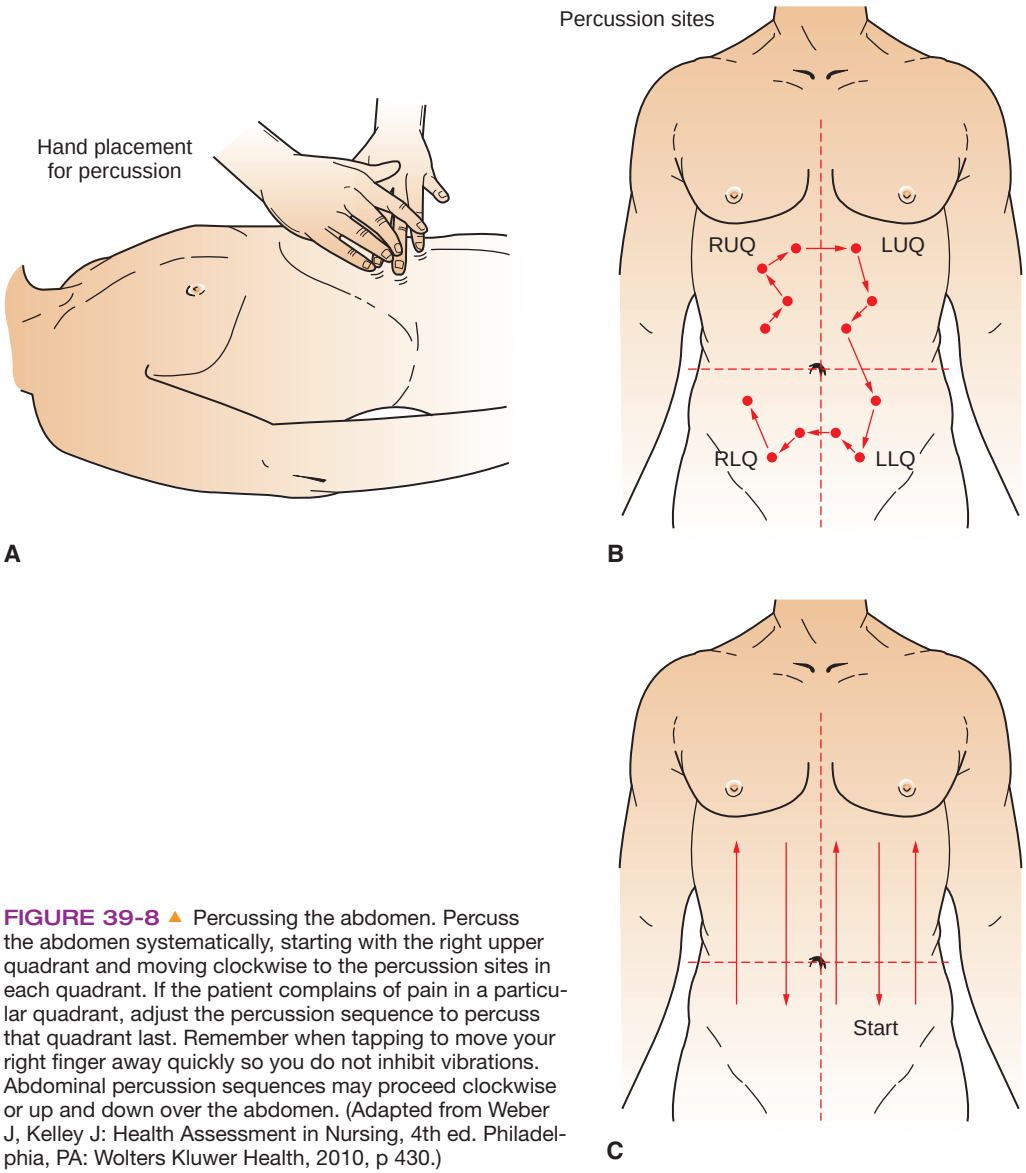


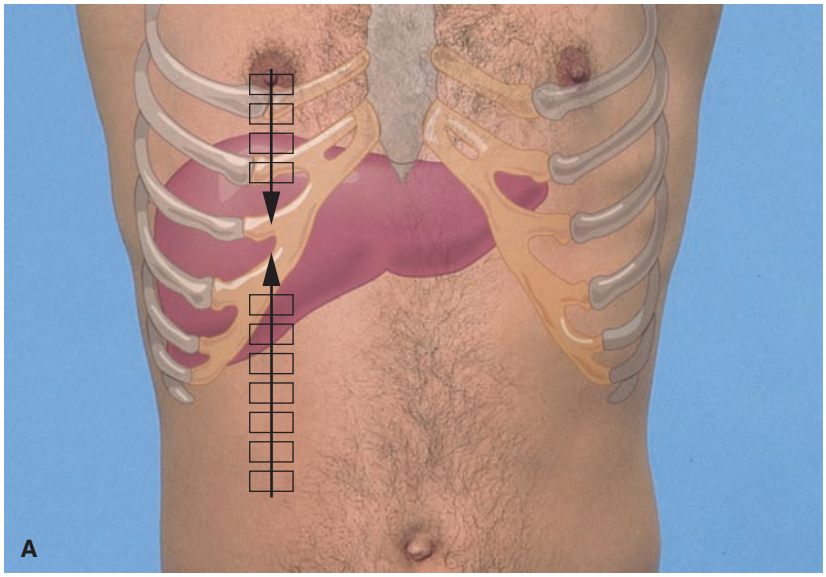
FIGURE 39-8 ▲ Percussing the abdomen. Percuss the abdomen systematically, starting with the right upper quadrant and moving clockwise to the percussion sites in each quadrant. If the patient complains of pain in a particular quadrant, adjust the percussion sequence to percuss that quadrant last. Remember when tapping to move your right finger away quickly so you do not inhibit vibrations. Abdominal percussion sequences may proceed clockwise or up and down over the abdomen. (Adapted from Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Wolters Kluwer Health, 2010, p 430.)

kidney is usually not palpable) and large masses (Fig. 39-11). The fingertips are used to depress the abdominal wall firmly to a depth of 7.5 cm (3 inch). Palpation in the epigastric area for the pulse of the aorta is warranted (Fig. 39-12). If the nurse finds an area of tenderness with light palpation, it is necessary to check rebound tenderness by quickly withdrawing the fingertips after depression. Rebound tenderness usually indicates inflammation of the peritoneum from an abdominal process, such as organ inflammation, infection, abscess formation, or perforated bowel (release of bowel contents into the abdomen). If the nurse palpates a mass, he or she notes its location, size, shape, consistency, type of border, degree of tenderness, presence of pulsations, and degree of mobility (fixed or mobile).[11]

The nurse palpates the spleen by standing at the patient's right side, reaching over the patient's abdomen. The left hand should be under the posterior ribs and pulling up gently on the patient, and the right hand should be below the left costal margin with the fingers pointing toward the patient's head. The nurse then asks the patient to inhale as the nurse presses inward and upward with the right hand while he or she provides support with the left hand (Fig. 39-13).

Palpation of the liver is easiest when one hand is under the patient at the level of the 11th rib while the other hand is on the abdomen at the level of the percussed liver edge dullness (Fig. 39-14). With the abdominal fingers pointing upward to lift the organ, the upper hand pushes down and upward to palpate the lower border of the liver. The liver edge should be firm and smooth. Finding an enlarged, nodular, or irregularly shaped liver warrants reporting.

Ballottement is the light, rapid tapping of the fingertips against the abdominal wall. This is used to elicit abdominal muscle resistance or guarding that may be missed with deep palpation or to detect the movement or bounce of a movable mass. In a patient with ascites, deep ballottement may be warranted. It is necessary to push the fingertips deeply inward in a rapid movement and then quickly release them, maintaining fingertip contact with the abdominal wall. Any movement of an organ lying beneath is felt, or a movable mass moves toward the fingertips.[3]



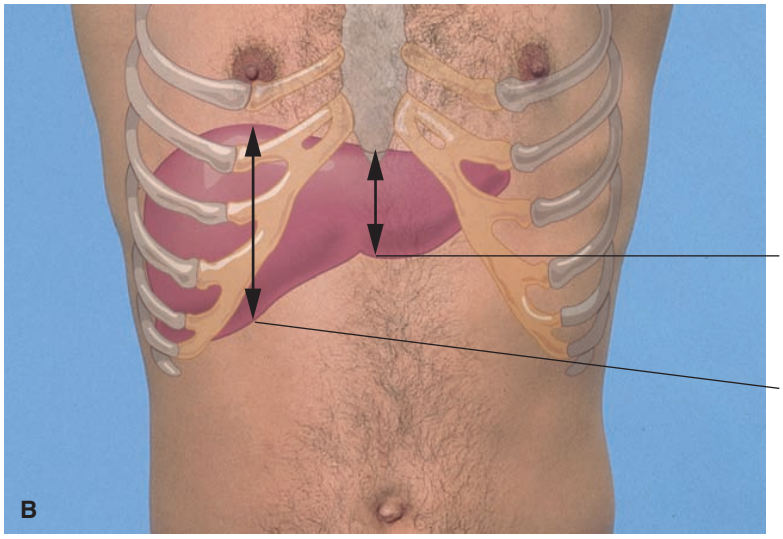


FIGURE 39-9 ▲ Percussing the liver. **A:** Determine the lower border of liver dullness in the midclavicular line. Next, identify the upper border of liver dullness in the midclavicular line. **B:** Measure in centimeters the distance between the lower border of liver dullness and the upper border of liver dullness. The distance is known as the vertical span of liver dullness. (From Bickley L: Bates' Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 440.)

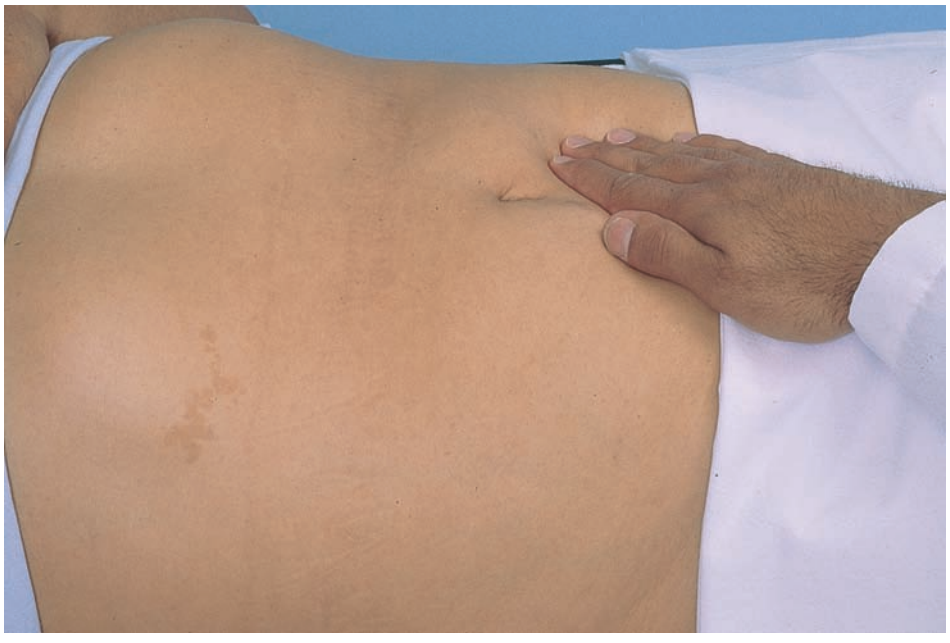


FIGURE 39-10 ▲ Performing light palpation. (From Bickley L: Bates' Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 438.)

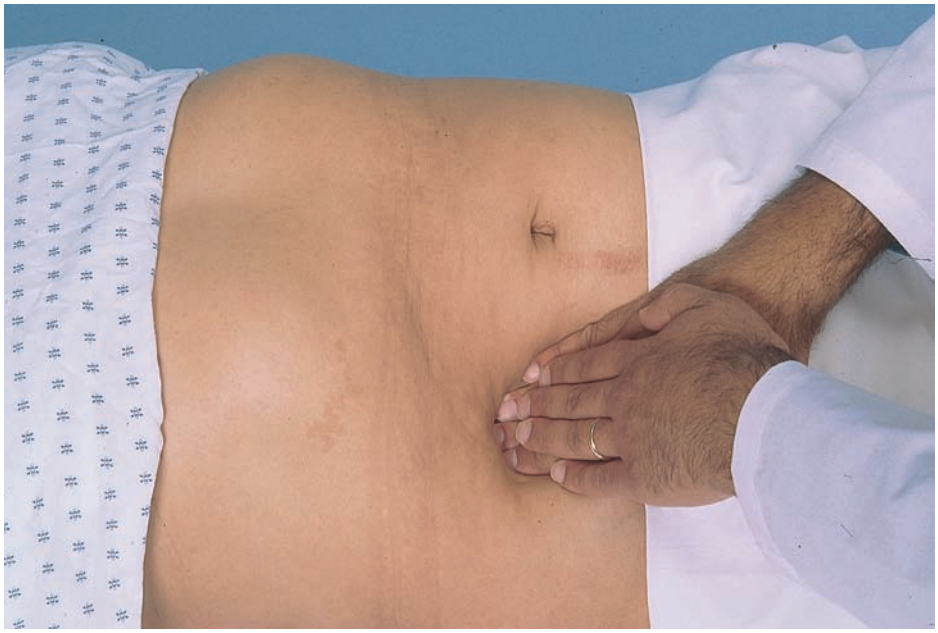


FIGURE 39-11 ▲ Two-handed deep palpation. (From Bickley L: Bates' Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 439.)

Anus and Rectum

Assessment of the anus involves inspection and palpation. The skin around the anus is normally darker than the surrounding area. The nurse should inspect for inflammation, lesions, skin tags or warts, obvious fissures, and hemorrhoids. Palpation of the area with a well-lubricated gloved finger for outpouchings, nodules, tenderness, irregularities, and fecal impaction is necessary. Alteration in elimination due to immobility, limited or no GI intake, opioids, or decreased intestinal peristalsis may result from a patient's disease or its treatment. Constipation may compound matters and, if left untreated, can lead to fecal impaction.[11] Astute nursing assessment is critical in preventing and treating constipation or fecal impaction in any patient.

To assess anal sphincter tone, it is necessary to slip a well-lubricated tip of the index finger of a gloved hand into the anal canal. The nurse asks the patient who can cooperate to bear down to tighten the external sphincter around the inserted finger. He or she assesses the tone; it should tighten, exert even pressure all around, feel smooth, and cause no discomfort to the patient. A lax sphincter may indicate a neurological deficit. A very tight sphincter may result from scarring, spasticity caused by a fissure or other lesion, inflammation, or anxiety about the examination. Inserting the finger farther allows palpation of the walls of the rectum. It is necessary to assess the walls for any nodules, masses, irregularities, polyps, or tenderness. The wall should feel smooth, even, and uninterrupted.[3]

▲ Nutritional Assessment

Adequate nutrition of patients in the ICU improves outcomes, while malnutrition is strongly associated with increased morbidity and mortality rates among critically ill patients. GI complications are one of the main reasons why nutritional requirements of critically ill patients are not met. Nutritional support should be started in intensive care patients as early as possible. Enteral feeding should be prioritized but it is often necessary to supplement this with parenteral feeding as most intensive care patients are at risk of malnutrition.[12]

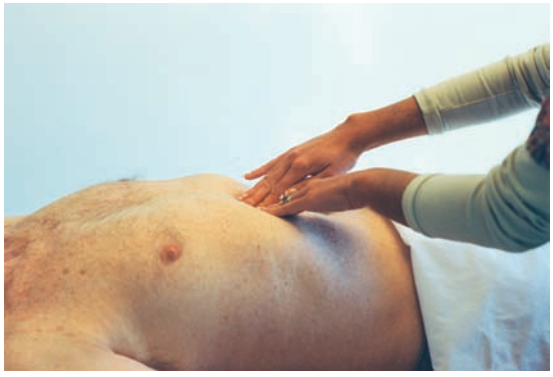


FIGURE 39-12 ▲ Palpating the aorta. (From Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Wolters Kluwer Health, 2010, p 436.)

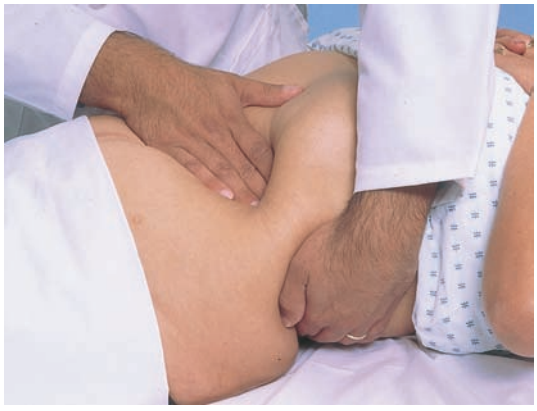


FIGURE 39-13 ▲ Palpating the spleen. (From Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Wolters Kluwer Health, 2010, p 436.)

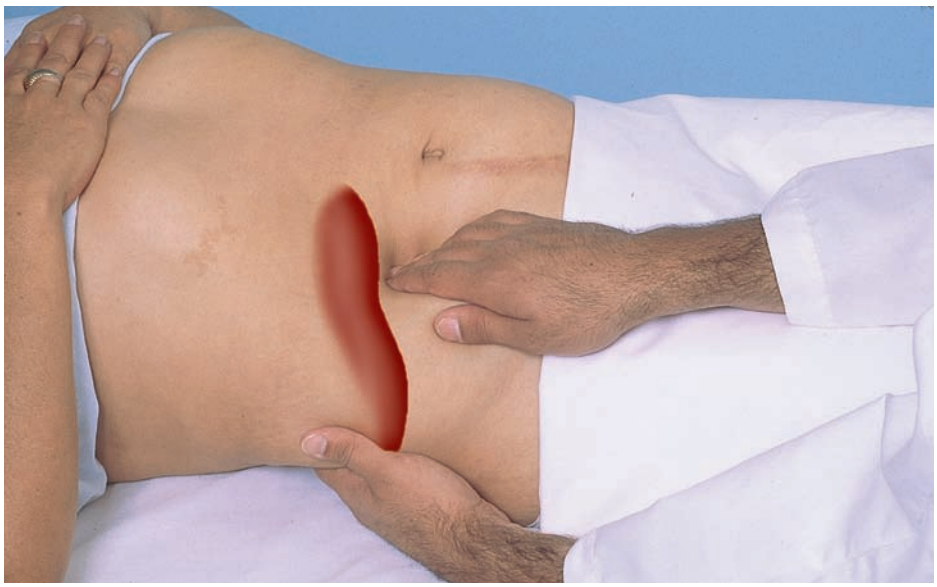


FIGURE 39-14 ▲ Bimanual technique for liver palpation. (From Bickley L: Bates' Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 441.)

A critically ill patient's nutritional status may fall anywhere on a continuum ranging from optimal nutrition to malnutrition. The critical care patient may have an inadequate dietary intake because of illness or the disorder that caused the hospitalization, particularly if the disorder is GI. In addition, critically ill patients are at risk for GI problems from treatments that cause damage to the GI system and reduce the body's ability to absorb nutrients. Optimal nutrient intake provides adequate energy and can protect from complications of disease.[13]

The nurse plays an important role in evaluating the nutritional status of the patients under his or her care. Certain signs and symptoms that suggest possible nutritional deficiency are easy to note because they are specific. Conversely, fluid changes, such as edema or effusions, can mask protein and fat loss. The fact that nutritional disturbances can be subtle and are frequently nonspecific makes the need for assessment important.[14]

Nutritional assessment of the patient refers to a comprehensive evaluation of nutritional status, whereas nutritional screening is the process of identifying patients at risk for malnutrition or who are presently malnourished.[15] The detection of malnutrition in the ICU is critical to appropriately address its contribution to outcomes. Patients who are malnourished generally have longer hospitalizations and higher morbidity, mortality, and costs of care than those who are well nourished.[16]

Nurses caring for patients in the ICU are integral to the screening of patients for nutritional deficiencies or needs. Nurses assist with a formal nutritional assessment by providing information to nutrition support service and even completing screening tools for nutrition risk assessment. Serial weight measurement is perhaps the single most important indicator of nutritional status and is the evaluation that the nurse performs most often.

An initial nutritional assessment may begin with cursory data, as dictated by the patient's condition. A registered dietitian or nutritionist or a nutritional support team may perform a more comprehensive nutritional assessment. The parameters of the nutritional assessment include anthropometric measurement, laboratory studies, physical examination, and dietary evaluation. Anthropometric measurements include height, weight, body mass index, triceps skinfold thickness, and midarm and arm muscle circumference.[15] Table 39-4 lists laboratory studies performed to evaluate nutritional status. Table 39-5 presents information about the physical examination and its interpretation in nutritional disorders.

The dietary evaluation may consist of a 24-hour recall to elicit all the foods and beverages consumed in the preceding 24 hours. However, this method may overestimate or underestimate a patient's usual caloric intake because the patient's recollection may not reflect long-term dietary habits. The nurse assesses the quantity and quality of food ingested by asking the patient to recall his or her normal daily intake pattern. This approach provides more information about intake patterns and tends to reflect long-term dietary habits with greater accuracy. It is also necessary to consider a patient's past or current patterns of food intake, or both, such as vegetarian or kosher dietary practices, as well as cultural background and social situation.[14] Special consideration of the geriatric patient is warranted; age-related changes in the GI system may affect dietary intake and maintenance of adequate nutrition (Box 39-2).

Screening tools are used for nutrition risk assessment and to plan nutrition treatment in the ICU.[15] When patients are critically ill, it is often necessary to elicit this information from the patient's family members or significant other.

A number of quick and efficient screening instruments have been developed to determine the risk or presence of malnutrition in the critically ill patient. The Subjective Global Assessment (SGA) is an example of a simple and reliable screening tool for nutritional assessment that may predict the patient's outcomes in the critical care setting (Fig. 39-15, p. 883). The SGA has been used in many settings for nutrition assessment.[15,17,18]

An important factor that influences nutritional status is nitrogen balance, a sensitive indicator of the body's gain or loss of protein. An adult is in nitrogen balance when the

Table 39-4 Laboratory Studies to Evaluate Nutritional Status

Study	Normal Findings	Clinical and Nursing Significance
Hemoglobin	Males: 13–18 g/dL Females: 12–16 g/dL	Main component of red blood cells (RBCs) used to transport oxygen; identifies iron-carrying capacity of the blood Helps identify anemia, protein deficiency, excessive blood loss, hydration status Elevated with dehydration; decreased in overhydration
Hematocrit	Males: 40%–52% Females: 36%–48%	Identifies volume of RBCs Decreased value with overhydration, blood loss, poor dietary intake of iron, protein, certain vitamins
Albumin	3.5–5.5 g/dL	Assesses protein levels in the body; requires functioning liver cells Decreased with protein deficiency, blood loss secondary to burns, malnutrition, liver/renal disease, heart failure, major surgery, infections, cancer Elevated with dehydration
Total protein	6–8 g/dL	Decreased with overhydration, malnutrition, liver disease
Prealbumin	15–30 mg/dL	Transport protein for thyroxine (T_4) Short half-life makes it more sensitive than albumin to changes in protein stores Decreased in malnutrition in critically ill or those with chronic disease
Transferrin	200–400 mg/dL	Transport protein for iron; synthesized in the liver; shorter half-life than albumin; reflects current protein status; a more sensitive indicator of visceral protein stores Elevated in pregnancy or iron deficiency Decreased in acute or chronic infection, cirrhosis, renal disease, cancer
Retinol-binding protein	3–6 mg/L	Rapidly responds to nutritional depletion due to short half-life Decreased in overhydration and liver disease
Total lymphocyte count	More than 2,000 mm^3	Indicator of immunocompetence Mild: 1,200–2,000 Moderate: 800–1,199 Severe: <800 May indicate malnutrition when no other cause apparent; may point to infection, leukemia, or tissue necrosis

nitrogen intake equals the nitrogen output (in urine, feces, and perspiration). Nitrogen balance is a sign of health. A positive nitrogen balance exists when nitrogen intake exceeds nitrogen output and indicates tissue growth, such as occurs during recovery from surgery, and rebuilding of wasted tissue. A negative nitrogen balance indicates that the tissue is breaking down faster than it is being replaced. In the absence of an adequate intake of protein, the body converts protein to glucose for energy. This can occur with fever, starvation, surgery, burns, and debilitating diseases. Malnutrition occurs when a patient is in negative nitrogen balance. Malnutrition, in turn, interferes with wound healing, increases susceptibility to infection, and contributes to an increased incidence of complications, a protracted hospitalization, and an extended bed confinement.[15]

The nutritional assessment directs the nutritional prescription and assists in the development of the interventions. Enteral or parenteral nutrition may be initiated if oral intake is prohibited or threatened for longer than a week. If the GI tract of the patient is functioning, enteral feeding is the intervention of choice. For people without a functioning GI tract, total parenteral nutrition may be the nutritional treatment of choice. See Chapter 40 for a discussion of enteral and parenteral nutrition. The level of interventions is dictated by the patient's baseline nutritional state, disease status, risk for malnutrition from treatment, and anticipated response to therapy.[12]

▲ Laboratory Studies

Because the oral cavity is the only part of the GI tract that is visible, it is essential to combine the information gleaned from the history and physical examination with the results of laboratory and diagnostic studies to assess the rest of the GI tract. Many laboratory studies help in the diagnosis of GI and abdominal disorders in the critically ill patient. Parameters evaluated include serum electrolytes; levels of end products of metabolism, enzymes, and proteins; and hematological parameters.

Laboratory Studies Relating to Liver Function

The liver is responsible for many functions, the most significant being bile formation and secretion, protein and fat metabolism, detoxification of many substances, and the production of clotting factors and enzymes. Table 39-6 summarizes common laboratory studies relating to liver function.

A single laboratory test or single value from any laboratory test does not give an accurate assessment of an organ's function. A series of values from a laboratory study and combinations of studies provide a more precise picture. For example, if a patient's liver enzymes and bilirubin are elevated but the alkaline phosphatase (AP) level is normal, this usually

Table 39-5 Physical Assessment Interpretation in Nutritional Disorders

Body System or Region	Sign or Symptom	Implications
General	Weakness and fatigue Weight loss	Anemia or electrolyte imbalance, decreased calorie intake, increased calorie use, or inadequate nutrient intake or absorption
Skin, hair, and nails	Dry, flaky skin Dry skin with poor turgor Rough, scaly skin with bumps Petechiae or ecchymoses Sore that will not heal Thinning, dry hair Spoon-shaped, brittle, or rigid nails	Vitamin A, vitamin B complex, or linoleic acid deficiency Dehydration Vitamin A deficiency Vitamin C or K deficiency Protein, vitamin C, or zinc deficiency Protein deficiency Iron deficiency
Eyes	Night blindness; corneal swelling, softening, or dryness; Bitot's spots (gray triangular patches on the conjunctiva) Red conjunctiva	Vitamin A deficiency Riboflavin deficiency
Throat and mouth	Cracks at the corner of mouth Magenta tongue Beefy, red tongue Soft, spongy, bleeding gums Swollen neck (goiter)	Riboflavin or niacin deficiency Riboflavin deficiency Vitamin B_{12} deficiency Vitamin C deficiency Iodine deficiency
Cardiovascular	Edema Tachycardia, hypotension	Protein deficiency Fluid volume deficit
GI	Ascites	Protein deficiency
Musculoskeletal	Bone pain and bow leg Muscle wasting	Vitamin D or calcium deficiency Protein, carbohydrate, and fat deficiency
Neurological	Altered mental status Paresthesia	Dehydration and thiamine or vitamin B_{12} deficiency Vitamin B_{12}, pyridoxine, or thiamine deficiency

Adapted from Lee R, Neiman D: Nutritional Assessment, 5th ed. New York, NY: McGraw Hill, 2010.

indicates injury to the hepatocytes, such as in hepatitis and cirrhosis. If the liver enzymes are within normal range and the bilirubin and AP levels are elevated, this usually indicates an extrahepatic biliary obstruction, such as in a distal common bile duct obstruction from pancreatic cancer or a clip occluding the common bile duct from a laparoscopic cholecystectomy misadventure.

Laboratory Studies Relating to Pancreatic Function

Table 39-7 lists serum laboratory tests that relate to pancreatic function. Amylase and lipase are digestive enzymes secreted by the pancreas. Serum amylase is found in the pancreas, parotid glands, intestine, liver, and fallopian tubes. Lipase is

BOX 39-2 CONSIDERATIONS FOR THE OLDER PATIENT

Age-Related Changes of the GI System

Oral Cavity and Pharynx
- Injury/loss or decay of teeth
- Atrophy of taste buds
- Decreased saliva production
- Reduced ptyalin and amylase in saliva

Esophagus
- Decreased motility and emptying
- Weakened gag reflex
- Decreased resting pressure of lower esophageal sphincter

Stomach
- Degeneration and atrophy of gastric mucosal surfaces with decreased production of HCl
- Decreased secretion of gastric acids and most digestive enzymes
- Decreased motility and emptying

Small Intestine
- Atrophy of muscle and mucosal surfaces
- Thinning of villi and epithelial cells

Large Intestine
- Decrease in mucous secretion
- Decrease in elasticity of rectal wall
- Decreased tone of internal anal sphincter
- Slower and duller nerve impulses in rectal area

Adapted from Smeltzer SC, Bare BG, Hinkle JL, et al (eds): Brunner & Suddarth's Textbook of Medical-Surgical Nursing, 12th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 983.

(Select appropriate category with a checkmark, or enter numerical value where indicated by "#.")

A. History

1. Weight change
   Overall loss in past 6 months: amount = #_____kg; % loss = #_____
   Change in past 2 weeks _____increase,
   _____no change,
   _____decrease.
2. Dietary intake change (relative to normal)
   _____No change
   _____Change _____duration = #_____weeks.
   _____type: _____suboptimal solid diet, _____full liquid diet,
   _____hypocaloric liquids, _____starvation.
3. Gastrointestinal symptoms (that persisted for > 2 weeks)
   _____none, _____nausea, _____vomiting, _____diarrhea, _____anorexia.
4. Functional capacity
   _____No dysfunction (e.g., full capacity)
   _____Dysfunction _____duration = #_____weeks.
   _____type: _____working suboptimally,
   _____ambulatory,
   _____bedridden.
5. Disease and its relation to nutritional requirements
   Primary diagnosis (specify)_____
   _____
   Metabolic demand (stress): _____no stress, _____low stress,
   _____moderate stress, _____high stress.

B. Physical (for each trait specify: 0 = normal, 1+ = mild, 2+ = moderate, 3+ = severe)
#_____loss of subcutaneous fat (triceps, chest)
#_____muscle wasting (quadriceps, deltoids)
#_____ankle edema
#_____sacral edema
#_____ascites

C. SGA rating (select one)
_____A = Well-nourished
_____B = Moderately (or suspected of being) malnourished
_____C = Severely malnourished

FIGURE 39-15 ▲ The Subjective Global Assessment (SGA) used to assess nutritional status. The final SGA rating is based on clinical judgment of the items and not a specific number. (Reprinted from the American Society for Parenteral and Enteral Nutrition (ASPEN): J Parenter Enter Nutr 11(1):8–13, 1987. ASPEN does not endorse the use of this material in any form other than its entirety.)

Table 39-6 Laboratory Studies Used to Evaluate Liver Function

Study	Normal Findings	Clinical and Nursing Significance
Bile Formation and Secretion		
Serum bilirubin		
Direct (conjugated)—soluble in water	0–5.1 micromol/L	Abnormal in biliary and liver disease; causes clinical jaundice
Indirect (unconjugated)—insoluble in water	0–14 micromol/L	Abnormal in hemolysis and in functional disorders of uptake or conjugation
Urine bilirubin	0	Urine is mahogany in color; shaking the specimen results in a light yellow foam; confirmed with Ictotest tablet or dipstick; false-positive results possible if patient is taking phenazopyridine (Pyridium).
Urobilinogen		Increased in cirrhosis, biliary obstruction with biliary tract infection, hemorrhage, and hepatotoxicity; decreased in biliary obstruction without biliary tract infection, hepatocellular damage, and renal insufficiency
Urine urobilinogen	Up to 0.09–4.23 micromol/24 h	Urine specimen is collected over a 2-h period after lunch and must be placed in a dark brown container and sent to the laboratory immediately to prevent decomposition.
Fecal urobilinogen	Up to 0.068–0.34 mmol/24 h	
Protein Studies		
Albumin	35–55 g/L	Decreased in cirrhosis, chronic hepatitis
Globulin	15–30 g/L	Increased in cirrhosis, chronic obstructive jaundice, viral hepatitis
Albumin/globulin ratio	1.5:1–2.5:1	Ratio reverses with chronic hepatitis or other chronic liver disease
Total serum protein	60–80 g/L	Individual protein measurements are of greater significance than total protein measurements.

(Continued on page 884)

Table 39-6 Laboratory Studies Used to Evaluate Liver Function (continued)

Study	Normal Findings	Clinical and Nursing Significance
Protein Studies		
Transferrin	220–400 mcg/dL	Decreased in cirrhosis, hepatitis, and malignancy; increased in severe iron deficiency anemia
Prothrombin time (PT) or International normalized ratio	11.0–14.0 s or 100% of control 0.8–1.2	Prolonged PT in liver disease will not return to normal with vitamin K administration, whereas prolonged PT resulting from malabsorption of fat and fat-soluble vitamins will return to normal with vitamin K administration.
Partial thromboplastin time	25.0–36.0 s	Increased with severe liver disease or therapy with heparin or other anticoagulants
Alpha-fetoprotein (AFP)	6–20 ng/mL	Elevated in primary hepatocellular carcinoma
Fat Metabolism		
Cholesterol	<200 mg/dL (adults)	Decreased in parenchymal liver disease; increased in biliary obstruction
High-density lipoprotein Men Women	 35–70 mg/dL 35–85 mg/dL	
Low-density lipoprotein	<130 mg/dL	
Very-low-density lipoprotein	25%–50%	
Liver Detoxification		
Serum alkaline phosphatase (AP)	20–90 units/L at 30°C	Level is elevated to more than three times the normal in obstructive jaundice, intrahepatic cholestasis, liver metastasis, or granulomas; also elevated in osteoblastic diseases, Paget's disease, and hyperparathyroidism
Ammonia	15–49 mcg/dL	An elevation indicates hepatocyte damage (liver converts ammonia to urea)
Enzyme Production		
Aspartate aminotransferase	8–20 units/L	Any elevation indicates hepatocyte damage.
Alanine aminotransferase	10–32 units/L	Any elevation indicates hepatocyte damage.
Lactate dehydrogenase	200–500 units/L	Any elevation indicates hepatocyte damage.
γ-Glutamyl transferase (GGT)	0–30 units/L at 30°C	An elevation in GGT along with an elevated AP usually indicates biliary disease; helpful in the diagnosis of chronic liver disease

Table 39-7 Laboratory Studies Used to Evaluate Pancreatic Function

Study	Normal Findings	Clinical and Nursing Significance
Serum amylase	25–125 units/L	In acute pancreatitis, the serum levels peak between 4 and 8 h after onset of condition, then fall to normal within 48–72 h; low levels usually indicate pancreatic insufficiency.
Urine amylase	2 h: 2–34 units 24 h: 24–408 units	Urine values 6–10 h behind serum values; low levels indicate pancreatic insufficiency.
Serum lipase	10–40 units/L (adults)	Elevated only in pancreatitis, markedly in acute pancreatitis and pancreatic duct obstruction; remains elevated after amylase returns to baseline
Serum glucose	65–110 mg/dL (fasting)	Patient must fast for 12 h before specimen is obtained.
Serum triglycerides	50–250 mg/dL	Patient must fast for 12 h before specimen is obtained; levels increased in alcoholic cirrhosis, diabetes mellitus (untreated), high-carbohydrate diet, hyperlipoproteinemia, and hypertension; levels decreased in malnutrition, vigorous exercise
Serum calcium Total Ionized	 8.2–10.2 mg/dL 4.65–5.28 mg/dL	 High total calcium levels seen in cancer of the liver, pancreas, and other organs Useful in tracking the course of disorders, such as cancer and acute pancreatitis
Fecal fat	2–5 g/24 h	Content of >6 g/24 h is suggestive of a decrease in the body's ability to absorb foods; indicative of pancreatic exocrine insufficiency as in chronic pancreatitis

Table 39-8 Other Selected Laboratory Studies Used in the Diagnosis of GI Disorders

Study	Normal Findings	Clinical and Nursing Significance
Stool specimen		
Occult blood	None	Positive test suggests possible malignancy.
Fat	2–5 g/24 h	Screening test for steatorrhea when malabsorption syndrome or pancreatic insufficiency is suspected
Ova and parasites	None	Positive test suggests infection.
Pus	None	Increased amount of pus may indicate ulcerative colitis, abscess, or anal or rectal fissure.
Pathogens	None	Common pathogens are *Salmonella typhi* (typhoid fever), *Shigella* (dysentery), *Vibrio cholerae* (cholera), *Yersinia* (enterocolitis), *Escherichia coli* and *Aeromonas* (gastroenteritis), *Staphylococcus aureus, Clostridium botulinum*, and *Clostridium perfringens* (food poisoning).
Urea breath test	Negative	Detects the presence of *Helicobacter pylori*
Hydrogen breath test	Negative	Determines the amount of hydrogen expelled in the breath after it is produced in the colon and absorbed into the blood; aids in the diagnosis of bacterial overgrowth in the intestine and short bowel syndrome

found primarily in the pancreas. In acute pancreatitis, serum amylase and lipase can be elevated four to six times the normal level, whereas in chronic pancreatitis, serum amylase and lipase levels may be normal or very low because the pancreas may no longer be producing the enzymes.

The pancreas produces insulin and glucagon, hormones that aid in the regulation of serum glucose levels. A disruption in normal pancreatic function or the presence of a tumor may alter the production of these hormones. Frequent blood glucose monitoring is warranted. Any elevation in the serum and urine glucose levels has a cascading effect on multiple body systems, which in turn affects the patient's overall condition.[19]

Other Laboratory Studies

Table 39-8 provides information about other selected laboratory studies that are used in the evaluation of GI disorders.

▲ Diagnostic Studies

The nurse caring for the critically ill patient coordinates the preparation for, and possibly the timing of, many diagnostic tests. The nurse prepares the patient, the family members, or both for the test by providing a thorough explanation of how the test is performed and what information the test is expected to yield. In addition, the nurse explains the need for informed consent to perform the test and answers any questions the patient or family members may have about the test. Table 39-9 summarizes the diagnostic studies for evaluating the GI tract, which can be divided into two categories, noninvasive and invasive.

Radiological and Imaging Studies

Body tissue has different densities that produce different shades of black and white on an x-ray. Bone tissue is high in density and appears white; air appears black; and soft tissue results in shades of gray. The stomach and intestines usually contain some air and appear darker. Solid organs, such as the pancreas, spleen, kidneys, or liver, appear grayer.[20]

Endoscopic Studies

The use of an endoscope is an important adjunct to radiographic studies because it allows direct observation of portions of the intestinal tract. A flexible fiberoptic endoscope is an instrument with a light and a lens at the end of a movable tip that can be manipulated through the intestinal tract by the operator. It also includes an instrument channel that allows for biopsy of lesions, such as tumors, ulcers, or areas of inflammation. Fluids can be aspirated from the lumen of the intestinal tract, and air can be insufflated to distend the intestinal tract for better observation. Cytology brushes and electrocautery snares can be passed through the scope. Special studies of the common bile duct and the pancreatic duct, by endoscopic retrograde cholangiopancreatography, use a side-viewing upper intestinal endoscope. Endoscopic ultrasonography with fine-needle aspiration is another procedure that uses an endoscope with an ultrasound probe and a biopsy needle at the end. The endoscope is inserted through an orifice and the ultrasound probe is used to bounce high-energy sound waves off internal organs and tissues that create a picture on a monitor. This allows the operator to place the needle to obtain cells from a mass or lymph nodes for biopsy.[21] Advances in

Table 39-9 Diagnostic Studies Used to Evaluate the GI Tract

Study	Description	Indications	Invasiveness	Preparation	Contrast
Abdominal film "flat plate of the abdomen"	Radiology test used to visualize a single flat plane; shows organ size, position, intactness, and normal gas patterns in the stomach, small intestine, and colon	Aids in diagnosis of intestinal obstruction, organ rupture, masses, foreign bodies, abnormal fluid or air ("stones, bones, gas, masses")	Noninvasive	None	No
Upper GI series (barium swallow)	Radiology test used to visualize the esophagus, stomach, and duodenum; barium enhances image; double-contrast study administers barium first followed by a radiolucent substance, such as air, to help coat bowel mucosa for better visualization of any type of lesion.	Aids in diagnosis of hiatal hernia, ulcers, tumors, foreign bodies, bowel obstruction	Noninvasive	NPO	Yes
Upper GI series with small bowel follow-through	Radiology test used to visualize the jejunum, ileum, and cecum	Aids in the diagnosis of tumors, Crohn's disease, Meckel's diverticulum	Noninvasive	NPO	Yes
Enteroclysis	Radiology test used to visualize entire small intestine; continuous infusion (through a duodenal tube) of air in a barium sulfate suspension along with methylcellulose fills the intestinal loops; transit of contrast filmed at intervals for progress through jejunum and ileum	Aids in diagnosis of partial bowel obstruction or diverticula	Invasive	NPO	Yes
Barium enema	Radiology test used to visualize the colon; barium enhances image; air may be introduced after the barium to provide a double-contrast study	Aids in diagnosis of polyps, tumors, fistulas, obstruction, diverticula, and stenosis	Noninvasive	Bowel cleansing	Yes
Gastric lavage	Aspiration of stomach contents and washing out of the stomach by a large gastric tube	Aids in diagnosis of upper GI bleeding; also used to arrest hemorrhage and prepare for further tests	Invasive	None	No
Paracentesis	Aspiration of peritoneal fluid	Laboratory studies (such as amylase and lipase to assess for pancreatitis), cytologic studies (to detect tumors), comfort measure (to alleviate accumulation of ascitic fluid)	Invasive	None	No
Peritoneal lavage	Irrigation of peritoneal cavity to examine irrigating fluid for blood	Blunt or penetrating trauma to the abdomen	Invasive	None	No

(continued on page 887)

Table 39-9 Diagnostic Studies Used to Evaluate the GI Tract (continued)

Study	Description	Indications	Invasiveness	Preparation	Contrast
Biopsy					
Percutaneous	A needle is placed through the skin to obtain tissue specimen for pathology evaluation	Aids in diagnosis of malignancy	Invasive	NPO	No
Fine-needle aspiration	A thin needle is used to obtain cells or minute tissue fragments from a suspect area for examination by light microscopy; usually guided by radiologists with fluoroscopy, ultrasound, computed tomography, or magnetic resonance imaging	Aids in diagnosis of malignancy	Invasive	NPO	No
Ultrasonography (sonogram)	Use of high-frequency sound waves over an abdominal organ to obtain an image of the structure	Aids in diagnosis of masses, dilated bile ducts, gallstones, and ascites	Noninvasive	NPO	No
Hepatobiliary scan	Intravenously injected radioisotope is primarily taken up by the liver and then secreted into the bile, allowing visualization of the biliary system, gallbladder, and duodenum (size, function, vascularity, and blood flow).	Aids in the diagnoses of common bile duct obstruction, acute and chronic cholecystitis, bile leaks, biliary dyskinesia, and biliary atresia; also used to evaluate liver transplant function	Noninvasive	NPO	Yes
Tagged red blood cell scan (technetium-labeled red blood cell scintigraphy)	Red blood cells are labeled with technetium and injected intravenously; images are obtained with a gamma camera that can identify areas of increased radioactivity as a site of slow or intermittent GI hemorrhage.	Aids in the diagnosis of GI bleeding	Noninvasive	None	Yes
Computed tomography (CT) scan	A radiological procedure that uses narrow x-ray beams to produce cross-sectional images of organs and tissues; can be performed with or without contrast medium; multidetector-row CT now provides more exact information even when combined with three-dimensional imaging, which can be rotated on a computer screen and allows different views for even more information.	Excellent for visualizing the abdomen, retroperitoneal structures, tumors, cysts, collection of fluid, air in a cavity, bleeding, or pulmonary embolism	Noninvasive	NPO	Yes/No

(continued on page 888)

Table 39-9 Diagnostic Studies Used to Evaluate the GI Tract (continued)

Study	Description	Indications	Invasiveness	Preparation	Contrast
Magnetic resonance imaging (MRI)	Diagnostic study that does not use radiation; obtains images by passing the patient through a tubular device that generates a powerful electromagnetic field; radiofrequency waves are transmitted into the patient in a controlled manner so that the patient's hydrogen ions (protons) emit radiofrequency signals that are processed by a computer to produce an image.	Useful in evaluating abdominal soft tissue and blood vessels, abscesses, fistulas, tumors, and sources of bleeding	Noninvasive	No metal devices attached to or implanted in the patient; patient must be able to lie quietly for a period of time.	No
Magnetic resonance cholangiopancreatography (MRCP)	Similar to MRI; because no contrast agent is required, MRCP is ideal for patients with allergies to iodine-based contrast medium.	Aids in the diagnosis of disorders affecting the pancreatic ducts and biliary tree	Noninvasive	No metal devices attached to or implanted in the patient	No
Percutaneous transhepatic cholangiography (PTC)	Radiological procedure done under fluoroscopy to examine the intrahepatic and extrahepatic biliary ducts after injection of contrast medium into the biliary tree through percutaneous needle injection	Helps to distinguish obstructive jaundice caused by liver disease from jaundice caused by biliary obstruction (eg, from a tumor, common bile duct injury, stones within the bile ducts, or sclerosing cholangitis)	Invasive	NPO	Yes
Percutaneous transhepatic biliary drainage (PTBD)	A biliary catheter is placed during a PTC; the biliary catheter may be placed to the obstruction or it may bypass the obstruction to allow the free flow of bile; catheter relieves jaundice and pruritus, improves nutritional status, allows easy access into the biliary tree for further procedures, and can be used as an anatomical landmark and stent at the time of surgery.	Biliary obstruction resulting in jaundice, cholangitis, sepsis, or pain	Invasive	NPO	Yes
Positron emission tomography (PET)	A computerized radiographic technique that uses radioactive substances to examine the metabolic activity of body structures PET scan can be combined with a CT scan so images can be acquired from both devices and can be taken sequentially at the same time for superimposed images.	Useful for precisely locating a tumor Provides metabolic activity revealed by the PET scan along with the anatomic information provided by the CT scan	Noninvasive	None	Yes
Angiography	A radiographic study of selected arteries and veins to see defects in the walls of the vessels; also used to evaluate blood flow through the vessels	Usually done when initial, noninvasive procedures are insufficient in revealing the cause of a suspected vascular defect	Invasive	NPO	Yes

Table 39-10 Endoscopic Studies Used to Evaluate the GI Tract

Study	Description	Indications	Invasiveness	Preparation	Contrast
Esophagogastroduodenoscopy	Endoscope passed through the mouth and advanced to visualize the esophagus, stomach, and duodenum; any abnormalities can be photographed and biopsied; bleeding areas may be cauterized and varices may be injected with sclerosing agents.	Helps to diagnose acute or chronic upper GI bleeding, esophageal or gastric varices, polyps, tumors, ulcers, esophagitis, gastritis, esophageal stenosis, and gastroesophageal reflux	Invasive	NPO	No
Colonoscopy	Flexible fiberoptic endoscope passed through the rectum and advanced to visualize the large intestine; any abnormalities can be photographed and biopsied; polyps can be removed and bleeding areas may be cauterized.	Helps to diagnose bleeding, diverticulosis, polyps, stricture, tumor, or inflammatory bowel disease (Crohn's disease or ulcerative colitis)	Invasive	Bowel cleansing	No
Proctoscopy (anoscopy)	Rigid scope passed through the rectum to visualize the mucosal surface of the anus and rectum	Helps to diagnose polyps, bleeding, tumors, and other defects	Invasive	None	No
Sigmoidoscopy	Flexible fiberoptic endoscope passed through the rectum and advanced to visualize the rectum, sigmoid colon, and proximal colon; any lesions can be biopsied.	Helps to diagnose tumors, polyps, diverticula, or bleeding	Invasive	Enema	No
Endoscopic retrograde cholangiopancreatography	Flexible fiberoptic endoscope inserted into the esophagus, passed through the stomach, and into the duodenum to visualize the common bile duct, hepatic bile ducts, and pancreatic ducts; the common bile duct and pancreatic duct are cannulated and contrast medium is injected into the ducts, permitting visualization and radiographic evaluation.	Can detect extrahepatic biliary obstruction (eg, from stones, tumors of the bile duct, strictures or injuries to the bile duct), intrahepatic biliary obstruction caused by stones or tumor; and pancreatic disease, such as chronic pancreatitis, pseudocysts, or tumors	Invasive	NPO	Yes
Endoscopic ultrasonography	Endoscopy and ultrasonography are used to visualize the GI tract; an ultrasonic transducer built into the distal end of the endoscope allows for high-quality resolution of the walls of the GI tract.	Useful in evaluating and staging tumors of the GI tract	Invasive	NPO	No

technology now allow specialized instruments direct intraductal visualization of all bile ducts and pancreatic ducts by steering the probe in four directions to access and inspect all four quadrants for diagnostic and therapeutic applications/interventions. This technique is called SpyGlass™.[22]

Table 39-10 describes endoscopic procedures used to evaluate the GI tract.

Other Diagnostic Studies

In addition to radiological and imaging studies and endoscopic procedures, other studies are specifically designed to aid in the diagnosis of GI disorders. Table 39-11 provides information about selected diagnostic studies used to diagnose specific GI disorders.

Table 39-11 Other Selected Diagnostic Studies Used in Diagnosing GI Disorders

Study	Description	Normal Findings
Gastric emptying studies	Liquid and solid components of a meal are tagged with a radionuclide marker. After ingesting the meal, the rate of passage of the radioactive substance out of the stomach is measured by a scintiscanner. Useful in diagnosing gastric motility disorders	Normal transit
Gastric analysis	Analysis of gastric juice yields information about the secretory activity of the gastric mucosa and the presence or degree of gastric retention, which is useful to help diagnose patients with pyloric or duodenal obstruction.	Normal contents
Gastric acid stimulation (usually performed in conjunction with gastric analysis)	Histamine or pentagastrin is given subcutaneously to stimulate gastric secretions. Gastric specimens are collected at intervals for analysis. Helps to determine the presence or absence of malignant cells	11–20 mEq/h after stimulation
Manometry	Measurement of pressures using a water-filled catheter connected to a transducer passed into the esophagus, stomach, colon, or rectum to evaluate contractility; useful in detecting motility disorders of the esophagus and lower esophageal sphincter; gastroduodenal, small intestine, and colonic manometry are used to evaluate delayed gastric emptying and gastric and intestinal motility disorders such as irritable bowel syndrome or atonic colon; anorectal manometry measures the resting tone of the internal anal sphincter and the contractility of the external anal sphincter, which is helpful in evaluation of chronic constipation or fecal incontinence.	Values differ at various levels of the intestine.
Gastric tonometry	Monitoring modality used to determine the perfusion status of the gastric mucosa using measurements of local PCO_2. The CO_2 diffuses from the mucosa of the stomach into the lumen of the stomach and then into the silicone balloon of the tonometer. The PCO_2 within the balloon serves as a proxy measure for gastric mucosal CO_2 ($PgCO_2$). In a normally perfused gastric mucosa, $PgCO_2$ is nearly equivalent to the $PaCO_2$. With hypoperfusion, the $PgCO_2$ increases and the gap between the $PgCO_2$ and the $PaCO_2$ increases. The gap is a very sensitive indicator of gastric hypoperfusion.	The $PgCO_2$ and the $PaCO_2$ are nearly equal.

▲ Clinical Applicability Challenges

CASE STUDY

Mr. H. is a 75-year-old male with a history of multiple primary cancers including prostate cancer treated with local radiation therapy, vocal cord cancer treated with partial laryngectomy, and colon cancer for which he had a subtotal colectomy with ileosigmoid anastomosis 1 year ago. The patient's other past medical and surgical history reveals mitral valve replacement for mitral valve regurgitation, atrial fibrillation, cerebrovascular accident with no residual deficits, type 1 diabetes mellitus, hypertension, and glaucoma.

Mr. H. received three rounds of adjuvant chemotherapy after his colon cancer surgery. A restaging CT scan of chest, abdomen, and pelvis showed no evidence of peritoneal or pulmonary lesions but a new lesion in the right lobe of the liver. PET/CT scan confirmed only site of metastases to be in the liver. Preoperative CEA level was 243, which was elevated from 81 three months ago.

The patient now presents to the ICU after partial hepatectomy for metastatic adenocarcinoma of the colon to the liver. Preoperative laboratory values reveal serum glucose ranges from 123 to 133 mg/dL, total protein 7.9, albumin 3.2, white blood count 6,710, hemoglobin 9.6, and hematocrit 35.9.

1. Discuss the risk factors for malnutrition for the patient in the case study.
2. What would be included in an educational plan for the patient and his family?
3. What abdominal assessment would the nurse perform when the patient in the case study is admitted to the ICU?

References

1. Ignatavicius DD, Workman ML: Medical-Surgical Nursing: Patient-Centered Collaborative Care, 6th ed. Philadelphia, PA: WB Saunders, 2009
2. Silen W: Cope's Early Diagnosis of the Acute Abdomen, 22nd ed. Oxford, UK: Oxford University Press, 2010
3. D'Amico D, Barbarito C: Health & Physical Assessment in Nursing. Englewood Cliffs, NJ: Prentice Hall, 2008
4. Leblond RF, DeGowin RL, Brown DD: DeGowin's Diagnostic Examination, 9th ed. New York, NY: McGraw Hill, 2009
5. Joseph NM, Sistla S, Dutta TK, et al: Ventilator-associated pneumonia: A review. Eur J Intern Med 21(5):360–368, 2010
6. Munro CL, Grap MJ, Jones DJ, et al: Chlorhexidine, toothbrushing, and preventing ventilator-associated pneumonia in critically ill adults. Am J Crit Care 18(5):428–438, 2009
7. Garcia R, Jendresky L, Colbert L, et al: Reducing ventilator-associated pneumonia through advanced oral-dental care: A 48-month study. Am J Crit Care 18(6):523–534, 2009
8. Feider LL, Mitchell P, Bridges E: Oral care practices for orally intubated critically ill adults. Am J Crit Care 19(2):175–183, 2010
9. Restrepo MI, Anzueto A, Arroliga AC, et al: Economic burden of ventilator-associated pneumonia based on total resource utilization. Infect Control Hosp Epidemiol 31(5):509–515, 2010
10. Estes MEZ: Health Assessment and Physical Examination, 4th ed. Canada: Thomson Delmar Learning, 2009
11. Goolsby MJ, Grubbs L: Advanced Assessment: Interpreting Findings and Formulating Differential Diagnoses. Philadelphia, PA: FA Davis, 2006
12. McClave SA, Martindale RG, Vanek VW, et al: Guidelines for the provision and assessment of nutrition support in the adult critically ill patient: Society of Critical Care Medicine (SCCM) and American Society of Parenteral and Enteral Nutrition (A.S.P.E.N.). J Parenter Enteral Nutr 33(3):277–316, 2009
13. Wischmeyer P: Pharmaconutrition and nutrition therapy in critical illness. Crit Care Clin 26(3):433–582, 2010
14. Hark L, Morrison G: Medical Nutrition and Disease: A Case-Based Approach, 4th ed. Malden, MA: Wiley-Blackwell, 2009
15. Sungurtekin H, Sungurtekin U, Oner O, et al: Nutritional assessment in critically ill patients. Nutr Clin Pract 23(4):635–641, 2008
16. Sheean PM, Peterson SJ, Gurka DP, et al: Nutritional assessment: The reproducibility of subjective global assessment in patients requiring mechanical ventilation. Eur J Clin Nutr 64(11):1358–1364, 2010
17. Makhija S, Baker J: The subjective global assessment: A review of its use in clinical practice. Nutr Clin Pract 23(4):405–409, 2008
18. Keith JN: Bedside nutrition assessment past, present, and future: A review of the Subjective Global Assessment. Nutr Clin Pract 23(4): 410–416, 2008
19. Gunst J, Van den Berghe G: Blood glucose control in the intensive care unit: Benefits and risks. Semin Dial 23(2):157–162, 2010
20. Federle MP, Jeffrey RB, Woodward PJ, et al: Diagnostic Imaging: Abdomen, 2nd ed. Canada: Amirsys Inc., Elsevier Saunders, 2009
21. Cotton PB: Advanced Digestive Endoscopy: Practice and Safety. Malden, MA: Wiley- Blackwell Publishing, 2008
22. Reavis KM, Melvin WS: Advanced endoscopic technologies. Surg Endosc 22(6):1533–1546, 2008

40 Patient Management: Gastrointestinal System

Valerie K. Sabol and Allison G. Steele

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Explain how the physiological stressors of illness and injury alter the body's needs for energy.
2. Describe the different forms of malnutrition.
3. Discuss enteral and parenteral nutrition with regard to indications, assessment, management, and complications.
4. Discuss common medications used for patients with gastrointestinal disorders.

Health and nutrition have a symbiotic relationship. Physiological stressors, such as illness and injury, alter the body's metabolic and energy demands. Although early identification and nutritional intervention can lessen morbidity and mortality risks in critically ill patients, the underlying disease process must be identified and corrected before the body can reverse abnormal nutrient metabolism. This chapter presents an overview of physiological stress and its effect on metabolism, types of malnutrition, and the indications, assessment, and management of enteral and parenteral nutrition support therapies and the complications associated with these therapies.

▲ Malnutrition

According to the laws of thermodynamics, energy can be neither created nor destroyed. Through the processes of metabolism, people obtain energy from the foods (or organic fuels) they consume. Metabolism has two parts: anabolism and catabolism. Anabolism is a building-up and repair process that requires energy. Catabolism consists of breaking down food and body tissues to liberate energy.

Glucose is the obligatory fuel of the body, and it is the primary fuel of the brain and nervous system in particular. The nervous system cannot store or synthesize glucose as a fuel source, so it relies on glucose extraction from the bloodstream. The liver regulates glucose entry into the circulatory system because it has the ability to both store and synthesize glucose. Excess glucose is converted and stored as either glycogen or fatty acids (triglycerides). Although glucose can be converted to fatty acids for storage, there is no pathway for the conversion of fatty acids back into glucose. Instead, fatty acids are used directly as a fuel source or are converted to ketones by the liver. After prolonged starvation, the body adapts to preserve vital proteins by using ketones, rather than glucose, as energy. Ketoacidosis occurs when ketone production exceeds utilization.

The pancreatic hormones glucagon and insulin have opposing functions in metabolic processes. Glucagon stimulates glycogenolysis (glycogen breakdown) and gluconeogenesis (glucose synthesis from other sources such as proteins) and increases lipolysis (fat breakdown and mobilization). Insulin, in contrast, helps transport glucose for storage into the cells and tissues, prevents fat breakdown, and increases protein synthesis.

Glycogenolysis is controlled by the hormone glucagon and the catecholamines epinephrine and norepinephrine, which are released from the adrenal medulla in times of stress. Once glucose and glycogen stores have been exhausted (usually within 8 to 12 hours), hepatic gluconeogenesis increases dramatically to meet metabolic demands. Hormones that stimulate gluconeogenesis include glucagon and the glucocorticoid hormone cortisol. If catabolic processes continue without the support of energy, amino acids, and essential nutrients, depletion of existing body stores compromises overall bodily health and function, and without intervention, malnutrition may develop.

The metabolic response to stress is characterized by increased release of cytokines (interleukin-1, interleukin-6, and tumor necrosis factor-α) and increased production of counter-regulatory hormones (catecholamines, cortisol, glucogen, and growth hormone).[1] These counter-regulatory hormones induce catabolism and oppose the anabolic effects of insulin. This results in hypermetabolism and hypercatabolism with loss of body energy stores through proteolysis, lipolysis, and glycogenolysis. Critical illness is typically associated with catabolic stress in which patients commonly demonstrate a systemic inflammatory response.

Approximately one third to one half of hospitalized patients have evidence of malnutrition.[2] Forty percent of patients experience considerable weight loss (more than 10 kg) during and after a stay in the intensive care unit (ICU).[3]

This unintentional weight loss may deplete vital nutrient reserves, which may predispose the patient to malnutrition. Malnutrition is associated with increased morbidity and mortality, delayed wound healing, increased length of hospitalization, increased complications, immunosuppression, and organ impairment.[2] Malnutrition from starvation alone can usually be corrected by replacing body stores of essential nutrients. However, malnutrition resulting from critical illness and disease processes that alter metabolism is not as easily rectified.

The degree of starvation and physiological stress determines the extent and type of malnutrition. The three major types of protein-energy malnutrition are marasmus, kwashiorkor, and protein–calorie malnutrition. Marasmus is a severe, cachectic process, whereby virtually all of the available fat stores have been exhausted from prolonged calorie deficiency. Severe muscle wasting is evident in marasmus; however, serum albumin levels may be within normal limits or only slightly reduced. Treatment requires nutrition and fluid volume replacement at slow rates to prevent the complications associated with sudden fluid shifts, electrolyte abnormalities, and cardiorespiratory failure.

In contrast to the adaptive response of relative protein sparing in marasmus, kwashiorkor and protein–calorie malnutrition are typically caused by an acute, life-threatening illness, such as surgery, trauma, or sepsis. Kwashiorkor is usually seen in children in developing countries who have had prolonged periods of protein malnutrition. Protein–calorie malnutrition is more commonly seen in developed countries and is due to depletion of fat, muscle wasting, and micronutrient deficiencies from acute and chronic illness. Typically, during periods of critical illness when the patient is relegated to NPO status (*nil per os*, nothing by mouth) for surgery, diagnostic testing, or a number of other medical complications, hypermetabolism and catabolism increase protein and energy demands. Although the critically ill patient may appear nourished, often this is due to the masking effects of generalized edema—the result of extracellular fluid shifts caused by low-protein oncotic pressures in the intravascular space. Other than edema, clinical signs of protein malnutrition include skin breakdown, poor wound healing, surgical dehiscence, or a combination of the three. Additionally, hair can easily be plucked, and hair remnants are often noted on the patient's pillowcase and sheets. Laboratory data reveal low serum albumin levels, and treatment requires aggressive repletion of protein stores. The fact that protein malnutrition is much easier to prevent than to treat reinforces the need for intense nursing vigilance over the patient's nutritional status.

Marasmus and kwashiorkor can coexist. Typically, this is seen when a patient with marasmus is exposed to an acute stressor such as surgery, trauma, or sepsis. Although each situation must be evaluated individually, aggressive protein and calorie replacement is often indicated. Regardless of the type (or types) of malnutrition, vigilant monitoring is crucial to the success of nutrition therapy.

▲ Nutritional Support

A nutritional assessment should be completed on all critically ill or injured patients early in their hospitalization to determine the need for nutritional support therapy. The timing of nutritional support therapy is based on evaluation of the preexisting nutritional status, the presence and extent of systemic response to inflammation, and the anticipated clinical course.[4,5] Nutritional support therapy delivered within 24 to 48 hours of admission, once patients are hemodynamically stable, is advocated.[5–7] Goals of care in nutritional support include the following: preventing and treating macronutrient and micronutrient deficiencies, maintaining fluid and electrolyte balance, maintaining immune function, preventing infection, and other complications associated with nutritional therapy, and improving patient morbidity and mortality. Meeting these goals involves a multidisciplinary approach that includes the nurse, physician, dietitian, and pharmacist. Sample nursing diagnoses are given in Box 40-1. After a dietitian determines nutritional needs, a method of delivering nutritional supplementation must be selected. In patients unable to meet their nutritional needs with oral intake, nutritional supplementation may be delivered by enteral or parenteral routes. Figure 40-1 outlines the decision-making process. Considerations for elderly patients are given in Box 40-2.

BOX 40-1 EXAMPLES OF NURSING DIAGNOSES

For the Patient With Gastrointestinal Problems

- Imbalanced Nutrition: Less Than Body Requirements
- Imbalanced Nutrition: More Than Body Requirements
- Risk for Aspiration related to reduced level of consciousness, depressed cough and gag reflexes, incompetent lower esophageal sphincter, delayed gastric emptying, displaced feeding tube
- Diarrhea related to altered dietary intake, malabsorption, concomitant drug therapy, type of formula, bacterial contamination, stress/anxiety
- Risk for Infection related to invasive procedure and delivery of high concentrations of glucose parenterally
- Disturbed Body Image
- Bowel Incontinence
- Risk for Imbalanced Fluid Volume
- Impaired Swallowing

Enteral Nutrition and Delivery

Enteral nutrition refers to any form of nutrition delivered to the gastrointestinal (GI) tract. For those patients with an intact GI tract, the enteral route is the preferred method of nutritional support. A clinical rule of thumb is, "If the gut works, use it."

The GI mucosa depends on nutrient delivery and adequate blood flow to prevent atrophy, thereby maintaining the absorptive, barrier, and immunological functions of the intestine. Enterocytes are tightly packed epithelial cells that line the intestinal lumen and function as a barrier to bacterial invasion. Gut-associated lymphoid tissue (GALT) lines the GI tract and is associated with maintenance of the immunological function of the mucosa. GALT produces immunoglobulin A (IgA), which is secreted across the GI mucosa in response to eating. IgA coats the luminal bacteria, preventing bacterial adherence to the enterocytes. Without food, the GI mucosa atrophies, and motility is impaired. In the event of atrophy, the tissue available to absorb nutrients decreases and GALT is impaired. Preservation of the intestinal mucosal integrity is

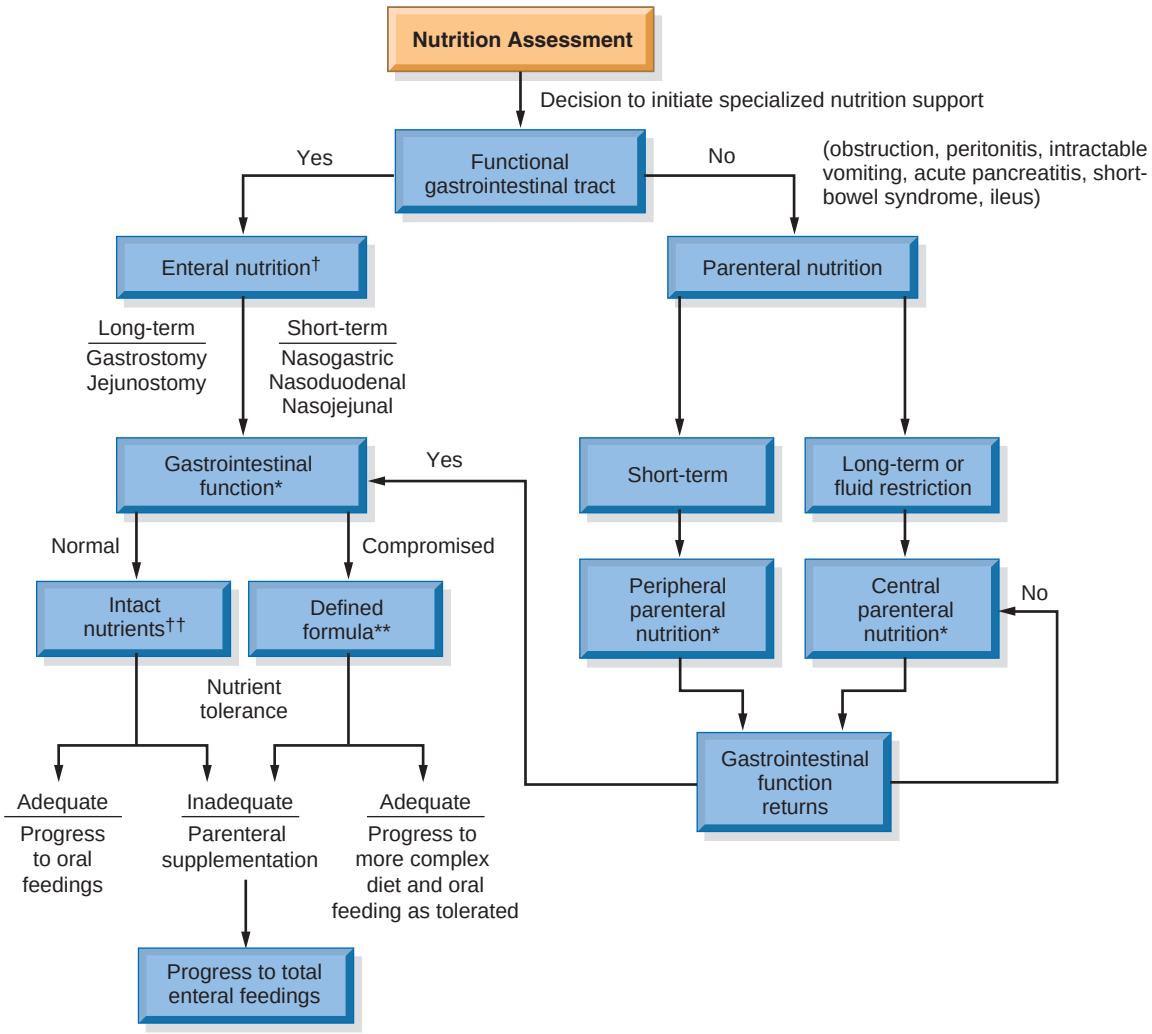


FIGURE 40-1 ▲ Route of administration of specialized nutrition support. *Formulation of enteral and parenteral solutions should be made considering organ function (eg, cardiac, renal, respiratory, hepatic). †Feeding may be more appropriate distal to the pylorus if the patient is at increased aspiration risk. ‡Elemental low-/high-fat content, lactose-free, fiber-rich, and modular formulas should be provided according to the patient's gastrointestinal tolerance. ††Polymeric, complete formulas, or pureed diets are appropriate. (Adapted from ASPEN Clinical Pathways and Algorithms for Delivery of Parenteral and Enteral Nutrition Support in Adults. Jacobs D (ed): Section II: Nutrition care process. J Parenter Enteral Nutr 26(1 Suppl):85A, 2002.)

also essential to preserve its function as a barrier. With atrophy, there is a loss of the tight junctions between enterocytes, resulting in increased mucosal permeability and decreased barrier function. This decreased barrier function can allow resident GI bacteria and endotoxins to enter the systemic circulation. This process, called bacterial translocation, can trigger immune and inflammatory responses that can lead to infection, sepsis, and multisystem organ failure. In addition to its trophic effects on the GI tract, enteral nutrition is associated with enhanced utilization of nutrients, decreased infectious complications, ease and safety of delivery, and lower cost.

Enteral nutrition is considered when the patient cannot or should not eat, intake is insufficient or unreliable, the patient has a functional GI tract, and access can be safely achieved. Mechanical obstruction is the only absolute contraindication to enteral feedings. In the setting of hemodynamic compromise (ie, the patient requires significant hemodynamic support including high-dose catecholamine agents, alone or in combination with large-volume fluid or blood product administration to maintain cellular perfusion), enteral nutrition should be withheld until the patient is fully resuscitated and/or stable.[5] Accordingly, each patient situation should be evaluated individually.

Enteral nutrition can be delivered through feeding tubes placed into the stomach or the small intestine. The expected duration of nutritional support, patient's overall condition, risk for aspiration, function of the GI tract, and placement technique should all be considered when deciding on which type of feeding tube to place. For most patient populations, nasoenteric tubes are widely used.

Nasoenteral Feeding and Feeding Tubes

A nasoenteric tube is indicated for short-term use in hospitalized patients, usually less than 4 to 6 weeks. Nasoenteral tubes are inserted through the nose and advanced through the

BOX 40–2 CONSIDERATIONS FOR THE OLDER PATIENT

Nutritional Requirements

- The risk for malnutrition increases as functional abilities decrease.
- The caloric needs of the elderly are generally less, secondary to decreased metabolism.
- Although protein requirements remain the same, it is important to monitor renal function.
- There is decreased ability to tolerate glucose loads.
- Atrophic gastritis occurs frequently in the elderly, which can result in decreased gastric acid secretion. The resultant achlorhydria or hypochlorhydria can lead to bacterial overgrowth and altered absorption of iron, vitamin B_{12}, folate, calcium, vitamin K, and zinc.
- Lactose intolerance increases with age; this intolerance to dairy products can contribute to osteopenia.
- Vitamin D deficiency in the elderly can be due to decreased dietary intake, decreased synthesis, or decreased exposure to sunlight.
- The elderly have less ability to regulate fluid balance, which places them at an increased risk for dehydration or overhydration.
- Encourage increased dietary fiber, fluids, and exercise to reduce the incidence of constipation.
- Decreased GI motility, exocrine function, and digestion or absorption may occur in the elderly.
- Physical changes in the jaw, including poor dentition or poorly fitting dentures, may interfere with mastication and adequate food intake.
- Swallowing may be more difficult because of decreased esophageal motility and decreased saliva production.
- Multiple medications or concomitant disease may contribute to anorexia or diminished sense of taste.

esophagus into the stomach (nasogastric tube), duodenum (nasoduodenal tube), or jejunum (nasojejunal tube). The tube is identified by the distal location of its tip. Most nasoenteric tubes are soft, flexible, small-bore polyurethane or silicone tubes that are 8 to 14 French in diameter and 20 to 60 inches in length, have markers to aid measurement, and are radiopaque to allow for radiographic confirmation of placement. The shorter lengths are used for nasogastric feedings, and the longer for nasoduodenal or nasojejunal feedings. As a general rule, the smallest-diameter tube of appropriate length is preferred because the smaller diameter has been associated with fewer complications and increased patient comfort. Small-diameter tubes may help prevent reflux and lessen the risk for aspiration because the small diameter reduces compromise of the lower esophageal sphincter. See Evidence-Based Practice Highlight 40-1 for information about the prevention of aspiration. In addition, small-diameter tubes cause less inhibition of swallowing, which is more comfortable for patients. Tubes made of polyvinyl chloride are less desirable because, over time, they can stiffen in the presence of acid, which can lead to patient discomfort and increased complications, such as tube perforation. Any nasally placed tube can cause sinusitis, erosion of the nasal septum or esophagus, otitis, vocal cord paralysis, epistaxis, or distal esophageal strictures, which may limit long-term use. Small, soft-bore tubes are less likely to cause these complications.

Most nasoenteric tubes have multiple ports staggered along their sides and tip, which minimize clogging and maximize flow. Many devices also have weighted tips and a stylet, which stiffens the tube to assist in placement. Another common feature of many nasoenteric tubes is a Y-port at the proximal tip, which allows for the administration of medications and irrigation without interrupting tube feeding.

NASOGASTRIC TUBES. Gastric feedings through a nasogastric tube are appropriate for patients who have intact gag and cough reflexes and adequate gastric emptying. Nasogastric tubes usually range from 8 to 12 French in diameter and 30 to 36 inches in length. Small-caliber nasogastric tubes are used solely for feeding, whereas large-caliber tubes can be used to decompress the stomach, monitor gastric pH, and deliver medications and feedings. Large-caliber nasogastric tubes are usually made of stiffer material and are often less comfortable for patients, possibly triggering self-extubation. These tubes are usually used to decompress and drain the stomach temporarily and are therefore typically for short-term use.

Advantages to gastric feeding include the ease of placement, the ease of checking residuals, and patient tolerability during enteral infusions. However, patients with nasogastric tubes are at the greatest risk for aspiration, especially when they are unconscious, mechanically ventilated, or otherwise unable to protect their airway. In a conscious patient, the mere physical appearance of the tube and associated discomfort may limit the clinical use of nasogastric tubes.

NASODUODENAL TUBES AND NASOJEJUNAL TUBES. Nasoduodenal tubes and nasojejunal tubes are thought to be better suited for long-term use than nasogastric tubes. Nasoduodenal tubes and nasojejunal tubes are advanced through the stomach, past the pylorus, and into the small intestine, usually in the third portion of the duodenum beyond the ligament of Treitz. In theory, the pyloric sphincter provides a barrier that reduces the risk for aspiration or regurgitation.

Transpyloric feeding can be given without regard to gastric emptying, providing an additional advantage over intragastric feeding. Candidates for transpyloric feedings include critically ill patients with a prior history of gastric aspiration, patients at risk for aspiration (such as ventilated patients), those with gastroparesis, those with gastric outlet obstruction, and patients with neurological conditions who are unable to protect their airway.

A common misconception is that enteral feedings should not be started if bowel sounds are absent. Bowel sounds are an indication of GI motility, not of absorption, and the presence or absent of bowel sounds or flatus is not required before the initiation of enteral feeding.[5,7] After injury and postoperatively, bowel sounds may not be detected for 3 to 5 days owing to gastric atony. The small bowel motility is less commonly impaired than the stomach or the colon and retains its absorptive and digestive capabilities, making it possible to accept enteral feedings immediately after surgery or trauma.

Nasoduodenal tubes and nasojejunal tubes range from 8 to 16 French in diameter and 152 to 240 cm in length. The length and diameter make it more difficult to check feeding residual because the lumen is smaller and tends to collapse

EVIDENCE-BASED PRACTICE HIGHLIGHT 40-1
Prevention of Aspiration

Expected Practice
- Maintain head-of-bed elevation at an angle of 30 to 45 degrees, unless contraindicated. (Level B)
- Use sedatives as sparingly as feasible. (Level C)
- For tube-fed patients, assess placement of the feeding tube at 4-hour intervals. (Level C)
- For patients receiving gastric tube feedings, assess for GI intolerance to the feedings at 4-hour intervals. (Level C)
- For tube-fed patients, avoid bolus feedings in those at high risk for aspiration. (Level E)
- Consult with physician about obtaining a swallowing assessment before oral feedings are started for recently extubated patients who have experienced prolonged intubation. (Level C)
- Maintain endotracheal cuff pressures at an appropriate level, and ensure that secretions are cleared from above the cuff before it is deflated. (Level B)

Supporting Evidence

Head-of-Bed Elevation
There is evidence that a sustained supine position (zero-degree head-of-bed elevation) increases gastroesophageal reflux and the probability for aspiration; for example, using a radioactive-labeled formula, endobronchial counts were higher when patients were lying flat in bed (0 degree) compared to when they were in a semirecumbent (45-degree) position.[2] Thus, elevating the head of the bed to an angle of 30 to 45 degrees, unless contraindicated, is recommended for patients at high risk for aspiration pneumonia (eg, a patient receiving mechanical ventilation and/or one who has a feeding tube in place).[3,4] Although effectiveness of the reverse Trendelenberg position in minimizing aspiration has not been studied, it is likely to produce similar results to an elevated backrest position.[4]

Sedation
Sedation causes reduced cough and gag reflexes and can interfere with the patient's ability to handle oropharyngeal secretions and refluxed gastric contents; in addition, sedation may slow gastric emptying.[5,6] To reduce the risk for aspiration, it is prudent to use the smallest effective level of sedation.

Assess Feeding-Tube Placement at Regular Intervals
Expert panels recommend that correct feeding-tube placement be verified at regular intervals to minimize the risk for aspiration.[3,4,7] If feedings are administered at the wrong site (such as the esophagus, or even the stomach of a patient who requires small-bowel feedings), the risk for aspiration is increased.[8] It is not uncommon for feeding tubes to become malpositioned during routine use. For example, in a study of 201 critically ill patients, it was found that the distal tips of 24 of 116 feeding tubes originally positioned in the small bowel were displaced upward into the GI tract (23 into the stomach and 1 into the esophagus).[9]

Assess for Gastrointestinal Intolerance to Tube Feedings
Tube-fed patients who experience frequent regurgitation and aspiration of gastric contents are at increased risk for poor respiratory outcomes.[1] Guidelines developed jointly by the Society of Critical Care Medicine and the American Society for Parenteral and Enteral Nutrition recommend that patients be monitored for tolerance to enteral feedings by noting abdominal distention, complaints of abdominal pain, observing for passage of flatus and stool, and monitoring GRVs.[10] Because gastric distention predisposes to regurgitation, it is recommended that GRVs be measured every 4 hours in critically ill patients.[4]

Practice varies widely in regard to GRVs; however, 200 and 250 mL are frequently cited values for initial concern.[11,12] In a study of 206 critically ill patients, two or more GRVs of at least 200 mL and one or more GRVs of at least 250 mL were found significantly more often in patients who experienced frequent aspiration.[11] Prokinetics are sometimes advocated to improve gastric emptying when GRVs exceed a stipulated value. Several sources recommend that feedings be stopped when GRVs exceed 500 mL.[4,7]

In a study of gastrointestinal (GI) symptoms in critically ill patients, investigators found that those with two or more simultaneous GI symptoms (such as high gastric aspirate volume, absent or abnormal bowel sounds, vomiting/regurgitation, diarrhea, bowel distension, and GI bleeding) were less likely to have successful enteral feedings than those with fewer than two GI symptoms (84% vs. 12.2%, respectively, $p < 0.001$).[13] Although bedside assessments for GI function such as GRVs and abdominal girth are difficult to evaluate,[14–16] they are frequently used in combination to provide an estimate of GI tolerance to enteral feedings. Small bowel feeding with the tube's ports situated at or below the Ligament of Treitz is strongly recommended for patients with persistent intolerance to gastric feedings and documented aspiration.[7]

Avoid Bolus Tube Feedings in Patients at High Risk for Aspiration
An expert panel has concluded that no recommendation can be made regarding the best type of formula delivery method (continuous or intermittent).[3] Also, no guidelines exist for bolus feedings. On the basis of logic, however, administering an entire 4-hour volume of formula over a period of a few minutes is more likely to predispose to regurgitation of gastric contents than is the steady administration of the same volume over a period of 4 hours.

Continuous feedings are used in most critical care units. Supportive of this action is a small study of neurologically impaired adult patients; aspiration was observed more frequently in those with intermittent feedings (3 of 17) than in those who received continuous feedings (1 of 17).[17] It is possible that the bolus method of feeding may decrease the lower esophageal pressure and thus predispose patients to reflux and aspiration.[18] Other researchers reported that adult burn patients who received continuous tube feedings had less stool frequency and less time required to reach nutritional goals than did intermittently fed patients.[19]

Swallowing Assessment Before Oral Feedings for Recently Extubated Patients
Tracheal intubation interferes with overall swallowing physiology.[20] Thus, it is reasonable to expect some degree of swallowing impairment when patients are initially extubated. A systematic literature review found that recently extubated patients were at increased risk for swallowing difficulties; more than 20% of the patients in many of the reviewed studies experienced dysphagia.[21]

Management of Endotracheal Tubes
A persistent low cuff pressure (<20 cm H_2O) predisposes patients to pneumonia, presumably by predisposing to aspiration of oropharyngeal secretions and/or refluxed gastric contents.[22] To minimize aspiration of secretions pooled above the endotracheal tube's cuff, hypopharyngeal suctioning should be performed before deflating the cuff.[3]

AACN Levels of Evidence

Level A Metaanalysis of quantitative studies or metasynthesis of qualitative studies with results that consistently support a specific action, intervention, or treatment

Level B Well-designed, controlled studies with results that consistently support a specific action, intervention, or treatment

(continued on page 897)

EVIDENCE-BASED PRACTICE HIGHLIGHT 40-1 (continued)

Prevention of Aspiration

Level C Qualitative studies, descriptive or correlational studies, integrative reviews, systematic reviews, or randomized controlled trials with inconsistent results
Level D Peer-reviewed professional and organizational standards with the support of clinical study recommendations
Level E Multiple case reports, theory-based evidence from expert opinions, or peer-reviewed professional organizational standards without clinical studies to support recommendations
Level M Manufacturer's recommendations only

Excerpted from American Association of Critical-Care Nurses Practice Alert. Available online at http://aacn.org. All references cited in this alert are available with the associated resources related to this chapter. Visit: http://thepoint.lww.com

on itself when aspirated. In addition, clogging of medications is more common than with nasogastric tubes. The primary disadvantage associated with nasoduodenal and nasojejunal tubes relates to the difficulty in initially placing the tubing tip past the pyloric sphincter.

Placing Nasoenteric Tubes

In most ICUs, skilled nurses or physicians routinely place nasoenteric tubes. Before placing a feeding tube, institutional policy and protocol should be reviewed because nasoenteric tube placement has many potential complications. Patients with a decreased level of consciousness, poor cough or gag reflex, or an inability or unwillingness to cooperate are at increased risk for pulmonary intubation. When a patient cannot cooperate or cough when the tube enters the bronchial tree, extra precautions must be taken to ensure proper placement. Feeding tubes placed in the bronchial tree can cause pulmonary hemorrhage or pneumothorax. A cuffed endotracheal tube (ETT) does not preclude accidental pulmonary intubation. Nasoenteric tubes can also be accidentally placed in the esophagus or, in patients with basilar skull fractures, in the intracranial space.

Nasogastric tube placement is usually easier than nasoduodenal or nasojejunal tube placement. When placing a nasoenteric feeding tube in the stomach, the nurse determines the length of tube insertion by measuring the distance from the tip of the nose, to the earlobe, to the tip of the xiphoid process. Before insertion, the nurse considers using a topical anesthetic or water-soluble lubricant to assist in placement. After placing the patient's bed in a high Fowler's position, the nurse slightly flexes the patient's head (if not clinically contraindicated) and passes the lubricated tip through the nares into the nasopharynx. While advancing the tube, the nurse asks the patient to swallow repeatedly. Having the patient sip water through a straw may also assist in tube placement (if not clinically contraindicated). Rotating the tube as it is advanced may also ease advancement.

When attempting to pass the nasoenteric tube tip past the pylorus, the nurse follows the same procedure described previously and then turns the patient to the right lateral decubitus position with the head of the bed at a 30- to 45-degree angle to take advantage of gravity and peristalsis. Nasoduodenal and nasojejunal tubes depend on gastric motility to carry the tip through the pylorus, but they have a tendency to coil in the stomach. Some nasoduodenal tubes and nasojejunal tubes have weights to aid in passage through the pylorus; however, the utility of the weighted tip is dubious. A promotility agent, such as metoclopramide or erythromycin, may be ordered before insertion because such a medication increases upper GI motility while relaxing the pylorus. Air insufflation, the process of inserting large amounts of air into the stomach, may also be helpful by distending the stomach and facilitating tube passage though the pylorus. If attempts to pass a nasoduodenal or nasojejunal tube are not successful within 24 hours, endoscopic or radiological assistance should be sought to advance the tip of the tube.

Before initiating tube feeding, proper tube placement must be confirmed by an abdominal radiograph. Feeding tubes placed surgically, by endoscopy, or under fluoroscopy do not require radiographic confirmation of placement. The external length of the tube is documented after placement is confirmed. The nurse marks the tube with tape or indelible ink at the point it enters the nares, rechecks tube placement before initiating intermittent feedings or medication administration and at least once each shift, and monitors tube placement during continuous tube feeding according to the institution's policy.

Auscultation, aspiration and inspection of aspirate, and pH testing have been used to monitor tube placement after initial placement is confirmed by an abdominal radiograph with varying degrees of accuracy. No one method is infallible, so using a combination of these methods is advised. Abdominal radiograph remains the gold standard. Injecting air into the tube and auscultation of the gastric bubble, although commonly used, is not an accurate method to verify initial tube placement. An air bubble sound can be transmitted to the epigastrium when the tube is in the esophagus. Although auscultation of insufflated air is not a reliable method to confirm initial feeding tube placement, it may still provide useful information. If no resistance is met, the tube is unlikely to be kinked, and if the patient immediately burps back air, the tip of the tube is probably in the esophagus.

Aspiration and inspection of the aspirate may help differentiate between gastric and intestinal placement, but not between intestinal and pulmonary placement. Fluid aspirated from the stomach is usually green, tan, brown, or bloody.[8] Small intestinal aspirate is usually golden yellow, clear, or bile colored and is often thicker than gastric aspirate.[8] Pulmonary fluid is usually tan, white, clear, or pale yellow and can closely mimic gastric or intestinal aspirates.[8] However, it is necessary to keep in mind that the diameter of small intestinal tubes may not allow withdrawal to check aspirate.

Measuring the pH of fluid aspirated from the feeding tube is another method of monitoring tube placement. The pH of esophageal secretions is usually 6.0 to 7.0, gastric aspirates 1.0 to 4.0, and intestinal contents 6.0 to 7.0.[8] However, the pH of gastric aspirate can be elevated with the infusion of enteral formulas, the use of acid-modifying medications, and the presence of bile reflux. The pH of both small intestinal aspirate and pulmonary fluid is usually greater than 6.0; therefore, if the pH of the aspirate is greater than 4.0, tube position cannot be determined based on pH alone.[8] For optimal results with pH testing, nothing that may alter the pH should be instilled in the tube for 60 minutes.

Capnometry and capnography detect carbon dioxide; these noninvasive monitoring techniques are used to monitor and evaluate respiratory function and ventilation. Observation of the presence or absence of a waveform is used to evaluate for accidental pulmonary placement of feeding tubes.

Suctioning and patient movement or coughing may potentially dislodge a feeding tube. If at any time tube location is in question, the nurse holds the tube feeding and requests an order for an abdominal radiograph to confirm placement. See Evidence-Based Highlight 40-2 for more information.

Securing Nasoenteric Tubes

Before securing any feeding tube, the nurse cleans the skin with alcohol to remove oils and dirt and considers applying a skin protectant to maintain skin integrity. Nasoenteric tubes should be secured in a way that avoids irritation or pressure on the nares, thereby preventing necrosis. The nurse allows the tube to hang straight from the nares and secures it to the bridge of the nose or the cheek with tape (or one of the many commercially available devices). For agitated or uncooperative patients, the nurse considers soft wrist restraints or mitts to avoid accidental self-extubation (refer to your institution's policy and procedure regarding the use of restraints). It is necessary to inspect the skin and nostrils every 4 to 8 hours for signs and symptoms of irritation, erythema, or skin breakdown. Patient comfort can be maximized by providing frequent mouth care and moistening of the nares.

Enterostomal Feeding and Feeding Tubes

If therapy is expected to last a month or more, a more permanent enterostomal device can be inserted through the abdomen into the stomach (gastrostomy) or jejunum (jejunostomy)[8] (Fig. 40-2). Enterostomal feeding tubes are also indicated when the nasal route is contraindicated and when the patient's swallowing is impaired or the oropharynx, larynx, or esophagus is obstructed. Enterostomal tubes are 18 to 28 French in diameter, made of silicone and polyurethane, and very durable.

GASTROSTOMY TUBES. Gastrostomy tubes have an internal retention bolster to prevent accidental dislodgment.

EVIDENCE-BASED PRACTICE HIGHLIGHT 40-2

Verification of Feeding Tube Placement (Blindly Inserted)

△ Expected Practice

- Use a variety of bedside methods to predict tube location *during* the insertion procedure:
  - Observe for signs of respiratory distress.
  - Use capnography if available.
  - Measure pH of aspirate from tube if pH strips are available.
  - Observe visual characteristics of aspirate from the tube.
  - Recognize that asucultatory (air bolus) and water bubbling methods are unreliable. (Level B)
- Obtain radiographic confirmation of correct placement of any blindly inserted tube prior to its initial use for feedings or medication administration.
  - The radiograph should visualize the entire course of the feeding tube in the GI tract and should be read by a radiologist to avoid errors in interpretation. Mark and document the tube's exit site from the nose or mouth immediately after radiographic confirmation of correct tube placement. (Level A)
- Check tube location at 4-hour intervals after feedings are started:
  - Observe four a change in length of external portion of the feeding tube (as determined by movement of the marked portion of the tube.
  - Review routine chest and abdominal x-ray reports to look for notations about tube location.
  - Observe changes in volume of aspirate from feeding tube.
  - If pH strips are available, measure pH of feeding tube aspirates if feedings are interrupted for more than a few hours.
  - Observe the appearance of feeding tube aspirates if feedings are interrupted for more than a few hours.
  - Obtain an x-ray to confirm tube position of there is doubt about the tube's location. (Level B)

△ Supporting Evidence

Bedside Methods to Determine Placement During Blind Tube Insertion

Signs of Respiratory Distress

- Symptoms such as coughing and dyspnea may occur when feeding tubes are inadvertently positioned in the airway, especially in patients with an impaired level of consciousness.[14–16] The occurrence of these signs should cause removal of the tube and a new insertion attempt.[17]

Capnography

- A carbon dioxide detector is helpful but is not sufficiently sensitive and specific to preclude the need for a confirmatory x-ray before initial use of a feeding tube.[22,23] In addition, a concurrently used CO_2 sensor failed to detect two of the four malpositioned tubes.[23] Also, a carbon dioxide sensor cannot determine where a feeding tube's tip ends in the GI tract (esophagus, stomach, or small bowel).[1]

pH and Appearance of an Aspirate

- Fasting gastric pH is usually 5 or less, even in patients receiving gastric-acid inhibitors.[17,24,25] Respiratory secretions typically have a pH greater than 6.[15,26] However, because gastric fluid occasionally has a high pH, the pH method is not sufficiently reliable to rule out the need for an x-ray to distinguish between gastric and respiratory tube placement.
- Small bowel secretions typically have higher pH values (≥6) than gastric juice; thus, observing for pH changes is useful in determining when a feeding tube has advanced from the stomach into the small bowel.[24,25,27] Using this method, it is often possible to limit the needed number of confirmatory x-rays to one.

(continued on page 899)

EVIDENCE-BASED PRACTICE HIGHLIGHT 40-2 (continued)

Verification of Feeding Tube Placement (Blindly Inserted)

- The pH method has no benefit in detecting placement of a feeding tube in the esophagus. Fluid withdrawn from the esophagus can be swallowed alkaline saliva or refluxed acid gastric juice.[28]
- In summary, while the pH method is helpful, it is not sufficiently accurate to eliminate the need for a confirmatory x-ray prior to first-time use of a feeding tube.
- Aspirate appearance is not sufficient to eliminate the need for a confirmatory radiograph prior to first-time use of a feeding tube; there is not confusion in differentiating between gastric and respiratory secretions.[6,15,16,30–36,37]

Listening Over Epigastrium for Air Insufflated Through Tube

- The auscultatory method is not reliable in distinguishing between respiratory and gastric placement or between gastric and small bowel placement.
- There are numerous anecdotal reports of blindly inserted tube entering the respiratory tract undetected by the auscultatory method, causing clinicians to assume that the tubes were correctly positioned in the stomach.[6,15,16,36,38–43] In a number of these cases, feedings or medications were administered and led to poor patient outcomes.[6,16,35,36,40,42–44]

Radiographic Confirmation

- A properly obtained and interpreted radiograph is recommended to confirm correct placement of any blindly inserted tube before its initial use for feedings or medication administration.[1,9,30,45,46,47] Because radiographs may be misinterpreted,[42,44,48] it is best to have a radiologist read the film to approve use of the tube for feedings.[1]
- Markings and documenting the tube's exit site at the time of radiographic confirmation of correct placement will be helpful in subsequent monitoring of the tube's location during its use for feedings.[49]

Checking Tube Location at Regular Intervals After Feedings Are Started

Feeding tube dislocation during feedings is a frequent problem.[49–51] Most often, it occurs when the tube is partially pulled out during movement or by an agitated patient.

Observing for Change in External Tube Length

- Observing for a change in length of the external portion of the feeding tube (as determined by movement of the marked portion of the tube) may be helpful in detecting tube dislocation.

Reviewing Routine Chest and Abdominal X-Ray Reports

- Reviewing routine chest and abdominal x-ray reports to determine if the radiologist has referred to feeding tube location can be quite helpful.

Observing for Changes in Volume of Feeding Tube Aspirates

- Observing the volume of fluid withdrawn from a tube at 4-hour intervals during continuous feedings or prior to each intermittent feeding may be helpful.[49] A sharp increase in residual volume may indicate displacement of a small-bowel tube into the stomach.
- Consistent inability to withdraw more than a few drops of fluid from the feeding tube may signal upward displacement into the esophagus.[28]
- It is often difficult to withdraw fluid from small-bore feeding tubes.[52] To avoid this problem, a proven method[53] calls for injecting 20 to 30 mL boluses of air into the tube with a large syringe (30 to 60 mL) and then slowly applying negative pressure to the plunger to withdraw fluid; it may be necessary to repeat the procedure several times.

Testing pH of Feeding and Observe the Appearance of Tube Aspirate if Feedings Are Off for Several Hours

- While feedings should never be interrupted solely for the purpose of pH testing, or observing the appearance of feeding tube aspirates, they are sometimes interrupted in preparation for tests or procedures. If the latter occurs, pH testing may be useful in distinguishing between gastric and small-bowel tube positions.[26,54] The pH method is of minimal benefit during continuous feedings because enteral formula buffers the pH of gastric and small bowel positions.[37] As indicated above, fasting gastric juice is usually grassy-green or clear and colorless, while small bowel juice is often bile-stained.

Listening Over Epigastrium for Air Insufflated Through the Tube

- The auscultatory method cannot distinguish between esophageal, gastric, or small bowl tube placement.

Obtain an X-Ray to Determine Tube Location if in Doubt

- When multiple bedside methods suggest that tube displacement has occurred, it is prudent to consider obtaining and x-ray to determine tube location.

Excerpted from American Association of Critical-Care Nurses Practice Alert. Available online at http://aacn.org. All references cited in this alert are available with the associated resources related to this chapter. Visit: http://thepoint.lww.com

Gastrostomy tubes may be used temporarily or for permanent feeding. If a gastrostomy tube is intended for permanent feedings, it may need to be replaced as the tube material deteriorates over time. Gastrostomy tubes may also be used for chronic gastric decompression. A low-profile gastrostomy device (LPGD), often referred to as a button, may be used to replace gastrostomy tubes in a mature gastrostomy tract, usually 3 to 6 months after initial placement or as an initial placement. LPGDs are anchored in the stomach and protrude through the abdomen, flush with the skin. These devices require a special extension adapter to connect with the tube-feeding bag, to check for residuals, and to use for decompression. This adapter may then be removed after use. Some LPGDs are equipped with a one-way antireflux valve to prevent leakage of gastric contents onto the skin. These devices are usually well accepted because they are durable, unlikely to irritate the skin, and difficult to dislodge. With agitated or confused adults who have a tendency to pull on their tubes, these advantages may be a benefit.

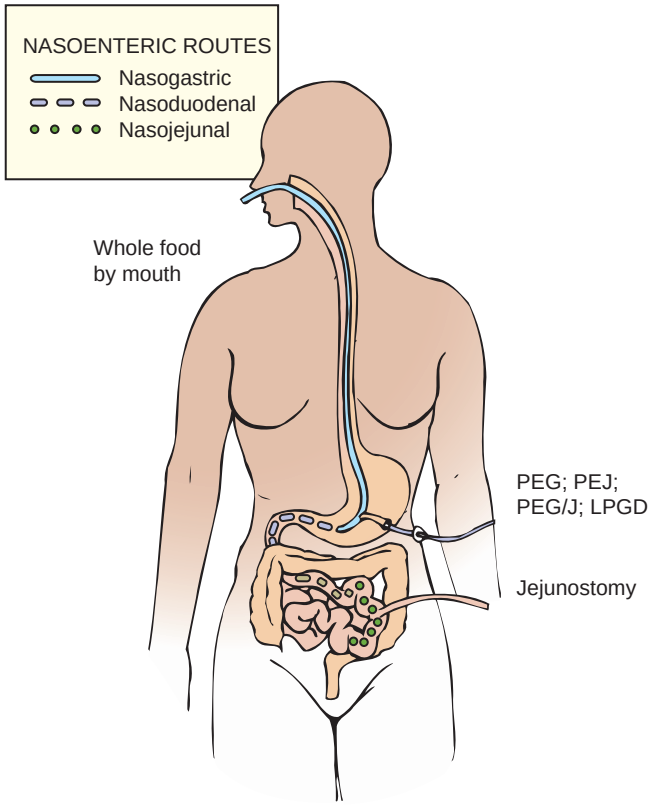


FIGURE 40-2 ▲ Possible routes for feeding. LPGD, low-profile gastrostomy device; PEG, percutaneous endoscopic gastrostomy; PEG/J, PEG modified with a jejunal extension tube; and PEJ, percutaneous endoscopic jejunostomy.

JEJUNOSTOMY TUBES. When gastric feedings are not possible or desired, jejunostomy tubes (J-tubes) are preferred for long-term feeding because they deliver enteral formula past the duodenum into the jejunum, decreasing pancreatic stimulation. J-tubes are indicated in patients who will benefit from jejunal feeding, particularly those with gastric disease, abnormal gastric emptying, upper GI obstruction or fistula, pancreatitis, or decreased gag reflex with significant risk for aspiration. J-tubes are contraindicated in patients with primary diseases of the small bowel (such as Crohn's disease) or radiation enteritis because of the increased risk for enterocutaneous fistula formation. A limitation of J-tubes is the potential for obstruction from the small diameter of the lumen.

Placing Enterostomal Tubes

Percutaneous endoscopic, open surgical, laparoscopic, and fluoroscopic techniques may be used to place a gastrostomy tube. J-tubes may be placed by percutaneous endoscopy or surgical methods. The patient's underlying disease and the physician's expertise need to be considered when selecting the appropriate placement technique.

PERCUTANEOUS ENDOSCOPIC GASTROSTOMY. Percutaneous endoscopic gastrostomy (PEG) has rapidly become the preferred method for placement of gastrostomy devices. A PEG may be performed at the bedside or in the endoscopy suite, using minimal sedation. Placement is through an abdominal incision using direct endoscopic visualization. Feeding may be administered as soon as 2 hours after placement.[8] Other advantages of PEG include increased comfort, decreased cost, and decreased recovery time. A candidate for a PEG must have an intact oropharynx and an esophagus free from obstruction. The only absolute contraindication to PEG is the inability to bring the gastric wall into apposition with the abdomen. Prior abdominal surgeries, ascites, hepatomegaly, and obesity may impede gastric transillumination and preclude placement of a PEG.

Complications of PEG are infrequent but include wound infection related to bacterial contamination by oral flora during insertion, necrotizing fasciitis, peritonitis, and aspiration. Pneumoperitoneum, a common finding after PEG placement, is not clinically significant unless accompanied by signs and symptoms of peritonitis. Prophylactic antibiotics are usually given 30 to 60 minutes before the procedure. Correct placement is then verified by endoscopy.

In patients with severe gastroesophageal reflux disease, gastroparesis, or increased risk for aspiration related to tube feeding, a PEG can be modified with a jejunal extension tube known as a PEG/J tube. The gastric lumen of a PEG/J tube is usually used for gastric decompression, and the jejunal lumen is used for the simultaneous delivery of enteral feeding. PEG/J tubes may decrease the risk for gastric aspiration; however, they do not necessarily provide the same protection against aspiration as jejunal tubes because the pylorus is compromised by the large catheter. The jejunal portion of a PEG/J tube may migrate back into the stomach and increase the risk for occlusion, gastric reflux, or aspiration. Both PEG and PEG/J tubes are held in place by internal and external retention devices. The internal device rests in the stomach, which prevents migration and leakage of gastric contents. The external retention device anchors the tube to the abdomen. These tubes have a high rate of mechanical dysfunction, which limits their long-term use.

SURGICAL GASTROSTOMY. Surgical gastrostomy tubes are inserted through an incision in the abdominal wall with the patient receiving general anesthesia. The stomach is usually sutured to the abdominal wall to create a permanent connection between the gastric and abdominal walls. Surgical placement of a gastrostomy is usually chosen if the surgeon wants to view the gastric anatomy clearly or as a secondary procedure during abdominal surgery. Disadvantages of surgical placement include the need for general anesthesia, increased recovery time, decreased comfort, and increased cost.

LAPAROSCOPIC GASTROSTOMY. A laparoscopically placed gastrostomy tube also requires general anesthesia or intravenous (IV) conscious sedation. Laparoscopic placement is usually reserved for patients with head, neck, or esophageal cancer. It is less invasive, is less painful, and usually involves fewer complications than a surgical gastrostomy.

FLUOROSCOPIC GASTROSTOMY. Direct percutaneous catheter insertion of a gastrostomy tube under fluoroscopy is indicated with high-grade pharyngeal or esophageal obstruction. Disadvantages to the use of fluoroscopy to place enterostomal devices include the inability to detect mucosal disease, the potential for prolonged exposure to radiation, the necessity of transport to the fluoroscopy suite, and increased cost.

Securing Enterostomal Tubes and Caring for the Enterostomy Site

Enterostomal tubes are secured to the abdominal wall to prevent dislodgment or migration of the tube, to avoid tension on the tubing, and to prevent the external retention device from digging into the skin. The length of the external tubing is documented to monitor for migration of the tubing.

To avoid tissue maceration, the insertion site is kept clean and dry by leaving it open to air (unless draining), and lifting or adjusting the tube is avoided for several days after the initial insertion. To avoid pulling the internal retention device taut against the gastric or intestinal mucosa, the amount of dressing between the device and the skin is limited. Any accumulated drainage may be cleaned with water. Serosanguineous drainage may be expected for 7 to 10 days after insertion. If no drainage is present, cleansing with soap and water is adequate. The skin around the insertion site and the retention device is assessed at least daily for skin breakdown, erythema, or drainage. The tissue usually heals within a month.

In-and-out play on the tubing is checked; it should be able to move one fourth inch to prevent erosion of gastric or abdominal tissue. If the anchor is too tight, the nurse should notify the physician immediately because this may indicate "buried bumper syndrome," a situation in which the retention device is imbedded in the tissue, thereby leading to mucosal or skin erosion. If a gastrostomy tube becomes accidentally dislodged, the nurse should notify the physician immediately so that the tube can be reinserted quickly before the tract closes.

Types and Delivery of Enteral Formulas

When selecting a tube feeding formula, nutrient requirements, the patient's clinical status, location of enteral access, GI function, cost, and duration must all be considered. Numerous tube feeding solutions are available for enteral nutrition, with many designed to assist in managing specific disease processes; however, no single formula is ideal for all patients. All contain proteins, carbohydrates, fats, vitamins, minerals, trace elements, and water. The difference lies in how these nutrients are structured and delivered. The dietary formula selected is based on the patient's ability to digest and absorb major nutrients, the total nutrient requirements, and fluid and electrolyte restrictions.

Polymeric solutions are the most commonly used formulas. These formulas are isotonic and can provide enough protein, carbohydrate, fat, vitamins, trace elements, and minerals to prevent nutritional deficiencies. They are considered nutritionally complete if given in enough volume to meet caloric needs. Standard formulas deliver 1 kcal/mL; some concentrated formulas may provide 2 kcal/mL.[9] The more concentrated formulas may be used for patients who require fluid restriction or in patients who have higher caloric requirements. All polymeric solutions contain intact proteins (most often meat, whey, milk, or soy proteins) that require normal pancreatic enzymes for digestion. Several disease-specific formulas are available.

Peptide (elemental or semielemental) formulas provide proteins as dipeptides, tripeptides, or free amino acids from hydrolysis of whey, milk, or soy proteins. Because peptides do not require pancreatic enzymes for digestion, elemental solutions are used when digestion is impaired, such as in pancreatic insufficiency, radiation enteritis, Crohn's disease, or short bowel syndrome secondary to surgical resection. Elemental solutions have no proven advantage in patients with normal gut function, are usually more expensive than polymeric formulas, and have an unpleasant taste.

Modular formulas contain individual nutrient components such as protein, carbohydrates, and fat that can be mixed or added to other formulas to individualize feedings to a patient's specific nutritional needs. The involvement of a dietitian is essential in the selection of these formulas.

Attention has recently focused on the role of enteral formulas that contain additional nutrients purported to enhance immune function. These formulas, referred to collectively as immunonutrition or immune-enhancing diets, have been reported to decrease infection rates, duration of mechanical ventilation, and length of hospitalization, but they have not been shown to affect mortality. Several nutrients that have attracted attention are glutamine, arginine, and omega-3 polyunsaturated fatty acids.

Glutamine is a nonessential amino acid that may become conditionally essential in critically ill adults.[9] Glutamine is an important fuel source for rapidly dividing cells, such as enterocytes, lymphocytes, and macrophages.[9] In addition, glutamine may improve immune function and reduce intestinal permeability.[9] Glutamine-enriched solutions should be considered in patients with burns and trauma.[5,7,9]

Another additive is arginine, which is also a nonessential amino acid that may become depleted in the critically ill. Arginine is a precursor of nitric oxide and is important in cell growth and proliferation, wound healing, and collagen synthesis.[9] Its role is controversial because increased nitric oxide production may increase tissue injury and trigger cardiovascular collapse in patients with sepsis or systemic inflammatory response syndrome.[5,10]

The omega-3 fatty acid is a precursor of prostaglandins, leukotrienes, and other inflammatory mediators. Use of an enteral formula with an antiinflammatory lipid profile fortified with omega-3 fatty acids or borage oil is recommended for patients with acute respiratory distress syndrome and severe acute lung injury.[5,7,10] These formulas have been shown to reduce the length of stay in the ICU, duration of mechanical ventilation, organ failure, and mortality when compared to standard formulas.[5]

When initiating enteral tube feedings, most clinicians recommend beginning with an isotonic formula at a slow rate, most often 20 to 30 mL/h, and increasing the rate incrementally every 8 to 12 hours until the goal rate is achieved. Dilution of formula may help tolerance but is not recommended because this may increase the time needed to meet the nutritional requirements.

Enteral feedings can be administered by bolus, gravity infusion, intermittent infusion, continuous infusion, or cyclic infusion. The tube tip location and tolerance generally dictate formula delivery. Gastric feedings are appropriate for patients who have intact gag and cough reflexes and adequate gastric emptying.

BOLUS FEEDINGS. Bolus feedings, considered the most natural method physiologically, are delivered by gravity by a large syringe in volumes as high as 400 mL over 5 to 15 minutes, three to five times a day.[8] The stomach is the

preferred site for bolus feedings. The stomach and pyloric sphincter regulate the outflow of feeding from the stomach. Bolus feedings allow for increased patient mobility because the patient is free from a mechanical device between feedings. Bolus feedings are usually initiated with 60 to 120 mL of full-strength formula every 8 to 12 hours until the goal is reached.[8] Unfortunately, as a result of high residuals, bolus feedings are usually not well tolerated and are often accompanied by nausea, bloating, cramping, diarrhea, or aspiration.

INTERMITTENT FEEDINGS. Intermittent feedings of 300 to 400 mL are administered by slow gravity drip four to six times a day over a period of 30 to 60 minutes. The stomach is the preferred site for intermittent infusion because of its capacity. Intermittent feedings are associated with a decreased risk for osmotic diarrhea. Advantages of intermittent feedings include freedom from dependence on a mechanical device and a power source, which can decrease cost and increase patient mobility.

CONTINUOUS FEEDINGS. If the tip of the nasoenteric tube is in the duodenum or jejunum, tube feedings must be delivered by infusion. Continuous infusions are administered over 24 hours with the aid of a feeding pump to ensure a constant flow rate. Continuous pump feedings are the preferred method for intestinal feeding because delivery that is too rapid may lead to "dumping syndrome," characterized by osmotic diarrhea, abdominal distention, cramps, hyperperistalsis, light-headedness, diaphoresis, and palpitations. When the tube is placed in the third portion of the duodenum, past the ligament of Treitz, continuous pump feedings are associated with decreased risk for aspiration. Initiate continuous feedings of full-strength formula at 10 to 40 mL/h advancing by 10 to 20 mL increments every 8 to 12 hours until goal is reached.[8] If the feeding is advanced slowly, the small bowel can usually tolerate feedings at a rate of 150 mL/h. The continuous method is best suited to the critically ill patient because it allows more time for nutrients to be absorbed in the intestine. Continuous infusion is often used in the ICU because there is decreased incidence of gastric distention and potential for aspiration. Continuous infusion tube feeding may also act prophylactically to prevent stress ulcers and metabolic complications. As with intermittent feedings, disadvantages include the dependence on a mechanical device and a power source.

CYCLIC FEEDINGS. Cyclic feedings are continuous feedings that deliver the total daily nutritional requirements in a shorter time frame, typically over 8 to 12 hours, to allow the patient freedom from 24-hour continuous feedings. Cyclic feedings of high density and high volume are typically given at night to allow hunger to develop during the day, but they may be given during the day if a patient has difficulty with regurgitation while lying supine. This schedule may assist the patient in progressing from enteral to oral consumption.

The ultimate goal is for patients to resume adequate oral intake. Enteral feeding may be discontinued when patients can drink enough liquid to maintain hydration and can eat two thirds of their nutritional requirements.

Complications of Enteral Nutrition

Although enteral nutrition is in general associated with fewer complications than parenteral nutrition, complications may still occur. These complications generally fall into GI, mechanical, metabolic, and infectious categories. Many of these complications can be prevented or treated by closely observing residuals and watching for signs and symptoms of gastric intolerance. See warnings in Evidence-Based Highlight 40-3.

GASTROINTESTINAL COMPLICATIONS. The patient's tolerance to enteral feeding depends on the rate of flow and the osmolality of the formula. Signs and symptoms of GI intolerance to enteral feeding include diarrhea, nausea, vomiting, abdominal

EVIDENCE-BASED PRACTICE HIGHLIGHT 40-3

Dye in Enteral Feeding

△ Expected Practice

- Dye should not be added to enteral feeding as a method for identifying aspiration of gastric contents.

△ Supporting Evidence

- Research has shown that dye in enteral feedings is often not visible in tracheal secretions following known aspiration events.[12,17] (Level B)
- A consensus statement from a multidisciplinary group of experts recommended that use of the dye method be discontinued since it lacks sensitivity for identifying aspiration of gastric contents.[3] (Level D)
- The addition of dye to enteral formula has been associated with several adverse events, including gastric bacterial colonization and diarrhea, systemic dye absorption, and death.[4,7,11,14] (Level A)
- The FDA issued a Public Health Advisory in 2003 based on reports of toxicity and death associated with dye in enteral feeding, although a direct causal relationship has not yet been definitively confirmed.[15] The majority of reported cases of toxicity and/or death occurred in patients with sepsis. (Level C)
- Use of glucose testing of tracheal aspirates is no longer recommended as a viable method to detect aspiration.3,18–20 (Level D, B)

AACN Levels of Evidence

Level A Meta-analysis of quantitative studies or metasynthesis of qualitative studies with results that consistently support a specific action, intervention, or treatment.
Level B Well-designed, controlled studies with results that consistently support a specific action, intervention, or treatment.
Level C Qualitative studies, descriptive or correlational studies, integrative review, systematic reviews, or randomized controlled trials with inconsistent results.
Level D Peer-reviewed professional organizational standards with clinical studies to support recommendations.
Level E Multiple case reports, theory-based evidence from expert opinions, or peer-reviewed professional organizational standards without clinical studies to support recommendations.
Level M Manufacturer's recommendations only.

Excerpted from American Association of Critical-Care Nurses Practice Alert. Available online at http://aacn.org. All references cited in this alert are available with the associated resources related to this chapter. Visit: http://thepoint.lww.com

discomfort, distention, and high residual returns. Food normally passes through the stomach at a rate of 2 to 10 mL/min; however, gastric emptying is delayed or absent in many critically ill patients. Unlike the stomach, the small intestine cannot act as a reservoir. If large residuals are withdrawn through a nasoduodenal tube or nasojejunal tube, the tube may have moved back into the stomach, and placement should be confirmed with an abdominal radiograph.

High Residuals. The monitoring of gastric residual volumes (GRVs) is a routine practice based on the assumption that GRVs are useful in predicting the risk for aspiration and pneumonia. However, studies have not shown a consistent relationship between GRV and aspiration; high GRVs do not indicate aspiration, and low GRVs do not preclude aspiration.[11] Additionally, there is no consensus among experts about what constitutes a high GRV, with reports varying from 100 to 500 mL. High GRVs have been thought to result from impaired gastric emptying caused by intolerance to enteral feedings; GRV is an imprecise measure of gastric emptying and may not take into account the volume of gastric and salivary secretions. In addition, it is difficult to determine whether gastric contents have been completely removed. Many clinicians stop tube feeding inappropriately, based on a single GRV of less than 400 to 500 mL. Although a GRV between 250 and 500 mL should raise suspicion of intolerance and cause the implementation of measures to reduce the risk of aspiration. One high value does not mean feeding failure, and automatic cessation of feeding can delay the patient's ability to meet his or her nutritional goals.[5,7] Tube feedings should not be stopped for GRVs less than 500 mL in the absence of other signs of intolerance.[5] The nurse must be sure to evaluate the clinical status of the patient before stopping tube feeding solely on the basis of one high GRV; the key is to monitor trends.[7,12] Residuals should be checked every 4 hours during the first 48 hours of gastric feeding, every 4 hours during continuous feedings and before initiating intermittent feedings.[1,8,11]

Tube feeding should be withheld if a patient demonstrates overt signs of regurgitation, vomiting, or aspiration. A common intervention involves holding the feeding for 1 to 2 hours and rechecking GRV every 1 to 2 hours until the GRV is less than 200 to 250 mL from a nasogastric tube or less than 100 mL from a gastrostomy tube, at which point feedings can be resumed. If GVR is greater than 250 mL after two measurements, providing a promotility agent should be considered.[1,8] This allows time for normal gastric emptying and reduces the risk for aspiration. It is necessary to remember that high infusion rates result in higher GRVs and to be aware of the institution's policy and protocol regarding high GRVs.

Nausea, Vomiting, and Bloating. Nausea, vomiting, and bloating are commonly associated with enteral feedings. Medications, rapid infusion rate, or improper tube placement may cause both nausea and vomiting. Nausea, vomiting, and bloating are most likely to occur when gastric emptying is delayed. The nurse carefully assesses medications that may contribute to these symptoms, and the medications should be eliminated, if possible. A change of formula, reduction in delivery rate, or addition of a prokinetic medication may also help.

Diarrhea. Diarrhea is the most common complication of enteral feedings; however, it is important to consider other etiologies before assuming that enteral feedings are the cause of diarrhea. Diarrhea in a patient receiving enteral feeding may result from the use of antibiotics or other diarrhea-inducing medications; altered bacterial flora; formula composition; intolerance to lactose, fat, or osmolality; a rate of infusion that is too high; hypoalbuminemia; or enteral formula contamination.

The liquid form of many medications may contain hypertonic sorbitol, which can have laxative effects. Antibiotics, antacids, magnesium, and prokinetic medications can also contribute to diarrhea. Antibiotics can contribute to diarrhea by causing overgrowth of *Clostridium difficile*. To assess for *C. difficile* infection, a stool sample is assessed for *C. difficile* toxin. Treatment options include antibiotic therapy with oral metronidazole, vancomycin, or cholestyramine (a bile acid sequestrant that binds the toxin). A patient who has received antibiotics should not receive antidiarrheals until *C. difficile* infection has been ruled out because diarrhea helps eliminate the toxin from the intestinal mucosa.

Bacterial overgrowth may cause diarrhea. Reduced gastric and small bowel motility may lead to small intestinal overgrowth, which can alter intestinal microflora. Acid suppression may also permit bacterial overgrowth because bacteria can colonize the GI tract when the gastric pH is greater than 6.0.

The infusion of enteral feedings too rapidly may cause diarrhea. Intolerance of lactose, fat, or osmolality may also lead to diarrhea. Reducing the infusion rate, changing to a peptide-based formula that is easier to digest, and giving an absorbing product, such as psyllium fiber (Metamucil), may help. The use of a fiber-containing formula may be helpful in bulking stools and correction of the diarrhea.

The composition of enteral formulas makes them an ideal medium for bacterial growth, which may result in diarrhea. Many organisms have been associated with enteral feedings, including coagulase-negative staphylococci, *C. difficile*, and Gram-negative bacilli, such as *Serratia*, *Klebsiella*, *Enterobacter*, *Proteus*, and *Pseudomonas* species. Bacterial contamination from the surface of the formula container, or even water added when the preparations are mixed or poured, may lead to diarrhea. Contamination of enteral feedings may also occur as a result of retrograde movement of bacteria from the patient's own GI tract. In addition, the aspiration of gastric residuals and the removal of guide wires from the feeding tube may contribute to contamination.

Contamination of the feeding administration set may also cause diarrhea. Breaks in the system should be minimized, and the use of a closed, prefilled, ready-to-hang solution should be considered to minimize contamination from pathogens. To prevent bacterial contamination, formulas that are reconstituted in advance should be immediately refrigerated and discarded within 24 hours if not used.[8] Formulas that are exposed to room temperature for longer than 4 hours should be discarded.[8] Sterile, premixed formulas should hang no longer than 8 hours.[8] The nurse checks the expiration date of the formula and discards the formula if it has expired. Closed system administration sets should be changed every 24 to 48 hours, and open system administration sets should be changed every 24 hours.[8] All practitioners should use good hand washing technique when handling equipment and wear gloves when handling feeding systems.

In patients receiving enteral feedings, a hyperosmotic formula may also contribute to diarrhea. If diarrhea decreases when the feedings are withheld, the formula may be the cause. After consultation with the dietitian, the nurse may consider changing the formula. Also, the nurse collects a stool sample to evaluate for an osmotic gap; this may help identify osmotic diarrhea.

Hypoalbuminemia may predispose patients to diarrhea by decreasing the osmotic pressure gradient. This decrease may lead to bowel edema and malabsorption. Any formula that is not absorbed may contribute to the diarrhea. Prealbumin level is monitored because it is a more reliable indicator of current nutritional status than serum albumin.

Constipation. Constipation associated with enteral feedings may be related to poor hydration, lack of fiber, bed rest, impaction, obstruction, and narcotics. Adequate hydration is ensured, and adding a stool softener should be considered along with minimizing narcotic administration, encouraging ambulation, and considering the addition of fiber to relieve constipation.

MECHANICAL COMPLICATIONS. Mechanical complications occur when the feeding tube becomes dislodged, occluded, or malpositioned.

Tube Dislodgment. Tube dislodgment by patients or staff accounts for most tube removals. Soft restraints or hand mitts should be considered for agitated patients to prevent accidental self-extubation (refer to your institution's policy regarding the use of restraints).

Tube Clogging. Precipitation of medications, clogging of pill fragments, or coagulation of formula may cause obstruction of any feeding tube, delaying the administration of nutrients and medications. To avoid clogging, the nurse flushes enteral feeding tubes every 4 hours during continuous feedings, before and after medication administration, after checking residuals, and when turning off feedings.[8] For flushing nasoenteric tubes, the nurse always uses a large 30- to 60-mL syringe to avoid rupturing the tube with excessive pressure and irrigates with 15 to 30 mL of sterile water.[8,13] The nurse frequently checks the enteral solution container for precipitation. Crushed tablets may leave a residue that blocks the tube. To prevent clogging, the nurse administers liquid medications when available. Flushing the tube before and after each medication, administration also helps avoid incompatibilities between medications and feedings and reduces the incidence of clogging. Medication should not be directly added to enteral formulas.[8,13]

An obstruction may exist if the formula does not flow by gravity, flushing an aspirate from the tube is not possible, or the occlusion alarm of the feeding pump sounds repeatedly. If an occlusion is suspected, the nurse uses a large piston syringe to flush the tube with warm water, using a gentle push–pull motion. Although many solutions have been proposed to assist in clearing an obstructed feeding tube, they offer no demonstrable benefit over tap water. A stylet should never be used to unclog a tube because of the risk for rupturing the feeding tube and perforating the esophagus, stomach, or small intestine. Recent studies show that pancreatic enzymes have been effective in unclogging a tube when water is unsuccessful, as long as the enzymes are activated before instillation.[8]

METABOLIC COMPLICATIONS. Multiple metabolic complications can accompany enteral nutrition. Fluid and electrolyte imbalance may occur because of fluid excess, fluid depletion by GI or renal losses, wound drainage, diuresis, fever, or inadequate free water intake. If dehydration is due to inadequate fluid intake, the nurse may need to give the patient extra fluid by bolus or by automatic flush using specialized feeding pumps. The average patient with good renal function needs 30 to 35 mL/kg of free water per day, if not medically contraindicated. Conversely, if cardiac or hepatic function is impaired, overhydration from enteral feedings may occur. The determination of the patient's baseline fluid requirements and accurate measurement of intake and output can help maintain fluid balance. The nurse should keep in mind that many of the patients seen in the ICU may be unable to convey feelings of thirst, secondary to intubation, or diminished levels of consciousness.

Hyperglycemia may occur if patients are being overfed, during hypermetabolic states, and as a result of steroid medications. Blood glucose is monitored during enteral therapy; a decrease in formula rate or concentration may help if hyperglycemia occurs. Bolus feeding may exacerbate hyperglycemia in patients with diabetes. If feedings are abruptly stopped, hypoglycemia is assessed, especially in patients receiving insulin.

INFECTIOUS COMPLICATIONS. Aspiration of enteral feeding resulting in hypoxia or pneumonia is a dreaded complication of enteral feeding. The incidence of aspiration of enteral formulas is as high as 50% to 75% in patients with ETTs. Loss of consciousness, mechanical ventilation, and many medications used in critically ill patients increase the risk for aspiration. To reduce this risk, the head of the bed should be maintained at a 30- to 45-degree angle.[5,8] If head of the bed elevation is medically contraindicated, a reverse Trendelenburg position is used unless medically contraindicated.[8] Intermittent or continuous feedings are used rather than rapid boluses because they allow the restoration of gastric pH, which can minimize gastric colonization. GRV is checked frequently, and signs of feeding intolerance are assessed. Feedings are discontinued at least 30 minutes before any procedure for which the patient must lie flat. If a procedure necessitates that the head of the bed be lowered, tube feedings may be withheld. The patient is returned to an elevated position, and the feedings are restarted promptly when the procedure is completed.[8] If a patient is intubated, secretions above the tube's cuff should be cleared before deflating the tube.

Pulmonary aspiration, although often subclinical, may be signaled by a low-grade fever, coughing, shortness of breath, rhonchi during or after enteral feeding infusions, and presence of a "sweet" formula odor emanating from tracheal or oral secretions during suctioning. The addition of Food, Drug, and Cosmetic (FD&C) blue dye No. 1 to enteral feeding formula to help visually identify aspirated feeding formula was a common practice for years. However, in 2003, the U.S. Food and Drug Administration (FDA) issued an FDA Public Health Advisory after several reports of toxicity, including bacterial colonization, diarrhea, systemic absorption, and death.[14] As a result, the American Association of Critical-Care Nurses Practice Alert advises that FD&C No. 1 dye should not be used in enteral feedings.[15] It is necessary that each nurse check his or her institution's policy and protocol regarding blue dye and its administration.

Checking tracheal suction fluid with a glucose strip and glucometer has been used to test for formula aspiration. Tracheobronchial secretions usually contain less than 5 mg/dL of glucose, so a reading greater than 20 to 25 mg/dL suggests aspiration, although there is some controversy concerning this procedure.

Parenteral (Intravenous) Nutrition

Parenteral nutrition is indicated when oral or enteral nutrition is not possible or when absorption or function of the GI tract is not sufficient (or is unreliable) to meet the nutritional needs of the patient. Before the inception of parenteral nutrition in the early 1960s, bowel rest was thought to be the cornerstone of treatment for many GI disorders. Today, except in cases of severe hemorrhagic pancreatitis, necrotizing enterocolitis, prolonged ileus, and distal bowel obstruction, some enteral nutrition is recommended to maintain gut integrity and function.[16] If a patient was healthy prior to critical illness with no evidence of protein–calorie malnutrition, use of parenteral nutrition should be reserved and initiated after the first 7 days of hospitalization.[5,7]

There are two types of parenteral (IV) nutrition: central and peripheral. Central parenteral nutrition, also known as total parenteral nutrition (TPN), is infused through a large central vein (Fig. 40-3). TPN has sometimes been referred to as "hyperalimentation" or "hyperal." These are not preferred terms because they imply that parenteral nutrition gives more nutrients than the patient may actually require. If TPN is expected to be needed for more than a few weeks, a more permanent device such a subcutaneously tunneled Hickman catheter or Port-a-Cath can be placed. Another central venous access device that can be used for long-term nutritional support is the peripherally inserted central catheter (PICC). A PICC is inserted peripherally into the basilic vein and advanced so that the tip of the catheter rests in the superior vena cava. Peripheral parenteral nutrition (PPN), unlike TPN, is infused into smaller, peripheral veins (eg, basilic vein) and is often used for short-term nutritional support (eg, 7 to 10 days) or as a supplement during transitional phases to enteral or oral nutrition (see Fig. 40-3). Because of the risks for phlebitis, concentrations of PPN formulas must not exceed 900 mOsm/L.[17] TPN differs from standard IV fluids in that all daily required nutrients are delivered to the patient in the form of macronutrients (carbohydrates, proteins, and fats) and micronutrients (electrolytes, vitamins, and trace minerals). Typically, the solution is infused at a constant rate over a 24-hour period to achieve maximal assimilation of the nutrients and to prevent hyperglycemia (or hypoglycemia). The aim of treatment is a continuous infusion that meets the caloric and nutritional requirements of the patient.

Critically ill patients often have issues with reliable IV access. TPN must be infused separately from other IV fluids, medications, and blood products because of the high risk for formula contamination and precipitation. Fortunately, the introduction of multiple-lumen catheters has greatly facilitated the care of patients who require multiple IV therapies by providing separate infusion ports and corresponding distal exit sites staggered along the catheter tubing. This separation prevents direct mixing of solutions before high-blood-volume dilution of the central veins. One port must be dedicated to exclusive use of TPN, whereas the remaining ports can be used for administering IV fluids and obtaining blood samples. When the central line is a single lumen, this lumen should be used exclusively for the infusion of TPN.

Composition of Parenteral Nutrition Formulas

TPN formulas typically contain three primary macronutrients: carbohydrates, lipids (fats), and amino acids (protein). This combination is called a mixed fuel source. When all three fuel sources are combined in one TPN bag, it is often referred to as a "3-in-1" admixture. To maintain strict sterility, the desired proportions of these nutrients are prepared under a laminar flow hood by a pharmacist. Because of differences in pharmacy equipment used to mix TPN, some facilities infuse lipids separately, usually in a glass bottle. Medications are not to be added to a TPN bag after it has been prepared by the pharmacist because of the risk for contamination

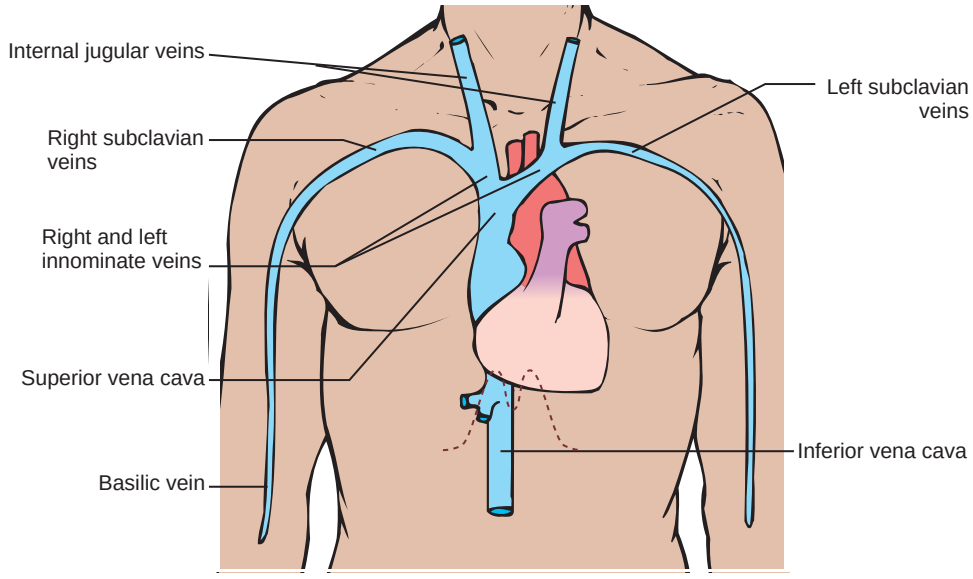


FIGURE 40-3 ▲ Venous anatomy for parenteral nutrition routes.

or precipitation of its contents. The current trend in TPN formulation is based on the specific needs of each patient; standard formulas are no longer widely prescribed.

CARBOHYDRATES. The primary source of energy in the body is carbohydrates (eg, dextrose). This macronutrient usually provides 40% to 60% of daily caloric requirements and is essential to central nervous system function. The most common and preferred source of carbohydrates is dextrose (D-glucose) because it is readily metabolized, stimulates the secretion of insulin, and is usually well tolerated in large quantities. Dextrose provides 3.4 kcal/g in IV form and contributes to most of the osmolality (or concentration) of the TPN solution. Initial TPN concentrations of dextrose may range from 50% to 70%, but final concentrations are diluted to approximately 25% to 30% after the addition of amino acids, lipid emulsions, and water.

Despite dilution, this concentration remains very high and requires delivery through a central venous catheter so that the higher blood volumes in the larger central veins are able to further dilute and disperse the solution. The superior vena cava is an excellent site for such delivery. Passage of a central venous catheter, by way of the subclavian vein into the superior vena cava, is the route of choice because it allows the patient the greatest freedom of movement without disturbing the insertion site. Jugular veins can also be used but may make it more difficult to keep dressings sterile, and these are not as comfortable for the patient because of limitations in neck movement. Regardless of the site, strict adherence to insertion and infection protocols must be followed, and radiologic verification of catheter tip placement is necessary before initial infusion. (Refer to your institution's central line care and infection control policies and procedures.)

The amount of dextrose prescribed in TPN is based on metabolic needs, which, once met, allows utilization of amino acids for protein synthesis rather than solely as an energy source. Adults require a minimum of 100 g/d of dextrose to perform vital metabolic activities; however, the maximal dose of dextrose varies based on an individual's needs, medical condition, and glucose tolerance. Recommendations suggest that dextrose must not exceed 7 g/kg/d.[18] One of the most common metabolic side effects of excessive dextrose concentrations is hyperglycemia, which often requires the use of insulin. Relatively tight blood glucose control is recommended to prevent complications associated with hyperglycemia. A protocol for moderately strict control of serum glucose between a range of 100 and 150 mg/dL may be appropriate.[5,7] In addition, excessive dextrose administration may put certain patients, such as those with pulmonary compromise, at risk for carbon dioxide retention and subsequent respiratory acidosis. Because one of the end products of dextrose metabolism is carbon dioxide, elevated levels may increase minute ventilation and hence the work of breathing. Overfeeding carbohydrates may make ventilator weaning difficult, if not impossible.

LIPIDS. Parenteral IV lipids, or fat emulsions, primarily contain long-chain linoleic and α-linolenic acids (essential fatty acids) from safflower and soybean vegetable oils. Lipid solutions also contain egg yolk phospholipids as emulsifiers, so it is important to check for a food allergy history before administration. Lipids provide a concentrated source of calories, 9 kcal/g, and they are important in maintaining connective tissue integrity and preventing fatty acid deficiency. Symptoms of fatty acid deficiency include rough, dry, scaly skin; nasolabial seborrhea; dull or dry hair, soft or brittle nails, poor wound healing, and diarrhea. Accordingly, patients should receive 2% to 4% of their daily energy requirements as linoleic acid and 0.25% to 0.5% of their daily energy requirements from α-linolenic acid.[18] The usual prescriptive dose of lipid emulsion infusion is approximately 1.0 to 1.3g/kg/d (not to exceed 2.5 g/kg/d) to supply up to 30% of the patient's caloric intake. Weekly administration of a 20% lipid emulsion (500 mL), however, is sufficient to prevent essential fatty acid deficiency in adults.[16,18]

Lipid emulsions are isotonic, and concentrations are available in 10%, 20%, and 30% solutions, providing 1.1, 2.0, and 2.9 kcal/mL, respectively.[17] The benefit of higher concentrations is that they provide a greater concentration of calories in less total fluid volume, an important consideration for many patients. In situations in which hyperglycemia has become problematic, dextrose solution concentrations and volumes may be reduced, and unless contraindicated, lipids concentrations and volumes can be increased. Lipids typically provide 15% to 30% of daily caloric intake; if delivery of lipids is higher than 30% of total caloric intake, vigilant monitoring for metabolic side effects is especially important. Baseline and weekly triglyceride trends monitor lipid tolerance. Elevated triglyceride levels exceeding 400 mg/dL suggest impaired lipid clearance and an increased risk for pancreatitis, so it is recommended that lipid emulsions be withheld until triglyceride levels return to normal.[19] Lipid concentrations may need to be adjusted for patients who may also be receiving additional lipids from sources other than TPN (eg, continuous infusion of propofol, a sedative delivered as a lipid emulsion). Lipid emulsions provide an excellent medium for bacterial growth, so increased manipulation and prolonged hang times are avoided. Adverse reactions to lipids include, but are not limited to, fever, chills, chest or back tightness, dyspnea, tachycardia, headache, nausea, and vomiting. If such reactions occur, the nurse stops the infusion immediately and reports the reaction to the physician and pharmacist. Long-term reactions to lipids include concerns over immune system suppression.[17] Before infusion, the nurse inspects lipid-containing TPN solutions for separation of the lipid solution, also known as cracking and coalescence. This loss of emulsion can be identified by yellow-brown marbling of the entire solution or as layering of oil at the surface of the TPN container. Such solutions are not safe for infusion and should be returned to the pharmacy for replacement.

AMINO ACIDS. All tissues require protein to maintain structure and facilitate wound healing. If protein intake is inadequate, the body becomes catabolic, seeking protein from skeletal muscle and vital organs. In TPN, protein is provided as a mixture of essential and nonessential crystalline amino acids, which are available in concentrations ranging from 5% to 15%. These concentrations supply approximately 15% to 20% of daily caloric needs. One gram of amino acids is equivalent to 1 g of protein, which provides 4 kcal/g. Adult amino acid requirements can range widely from 0.8 to 2.5 g/kg/d, and those patients with burns, wounds, draining fistulas, renal failure, or hepatic failure may need frequent adjustments in the amount of amino acids they receive.[16] For patients with renal disease, solutions with a higher concentration of essential amino acids are available. For patients with hepatic failure or hypercatabolic conditions, formulas with branched-

chain amino acids may be used. These formulas spare the breakdown of other muscle proteins to use as energy, possibly reducing the incidence of hepatic encephalopathy.

MICRONUTRIENTS. Vitamins, trace minerals, and electrolytes are considered micronutrients. Unfortunately, the U.S. Recommended Dietary Allowance requirements do not apply to parenteral nutrition for several reasons. First, the liver and GI tract absorptive processes are bypassed, resulting in elimination of these micronutrients through the urine without their being utilized. Second, many diseases alter the gut's ability to absorb fat-soluble vitamins and vitamin B_{12}. Finally, many nutrients adhere to the plastic tubing and IV solution bags or are destroyed by exposure to light and oxygen (especially vitamin A) before reaching the bloodstream.

With these factors in mind, standard aqueous multivitamin preparations have been created and provide higher levels of thiamine, pyridoxine, ascorbic acid, and folic acid.[20] Unfortunately, hypermetabolic conditions of critical illness can exacerbate deficiencies that require additional monitoring and potential supplementation; individual vitamin products are available for supplements as needed. Until recently, vitamin K was the only vitamin not included in the multivitamin preparation; it is provided by adding up to 10 mg/wk of the vitamin to the TPN solution (unless contraindicated by anticoagulation treatment). Because some parenteral formulas may now contain vitamin K, weekly supplementation is no longer necessary, but it is important to continue to monitor coagulation studies, especially if the patient is receiving anticoagulation treatment. Unlike patients who require additional nutrients, patients with liver or kidney disease may need to receive lower doses of certain vitamins.

Trace mineral elements are required to maintain biochemical homeostasis. They come in a variety of commercial mixtures but typically include chromium, copper, manganese, selenium, and zinc. Parenteral iron is not added to TPN solutions because of stability issues and the potential for adverse effects, and it may need to be supplemented in long-term therapy.

Most electrolyte standard mixtures contain sodium, potassium, calcium, magnesium, phosphorus, chloride, and acetate. Sodium bicarbonate is not added to TPN as precipitation with the other electrolytes may occur. Instead, acetate is used because it can be converted by the liver to bicarbonate. Depending on the patient's underlying disease process and physical assessment findings, specific electrolyte concentrations can be adjusted daily in the TPN solution. If an electrolyte deficiency is detected after the TPN solution has been prepared, or while infusing, additional IV supplements can be given separately by IV piggyback administration. Electrolyte supplements should never be added to the TPN bag after the pharmacist has formulated it because this would compromise the sterility of the solution and may cause the solution to precipitate.

MEDICATIONS. While preparing the TPN solution, the pharmacist can add medications, many of which are often necessitated by the TPN therapy itself. For instance, although insulin drips are now the current trend in managing hyperglycemia, insulin can be added to the TPN solution. Additionally, heparin can be added to reduce fibrin buildup along the catheter tip. The clinician ordering the medications consults with the pharmacist to ensure compatibility.

Complications of Parenteral Nutrition

Complications can be divided into three main categories: metabolic, infectious, and mechanical.

METABOLIC COMPLICATIONS. TPN has been recognized as a cause of severe morbidity and life-threatening complications.[21] This is often related to the infusion amount and flow rate. Specific complications include hepatic steatosis (fatty liver), intrahepatic and extrahepatic cholestasis (suppression of bile flow), and cholelithiasis (formation of gallstones). Although the exact mechanisms of these hepatic disorders are not completely understood, it has been observed that cholestasis is less likely to occur if some form of enteral feeding is maintained.[21] GI atrophy, and all of its associated complications, may occur from disuse. If not contraindicated, oral or enteral feedings should be initiated as soon as possible.

It is important to understand that many metabolic complications stem from the patient's underlying disease processes or from imprudent formula administration. Some metabolic disturbances can be prevented by checking each bag of parenteral nutrition solution for transcription accuracy, monitoring the IV pump for infusion accuracy, and monitoring the patient's response to therapy. Virtually any metabolic disturbance can occur during parenteral nutrition infusion: the most common metabolic complications include hyperglycemia, hypoglycemia, hypophosphatemia, hypokalemia, hypomagnesemia, and hypocalcemia. These metabolic disturbances, coupled with rapid fluid shifts and imbalances, may lead to a disorder called refeeding syndrome, which is discussed later.

Hyperglycemia. Hyperglycemia, or a blood glucose elevated over 220 mg/dL, can occur if the pancreas does not respond to the increased glucose load. Although hyperglycemia can be caused by either enteral or parenteral feedings, it is more commonly seen in patients receiving parenteral nutrition. Even slightly elevated blood glucose levels can impair the function of lymphocytes, leading to immunosuppression and increased risk for infection. Elevated glucose concentrations have been shown to reduce neutrophil chemotaxis and phagocytosis and may be an independent risk factor for short-term infections.[4] If the renal threshold for glucose reabsorption is exceeded, osmotic diuresis results in subsequent dehydration and electrolyte imbalances. Glycemic control can be achieved by increasing the amount of insulin in the TPN solution, by maintaining a continuous insulin drip during TPN administration, or by administering sliding-scale insulin subcutaneously at regular intervals. Once TPN is discontinued, insulin requirements become notably less or nonexistent. If new TPN solution is temporarily unavailable, administration of 10% dextrose in water ($D_{10}W$) is recommended to prevent rebound hypoglycemia. In addition, if a solution is "behind schedule," the infusion rate should not be increased to make up time because this may cause sudden metabolic fluctuations and fluid overload.

Refeeding Syndrome. Refeeding syndrome is one of the most critical complications that occur with the initiation of TPN. This condition is characterized by rapid shifts in electrolytes (phosphorus, potassium, magnesium, and calcium), glucose, and volume status within hours to days of nutrition implementation. Parenterally delivered glucose loads stimulate insulin release, which in turn stimulates intracellular uptake of phosphorus, glucose, and other electrolytes

for anabolic processes. Despite relatively normal serum phosphorus levels on standard laboratory reports, intracellular stores are markedly depleted in malnourished catabolic patients; severe hypophosphatemia (<1 mg/dL) can lead to neuromuscular, respiratory, and cardiac dysfunction. Low serum levels of potassium, magnesium, and calcium can precipitate cardiac dysrhythmias. The increased intravascular fluid volumes associated with parenteral nutrition can strain the viscerally depleted heart and possibly induce heart failure and myocardial damage. Risk factors for refeeding syndrome include marasmus, chronic alcoholism, anorexia nervosa, rapid refeeding, and excessive dextrose infusion.

Prevention of refeeding syndrome includes repletion of phosphorus, potassium, magnesium, and calcium before TPN initiation, limiting initial dextrose dosing, and titrating total volume and rate to evaluate for fluid overload and potential cardiac decompensation. Daily monitoring of phosphorous, potassium, and magnesium is recommended.[22] Weight-based phosphorus repletion algorithms (eg, 0.32 to 1.0 mmol/kg) have been shown to be highly efficacious in correcting hypophosphatemia during nutrition support therapy.[22] It is of paramount importance that the critical care nurse takes accurate intake and output measurements and daily weights because adequate parenteral nutrition often means giving 1.5 to 3 L of fluid/d in addition to other therapies. Progressive weight gain could be an early indicator of poor fluid tolerance.

INFECTIOUS COMPLICATIONS. Both the solution and the indwelling catheter are prime sites for infection because of the high glucose content. Any break in the system is a nidus for infection that can progress to a systemic infection if left unchecked. After initial preparation by a pharmacist, the access hubs on the bag of TPN are often covered with tape as a reminder that no additional solutions or medications are to be added.

At the bedside, the nurse changes the TPN solution bag and tubing according to institution policy, usually every 24 hours. He or she redresses the catheter insertion site per institution policy as well, usually every 24 to 72 hours, using either a sterile transparent or gauze dressing. Frequent dressing changes, however, have been shown to increase bacterial colonization. Fortunately, transparent dressings allow for easier observation of the catheter entrance site; visible inflammation indicates significant bacterial colonization and the need for prompt catheter removal.[23] It is important to check institutional policies and procedures regarding central and peripheral line dressing changes.

At the time of the dressing change, the nurse examines the site for signs of leakage, erythema, and/or inflammation, and then cleanses the site with an antibacterial solution to remove pathogenic organisms. Researchers have shown that chlorhexidine solution is a more effective local antiseptic than povidone-iodine solutions.[24,25] The use of impregnated chlorhexidine/silver sulfadiazine or minocycline/rifampin catheters could reduce the incidence of central venous catheter–related infections.[26] The presence of a tracheostomy or other open draining wounds near the IV insertion site requires special precautions to prevent site contamination.

Potential for infection can be minimized by meticulous catheter care. In critically ill patients, catheter-related infections range from local inflammation to systemic bloodstream infection and sepsis. Central venous catheters are a leading source of nosocomial bloodstream infection with an estimated 10% mortality.[23] If fever, rigors, or chills coincide with parenteral infusion, catheter-related sepsis should be suspected; slowing or stopping the infusion may cause fever to abate. Treatment of an infection may involve local topical antibiotics, systemic antibiotics, and, in many cases, catheter removal. If catheter sepsis is suspected, the catheter tip is usually cultured to identify the offending organism to ensure appropriate antibiotic coverage.

Mechanical Complications

Mechanical complications include those associated with central venous catheter insertion, such as trauma to the vessel, pneumothorax, catheter occlusion, thrombosis, and venous air embolism. After insertion of a central catheter, a chest radiograph is the standard method of confirming correct placement. If there is a clinical suspicion of catheter tip migration or other potential complications, further diagnostic testing is indicated.

Trauma to vessels and pneumothorax are complications that may warrant surgical intervention, insertion of a chest tube or tubes, or both. Catheter occlusion can simply be a result of the catheter tip lodging against the vessel wall or being physiologically "pinched" between the clavicle and first rib. Occlusion can also occur from fibrin buildup, blood or lipid deposition, drug precipitates, and catheter breakage. Another type of occlusion, "withdrawal occlusion," is an occlusion that allows infusion of a solution but prevents blood withdrawal. The addition of 6,000 units of heparin in the daily parenteral formula in hospitalized patients with temporary catheters reduces the risk of fibrin sheath formation and catheter infection.[4] Thrombosis formation in the lumen of the vessel often results from mechanical irritation (such as from traumatic catheter insertion), a small lumen, an extended duration of catheter use, the catheter material, or malpositioning. Nurses need to be aware that patients may have a thrombosis and may be asymptomatic, yet complain of vague head and eye swelling on the affected side. Vigilant assessments during parenteral nutrition administration are recommended. Treatment includes catheter removal, systemic anticoagulation, and thrombolytic therapy.

A venous air embolism is another serious complication; rapid introduction of air into the venous circulation can be fatal.[27] In a review of all patients in the literature with a cerebral air embolism associated with central venous catheters from 1975 to 1988, 54% occurred secondary to disconnection of the catheter, 31% occurred during removal of the catheter, and 15% occurred during insertion.[27] Any disruption of the closed catheter system (usually during line connection changes, when hanging a new bag of TPN, or in an accidental tubing disconnection) can increase the risk for an air embolism. If such an incident occurs, the patient will most likely experience acute, centrally located chest pain, dyspnea, and hypotension. Immediate nursing interventions include clamping the tubing of the catheter or occluding the catheter hub, attempting to aspirate air directly from the venous line (ie, for patients who have central venous access properly placed in the right atrium, attempts may be made to aspirate air bubbles from the distal port), administering 100% oxygen via a facemask, and positioning the patient head down on the left side (Durant's maneuver).[27] This position allows air to rise to the level of the right ventricle, away from

the pulmonary vasculature. Prevention of an air embolism can be facilitated by having the patient perform the Valsalva maneuver or simply hum audibly during line changes. In ventilator-dependent patients, positive intrathoracic pressure can be created by initiating mechanical lung inflations or "breaths." Finally, use of sterile occlusive dressings (eg, petrolatum gauze) over the catheter entrance site is an effective measure in preventing air from entering the track after the catheter has been removed.

Tapering Parenteral Nutrition

Tapering (gradually reducing) TPN is often initiated for those patients who are able to resume safely (and tolerate) approximately 50% to 75% of their nutritional needs by enteral or oral nutrition. In such instances, a calorie count is essential to be certain that the patient's nutritional needs are being met. If the parenteral nutrition needs to be interrupted or is to be discontinued, the infusion rate is decreased by half for 30 to 60 minutes. This allows for a plasma glucose response and prevention of rebound hypoglycemia.[19] Checking blood glucose levels for 30 to 60 minutes after discontinuation helps the nurse identify and manage immediate glucose abnormalities.

In situations in which poor prognosis does not warrant aggressive nutritional support, emotional and ethical dilemmas may surface for many nurses because feeding and hydration have long been basic tenets of nursing care. Although many institutions may have protocols in place regarding parenteral nutrition, treatment decisions and plans of care should be discussed on an individual basis. Frequent, ongoing discussions between the patient, family, and the health care team are imperative to providing the best possible care to each patient.

Role of the Nurse in Nutritional Support

Nurses are responsible for obtaining initial "dry weight" and weekly weight measurements, vital signs, intake and output measurements, and laboratory data and for providing enteral tube and IV catheter care throughout the duration of nutrition support therapies. Many complications, whether from enteral or parenteral nutrition, can be prevented by vigilant observation and care. If the patient is awake and alert, the patient's subjective assessment of tolerance can be very informative. The nurse obtains more objective signs of feeding tolerance through abdominal examinations, which assess bowel sounds and changes in abdominal girth. Also, the nurse monitors and records volume and frequency of both urine and stool.

The nurse must also monitor for clinical signs of dehydration (thirst, dry mucous membranes, tachycardia, and poor skin turgor) and fluid excess (peripheral edema and adventitious lung sounds). Early detection and subsequent interventions may prevent the occurrence of excessive fluid shifts and cardiac compromise. This is of special concern if the patient is severely malnourished, which may precipitate refeeding syndrome and other untoward complications. Meticulous feeding tube and IV catheter care are critical to preventing local and systemic forms of infection.

Care also includes providing information and emotional support to the patient and family. Examples include explaining the procedure, what to expect, risks, and expected outcomes (Box 40-3).

BOX 40-3 TEACHING GUIDE Living With Nutritional Support

General Care: Enteral Nutrition
- Administer enteral formulas as prescribed.
- Know potential complications and appropriate treatments.
- Avoid activities that may result in high impact or stress at the insertion site and report any activity that may have damaged the enteral access site.
- Return to previous activities (eg, work, leisure, sexual activity) after obtaining physician consent.

General Care: Parenteral Nutrition
- Administer parenteral formulas as prescribed.
- Monitor blood glucose levels closely to help determine tolerance for parenteral solutions.
- Know the potential complications and appropriate treatments.
- Avoid activities that may result in high impact or stress at the insertion site and report any activity that may have damaged the parenteral access site.
- Return to previous activities (eg, work, leisure, sexual activity) after obtaining physician consent.

Signs of Infections
- Understand the rationale for aseptic technique.
- Notify the nurse of symptoms of fever, localized warmth, redness, pain, or drainage at the feeding tube or IV insertion site.

Medications
- Follow instructions regarding medications.
- Know the names of medications and the dose, frequency of administration, side effects, and use of each medication.
- Know the proper technique of administering medications through the feeding tube and proper flushing technique.
- Never add medications to TPN solutions—they should be added by the supplier because of risk for contamination or precipitation of the formula.

Safety Measures
- Inform other health care providers about enteral or parenteral access devices and notify them about any medications that the patient may be taking.

Follow-Up Care
- Report any problems to the home care nurse.
- Adhere to schedule for follow-up visits with patient's physician or clinic.
- Ensure that patient/care giver learning includes determining procedures and risks, identifying patient and equipment problems early, troubleshooting, and following up with the health care provider.
- Refer to and communicate with home care services.
- Provide written instructions for patient.
- If possible, do not change the amount or rate of nutrition support on the day of discharge to home.

▲ Pharmacological Management of Gastrointestinal Disorders

Table 40-1 summarizes many of the common GI medications administered to critically ill patients who are concurrently receiving nutrition support therapy.

Table 40-1 Common Gastrointestinal Medications

Medication	Mechanism of Action	Indication	Common Adverse Effects	Comments
Antacids				
Aluminum carbonate	Neutralization of gastric acid; binding phosphates in the GI tract	Symptomatic relief of gastric irritation, prevention of urinary phosphate stone development, binding of phosphate in chronic renal failure	Fecal impaction, cramps, constipation, hypophosphatemia (when given in excessive doses)	Monitor phosphorus levels.
Aluminum hydroxide (Amphojel, AlternaGEL)	Neutralization of gastric acid; binding phosphates in the GI tract	Symptomatic relief of gastric irritation, hyperphosphatemia in chronic renal failure	Constipation, hypophosphatemia (when given in excessive doses)	Less phosphate binding than aluminum carbonate. Monitor phosphorus levels.
Calcium carbonate (Tums, Caltrate)	Neutralization of gastric acid	Symptomatic relief of gastric irritation, calcium supplementation	Headaches	Usually well tolerated. Monitor calcium and phosphorus levels.
Magnesium hydroxide (Milk of magnesia)	Neutralization of gastric acid	Symptomatic relief of gastric irritation, hypomagnesemia, constipation	Hypermagnesemia, abdominal cramping and diarrhea (with high doses)	Monitor magnesium levels.
Dihydroxyaluminum, sodium carbonate (Rolaids)	Neutralization of gastric acid; reduction of pepsin	Symptomatic relief of gastric irritation	Constipation	Use with caution in sodium-restricted patients.
Histamine Type 2 (H_2) Receptor Antagonists				
Cimetidine (Tagamet)	Inhibition of histamine at H_2 receptor sites on gastric parietal cells, which inhibits gastric acid secretion	GERD, PUD, acid hypersecretory states	Confusion, headaches, diarrhea	May cause rare blood dyscrasias. Monitor CBC.
Ranitidine (Zantac)	Inhibition of histamine at H_2 receptor sites on gastric parietal cells, which inhibits gastric acid secretion	GERD, PUD, acid hypersecretory states	Headaches, dizziness, constipation	May cause hepatotoxicity and rare blood dyscrasias.
Famotidine (Pepcid)	Inhibition of histamine at H_2 receptor sites on gastric parietal cells, which inhibits gastric acid secretion	GERD, PUD, acid hypersecretory states	Headaches, dizziness	May cause seizures, bronchospasm, constipation, or thrombocytopenia. Monitor CBC.
Nizatidine (Axid)	Inhibition of histamine at H_2 receptor sites on gastric parietal cells, which inhibits gastric acid secretion	GERD, PUD, acid hypersecretory states	Dizziness, headaches, diarrhea	
Proton Pump Inhibitors				
Omeprazole (Prilosec), lansoprazole (Prevacid), rapeprazole (Aciphex), pantoprazole (Protonix), esomeprazole (Nexium)	Suppression of gastric acid secretion by inhibition of H^+, K^+-ATPase pump (proton pump) of parietal cells, blocking final step in acid production	Reflux esophagitis, treatment of gastric and duodenal ulcers, pathological hypersecretory states (Zollinger-Ellison syndrome)	Headaches, diarrhea, abdominal pain	Less common side effects include nausea, vomiting, and dizziness.

(continued on page 911)

Table 40-1 **Common Gastrointestinal Medications** (continued)

Medication	Mechanism of Action	Indication	Common Adverse Effects	Comments
Pancreatic Enzymes				
Pancreatin (Creon, Donnazyme, Ultrase)	Assistance in the digestion of carbohydrates, fats, and proteins	Replacement in pancreatic enzyme deficiencies, cystic fibrosis	Nausea, diarrhea, cramping, anorexia, hypersensitivity reactions, perianal irritation	
Pancrealipase (Pancrease, Viokase)	Assistance in the digestion of carbohydrates, fats, and proteins	Replacement in pancreatic enzyme deficiencies, steatorrhea of malabsorption, cystic fibrosis, postgastrectomy, or postpancreatectomy	Nausea, diarrhea, cramping, anorexia, hypersensitivity reactions, perianal irritation	
Antidiarrheals				
Attapulgite (Kaopectate)	Absorption of toxins produced by bacterial and GI irritants; decrease in gastric motility and stool water content	Diarrhea	Increased potassium loss, interference with absorption of medications	
Bismuth subsalicylate (Pepto Bismol)	Slowing of motility; antimicrobial activity against GI microbes; antisecretory sensitivity	Diarrhea, prophylaxis of traveler's diarrhea	Tongue discoloration, dark stools	Assess electrolytes if diarrhea persists. Use cautiously in patients using other salicylates.
Cholestyramine (Questran)	Absorption of bile salts, which can cause diarrhea; absorption of *Clostridium difficile* toxin	Diarrhea caused by bile salts or *C. difficile*	Constipation	Because it may alter absorption of other medications, administer other medications at least 1 h before cholestyramine.
Loperamide (Imodium)	Slowing intestinal motility, including peristalsis	Acute and chronic diarrhea	Abdominal distention, constipation, drowsiness, dizziness, nausea, vomiting	
Tincture of opium (Paregoric, DTO)	Decrease in GI motility and peristalsis, decrease in digestive secretions	Acute diarrhea, relief of abdominal cramping	Drowsiness, lightheadedness, bradycardia	Side effects are related to opioid content. Other possible reactions include allergic reactions, vomiting, dizziness, sweating, constipation, and habituation.
Laxatives				
Bowel Evacuants				
Polyethylene glycol with electrolytes (Colyte, NuLytely, GoLYTELY)	Nonabsorbable solution that acts like an osmotic agent	Bowel cleansing before colonoscopy or bowel surgery	Transient bloating, nausea, cramping	
Bulk-Forming Agents				
Calcium polycarbophil (Fibercon), methylcellulose (Citracel), psyllium (Metamucil)	Nondigestable plant cell wall draws water into the feces and softens stool; absorption of excess water in the stool	Diarrhea, constipation	Flatulence, impaction (if feces are obstructed)	Generally well tolerated.

(continued on page 912)

Table 40-1 Common Gastrointestinal Medications (continued)

Medication	Mechanism of Action	Indication	Common Adverse Effects	Comments
Laxatives				
Lactulose (Cephulac, Enulose)	Hyperosmolality draws water into the intestinal lumen, increasing stool water content and softening stool; prevention of absorption of ammonia in the colon	Constipation, prevention and treatment of hepatic encephalopathy	Flatulence, cramping, impaction (if feces is obstructed)	For use in prevention and treatment of hepatic encephalopathy, titrate dose to two to three loose stools a day. Monitor serum ammonia levels.
Polyethylene glycol (MiraLax)	Nonabsorbable solution that acts like an osmotic agent	Constipation	Nausea, abdominal bloating, cramping, diarrhea	
Saline Laxatives				
Magnesium citrate (Citrate of Magnesium)	Magnesium and sodium salts are poorly absorbed, drawing water into the intestinal lumen	Constipation, cleansing of the colon before examination	Cramps, flatulence, nausea, vomiting	Do not use in renal disease. Observe for hypermagnesemia (watching for thirst, drowsiness, dizziness).
Sodium biphosphate (Fleet Phospha-soda, Fleet Enema)	Increase in water absorption in the small intestine through osmosis	Constipation, acute bowel evacuation before a bowel or colon examination	Nausea, cramps	May precipitate or exacerbate cardiac, renal, or seizure disorder.
Stimulants				
Bisacodyl (Dulcolax)	Increase in peristalsis by direct effect on nerve endings in colonic mucosa	Constipation, evacuation of bowel before examination		Can cause habituation with gradual lessening effect in long-term use. Administered orally onset is 6–10 h; Administered rectally, onset is 15–60 min.
Cascara	Increase in propulsive movements through chemical irritation of the colon	Constipation	Discoloration of urine (red or yellow-brown)	May cause habituation. Onset is 6–10 h.
Senna (Senokot, SenokotXTRA)	Stimulation of propulsion	Constipation	Discoloration of urine	Natural product from cassia
Phenolphthalein (Ex-Lax)	Stimulation of peristalsis (similar to bisacodyl)	Constipation		May cause an allergic reaction; discontinue use if rash develops.
Stool Softeners				
Docusate sodium (Surfak, Colace)	Increase in the penetration of the feces by water and fat; softening of the stool	Constipation	Cramping, diarrhea	Prolonged or excessive use may cause habituation or electrolyte abnormalities.
Stimulant/Stool Softeners				
Glycerin	Drawing water into the colon (by high osmotic pressure)	Constipation	Headaches, nausea, vomiting	
Antiemetics				
Trimethobenzamide (Tigan)	Inhibition of the chemoreceptor trigger zone, which then inhibits the vomiting center	Symptomatic relief of nausea and vomiting	Hypersensitivity, drowsiness, hypotension, diarrhea, depression, vertigo	

(continued on page 913)

Table 40-1 **Common Gastrointestinal Medications (continued)**

Medication	Mechanism of Action	Indication	Common Adverse Effects	Comments
Antiemetics				
Prochlorperazine (Compazine)	Blocking of dopamine receptors in the chemoreceptor trigger zone in the brainstem	Nausea, vomiting	Extrapyramidal side effects such as drowsiness, blurred vision, tachycardia, and respiratory depression	
Promethazine (Phenergan)	Competition with histamine in blood vessels and GI and respiratory systems to decrease allergic responses	Nausea, vomiting, motion sickness, sedation	Dizziness, drowsiness, constipation, urinary retention	Other reactions may include thrombocytopenia, agranulocytosis, and hemolytic anemia.
Dolasetron (Anzemet)	Blocking serotonin ($5\text{-}HT_3$) receptors in the chemoreceptor trigger zone and GI tract	Nausea and vomiting associated with chemotherapy, prevention and treatment of postoperative nausea and vomiting	ECG changes, hypertension, abdominal pain, diarrhea, urinary retention	
Granisetron (Kytril)	Blocking serotonin ($5\text{-}HT_3$) receptors in the chemoreceptor trigger zone and GI tract	Nausea and vomiting associated with chemotherapy and radiation	Headache, constipation, asthenia	Use with caution in patients with liver disease.
Ondansetron (Zofran)	Blocking serotonin ($5\text{-}HT_3$) receptors in the chemoreceptor trigger zone and GI tract	Nausea and vomiting associated with chemotherapy, prevention of postoperative nausea and vomiting	Diarrhea, bronchospasm, fatigue, constipation	
Other				
Sucralfate (Carafate)	Formation of a protective covering at ulcer site	Short-term treatment of peptic ulcers	Constipation	Because it may alter absorption of other medications, patient should take other medications at least 2 h before sucralfate.
Metoclopramide (Reglan)	Stimulation of upper GI motility; decrease in inhibitory tone	Diabetic gastroparesis, delayed gastric emptying, short-term treatment of GERD, prevention of postoperative nausea and vomiting, facilitation of small bowel feeding tube placement	Diarrhea, constipation, drowsiness, restlessness	May occasionally have extrapyramidal side effects.
Misoprostol (Cytotec)	Prostaglandin analog increases bicarbonate and mucus release and decreases acid secretions	Prevention of aspirin- and NSAID-induced ulcers	Diarrhea, nausea, vomiting, flatulence	Use with caution in pregnant women and in women of childbearing age; it increases uterine contractions, which may cause abortion.
Octreotide (Sandostatin)	Synthetic analog of somostatin, inhibition of the secretion of gastrin, vasoactive intestinal peptide, insulin, glucagon, motilin, secretin, and pancreatic polypeptides	Secretory diarrhea, acute variceal hemorrhage	Edema, flushing, dizziness, headache, abdominal pain, constipation, diarrhea, hyperglycemia, hypoglycemia	Monitor blood glucose and adjust insulin requirements.

CBC, complete blood count; ECG, electrocardiogram; GERD, gastroesophageal reflux disease; NSAIDs, nonsteroidal anti-inflammatory drugs; PUD, peptic ulcer disease.

▲ Clinical Applicability Challenges

CASE STUDY

K.M. is a 36-year-old man with a history of a gunshot wound to the abdomen status post multiple small bowel resections, which has resulted in short bowel syndrome. Because he cannot meet his caloric and protein requirements, protein–calorie malnutrition has resulted, and he now requires chronic TPN to meet his nutritional needs. A PICC is placed, and home TPN is initiated.

K.M. presents to the emergency department 4 months later with complaints of fever and chills. His vital signs include an oral temperature of 101.8°F; pulse 115 beats/min, blood pressure 102/58 mm Hg, and respirations 22 breaths/min. Point of care blood glucose level is 217 mg/dL.

1. What physical examination findings would you expect to find?
2. What diagnostic tests should be evaluated?
3. What is the most likely management of this patient?
4. What discharge teaching should this patient receive?

References

1. Btaiche IF, Chan LN, Pleva M, et al: Critical illness, gastrointestinal complications, and medication therapy during enteral feeding in critically ill Adult Patients. Nutr Clin Pract 25(1):32–49, 2010
2. Heimburger DC: Malnutrition and nutritional assessment. In Fauci AS, Braunwald E, Kasper DL, et al (eds): Harrison's Principles of Internal Medicine, 17th ed. New York, NY: McGraw-Hill, 2008, pp 450–454
3. Leaf DA: Emerging trends in novolitional nutrition support: The role of parenteral nutrition. J Clin Outcomes Manage 17(1):31–34, 2010
4. Bistrian BR, Driscoll DF: Enteral and parenteral nutrition therapy. In Fauci AS, Braunwald E, Kasper DL, et al (eds): Harrison's Principles of Internal Medicine, 17th ed. New York, NY: McGraw-Hill, 2008, pp 455–462.
5. McClave SA, Martindale RG, Vanek VW, et al; the ASPEN Board of Directors, & American College of Critical Care Medicine: Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient. JPEN J Parenter Enteral Nutr 33(3):277–316, 2009
6. Khalid I, Doshi P, DiGiovine B: Early enteral nutrition and outcomes of critically ill patients treated with vasopressors and mechanical ventilation. Am J Crit Care 19(3):261–268, 2010
7. Martindale RG, McClave SA, Vanek VW; American Collage of Critical Care Medicine an; and the A.S.P.E.N. Board of Directors. Guidelines for the provision and assessment of nutritional support therapy in the adult critically ill patients: Society of Critical Care Medicine and the American Society for Parenteral and Enteral Nutrition: executive summary. Crit Care Med 37(5):1757–1761, 2009
8. Bankhead R, Boullata J, Brantley S, et al; ASPEN Board of Directors. Enteral nutrition practice recommendations. JPEN J Parenter Enteral Nutr 33(2) 122–167, 2009
9. Chen Y, Peterson SJ: Enteral nutrition formulas: which formula is right for your adult patient? Nutr Clin Pract 24(3):344–355, 2009
10. Marik PE, Zaloga GP. Immunonutrition in critically ill patients: a systemic review and analysis of the literature. Intens Care Med 34 1980–1990
11. Metheny NA, Schallom L, Oliver DA, et al: Gastric residual volume and aspiration in critically ill patients receiving gastric feedings. Am J Crit Care 17(6):512–520, 2008
12. Johnson AD: Assessing gastric residual volumes. Crit Care Nurse 29(5):72–73, 2009
13. Wohlt PD, Zheng L, Gunderson S, et al: Recommendations for the use of medications with continuous enteral nutrition. Am J Health Syst Pharm 66:1458–1467, 2009
14. U.S. Food and Drug Administration Center for Food Safety and Applied Nutrition: FDA Public Health Advisory: 2003. Reports of blue discoloration and death in patients receiving enteral feeding tinted with the dye, FD&C blue No. 1. Retrieved November 13, 2006, from http://www.cfsan.fda.gov/%7Edms/col-ltr2.html
15. American Association of Critical Care Nurses: 2005 Practice alert: Dye in enteral feeding. Retrieved November 13, 2006, from http://www.aacn.org/AACN/practceAlertnsf/Files/DEF/$files/dyeinenteralfeeding.pdf
16. Zeigler TR: Parenteral nutrition support in the critically ill patient. N Engl J Med 361(11):1088–1097, 2009
17. Miller SJ: Parenteral Nutrition. U.S. Pharmacist 31(7):10–20, 2006
18. Mirtallo J, Canada T, Johnson D, et al: Safe Practices for Parenteral Nutrition. JPEN J Parenter Enteral Nutr 28(6):S39–S70, 2004
19. Peterson S, Chen Y: Approach to parenteral nutrition. Curr Drug Saf 5(1):33–40, 2010
20. Buchman AL, Howard LJ, Guenter P, et al: Micronutrients in parenteral nutrition: Too little or too much? The past, present, and recommendations for the future. Gastroenterology137:S1–S6, 2009
21. Guglielmi FW, Regano N, Mazzuoli S, et al: Cholestasis induced by total parenteral nutrition. Clin Liver Dis 12:97–110, 2008
22. Tresley J, Sheean PM: Refeeding syndrome: Recognition is the key to prevention and management. J Am Diet Assoc 108(12):2105–2108, 2008
23. Sudharma Ranasinghe J, Lee AJ, Birnbach DJ: Infection associated with central venous or epidural catheters: How to reduce it? Curr Opin Anaesthesiol 21:386–390, 2008
24. Garnacho-Montero J, Aldabo-Pallas T, Palomar-Martinez M, et al: Risk factors and prognosis of catheter-related bloodstream infection in critically ill patients: A multicenter study. Intensive Care Med 34:2185–2193, 2008
25. Casey AL, Mermel LA, Nightingale P, et al: Antimicrobial central venous catheters in adults: A systematic review and meta-analyses. Lancet Infect Dis 8:763–776, 2008
26. Dave P, Cartwright AF, Subhani JM: Complication rates for central venous catheters used for parenteral nutrition. Proc Nutr Soc 69 (OCE2):E162, 2010
27. Scruggs JE, Joffe A, Wood KE: Paradoxical air embolism successfully Treated with hyperbaric oxygen. J Intensive Care Med 23(3):204–209, 2008

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

41 Common Gastrointestinal Disorders

Allison G. Steele and Valerie K. Sabol

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Examine the pathophysiological concepts that help define acute gastrointestinal bleeding (GIB), obstruction and ileus, acute pancreatitis (AP), hepatitis, and complications of liver disease.
2. Compare and contrast the pertinent history, physical examination, and diagnostic study findings for acute GIB, obstruction and ileus, AP, hepatitis, and cirrhosis.
3. Discuss laboratory studies that are useful in the diagnosis and management of acute GIB, obstruction and ileus, AP, hepatitis, and complications of liver disease.
4. Analyze the similarities and differences in caring for patients with acute GIB, obstruction and ileus, hepatitis, and complications of liver disease.
5. Explore the nursing role in assessing, managing, and evaluating a plan of care for patients with acute GIB, obstruction and ileus, AP, hepatitis, and complications of liver disease.

The critical care nurse will inevitably provide care for patients with common yet serious disorders of the gastrointestinal (GI) tract. Some of these disorders include gastrointestinal bleeding (GIB), intestinal obstructions, and complex inflammations, such as pancreatitis and hepatitis.

▲ Acute Gastrointestinal Bleeding

Acute GIB is a common, and potentially lethal, medical emergency seen in people admitted to the intensive care unit (ICU). There are 300,000 hospital admissions each year in the United States as a result of acute GIB.[1–4] The 10% mortality rate associated with acute GIB has remained constant over the past half century despite advances in diagnosis and treatment.[1–3] This constant mortality rate may result from the prevalence of comorbid disease in older adults and the widespread use of nonsteroidal anti-inflammatory drugs (NSAIDs). The cause of death is rarely from exsanguination but rather from the exacerbation of other medical illnesses. Prompt recognition and treatment of patients experiencing acute GIB requires a team approach.

Acute GIB is differentiated into upper and lower GIB. The ligament of Treitz at the junction of the duodenum and jejunum is the anatomic division between the upper and lower GI tracts. Upper GIB occurs from a source in the esophagus, stomach, or duodenum. Lower GIB occurs from a source in the jejunum, ileum, colon, or rectum. GIB from a lower GI source is less common than upper GIB.

Upper Gastrointestinal Bleeding

Etiology

The possible causes of acute upper GIB are listed in Box 41-1. A complete discussion of this list is beyond the scope of this chapter. The most commonly seen causes of acute GIB in the ICU are discussed in the following sections. See Spotlight on Genetics 41-1 for a discussion of Crohn's disease.

Peptic Ulcer Disease

Peptic ulcer disease, which includes both gastric and duodenal ulcers, accounts for approximately 40% to 60% of acute upper GIB.[1,2,5–7] The epithelial cells of the gastroduodenal mucosa are protected from the potentially damaging effects of gastric secretions, medications, alcohol, and bacteria by several protective mechanisms. These cells secrete mucins, phospholipids, and bicarbonate, which create a pH gradient between the acidic gastric lumen and the cell surface. Prostaglandins enhance this mucosal protection by increasing mucosal secretion, increasing bicarbonate production, maintaining mucosal blood flow, and enhancing the resistance of gastroduodenal cells to injury. In addition, the tight junctions of the epithelial cells resist diffusion. When these protective factors are overwhelmed by aggressive factors, the integrity of the gastric or duodenal mucosa is interrupted, which can result in peptic ulcer disease. Bleeding from peptic ulcer disease occurs when the ulcer erodes into the wall of a blood vessel.

BOX 41-1 Common Causes of Acute Gastrointestinal Bleeding (GIB)

Upper Gastrointestinal Bleeding

Esophageal Source
- Varices
- Esophagitis
- Ulcers
- Tumors
- Mallory-Weiss tears

Gastric Source
- Peptic ulcers
- Gastritis
- Tumors
- Angiodysplasia
- Dieulafoy's lesions

Duodenal Source
- Peptic ulcers
- Angiodysplasia
- Crohn's disease
- Meckel's diverticulum

Lower Gastrointestinal Bleeding
- Malignant tumors
- Polyps
- Ulcerative colitis
- Crohn's disease
- Ischemic colitis
- Infectious colitis
- Angiodysplasia
- Diverticulosis
- Hemorrhoids
- Massive upper gastrointestinal hemorrhage

SPOTLIGHT ON GENETICS 41-1

GI SYSTEM—CROHN'S DISEASE
- Crohn's disease is a complex, chronic disorder that primarily affects the digestive system. This condition typically involves abnormal inflammation of the intestinal walls, particularly in the lower part of the small intestine and portions of the large intestine and is most common in Western Europe and North America, where it affects 100 to 150 in 100,000 people.
- The *IL23R* gene is associated with Crohn's disease, and variety of genetic and environmental factors likely play a role in causing Crohn's disease. Although researchers are studying risk factors that may contribute to this complex disorder, many of these factors remain unknown.
- Crohn's disease may result from a combination of certain genetic variations, changes in the immune system, and the presence of bacteria in the digestive tract. Recent studies have identified variations in specific genes, including *ATG16L1*, *IL23R*, *IRGM*, and *NOD2*, that influence the risk of developing Crohn's disease. These genes provide instructions for making proteins that are involved in immune system function. Variations in any of these genes may disrupt the ability of cells in the intestine to respond normally to bacteria. An abnormal immune response to bacteria in the intestinal walls may lead to chronic inflammation and the digestive problems characteristic of Crohn's disease.
- Sequence analysis of the entire coding region or targeted mutation analysis is available in the diagnosis of Crohn's disease.

Genetic Home Reference-http://ghr.nlm.nih.gov, accessed July 14, 2011

Ruthruff B: Clinical review of Crohn's disease. J Am Acad Nurse Pract 19(8):392–397, 2007.

The primary risk factor for peptic ulcer disease is infection with the bacterium *Helicobacter pylori*. *H. pylori* infection has been associated with 90% of duodenal ulcers and 75% of gastric ulcers. *H. pylori* is a Gram-negative, spiral, flagellated rod that colonizes the mucous layer overlying the gastric epithelium. The flagellum of *H. pylori* facilitates the bacterium's ability to move and adhere to the mucous layer. *H. pylori* produces urease, which converts urea to ammonia and carbon dioxide. The ammonia buffers the acid surrounding the bacterium, creating a more hospitable environment that allows the bacterium to thrive in the acidic stomach. *H. pylori* infection predisposes the mucosa to damage by disrupting the mucous layer, liberating enzymes and toxins, and adhering to the epithelium. Inflammation is furthered by a host immune response. This chronic inflammation usually results in an asymptomatic chronic gastritis. However, in some instances, ulceration develops.

In the absence of *H. pylori* infection, the ingestion of aspirin or NSAIDs accounts for most cases of peptic ulcer disease. The ingestion of aspirin and NSAIDs may directly injure the mucosal layer. Ingestion enhances mucosal permeability and allows back-diffusion of acid. Systemic effects of chronic aspirin or NSAID use include inhibition of prostaglandin synthesis by the gastroduodenal mucosa, which decreases production of mucus and bicarbonate and also decreases mucosal blood flow. This alteration in mucosal cytoprotection may lead to the development of an ulcer. Upper GIB related to NSAIDs is more common in older patients. Cigarette smoking may also predispose individuals to peptic ulcer disease, and it is linked to prolonged healing rates and high ulcer reoccurrence.

Stress-Related Erosive Syndrome

Stress-related erosive syndrome, also called erosive gastritis, stress ulceration, and hemorrhagic gastritis, is a common cause of acute GIB in critically ill patients. Stress ulcers are different from the ulcers of peptic ulcer disease; they tend to be more numerous, shallower, and more diffuse. These ulcers may develop in the stomach, duodenum, and esophagus within hours of injury. They are usually shallow and cause oozing from superficial capillaries but may erode into the submucosa and cause massive hemorrhage.

The risk for developing a stress ulcer depends on the severity and type of illness (Box 41-2). The common feature of these risk factors is the relationship to physiological stress. Decreased perfusion of the stomach mucosa is probably the main mechanism of ulcer development. This contributes to impaired secretion of mucus, low mucosal pH, poor mucosal cell regeneration, and decreased tolerance to acidic gastric secretions.

Esophageal Varices

Portal hypertension usually develops as a result of cirrhosis, from increased resistance in the portal venous system caused by disruption of the normal liver lobular structure.

BOX 41-2 PATIENT SAFETY

Risk Factors for Stress-Related Erosive Syndrome

- Hypotension or shock
- Coagulopathy
- Respiratory failure requiring mechanical ventilation
- Sepsis
- Hepatic failure
- Renal failure
- Multiple or severe trauma
- Burns over 35% of the total body surface area
- Post–organ transplantation status
- Head or spinal cord injury
- History of peptic ulcer disease or upper GIB
- Prolonged stay in intensive care unit

This resistance impedes blood flow into, through, and out of the liver. In response to portal hypertension, collateral veins develop to bypass the increased portal resistance in an attempt to return blood to systemic circulation. As pressure rises in these veins, they become tortuous and distended, forming varicose veins or varices.

Esophageal varices account for 10% to 25% of acute upper GIB.[1,6] Varices are present in 50% of patients with cirrhosis at the time of diagnosis.[1,8–10] Varices may develop in the esophagus, stomach, duodenum, colon, rectum, or anus. The most clinically significant site of varices is the gastroesophageal junction because of the propensity of varices in this area to rupture, resulting in massive GI hemorrhage. The mortality rate associated with variceal bleeding is 15% to 50% with each episode.[6,8,10,11]

Mallory-Weiss Tears

Mallory-Weiss tears account for approximately 10% of acute upper GIB.[1] Mallory-Weiss tears are lacerations that occur in the distal esophagus, at the gastroesophageal junction, and in the cardia of the stomach. Bleeding from Mallory-Weiss tears occurs when the tear involves the underlying venous or arterial bed. Mallory-Weiss tears are strongly associated with heavy alcohol use or recent binge drinking and a prior history of forceful vomiting or retching, or violent coughing. Patients with portal hypertension have an increased risk for bleeding from Mallory-Weiss tears.

Dieulafoy's Lesions

Dieulafoy's lesions are vascular malformations of unusually large submucosal arteries, which lie in close contact with the mucosal surface. They can be found anywhere in the GI tract but are most likely to be found in the proximal stomach. Because of the large size of the artery, bleeding from a Dieulafoy's lesion may be massive and recurrent. When bleeding ceases, a Dieulafoy's lesion can be difficult to identify because there is no associated ulcer, and it is likely to be the origin of many upper GIBs of unknown cause.

Clinical Presentation

Regardless of the cause, patients with acute upper GIB have a clinical presentation consistent with the amount of blood loss. A patient's response to blood loss depends on the amount and rate of blood loss, age, degree of compensation, comorbidities, and rapidity of treatment. Patients with minimal loss may present with anemia and no further symptoms, whereas patients with rapid and severe loss may present with signs and symptoms of shock. If blood loss is moderate, the sympathetic nervous system responds with a release of the catecholamines epinephrine and norepinephrine, which initially cause an increase in heart rate and peripheral vascular vasoconstriction in an attempt to maintain an adequate blood pressure. Orthostatic changes (a decrease in blood pressure >10 mm Hg with a corresponding heart rate increase of 20 beats/min in the sitting or standing position) imply volume depletion of 15% or more.

With severe blood loss, signs and symptoms of shock appear. The release of catecholamines triggers the blood vessels in the skin, lungs, intestines, liver, and kidneys to constrict, thereby increasing the volume of blood flow to the brain and heart. Because of the decreased flow of blood in the skin, the patient's skin is cool to the touch. With decreased blood flow to the lungs, hyperventilation occurs to maintain adequate gas exchange.

The classic hallmarks of GIB are hematemesis, hematochezia, and melena. Patients with upper GIB usually present with hematemesis, the vomiting of fresh, unaltered blood or "coffee-ground" material; melena, the passage of foul-smelling, black, tarry, sticky stool; or both. A patient who presents with hematemesis is usually bleeding from a source above the ligament of Treitz. Reverse peristalsis is seldom sufficient to cause hematemesis if the bleeding point is below this area. The classic coffee-ground emesis associated with upper GIB results from the partial decomposition of the blood from contact with gastric secretions. Gastric acid converts bright red hemoglobin to brown hematin, accounting for the coffee-ground appearance of the drainage. Maroon or bright red blood results from profuse bleeding and little contact with gastric juices.

Melena is black from the breakdown of the blood in transit and suggests a long transit time through the GI tract. Melena is indicative of upper GIB in 90% of cases.[1] It may take several days after bleeding cessation for melenic stools to clear. After upper GIB, hemoccult stool test results may remain positive for 1 to 2 weeks. Melena should not be confused with greenish stool that results from iron ingestion or black stool caused by the ingestion of bismuth subsalicylate (Pepto-Bismol).

Hematochezia, the passage of maroon or bright red blood that may be mixed with stool, usually indicates bleeding from a lower GI source. Uncommonly, hematochezia can occur in the setting of massive, rapid hemorrhage from the upper GI tract, where the large amount of blood acts as a cathartic, resulting in rapid transit through the GI tract.

Occult GIB refers to small amounts of blood loss, which is not apparent to the patient. Obscure GIB refers to obvious bleeding with no easily identifiable source on routine examination.

Assessment

HISTORY. A prompt, careful, focused history may suggest the underlying cause of GIB. A history of epigastric pain or dyspepsia or a medical history of peptic ulcer disease is suggestive of peptic ulcer disease. A medical history of GIB should be elicited because most upper GI bleeds rebleed

from the same site. Heavy alcohol use increases the likelihood of cirrhosis and bleeding from esophageal varices. Patients with a history of tobacco use have a greater risk for duodenal ulcers. Underlying medical conditions may suggest an underlying cause; patients with renal failure frequently bleed from arteriovenous malformations. Vomiting, coughing, or retching before bleeding suggests a Mallory-Weiss tear. Prior use of NSAIDs or aspirin increases the risk for gastroduodenal ulcers and the likelihood of bleeding from these ulcers.

PHYSICAL EXAMINATION. The physical examination is directed initially to the assessment of hemodynamic stability with ongoing assessment of vital signs. Tachycardia and orthostatic hypotension indicate dehydration secondary to blood loss or vomiting. Orthostatic hypotension, syncope, lightheadedness, and tachycardia are suggestive of a greater than 15% blood volume loss and are predictive of a poor outcome.[1,3] If 40% of blood volume is lost, hypotension and hypovolemia occur, with decreased perfusion of the brain and heart.[1] Therefore, assessing for signs and symptoms of poor tissue perfusion, such as angina, cyanosis, and altered mental status, is important. A baseline electrocardiogram is critical in patients with known cardiac disease because blood loss may precipitate cardiac ischemia. A loss of circulating blood volume may also result in decreased cerebral perfusion. The nurse should be alert to signs of agitation or confusion, which may signal cerebral hypoperfusion. The abdomen is assessed for bowel sounds; abdominal tenderness; the presence of guarding, rigidity, or abdominal masses; and the stigmata of liver disease. Splenomegaly, ascites, and caput medusae suggest liver disease. A tender, board-like abdomen is suggestive of peritonitis, possibly as a result of perforation. A rectal examination is essential to assess for hematochezia and melena.

LABORATORY STUDIES. Laboratory studies can help determine the extent of bleeding and can often provide a clue to the etiology. Common laboratory abnormalities for the patient with acute GIB are listed in Box 41-3. The initial hematocrit and hemoglobin may not accurately reflect initial blood loss because plasma volume is lost in the same proportion as red blood cells (RBCs). Within 24 to 48 hours of the initial bleeding, redistribution of plasma from the extravascular to the intravascular space results in a decreased hematocrit. Fluids administered during resuscitation contribute to the hemodilution. Leukocytosis and hyperglycemia may reflect the body's response to stress. Hypokalemia and hypernatremia may result from loss through emesis. An elevated blood urea nitrogen (BUN) level reflects a large protein load from the breakdown of blood. A high BUN/creatinine ratio suggests an upper GI source of bleeding.[1] Coagulopathy with a prolonged prothrombin time (PT) can indicate liver disease or concurrent long-term anticoagulant therapy. Thrombocytopenia may be present in patients with cirrhosis and portal hypertension with splenomegaly. If large amounts of blood are lost, metabolic acidosis occurs as a result of anaerobic metabolism. Severe blood loss can result in hypoxemia because of decreased circulating hemoglobin with impairment of oxygen transport to cells.

BOX 41-3 Typical Laboratory Abnormalities in a Patient With Acute Gastrointestinal Bleeding

- Decreased hemoglobin and hematocrit
- Mild leukocytosis and hyperglycemia
- Elevated blood urea nitrogen (BUN) level
- Hypernatremia
- Hypokalemia
- Prolonged prothrombin time (PT)/partial thromboplastin time (PTT)
- Thrombocytopenia
- Hypoxemia

Management

RESUSCITATION. The initial management of any patient with acute upper GIB is directed at fluid resuscitation to reverse the effects of blood loss. Supplemental oxygen is provided to any patient with acute GIB to promote oxygen saturation and transport as well as to prevent ischemia and dysrhythmias. Intubation may be required for actively bleeding patients at high risk for aspiration, those with a diminished mental status, and those in respiratory distress. Patients with acute upper GIB should be given nothing by mouth (NPO) because urgent endoscopy or surgery may be required. A Foley catheter is inserted to monitor urine output as an indication of the adequacy of fluid resuscitation. All patients with hemodynamic instability, a drop in hematocrit, transfusion requirements greater than 2 units of packed red blood cells (PRBCs), or active bleeding may warrant an ICU admission.

Volume Resuscitation. Patients with acute GIB require immediate intravenous (IV) access with at least two large-bore (14- to 16-gauge) IV catheters or central access. A type and cross-match should be sent early in the course of the bleeding because blood losses of greater than 1,500 mL require blood replacement in addition to fluids. While awaiting cross-matched blood, lactated Ringer's or normal saline solution is infused to restore circulating volume and to prevent the progression to hypovolemic shock. PRBCs should be transfused for a hemoglobin of 7 g/dL or less to reestablish the oxygen-carrying capacity of the blood.[12] Other blood products, such as platelets and clotting factors, are ordered according to results of laboratory tests and the patient's underlying condition. Calcium replacement may be necessary if large numbers of banked RBCs are transfused because the citrate in banked blood products can bind calcium and lead to hypocalcemia. A pulmonary artery catheter or central venous catheter may be useful to help avoid overresuscitation in patients with underlying renal or cardiac disease. In patients with a coagulopathy, vitamin K can be given in the form of phytonadione (AquaMEPHYTON), 10 mg intramuscularly or very slowly IV, in an attempt to restore the PT to normal. For patients receiving anticoagulants, correction of coagulopathy is recommended but should not delay endoscopy.[12] Fresh frozen plasma is ordered to correct the abnormality if rapid correction of the abnormality is warranted.

Vasoactive drugs may be used until fluid balance is restored to maintain blood pressure and perfusion to vital body organs. Dopamine, epinephrine, or norepinephrine may be ordered to stabilize the patient until definitive treatment can be undertaken.

Nasogastric Intubation. A large-bore nasogastric tube is placed in all patients with GIB to aspirate and lavage gastric contents. A nasogastric tube documents the presence and activity of bleeding. The color of gastric aspirate is prognostically significant. Coffee-ground or black nasogastric drainage with melenic stools indicates a slow bleed, whereas bright red nasogastric drainage and bright red blood in the stools signify a rapidly bleeding upper GI source.

A nasogastric tube is also useful for decompression and lavage. Lavage helps clear blood from the stomach, which allows better visualization to identify the source of bleeding during endoscopy. Iced lavage should be avoided because it is uncomfortable, fails to control bleeding, can significantly decrease core body temperature, and can trigger cardiac dysrhythmias. Lavage should be performed with tap water or saline. A total of 250 to 500 mL is instilled through the nasogastric tube and then removed with a syringe or by intermittent wall suction until gastric secretions are clear. Nasogastric tubes are usually removed after lavage of stomach contents unless the patient is actively bleeding or is experiencing severe nausea and vomiting because a nasogastric tube may injure the gastric mucosa and contribute to bleeding.

Acid-Suppressive Therapy. Acid impairs platelet aggregation and clot formation and promotes fibrinolysis.[6,7,13] Patients with acute upper GIB should be treated with acid-suppressive therapy to decrease the risk for recurrent bleeding, particularly from peptic ulcers. High-dose proton pump inhibitors (PPIs) (omeprazole, lansoprazole, esomeprazole, pantoprazole, rabeprazole) should be used to maintain a gastric pH greater than 6.0.[6,7] PPI therapy should be maintained for at least 72 hours after hemostasis has been achieved to prevent lysis of clots.[6,7] PPI therapy can be given either IV or PO. In the United States, both pantoprazole and esomeprazole are available for IV infusion.

Acid-suppressive therapy with histamine (H_2)-antagonistic drugs (H2RAs) (cimetidine, ranitidine, famotidine, nizatidine) is not recommended for patients with acute nonvariceal bleeding.[12] H2RAs may be used as prophylactic therapy in patients at high risk for stress-related erosive syndrome, but their use is limited by the rapid development of tolerance.[14,15]

Antacids may also be ordered, but their use is limited because of frequent dosing requirements and potential side effects. Antacids act as a direct alkaline buffer and are administered to control gastric pH. Sucralfate, a basic aluminum salt of sucrose octasulfate, acts locally as a cytoprotective drug and can be ordered for stress-related erosive syndrome prophylaxis.

Pharmacotherapy for Decreasing Portal Hypertension. Even before a bleeding source is identified, decreasing portal pressure with vasopressin or octreotide should be considered for patients in whom variceal hemorrhage is suspected. Vasopressin (Pitressin) decreases portal hypertension by constriction of the splanchnic arteries, which reduces portal blood flow. Vasopressin should be administered through a central line. Complications of vasopressin therapy can limit its use. Vasopressin reduces coronary blood flow and increases blood pressure, which increases oxygen demand, and causes coronary artery constriction, which can potentially result in multiple cardiac dysrhythmias. Because vasopressin also reduces blood flow to the mesenteric circulation, bowel ischemia can develop. To minimize these potential side effects, vasopressin should be given concurrently with IV, sublingual, or topical nitroglycerin, which reduces its systemic effects.

Somatostatin is a natural polypeptide that lowers portal venous pressure by vasoconstriction of splanchnic circulation. Somatostatin causes selective vasoconstriction of the splanchnic circulation and is associated with fewer systemic side effects than vasopressin. IV infusion is necessary because of its short half-life.

Octreotide (Sandostatin), a synthetic analog of somatostatin with similar hemodynamic properties but a longer half-life, is available in the United States. Octreotide causes a decrease in splanchnic blood flow with a resultant decrease in intravariceal pressure, decreases secretion of gastric acid and pepsin, and stimulates mucous production. Octreotide is usually given as a 50 to 100 mcg IV bolus followed by 50 mcg/h for 3 to 5 days. The effects of octreotide are similar to vasopressin with concurrent nitroglycerin infusion without the impact on hemodynamics or cardiac output.

DEFINITIVE DIAGNOSIS. After patients with acute upper GIB are resuscitated, endoscopy is considered. Endoscopy can be performed urgently at the bedside and is the procedure of choice for the diagnosis and treatment of acute upper GIB. Endoscopy within 12 to 24 hours of the initial bleeding has the best results. Early endoscopy is essential in acute GIB because the treatment is directed by the cause. Endoscopy allows the identification of the bleeding site between 90% and 95% of the time because direct mucosal inspection is possible.[7] The patient's hemodynamic status and endoscopic appearance provide prognostic value. The presence of active bleeding, a nonbleeding visible vessel, adherent clot, ulcer size greater than 2 cm, and ulcer location on the posterior lesser gastric curvature or posterior duodenal wall indicate a high risk for of rebleeding with medical therapy alone.[7,12] Vital signs must be monitored closely during endoscopy. The left lateral decubitus position decreases the risk for aspiration from active bleeding.

When diagnostic endoscopy is unsuccessful because of massive hemorrhage, angiography can be used to define the site of bleeding or abnormal vasculature. Angiography can detect bleeding rates as low as 0.5 to 1.0 mL/min.[3] Angiography is insensitive in the detection of venous bleeding.

Barium studies, such as an upper GI series, are of no value in acute upper GIB. These studies lack therapeutic capability and preclude endoscopy and angiography because of retained barium. Barium studies are also often inconclusive if there are clots or superficial bleeding in the stomach.

THERAPEUTIC INTERVENTION. In addition to its use in diagnosis, endoscopy is the procedure of choice for treating a GIB. If this fails to meet the needs of the patient, then additional therapeutic options are available.

Endoscopy. In 90% of cases, endoscopic therapy results in hemostasis, although 20% to 25% of sites may rebleed within 72 hours.[5,6,16] Multiple therapeutic options are available, including injection sclerotherapy, thermal coagulation, the placement of hemostatic clips, and endoscopic variceal ligation (EVL). The optimal technique depends on multiple variables, including the type and appearance of the lesion and the experience of the endoscopist.

The primary methods of endoscopic control of upper GI hemorrhage from peptic ulcers include injection therapy and thermal methods. Injection therapy consists of the injection of an agent such as epinephrine around and into the bleeding vessel. Thermal methods include heater probe and bipolar electrocoagulation (where a probe is applied with pressure to heat and seal the bleeding vessel). Hemostatic clips, called endoclips, have also been used successfully to ligate bleeding blood vessels within a lesion.

EVL is the treatment of choice for variceal bleeding. In EVL, a rubber band is placed endoscopically around the base of each varix. This causes coagulative necrosis and sloughing of thrombosed varices. EVL can control acute variceal bleeding in 90% of cases, with a reduction in the rate of rebleeding to between 10% and 20%.[11,16] An alternative to EVL is sclerotherapy. Injection sclerotherapy involves injecting the varices with a sclerosing agent to stop the bleeding. These agents cause local tamponade and vasoconstriction, causing necrosis and eventual sclerosis of the bleeding vessel. Acute hemostasis rates are similar to those with EVL, but sclerotherapy is associated with a higher complication rate.

Angiography. Most cases of GIB resolve spontaneously or can be controlled during endoscopy. However, those patients with persistent bleeding may require angiography to control the source of bleeding. During angiography, arterial GIB can be controlled by the infusion of intra-arterial vasopressin or by the embolization of the artery by an interventional radiologist. If therapeutic endoscopy fails, this is a useful therapeutic option, particularly in those who are critically ill and poor surgical candidates.

Intra-arterial vasopressin causes a generalized vasoconstriction that produces a rapid reduction in local blood flow. Patients should be monitored closely for dysrhythmias and fluid retention with resultant hyponatremia. Repeat angiography is performed after the initial infusion, and the dose can then be titrated as needed. Once bleeding is controlled, this infusion may be continued in the ICU for 24 to 36 hours and then tapered over 24 hours. Patients should have cardiac monitoring during vasopressin therapy to watch for cardiac dysrhythmias. Nitroglycerin patches or drips may be used to counteract any ischemic changes.

Embolization of a bleeding vessel consists of occluding the vessel with material that can be either temporary or permanent. Biodegradable long-acting gelatin sponges are commonly used. These sponges cause hemostasis on contact when injected into the vessel. Steel coils, balloons, and silk thread can be used to block an artery mechanically, resulting in permanent occlusion. Uncommon complications include bowel ischemia, secondary duodenal stenosis, and gastric, hepatic, or splenic infarction.[12]

Balloon Tamponade. Variceal bleeding unresponsive to endoscopic therapy can be temporarily controlled with balloon tamponade in 60% to 90% of cases.[10,16] Most esophagogastric tubes have two balloons, one for the stomach and one for the esophagus, and a distal port for gastric drainage. The Sengstaken-Blakemore tube is the most widely used (Fig. 41-1).

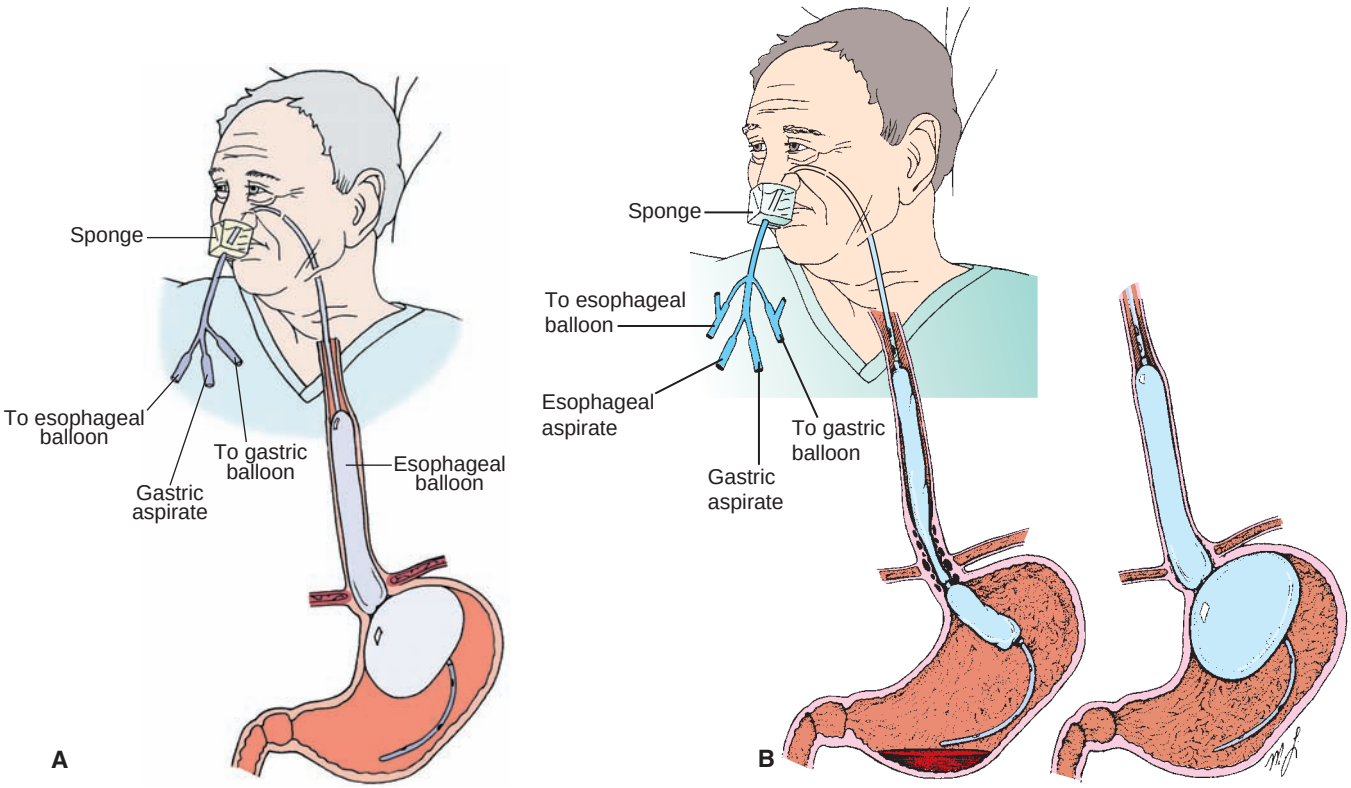


FIGURE 41-1 ▲ Comparison of two types of esophageal tamponade tubes. **A:** The Sengstaken-Blakemore tube is the best known. An additional tube must be placed in the proximal esophagus. **B:** The Minnesota esophagogastric tamponade tube includes an esophageal aspirate lumen.

With the use of balloon tamponade, pressure is exerted on the cardia of the stomach and against the bleeding varices. The tube is inserted to at least 50 cm to ensure gastric intubation. The gastric balloon is then slowly inflated with 250 to 300 mL of air, and gentle traction is applied until the gastric balloon fits snugly against the cardia of the stomach. Position is then confirmed by radiography. Traction is then placed on the tube where it enters the patient by means of a piece of sponge rubber, as shown in Figure 41-1, or by traction fixed to a head helmet device or the foot of the bed. If chest pain occurs, the gastric balloon must be deflated immediately because it may have shifted into the esophagus.

If bleeding continues, the esophageal balloon is inflated to a pressure of 25 to 39 mm Hg and maintained at this pressure for 24 to 48 hours. Although pressure for longer than 24 hours may be needed to control bleeding, it can cause edema, esophagitis, ulcerations, or perforation of the esophagus. After bleeding is controlled, the balloon is maintained and inflated for no longer than 12 to 24 hours to decrease the risk for gastric ischemia and necrosis. Unfortunately, rebleeding often occurs after balloon deflation unless additional therapeutic measures are taken.

A nasogastric tube should be placed in patients with a Sengstaken-Blakemore tube to aspirate oral and nasopharyngeal secretions that collect above the esophageal balloon, preventing aspiration of these secretions into the lungs. The Minnesota esophagogastric tamponade tube (see Fig. 41-1) has a suction port above the esophageal balloon in addition to the usual ports (two balloon, one gastric suction) of the Sengstaken-Blakemore tube. Nursing interventions for the patient with an esophageal tamponade tube are given in Box 41-4.

Transjugular Intrahepatic Portosystemic Shunt. A transjugular intrahepatic portosystemic shunt (TIPS) is a radiologic procedure that creates an intrahepatic shunt in an attempt to decrease portal pressure. The placement of a TIPS may be considered if other methods of managing esophageal varices fail.

Surgery. In the era of endoscopic therapy and PPIs, surgery is rarely used for the control of GIB. The indications for surgical intervention are severe hemorrhage unresponsive to initial resuscitation, massive bleeding that is immediately life threatening, unavailable or failed endoscopic therapy, perforation, obstruction, suspicion of malignancy, or continued bleeding despite aggressive medical therapies.

Surgical options for a bleeding peptic ulcer depend on the age and the condition of the patient, as well as on the location, size, and anatomy of the bleeding source. Emergency surgery of a bleeding duodenal ulcer may be a simple suturing (eg, oversew) of the ulcer. Bleeding duodenal ulcers can also be treated using one of the following procedures:

- Truncal vagotomy and pyloroplasty with suture ligation of the ulcer
- Truncal vagotomy and antrectomy with resection or suture ligation of the ulcer
- Proximal gastric vagotomy with duodenotomy and suture ligation of the ulcer

Bleeding gastric ulcers are commonly treated with one of the following procedures:

- Truncal vagotomy and pyloroplasty with wedge resection of the ulcer
- Antrectomy with wedge excision of the proximal ulcer
- Distal gastrectomy with or without truncal vagotomy
- Wedge resection of the ulcer

A vagotomy involves severing the vagus nerve, which innervates the gastric cells. This results in decreased gastric acid secretion. A truncal (gastric) vagotomy selectively cuts the vagus distribution to the stomach. A pyloroplasty is necessary in conjunction with the vagotomy because denervation of the vagus nerve affects gastric motility. A pyloroplasty allows for continued gastric emptying. An antrectomy removes acid-producing cells in the stomach. A Billroth I procedure includes a vagotomy and antrectomy with anastomosis of the stomach to the duodenum. A Billroth II procedure involves a vagotomy, resection of the antrum, and anastomosis of the stomach to the jejunum (Fig. 41-2). A gastric perforation can be surgically treated by simple closure or use of a patch to cover the mucosal hole.

Surgical decompression of portal hypertension can be used in patients with esophageal or gastric varices that are unresponsive to medical and endoscopic therapy. In this surgery, a portosystemic shunt is created, connecting the portal vein and the inferior vena cava to divert blood flow into the vena cava to decrease pressure.

BOX 41-4 NURSING INTERVENTIONS

For the Patient With an Esophagogastric Balloon Tamponade Tube

- Explain the purpose of the tube and the procedure to the patient.
- Lubricate and chill the tube as directed by the manufacturer.
- Identify and label the lumens of the tube.
- Check the patency of each lumen before insertion of the tube.
- Lavage the patient's stomach before insertion of the tube.
- Monitor the patient while the physician inserts the tube.
- Elevate the head of the bed to 30 degrees to prevent reflux.
- When a Sengstaken-Blakemore tube is in place, perform oropharyngeal suction frequently to prevent aspiration, *or* place a second nasogastric tube, if ordered, above the esophageal balloon to control secretions and prevent aspiration.
- Suction the esophageal port when a Minnesota tube is used.
- Maintain balloon pressure and traction.
- Maintain balloon position.
- Clean and lubricate the patient's nostrils frequently to prevent tube-caused pressure areas.
- Irrigate the nasogastric port every 2 hours to ensure patency and to keep the stomach empty.
- Teach the patient to avoid coughing or straining, which increases intra-abdominal pressure and predisposes to further bleeding.
- Have a second nasogastric tube, suction, and scissors available at the bedside.
- If the gastric balloon ruptures, the tube can rise into the nasopharynx, obstructing the airway. If this occurs, cut the tube immediately to deflate the balloon rapidly.
- Cut and remove the tube whenever there is a question of respiratory insufficiency or aspiration.
- Restrain the patient's arms if the patient is at risk for pulling out the tube. Agitation, confusion, and restlessness are risk factors.
- Assess for complications, including rupture or deflation of the balloon, pulmonary aspiration, and esophageal rupture.

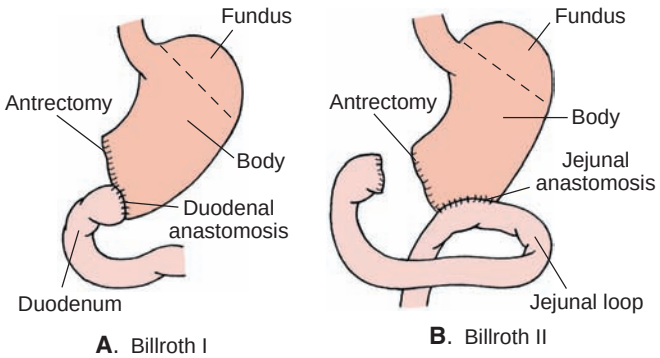


FIGURE 41-2 ▲ **A:** The Billroth I procedure includes a vagotomy and antrectomy with anastomosis of the stomach to the duodenum. **B:** The Billroth II procedure includes a vagotomy, antrectomy, and anastomosis of the stomach to the jejunum.

Medical Management. Once bleeding is controlled, management focuses on treating the underlying cause of the acute upper GIB and preventing rebleeding. For patients with peptic ulcer disease, eradication of *H. pylori* and elimination of NSAID use increase the healing rate and markedly reduce the recurrence of rebleeding. Patients treated for *H. pylori* should be retested to document eradication. PPI therapy should be continued at discharge based on etiology of bleeding. A combination of a PPI and a COX-2 inhibitor is recommended for use in patients who had previous bleeding from an ulcer and who require long-term aspirin or NSAID use.[12] Recurrent variceal hemorrhage occurs in approximately 70% of untreated patients within a year.[9,16] Esophageal varices can be obliterated in subsequent endoscopy sessions, and beta blockade with propranolol (Inderal) or nadolol (Corgard) to reduce portal pressure should be instituted to decrease the rebleeding rate. The dose should be titrated to achieve a 25% decrease in resting heart rate or a heart rate of about 55 beats/min as the desired end point.[6,9,11] The addition of isosorbide mononitrate may further decrease the risk for rebleeding. The use of prophylactic antibiotics in patients with acute bleeding varices has been shown to decrease the risk of rebleeding. Cessation of alcohol ingestion is imperative. See Box 41-5 for nursing interventions in the care of the patient with acute GIB.

BOX 41-5 NURSING INTERVENTIONS

For the Patient With Acute Gastrointestinal Bleeding

- Maintain a patent airway, elevate the head of the bed, and have suction available at the bedside to prevent aspiration of emesis or blood.
- Administer oxygen therapy to treat hypoxia that may result from decreased hemoglobin levels.
- Monitor pulse oximetry values.
- Assess and document signs and symptoms of shock, such as restlessness; diminished peripheral pulses; or cool, pale, or moist skin. Assess and document vital signs, urinary output, hemodynamic values, and oxygen saturation (SaO_2).
- Assess and document electrocardiographic monitoring and heart, lung, and bowel sounds.
- Assist with the placement of a central venous pressure (CVP) catheter or a pulmonary artery catheter.
- Monitor and document CVP, pulmonary artery pressure, pulmonary artery occlusion pressure, cardiac output, and systemic vascular resistance.
- Maintain IV access and administer IV fluids and blood products as ordered.
- Insert a nasogastric tube and lavage as ordered.
- Monitor gastric pH; consult with physician about specific pH range and antacid administration.
- Administer antisecretory medications as ordered to reduce gastric acid secretion.
- Administer vasopressin or octreotide as ordered.
- Maintain accurate intake and output every 1 to 2 hours and PRN.
- Record urine, nasogastric drainage, and emesis.
- Monitor electrolytes, which may be lost with fluids or altered due to fluid shifts, and report abnormal values.
- Monitor hemoglobin, hematocrit, red blood cell (RBC) count, PT, PTT, and BUN level and report abnormal values.
- Provide mouth care as needed.
- Explain all procedures to the patient.
- Prepare the patient for diagnostic procedures and therapeutic interventions.
- Monitor the patient for potential complications of endoscopy or colonoscopy, which include perforation, sepsis, pulmonary aspiration, and induced bleeding.
- Teach the patient the importance of seeking medical intervention if signs or symptoms of bleeding recur.
- Encourage smoking cessation and avoidance of alcohol.

Lower Gastrointestinal Bleeding

Etiology

Common causes of lower GIB are listed in Box 41-1 on page 917. Most cases of acute lower GIB that require ICU admission result from diverticulosis or angiodysplasia, although bleeding from neoplasm, colitis, inflammatory bowel disease, and hemorrhoids is also seen.

Diverticulosis

Diverticula are sac-like protrusions in the colon wall that usually develop at the point where arteries penetrate the colon wall. These vessels are separated from the bowel lumen only by the mucosa and are subsequently prone to injury. Diverticular bleeding accounts for 30% to 50% of all cases of acute lower GIB, with greater incidence in the elderly.[17–19] Most patients with diverticular bleeding will stop bleeding spontaneously, but up to 25% of bleeding may be massive, resulting in hemorrhage and the need for surgery.[18] Risk factors for diverticular bleeding include a diet low in fiber, aspirin and NSAID use, advanced age, and constipation.

Angiodysplasia

Angiodysplasia, also called arteriovenous malformation or angioma, is the term used to describe dilated, tortuous submucosal veins, small arteriovenous communications, or enlarged arteries. The walls of the vessels lack smooth muscle and are composed of endothelial cells. The incidence of angiodysplasia increases with age, owing to degeneration of the vessel walls; most cases occur in people older than 50 years, and two thirds occur in those older than 70 years. Acute lower GIB from angiodysplasia accounts for up to 20% of cases.[18] Angiodysplasia can occur anywhere in the colon, although

it most often occurs in the cecum or ascending colon. As opposed to bleeding from diverticula, bleeding from angiodysplasia may be venous or arteriovenous in nature and is therefore usually less severe than bleeding from diverticular disease, which is arterial. Angiodysplasia is a common cause of lower GIB in patients with renal disease.

Clinical Presentation

Acute lower GIB is defined by the presence of hemodynamic instability and the passage of hematochezia. Patients with diverticular bleeding usually describe the sudden onset of painless maroon or bright red hematochezia, although rarely, melena can occur. Diverticular bleeding is often painless, although patients may complain of cramping (which results from colonic spasm secondary to intraluminal exposure to blood). Blood loss from angiodysplasia usually presents as painless hematochezia.

If lower GIB is chronic, patients may present with iron deficiency anemia and symptoms related to the anemia, such as weakness, fatigue, or dyspnea on exertion. Massive bleeding from hemorrhoids is rare but can occur in patients with rectal varices from portal hypertension.

Assessment

HISTORY. Relevant findings in the medical history include abdominal surgery; a previous bleeding episode; peptic ulcer disease; inflammatory bowel disease; radiation to the abdomen or pelvis; or cardiopulmonary, renal, or liver disease. Knowledge of the patient's current medications and the existence of any allergies can also assist in diagnosis. A history of associated symptoms, including abdominal pain, fever, rectal urgency, tenesmus, weight loss, or a change in bowel habits or stool, should be elicited. The color and consistency of stool should be determined; in brisk bleeding, frequent red or maroon stools are more likely, and brown or infrequent stools are unlikely. The age of the patient may give a clue to diagnosis, because the risk for bleeding from diverticula and angiodysplasia increases with age.

PHYSICAL EXAMINATION. Often the physical examination findings are unremarkable. Vital signs are closely monitored to assess for hemodynamic instability. A palpable mass may reveal a neoplasm. A rectal examination is essential to assess for hematochezia and melena and exclude the possibility of bleeding hemorrhoids, which can occasionally present as a hemorrhage.

LABORATORY STUDIES. The initial laboratory studies include a complete blood count, serum electrolytes, BUN and creatinine levels, and PT and partial thromboplastin time (PTT). As in acute upper GIB, type and cross-match is mandatory before RBC transfusion.

Management

RESUSCITATION. The management of acute lower GIB requires aggressive fluid resuscitation, as described for acute upper GIB. Patients with hematochezia should have a nasogastric tube inserted to exclude an upper GI source of bleeding because 10% of suspected lower GIB occurs from upper GI sources.[6] The presence of bloody aspirate confirms an upper GI source of bleeding. However, the absence of blood does not exclude an upper GI source because bleeding from a site in the duodenum may not reflux into the stomach. Nasogastric aspirate that reveals bile without blood is unlikely in bleeding from an upper GI source. Once it is determined that bleeding is coming from a lower GI source, colonoscopy is the procedure of choice for both diagnosis and treatment.

DEFINITIVE DIAGNOSIS. Colonoscopy is the test of choice for the evaluation of lower GIB. It has a diagnostic accuracy of up to 95% in affected patients. Other advantages of colonoscopy are the ability to locate the source of the bleeding precisely, the ability to perform biopsies, and the potential for therapeutic intervention. Before colonoscopy in the acute care setting, the colon needs to be cleansed with 4 L of polyethylene glycol solution given orally or by nasogastric tube until the waste is clear. For those patients in whom bleeding has stopped, it is reasonable to perform colonoscopy on an elective rather than emergent basis. If a source of bleeding is identified during colonoscopy, therapeutic options include thermal coagulation or injection with epinephrine or other sclerosants, as discussed previously.

Endoscopy. Upper endoscopy should be performed if colonoscopy is unable to distinguish a lower GI source.

Radionucleotide Imaging. When colonoscopy fails to identify a bleeding source, radionucleotide scanning can detect bleeding that occurs at rates as low as 0.1 mL/min.[18] This is more sensitive than angiography but less specific than either colonoscopy or a positive angiogram. The two types of scanning available are the technetium (^{99m}Tc)–sulfur colloid and ^{99m}Tc pertechnetate–labeled autologous RBCs. Unfortunately, both of these techniques provide poor localization because of the peristaltic action of the bowel. However, these scans may be useful before angiography because a positive scan can aid in localizing the bleeding.

Angiography. Angiography is reserved for patients with massive, ongoing bleeding when endoscopy is not an acceptable option or with recurrent or persistent bleeding from a source not identified on colonoscopy. Angiography requires the active blood loss of 0.5 to 1.0 mL/min to localize a bleeding site because the contrast in the arterial system is present for only a short time.[18] A positive angiogram is associated with a high likelihood for surgical intervention. When an active source is identified, arteriographic intervention with intra-arterial vasopressin or embolization may be used. However, embolization with gelatin sponges, microcoils, or polyvinyl alcohol particles is replacing vasopressin because of the high incidence of complication and rebleeding after stopping the infusion. The nurse must be aware of the potential complications associated with arteriography, which include allergy to contrast medium, contrast-induced renal failure, bleeding from the arterial puncture site, and even embolism from thrombus.

SURGICAL INTERVENTION. Surgical management of lower GIB is indicated for massive or recurrent bleeding and in those patients with high transfusion requirements. An exploratory laparotomy to identify the source of the bleeding is often performed. A segmental bowel resection with a primary anastomosis is often necessary for definitive treatment of lower GIB. In patients who are unstable, a stoma and

mucous fistula may be created. In those patients with severe lower GIB without a localized source, a blind total colectomy may be the operative choice. Surgical management of diverticular bleeding is indicated if bleeding is not controlled with endoscopic or angiographic means or in patients with recurrent bleeding from the same segment.

▲ Intestinal Obstruction and Ileus

Intestinal obstruction occurs when the passage of intestinal contents through the lumen is impaired. This can result from either mechanical (anatomical) or nonmechanical causes. Intestinal obstruction is classified as either partial or complete, depending on the degree of obstruction. In a simple obstruction there is no ischemia, whereas in cases of strangulated obstruction, ischemia is present. A closed-loop obstruction describes a mechanical obstruction with a proximal and distal occlusion of the affected intestinal segment.

Bowel obstruction can occur in both the small and large bowel. The small bowel is most commonly affected, with the ileum as the most common site of obstruction. In large bowel obstruction, the sigmoid colon is the most common site of obstruction. The location of the obstruction, the degree of obstruction, and the presence of ischemia are important distinctions because treatment varies. Prompt recognition of bowel obstruction is important for the nurse because intestinal obstruction can progress to bowel strangulation, infarction, and perforation and result in potentially life-threatening peritoneal and systemic infection. The mortality rate associated with a strangulated obstruction is high.

The causes of mechanical obstruction are varied and classified as extrinsic, intrinsic, and intraluminal (Box 41-6). Extrinsic lesions occur outside of the bowel. Examples of extrinsic lesions are adhesions, hernias, volvulus (twisting of a segment of the bowel on itself), and masses. Intrinsic lesions extend into the bowel wall. Diverticulitis, neoplasms, and radiation enteritis are examples of intrinsic lesions. Intraluminal causes of obstruction can result from the ingestion of foreign bodies, intussusception, and neoplasms.

Small Bowel Obstruction

Etiology

Adhesions are the most common cause of small bowel obstruction (SBO) in adults and account for 50% to 75% of obstructions.[20–22] Adhesions most commonly occur after laparotomy for colectomy, appendectomy, or gynecological procedures. Adhesions can also develop after abdominal radiation, ischemia, or infection, or as the result of foreign bodies. Adhesions may develop only days after surgery and as late as 10 to 20 years later. Adhesive bands can form and contract and, in time, may entrap a loop of bowel.

Hernias are the cause of SBO in 10% to 15% of cases.[21,22] SBO secondary to hernia carries a high risk for complete obstruction and strangulation. The herniation of a portion of the bowel after laparotomy is called a Richter's hernia. The occurrence of SBO in the absence of previous laparotomy should suggest hernia as the cause.

BOX 41-6 Causes of Mechanical Obstruction

Extrinsic Lesions
- Adhesions and congenital bands
- Hernias
  - External hernias
  - Internal hernias
  - Diaphragmatic hernias
  - Pelvic hernias
- Volvulus
  - Gastric
  - Midgut
  - Cecal
  - Sigmoid
- Extrinsic masses
  - Benign or malignant tumors
  - Abscesses
  - Aneurysms
  - Hematomas
  - Endometriosis

Intrinsic Lesions
- Benign and malignant neoplasms
  - Adenocarcinomas
  - Lymphomas, lymphosarcomas
  - Carcinoid tumors
- Inflammatory conditions
  - Tuberculous enteritis, Crohn's disease
  - Strictures secondary to potassium chloride, nonsteroidal anti-inflammatory drugs, and ischemia
  - Radiation injury, caustic ingestants
  - Eosinophilic gastroenteritis, ameboma
  - Diverticulitis, pelvic inflammatory disease
- Intussusception
- Congenital defects
  - Hypertrophic pyloric stenosis, annular pancreas
  - Intestinal atresia/agenesis
  - Malrotation/volvulus
  - Intestinal duplication, mesenteric cysts
  - Meckel's diverticulum
  - Hirschsprung's disease
- Hematoma
  - Abdominal trauma
  - Thrombocytopenia
  - Henoch-Schönlein purpura

Intraluminal Causes
- Meconium ileus
- Barium impaction
- Fecal impaction
- Gallstone ileus
- Gastric bezoars
- Foreign bodies

From Yamada T, Alpers DH, Laine L, et al (eds): Textbook of Gastroenterology, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2003, p 834.

Tumors are uncommon in the small bowel, and primary neoplasms of the small bowel account for less than 5% to 10% of SBO.[21] Luminal compression of the small bowel or local invasion by gastric, pancreatic, colonic, and gynecological cancers can cause extrinsic compression, which accounts for most cases of SBO that result from malignancy. Intraluminal strictures that result from Crohn's disease, radiation therapy, ischemia, and certain drugs, such as enteric-coated potassium chloride or NSAIDs, are other possible causes of SBO.

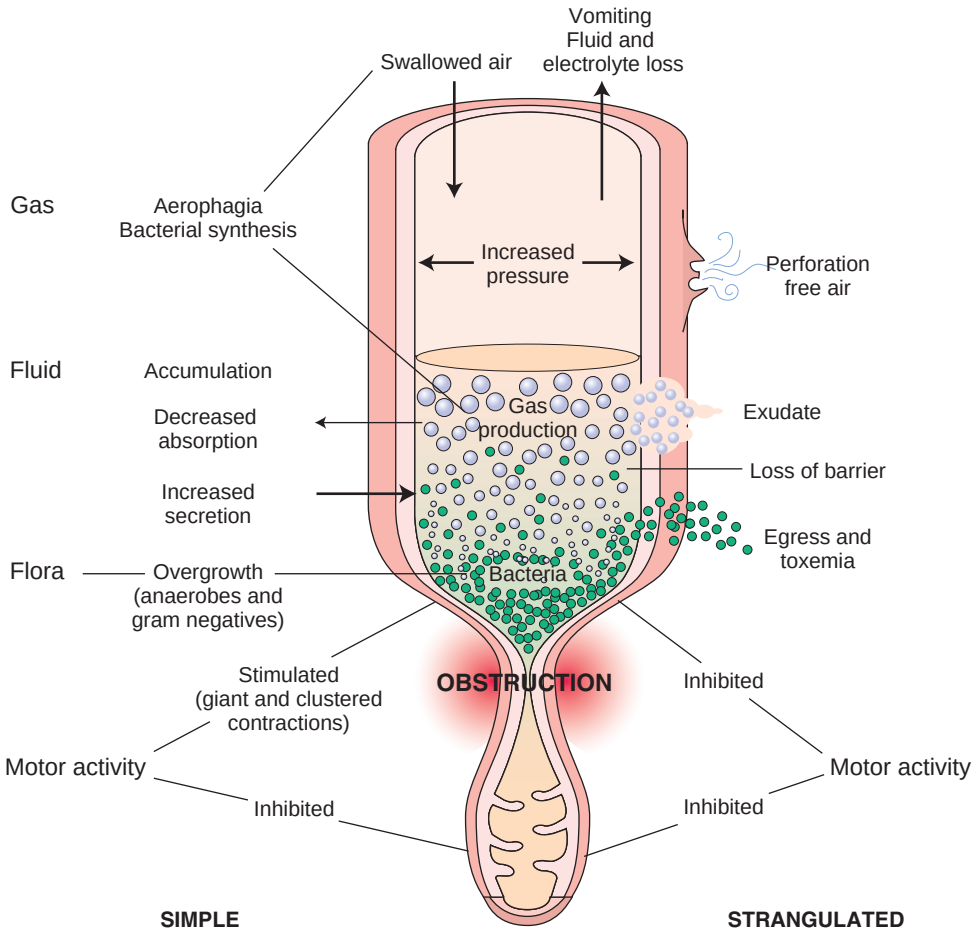


FIGURE 41-3 ▲ The pathophysiology of simple obstruction (**left**) and strangulated obstruction (**right**) in the small intestine. (From Yamada T, Alpers DH, Laine L, et al [eds]: Textbook of Gastroenterology, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2003, p 830.)

Pathophysiology

In SBO, large amounts of fluid and swallowed air accumulate in the intestinal lumen proximal to the obstruction, causing distention (Fig. 41-3). Fluid accumulates from oral intake; swallowed saliva; and gastric, biliary, and pancreatic juices. Swallowed air has a high nitrogen content and is poorly absorbed from the lumen.

As the obstruction continues, the bowel wall and lumen become edematous and distended. Increased intraluminal pressure leads to increased capillary permeability and movement of fluid and electrolytes into the abdominal cavity. This extravasation of fluid and electrolytes into the peritoneal cavity, combined with fluid lost through vomiting, can lead to hypovolemia, hypokalemia, and hyponatremia. Peristalsis decreases, and the normal functions of the intestine decrease or halt. In the absence of normal intestinal motility, bacterial overgrowth occurs. If oral intake continues, bacterial fermentation can contribute to gas accumulation. Within hours of acute obstruction, the contents of the lumen proximal to obstruction become malodorous and feculent because of this bacterial overgrowth.

Clinical Presentation

The severity of symptoms is related to the site and degree of obstruction, duration, and the presence and severity of ischemia (Table 41-1).Patients with SBO usually complain of the acute onset of intermittent, crampy, periumbilical pain. Bursts of peristalsis above the obstruction cause pain. The pain is often more severe the more proximal the obstruction. Patients with an incomplete obstruction often describe crampy abdominal pain after meals. The pain in incomplete obstruction may be exacerbated by the ingestion of high-fiber meals. Patients with a closed loop obstruction may describe pain out of proportion to physical findings.

In patients with proximal SBO, vomiting occurs frequently and early in the course of obstruction. The emesis is usually bilious, and vomiting often relieves the pain by deflating the distended bowel. Minimal abdominal distention usually accompanies proximal SBO.

In distal SBO, moderate abdominal distention and intermittent or constant pain are often present. Vomiting is intermittent. In ileal SBO, the emesis may be feculent secondary to bacterial overgrowth.

In strangulated SBO, the pain is more localized and may be steady and severe. When vomiting is protracted, dehydration and hypovolemia may occur.

Fever may be present secondary to an inflammatory process or in response to bowel ischemia or perforation. Constipation is also a common complaint, although patients may continue to pass gas and stool as the bowel distal to the obstruction empties. Obstipation is an important indicator of complete obstruction, but patients with a complete bowel obstruction evacuate the contents distal to the obstruction. Depending on the duration and severity of the obstruction, hemodynamic instability may develop as the result of massive fluid trapping in the lumen with leakage into the peritoneum.

Table 41-1 Clinical Features of Ileus and Obstruction Dependent on Anatomic Site

Feature	Ileus	Site of Obstruction			
		Gastric Outlet	Distal Duodenum	Jejunoileal	Colon
Pain	Mild	Mild	Mild	Moderate	Severe
Distention	Moderate to severe	Mild	Mild	Moderate	Severe
Emesis					
Amount/frequency	Small, infrequent	Copious, frequent	Copious, frequent	Smaller/less frequent	Uncommon
Nature	Sour, bilious	Clear, sour, HCl, KCl	Bile-stained, bitter, NaCl, $NaHCO_3$	Malodorous, feculent	Variable
Acid–base imbalance	Variable	Metabolic alkalosis	Metabolic acidosis	Dehydration, hypotension	Usually not severe

HCl, hydrogen chloride; KCl, potassium chloride; $NaHCO_3$, sodium bicarbonate; NaCl, sodium chloride.

From Yamada T, Alpers DH, Laine L, et al (eds): Textbook of Gastroenterology, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2003, p 833.

Assessment

HISTORY. A careful history provides clues to etiology. A medical history of previous abdominal surgery or trauma increases the risk for adhesions. Other pertinent medical history findings include inflammatory bowel disease, diverticulitis, abdominal or pelvic radiation, peptic ulcer disease, pancreatitis, and previous obstruction or cancer. Correlation to menses suggests endometriosis. A complete medication history is also essential. Patients with a psychiatric history should be questioned about ingestion of foreign objects.

PHYSICAL EXAMINATION. Patients with SBO often appear acutely ill. Inspection of the abdomen often reveals visible peristalsis and distention. Patients with a proximal SBO may have epigastric or periumbilical tenderness, whereas those with distal SBO often have more diffuse tenderness. Bowel sounds are usually hyperactive in the early course of the obstruction, then high-pitched and tinkling with loud rushes as peristaltic waves attempt to push intestinal contents past the obstruction. Bowel sounds decrease as the obstruction progresses and the bowel fatigues. Tachycardia, orthostatic hypotension, poor skin turgor, or dry mucous membranes may indicate dehydration. A palpable mass may represent a neoplasm or volvulus. It is necessary to perform a rectal examination to assess for blood, fecal impaction, or mass. Inspection may reveal scars and external hernias. Hepatomegaly, liver masses, and palpable periumbilical, inguinal, or supraclavicular lymphadenopathy suggest malignancy. Abdominal tenderness and palpable masses may suggest abscess. Fever, rigors, and declining clinical status suggest bowel strangulation. Borborygmi, rumbling, gurgling, and tinkling noises produced by hyperactive intestinal peristalsis are often audible and may correlate with abdominal cramping. If rebound tenderness is present, observe for signs and symptoms of shock because perforation is a possibility. Percussion of the abdomen may reveal resonance or tympany from fluid trapped in the intestine. Shifting dullness to percussion indicates ascites. Palpate for inguinal, femoral, and umbilical hernias. A tender mass at the site of a hernia suggests the etiology. Tachycardia, tachypnea, altered mental status, oliguria, and hypotension may all be present in hypovolemia.

LABORATORY STUDIES. There is no single laboratory value that is diagnostic for SBO. A mild leukocytosis is present with simple obstructions, whereas significant leukocytosis suggests strangulation. In proximal obstruction, potassium, sodium, hydrogen, and chloride may be lost in emesis, resulting in metabolic alkalosis. BUN, creatinine, sodium, and osmolality levels reflect the fluid and electrolyte shifts that occur as fluid leaks out of the intestine and electrolytes are either reabsorbed or lost. As dehydration increases, the hemoglobin and hematocrit levels are elevated, reflecting hemoconcentration. In ischemia or strangulation, amylase, lipase, alkaline phosphatase, creatine phosphokinase, aspartate aminotransferase (AST), alanine aminotransferase (ALT), and lactate dehydrogenase levels may rise. Often, heme-positive stools are present in ischemia or carcinoma. Metabolic acidosis suggests severe hypoxemia from hypoperfusion, and metabolic acidosis refractory to fluid resuscitation suggests strangulation.

IMAGING STUDIES. Various imaging studies are available to confirm diagnoses.

Radiography. When SBO is suspected, abdominal radiographs with the patient in upright, flat, and side-lying positions can confirm the diagnosis of obstruction, localize the site of obstruction, and assist in determining the degree of obstruction. Pneumoperitoneum seen in upright films suggests intestinal perforation. Normally, there is little air in the small bowel. In complete SBO, gas and fluid accumulate proximal to the obstruction. Multiple air–fluid levels may be visible, with a stepladder pattern that demonstrates multiple loops of bowel with different air levels. Distal to the obstruction, the bowel lumen empties and collapses within 12 to 24 hours. Successive films may also confirm the diagnosis, but distinguishing between a small and large bowel obstruction or ileus is difficult.

Barium studies may be helpful in the diagnosis of obstruction if plain films are nondiagnostic. Contrast-enhanced studies can differentiate between a complete or partial obstruction. Barium is the agent of choice if SBO is suspected because it provides a better contrast than water-soluble material. In SBO, the large amount of water present proximal to the obstruction dilutes water-soluble contrast. However, if there is any question about bowel perforation, barium should

be avoided because free barium in the peritoneum can cause significant inflammation. If colonic obstruction is suspected, a limited barium enema is used for diagnosis before barium is given by mouth.

Computed Tomography. Abdominal computed tomography (CT) can help identify obstructive lesions, neoplasms, hernias, and signs of ischemia. Abdominal CT with oral or IV contrast medium can help differentiate mechanical obstruction from pseudo-obstruction. Oral contrast seen in the colon on CT 12 hours after ingestion indicates incomplete SBO, whereas nonvisualization of oral contrast in the colon within 12 hours indicates complete SBO.[21] The CT diagnosis of a complete SBO requires a transition zone between dilated and collapsed loops of bowel, suggesting the point of obstruction. CT is less sensitive in the diagnosis of a partial SBO. CT can also assess the entire abdomen, which can suggest alternative diagnoses and identify any complications associated with obstruction. CT is also accurate in determining the presence of strangulation or a closed-loop obstruction.

Endoscopy. Direct visualization with endoscopy may confirm the obstruction in the colon or proximal small bowel and aid in determining the type of obstruction.

Management

MEDICAL MANAGEMENT. When possible, obstructions, especially incomplete obstructions, are treated medically rather than surgically. Oral food and fluid are withheld (ie, the patient is put on NPO status), and a nasogastric tube is placed to decompress the stomach or duodenum. Fluid and electrolytes are aggressively supplied IV with lactated Ringer's or saline solution. When possible, the underlying causes are treated. Total parenteral nutrition (TPN) may be required to provide nutritional support. A Foley catheter is inserted to allow continual assessment of the fluid replacement. In patients with renal or cardiac disease, a central venous pressure (CVP) or pulmonary artery catheter may guide fluid replacement.

About 90% of SBO resolve spontaneously, and if patients continue to pass gas and stool, supportive management is continued.[21] If patients show no improvement within 24 to 48 hours, or if fever or rebound tenderness occurs, a surgical evaluation is indicated. All patients with intestinal obstruction should be watched closely for signs and symptoms that reflect sepsis, perforation, ischemia, necrosis, or gangrene. Broad-spectrum antibiotics are started immediately when strangulation or sepsis is suspected. The mortality rate associated with bowel ischemia resulting from obstruction is high.

SURGICAL MANAGEMENT. Acute complete SBO is a surgical emergency. Acute complete SBO is suspected when the patient fails to pass gas, and stool and gas are not evident in the distal intestine on radiography. An acute complete SBO is accompanied by the risk for bowel strangulation. Patients with strangulated bowel, volvulus, and incarceration of bowel loop in a hernia or a closed-loop obstruction require immediate surgery. In addition, those patients who fail conservative therapy or experience a decline in clinical status warrant surgical intervention.

Surgical procedures include laparoscopic lysis of adhesions, reduction of volvulus, resection of the involved and surrounding area of bowel with impaired blood supply, bowel decompression, and possible ostomy. These patients may require a second surgery to assess bowel viability.

Colonic Obstruction

Etiology

Carcinoma, sigmoid diverticulitis, and volvulus are the three most common causes of colonic obstruction and together account for the majority of colonic obstructions. Malignancy is the most common cause of colonic obstruction in the United States, accounting for approximately 60% to 75% of cases.[21,23] Patients with colorectal cancer present with a colonic obstruction between 10% and 20% of the time.[21] Malignancy that causes colon obstruction occurs most commonly in the sigmoid colon. Extrinsic compression or colonic invasion from pelvic tumors may also cause a colon obstruction. Diverticulitis can cause strictures in the colon that can lead to mechanical obstruction, resulting in approximately 10% of cases of colonic obstruction.[21] Volvulus, which occurs most commonly in the sigmoid colon and the cecum, causes 10% to 15% of colonic obstructions in the United States.[21] A closed-loop obstruction is usually produced with volvulus and carries a high incidence of strangulation. Other causes of colon obstruction include anastomotic or inflammatory strictures.

Pathophysiology

When the ileocecal valve is competent, a closed-loop obstruction can occur because the cecum does not allow decompression of fluid and gas into the small bowel. As fluid and gas accumulate, the intraluminal pressure increases, and the colonic wall can become ischemic if this pressure exceeds the capillary pressure. In some cases, the cecum may become so severely distended that it inhibits intramural blood flow, which can result in necrosis and gangrene. In colonic obstruction, the normal colonic flora produces methane and ammonia, which contribute to the distention. Dehydration results when secretions are sequestered in the colon.

Patients with colonic obstruction have changes in intestinal flora and translocation of bacteria in mesenteric lymph nodes. This is the most likely cause of septic complications of colonic obstruction.

Clinical Presentation

The clinical presentation of patients with colonic obstruction depends on the degree of obstruction, the cause, the presence of comorbidity, the presence of closed-loop obstruction, and the competency of the ileocecal valve. Patients with colonic obstruction typically present with abdominal pain, distention, and progressive obstipation. The pain may be colicky or severe and unremitting if peritonitis is present. Severe, constant pain suggests gangrenous bowel. If vomiting occurs, it tends to be late in the course of obstruction, especially in patients with a competent ileocecal valve. Patients with volvulus may present with a sudden onset of marked abdominal distention. Patients with obstruction from colon cancer may describe a gradual development of symptoms like altered bowel habits or a change in stool caliber. Dehydration results when secretions become sequestered in the colon. Patients with a competent ileocecal valve may have greater

distention, which increases the risk for ischemia and perforation because an incompetent ileocecal valve allows decompression into the small intestine. Most patients with colonic obstruction complain of constipation; however, diarrhea may be present if stool is leaking past an obstruction. Patients may complain of dyspnea if diaphragmatic excursion is compromised by abdominal distention.

Assessment

HISTORY. A history of altered bowel movements, blood in the stool, or iron deficiency anemia is suggestive of carcinoma, as are weight loss and anorexia. Diverticulitis typically presents with left lower quadrant pain and associated fever. There may be a change in bowel habits as well. Bleeding is not usually associated with diverticulitis. A history of laxative use and constipation is common in patients with volvulus.

PHYSICAL EXAMINATION. Abdominal distention is common, and tympany is present with percussion. Signs of dehydration including tachycardia, hypotension, poor skin turgor, and dry mucous membranes may be seen. Bowel sounds are usually hyperactive initially but become progressively hypoactive. Abdominal masses and signs of peritoneal irritation may be elicited. The abdomen may be diffusely tender. Guarding or rebound tenderness suggests peritonitis. Ascites and hepatomegaly may be present in patients with colon cancer with liver metastasis. A rectal examination may be helpful in identifying rectal cancer. High fever and tachycardia, regardless of rehydration or the presence of peritoneal signs, suggest strangulation and warrant urgent surgical evaluation.

LABORATORY STUDIES. Iron deficiency anemia may be present if obstruction results from neoplasm. Marked leukocytosis suggests diverticulitis, ischemia, or perforation.

IMAGING STUDIES. Plain abdominal films in the supine and upright positions identify the site of obstruction and determine the degree of obstruction. Obstruction in patients with a competent ileocecal valve causes dilation confined to the colon. Small bowel distention may be visible on abdominal films in patients with acute colonic obstruction and in patients with intact ileocecal valves. Abdominal films may also suggest volvulus.

If a contrast enema is given, water-soluble contrast medium rather than barium may be considered. Barium should never be given orally unless a barium enema, CT scan, or colonoscopy has ruled out colonic obstruction. Oral barium accumulates proximal to the colonic obstruction, and water will continually be extracted, possibly causing a barium impaction. In patients with suspected volvulus, a water-soluble contrast enema may demonstrate a point of torsion.

CT may be valuable in distinguishing between an anatomic obstruction and a pseudo-obstruction. CT can also diagnose other causes of colonic obstruction, such as inflammation as a result of colitis or diverticulitis and perforation as a result of colorectal cancer.

Management

MEDICAL MANAGEMENT. The medical management of the patient with acute colonic obstruction is similar to that of the patient with SBO. Medical management focuses on fluid and electrolyte replacement. Oral intake is limited, or the patient is placed on NPO status. Nasogastric suction may assist in decompression of abdominal distention. A rectal tube may decompress the distal colon but has little effect on the proximal colon. Colonic decompression in the setting of a volvulus can be attempted by colonoscopy.

SURGICAL MANAGEMENT. Colonic obstruction usually requires surgery. The goals of surgery are to decompress the colon and to treat the obstructive lesion. Surgical management of the colonic obstruction is warranted if the patient fails to improve with medical management, the patient's clinical status deteriorates, or the patient has a complete colonic obstruction with a competent ileocecal valve. For obstruction of the left colon, operative decompression followed by primary anastomosis after intraoperative lavage is the treatment of choice. For obstruction in the transverse and right colon, primary resection and anastomosis can also be performed safely. Primary anastomosis should be avoided in a patient with an unprepared colon.

ENDOSCOPIC THERAPY. Stents that are placed endoscopically can be used as a temporary measure before surgical resection of obstruction from malignancy or as a palliative measure in nonoperative colorectal cancer. Endoscopic laser therapy, argon plasma coagulation, and snare polypectomy may be used to debulk obstructing tumors in patients unwilling or unable to have surgery.[23]

Ileus

Ileus, often called paralytic ileus or adynamic ileus, is the failure of intestinal contents to pass because of decreased peristalsis activity in the absence of mechanical obstruction. Ileus can have intra-abdominal or extra-abdominal causes (Box 41-7), many of which are likely to be seen in the ICU setting. Acute colonic pseudo-obstruction (ACPO), also called acute colonic ileus and Ogilvie's syndrome, is a variant of ileus characterized by massive colonic dilation in the absence of mechanical obstruction.

Etiology

Postoperative ileus (a transient inhibition of normal GI motility that usually lasts for 3 to 5 days after surgery) is the most common cause of a delayed discharge after surgery, abdominal or otherwise.[24,25] An underlying disease is present in the majority of ileus. Causes of ileus include metabolic abnormalities (electrolyte disturbances, diabetic ketoacidosis, uremia, heavy metal poisoning), drugs (narcotics, catecholamines, antihistamines, calcium channel blockers, adrenocorticotropic hormones, anticholinergics), and local or systemic inflammation (sepsis, peritonitis, ischemia, pancreatitis). Ileus may also be present after spinal cord injury. Blood-borne toxins, abnormalities in acid–base balance, electrolyte disturbances, and decreased oxygen supply are all possible causes of ileus.

Pathophysiology

Although the etiology of ileus can be defined, the pathophysiology of ileus is poorly understood. Postoperative ileus has been widely studied, and multiple mechanisms are thought

BOX 41-7 Causes of Adynamic Ileus and Acute Colonic Pseudo-Obstruction

Intra-Abdominal Causes

Reflex Inhibition
- Laparotomy
- Abdominal trauma
- Renal transplantation

Inflammatory Conditions
- Perforated viscus or penetrating wounds
- Bile peritonitis
- Chemical peritonitis
- Intraperitoneal hemorrhage
- Toxic megacolon
- Familial Mediterranean fever
- Acute pancreatitis
- Acute cholecystitis
- Celiac disease
- Inflammatory bowel disease

Acute Irradiation Injury
- Abdominal irradiation

Infectious Processes
- Bacterial peritonitis
- Appendicitis
- Diverticulitis
- Herpes zoster virus
- Anorectal herpes simplex virus

Ischemic Processes
- Arterial insufficiency
- Venous thrombosis
- Mesenteric arteritis
- Strangulation obstruction

Retroperitoneal Processes
- Ureteropelvic stones
- Pyelonephritis
- Retroperitoneal hemorrhage
- Pheochromocytoma
- Malignancy (Ogilvie's syndrome)

Extra-Abdominal Causes

Reflex Inhibition
- Craniotomy
- Spine or pelvic fractures
- Myocardial infarction
- Coronary bypass
- Open heart surgery
- Pneumonia, pulmonary embolus
- Burns
- Black widow spider bites

Drug-Induced
- Anticholinergic/ganglionic antagonists
- Opiates
- Chemotherapeutic agents
- Tricyclic antidepressants
- Phenothiazines

Metabolic Abnormalities
- Septicemia
- Electrolyte imbalance
- Heavy metal poisoning (lead, mercury)
- Porphyria
- Uremia
- Diabetic ketoacidosis
- Sickle cell disease
- Pulmonary failure

From Yamada T, Alpers DH, Laine L, et al (eds): Textbook of Gastroenterology, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2003, p 836.

to play a role, including sympathetic neural reflexes that inhibit normal bowel motility, local and systemic inflammatory mediators that result in bowel edema, and changes in neural and hormonal transmitters.[24,25] The effects of anesthesia, combined with inflammation or ischemia in the operative area, may also interfere with nerve conduction. Opioid narcotics may also contribute to postoperative ileus because they decrease propulsive motility of the intestine. In ileus, peristalsis ceases or is decreased, and distention of the intestine occurs as gas, fluid, and electrolytes accumulate in a process similar to that seen in mechanical obstruction.

Clinical Presentation

Patients with ileus may complain of diffuse abdominal discomfort and distention (see Table 41-1, p. 927). ACPO is more common in men and patients 60 years of age and older.[24] Nausea and vomiting are often predominant in patients with postoperative ileus. Vomiting is frequent, and the emesis usually contains gastric contents and bile. The vomiting of feculent material is rare. The pain is usually less intense than in small bowel or colonic obstruction. The patient with ileus also complains of constipation and usually denies the passage of flatus. Other common symptoms are nausea, anorexia, hiccups, and bloating.

Assessment

HISTORY. A history of thyroid or parathyroid disease, heavy metal exposure, diabetes mellitus, and scleroderma should be elicited to identify underlying causes.

PHYSICAL EXAMINATION. Abdominal distention is often prominent in ileus. Auscultation of the abdomen usually reveals infrequent or absent bowel sounds. The abdomen is usually resonant to percussion secondary to air in the dilated loops of intestine. Abdominal distention may cause labored breathing. Abdominal girth is assessed at frequent intervals. Peritoneal signs may indicate impending perforation. Tachycardia, orthostatic hypotension, poor skin turgor, or dry mucous membranes may indicate dehydration.

LABORATORY STUDIES. Electrolyte abnormalities commonly associated with ileus are similar to those seen in patients with mechanical obstruction.

IMAGING STUDIES. Abdominal radiography that shows massive colonic dilation confirms the diagnosis of ileus. In ileus, gas and fluid accumulate in loops of mildly dilated bowel proximal or adjacent to the site of an acute inflammatory process, such as appendicitis or pancreatitis. These loops are involved in localized ileus and are called sentinel loops. In ACPO, the entire colon becomes dilated, with the cecal diameter the greatest. Chest radiography may help identify pneumonia or other causes of ileus. Contrast-enhanced enema studies can be used to differentiate complete obstruction from partial obstruction and ileus. CT of the abdomen may identify causes that can contribute to ileus. Ultrasonography has no role in the diagnosis of ileus because the dilated loops of bowel prevent imaging.

Management

Treatment of ileus focuses on management of underlying causes. Because ileus may present in much the same way as mechanical obstruction, exclusion of mechanical causes is necessary. Treatment usually consists of supportive care. Patients with ileus have traditionally been placed on NPO status, although recent studies suggest that early feeding is safe. Fluid and electrolyte replacement is directed by clinical status and laboratory values as needed. Nasogastric suction limits the collection of swallowed air that can contribute to abdominal distention. Medications that can adversely affect colonic motility including narcotics and anticholinergics should be discontinued when possible. Laxative use should be avoided because these agents can provide a substrate for bacterial fermentation, which results in further gas accumulation. In addition, patients should be mobilized and encouraged to get out of bed if they are ambulatory.

If patients show no improvement in 3 to 5 days, a further search for underlying causes is initiated. Neostigmine has been effective in treating colonic ileus not responsive to conservative therapy. Neostigmine is a parasympathomimetic that can correct the autonomic imbalance thought to contribute to ileus. Neostigmine can cause bradycardia and dysrhythmias, so careful cardiac monitoring is required. Prokinetic medications, such as metoclopramide (Reglan) and erythromycin, have not been found to be effective in treating ileus.

Therapeutic interventions for decompressing the colon include colonoscopy, open or percutaneous cecostomy, and a decompression colostomy. Colonoscopy is the procedure of choice for decompression in patients who do not respond to conservative or medical therapy.

Surgery is indicated for patients who fail conservative management or in patients who develop a perforation or evidence of ischemia.

▲ Acute Pancreatitis

Acute pancreatitis (AP) is an acute inflammation of the pancreas that can also involve surrounding tissues, remote organs, or both. There are approximately 300,000 hospital admissions a year in the United States caused by AP.[26,27] AP refers to an acute attack in a previously healthy individual, with resolution of symptoms after the attack. Chronic pancreatitis refers to repeated attacks with continued symptoms. AP can be mild or severe. Mild AP is not associated with organ dysfunction or complications, and recovery is usually uneventful. Approximately 10% to 20% of patients with AP develop severe pancreatitis, also called necrotic or hemorrhagic pancreatitis.[26] In severe AP, there is extensive fat necrosis in and around the pancreas, pancreatic cellular necrosis, and hemorrhage in the pancreas. Severe AP is associated with local and systemic complications. The incidence of AP varies among populations based on the prevalence of precipitating factors, such as alcohol use and gallstone disease.

Etiology

There are multiple causes of AP (Box 41-8). Gallstones and excessive alcohol use together account for 70% to 80% of cases.[26–28]

Gallstones are responsible for 40% of cases.[26,27] Gallstones and biliary sludge may become lodged as they pass through biliary system, blocking pancreatic secretions from emptying into the duodenum. Reflux of bile into the pancreatic duct resulting from this obstruction is thought to be the inciting factor. Gallstone pancreatitis is more common in women.

Alcoholism is the second leading cause of pancreatitis and accounts for 35% of the cases of AP.[26,27] The exact mechanism by which alcohol induces AP is unknown. Alcohol may cause overstimulation of the pancreas and activation of pancreatic secretions within the pancreas. Alcohol may also have a direct toxic effect. Another theory is that alcohol causes sphincter of Oddi spasm, which causes pancreatic enzymes to back up into the pancreas.[26,27] Alcoholic pancreatitis is more common in men. AP is rarely the result of binge drinking unless the pancreas is already damaged by chronic alcohol use. Drinking five to eight alcoholic beverages a day for more than 5 years is a risk factor for the pancreatitis.[26,27]

BOX 41-8 Major Causes of Acute Pancreatitis

- Biliary disease: gallstones or microlithiasis, common bile duct obstruction, biliary sludge
- Pancreas divisum
- Alcohol abuse
- Drugs: thiazide diuretics, furosemide, procainamide, tetracycline, sulfonamides, azathioprine, 6-mercaptopurine, angiotensin-converting enzyme inhibitors, valproic acid
- Hypertriglyceridemia
- Hypercalcemia
- Idiopathic
- Miscellaneous (postoperative, ectopic pregnancy, ovarian cyst, total parenteral nutrition)
- Abdominal trauma
- Endoscopic retrograde cholangiopancreatography
- Infectious processes

Metabolic causes of AP include hypercalcemia and hypertriglyceridemia. Hypertriglyceridemia accounts for up to 4% of all cases of AP.[26–28] Many drugs, including diuretics, sulfonamides, metronidazole, aminosalicylates, and estrogen, can precipitate AP as a result of toxic metabolites or a drug reaction. Idiopathic pancreatitis is associated with pregnancy, the administration of TPN, or major surgery. Pancreatitis has also occurred after blunt or penetrating abdominal trauma or after endoscopic manipulation of the ampulla of Vater. Other possible precipitating factors include infectious processes, such as mumps, staphylococcal infection, scarlet fever, and viral infections, as well as the congenital variant of pancreas divisum. Pancreatitis may occur as an isolated event, or the patient may suffer repeated attacks.

Pathophysiology

The acinar cells of the pancreas synthesize and secrete digestive enzymes to assist in the breakdown of starch, fat, and proteins. Under normal circumstances, these enzymes remain inactive until they enter the duodenum. As pancreatic juice enters the duodenum, trypsinogen is activated by enterokinase into its active form, trypsin.

In AP, pancreatic enzymes become prematurely activated in the pancreas. This premature activation results in autodigestion of the pancreas and the peripancreatic tissue. The exact mechanism by which pancreatic enzymes become activated and initiate autodigestion is not fully understood. However, the activation of trypsinogen is thought to be the critical event that promotes the activation of other injurious enzymes, including elastase, kinases, and phospholipase A. Elastase can cause dissolution of elastic fibers in blood vessels, which can lead to hemorrhage. Activated kinins cause systemic vasodilation and increased vascular permeability, which promotes edema. Phospholipase A causes necrosis of the pancreas and the surrounding fatty tissue.

Pancreatic enzymes, vasoactive substances, hormones, and cytokines released from the injured pancreas cause a cascade of events that can lead to edema, vascular damage, hemorrhage, and necrosis. Systemic effects mediated by the immune system can lead to a systemic inflammatory response syndrome (SIRS), which can result in distant organ damage and multisystem organ failure. This immune response is independent of the event that causes AP but is responsible for the majority of the morbidity and mortality associated with it.

Clinical Presentation

Abdominal pain is the hallmark of AP and occurs in 95% of cases.[27] The severity of the pain correlates to the degree of pancreatic involvement. The pain is usually deep and boring, midepigastric or periumbilical, with radiation to the back, but it may radiate to the spine, flank, or left shoulder. The pain usually begins abruptly and increases in intensity over several hours. It is usually steady, but can be exacerbated by intake. Pain associated with gallstone pancreatitis may be more localized to the right upper quadrant, more colicky, and more variable in intensity. The pain is usually exacerbated when the patient lies supine and is usually relieved when the patient sits and leans forward or lies in a fetal position. Patients are often restless and agitated. Nausea and vomiting without pain relief is present 90% of the time.[27] Tachycardia, abdominal distention, and hypotension are other common symptoms. A low-grade fever may or may not be present. A persistent fever may indicate complications, such as peritonitis, cholecystitis, or intra-abdominal abscess.

The diagnosis of AP is often challenging because AP can mimic many other conditions. The differential diagnosis includes gastritis, perforated duodenal or gastric ulcers, acute SBO, ruptured ectopic pregnancy, sickle cell crisis, acute cholecystitis, mesenteric artery occlusion, and ruptured aortic aneurysm. Diagnosis is made on the basis of the patient's clinical presentation, history, physical examination findings, and the results of laboratory and radiographic studies (Box 41-9).

BOX 41-9 Clinical Manifestations of Acute Pancreatitis

Physical Examination Findings
- Abdominal pain
- Low-grade fever
- ± Jaundice
- Abdominal guarding or distention
- Paralytic ileus
- Grey Turner's sign
- Cullen's sign
- Nausea or vomiting without relief

Laboratory Findings
- Elevated serum and urine amylase
- Elevated serum lipase
- Elevated white blood cell (WBC) count
- Hypokalemia
- Hypocalcemia
- Elevated bilirubin, aspartate aminotransferase (AST), and PT (with liver disease)
- Elevated alkaline phosphatase level (with biliary disease)
- Hypertriglyceridemia
- Hyperglycemia
- Hypoxemia

Assessment

History

A careful history can provide important clues to diagnosis. A history of biliary tract disease, alcohol intake, diabetes, and medication use should be elicited to identify precipitating causes. A family history of AP may suggest hereditary causes. The patient may report anorexia, weight loss, nausea, vomiting, or abdominal distention. Assessment of the location, duration, quality, quantity, and precipitating factors of pain is important to help identify potential causes.

Physical Examination

Diffuse abdominal tenderness and guarding may be present during abdominal palpation. The upper abdomen may be distended and tympanic to percussion. Bowel sounds may be hypoactive or absent, owing to decreased intestinal mobility or paralytic ileus. Jaundice may be present in gallstone disease or from obstruction of the biliary tree from pancreatic edema. Ascites or palpable abdominal masses may be present. Patients with severe acute hemorrhagic pancreatitis may have signs of dehydration and hypovolemic shock. These signs may worsen when fluid is lost into the bowel lumen because of a paralytic ileus. The presence of a bluish discoloration of the lower abdominal flanks (Grey Turner's sign) or around the umbilical area (Cullen's sign) indicates hemorrhagic pancreatitis and an accumulation of blood in these areas. These findings are rare, but if they occur, the findings usually do not appear until 48 hours or more after onset of symptoms.

Laboratory Studies

No single laboratory study is diagnostic of AP; however, elevations of serum amylase and lipase enzymes are often seen in AP (see Table 39-7, p. 884). These enzymes are released as the pancreatic cells and ducts are destroyed. Serum amylase levels rise within 2 to 12 hours of the onset of symptoms and gradually return to baseline within 3 to 5 days in AP. In mild pancreatitis, amylase levels can be close to normal. If a few days have elapsed since symptoms began, amylase values can also be normal even with an active inflammatory process in the pancreas. The sensitivity of serum amylase is limited in patients with hypertriglyceridemia and in patients with an acute chronic alcoholic pancreatitis.[27] The specificity of serum amylase is decreased in biliary tract disease, tumors, salivary gland lesions, cerebral trauma, gynecological disorders, and renal failure. However, serum amylase levels that are more than three times the upper limits of normal are highly specific for pancreatitis.[26,27]

Compared with serum amylase levels, serum lipase levels rise later and remain elevated. Serum lipase levels usually

rise within 4 to 8 hours of the onset of symptoms, peak at 24 hours, and return to normal after 8 to 14 days. Because the serum lipase level stays elevated longer, it is a useful test in diagnosis if there is a delay in examination. Like amylase, serum lipase levels may be elevated in patients who have intra-abdominal inflammation or renal insufficiency.

Elevations of isoenzymes, urinary amylase, and the amylase values of pleural fluid and paracentesis drainage support the presence of AP. Leukocytosis, hypokalemia, hypocalcemia, and hypertriglyceridemia may be present but are not specific to AP. Leukocytosis frequently results from infection, stress, or dehydration. Persistent vomiting may result in hypokalemia. Hypocalcemia may indicate the presence of pancreatic fat necrosis because calcium binds with fatty acids during tissue necrosis. In addition, trypsin inactivates parathyroid hormone, which is needed for calcium absorption. Hyperglycemia may result from decreased insulin release from damaged beta cells, increased glucagon release, and the stress response. Hemoconcentration may occur as fluid is lost into the peritoneal space. Elevations in serum bilirubin, AST, and PT are common in the presence of concurrent liver disease. A greater than threefold elevation in ALT suggests biliary pancreatitis.[27] Alkaline phosphatase is elevated with biliary tract disease. Triglyceride levels associated with AP are usually greater than 1,000 mg/dL.[28]

Imaging Studies

Radiographs of the chest and abdomen are useful to exclude other causes of abdominal pain, including intestinal ileus, perforation, pericardial effusion, and pulmonary disease.

Abdominal ultrasonography is of limited use in visualization of the pancreas because of intestinal gas and adipose tissue. Abdominal ultrasonography is used to evaluate the biliary tree for gallstones, sludge, or ductal dilation as the etiology of pancreatitis. CT is the best imaging study to confirm the diagnosis and determine the severity of AP. CT can visualize the size of the pancreas and identify the presence of peripancreatic fluid, pancreatic pseudocysts, and abscesses. Dynamic CT done with contrast can help identify areas of necrosis in the pancreas. CT findings of extensive necrosis have correlated with a high risk for pancreatitis-related infection and death. Sequential CT allows for assessment of progressive disease or resolution. CT can also demonstrate fluid collection and areas of necrosis and can be used to guide percutaneous needle aspiration for culture.

Magnetic resonance cholangiopancreatography may have a sensitivity of more than 90% for bile duct stones. It can be used in patients who are pregnant and those who have allergies to the contrast used in CT or those with renal disease. Endoscopic retrograde cholangiopancreatography plays a role in locating and removing stones in the common bile duct if gallstone pancreatitis is present.

Tools for Predicting Severity

AP is self-limiting and mild in 80% to 90% of patients, resolving spontaneously within 5 to 7 days.[26] These patients usually require conservative care. However, in 10% to 20% of patients with AP, increased intrapancreatic and extrapancreatic inflammation results in a systemic inflammatory response. Although the mortality rate for severe AP is 10%, this value rises to 30% or more when there are complications.[26] Multiple assessment tools have been developed in attempts to identify patients who are likely to develop severe AP so that aggressive treatment and surveillance can decrease complications and mortality.

BOX 41-10 Ranson's Criteria for Acute Pancreatitis

Evaluate on admission or on diagnosis:
- Age more than 55 years
- Leukocyte count more than 16,000/mL
- Serum glucose more than 200 mg/dL
- Serum lactate dehydrogenase more than 350 IU/mL
- Serum AST more than 250 IU/dL

Evaluate during initial 48 hours:
- Fall in hematocrit more than 10%
- BUN level rise more than 5 mg/dL
- Serum calcium less than 8 mg/dL
- Base deficit more than 4 mEq/L
- Estimated fluid sequestration more than 6 L
- Arterial PaO_2 less than 60 mm Hg

Ranson's criteria have been widely used to assess the severity of AP (Box 41-10). Ranson's criteria consist of multiple clinical criteria used to identify those patients at risk for increased morbidity and mortality. The criteria assessed at admission indicate the severity of the acute inflammatory response, and the criteria assessed at 48 hours evaluate systemic effects. Three or more signs identified at the time of admission or during the initial 48 hours are predictive of severe AP, with an associated mortality rate of 10% to 20%.[26] Six or more have a corresponding mortality rate of 50%.[26] Ranson's criteria have a greater than 90% accuracy rate and are useful clinically in identifying high-risk patients. The primary disadvantage to Ranson's criteria is the 48-hour delay before the assessment is completed.

The Acute Physiology and Chronic Health Evaluation II (APACHE II) score has also been studied and found to be useful in predicting severity of AP (Table 41-2). The APACHE II uses the worst values of physiological measures, age, and previous health status at admission, at 24 hours, and at 48 hours to predict severity of AP. An APACHE II score on admission of eight or more predicted 68% of severe attacks.[26] An advantage of the APACHE II score is that it can be used daily. An APACHE II score that increases during the first 48 hours strongly suggests severe pancreatitis. Both Ranson's criteria results and APACHE II scores are comparable at 48 hours.

Extravasation and third spacing seen in severe AP can cause significant intravascular volume depletion. This depletion can lead to decreased perfusion of the pancreas and cause pancreatic necrosis. Some experts have proposed that hemoconcentration, as detected by an elevated serum hematocrit, is a reliable predictor of necrotizing pancreatitis; however, there is no consensus.

The presence of peripancreatic inflammation, peripancreatic fluid collection, and extent of pancreatic necrosis found on CT have been shown to predict severity of AP. The CT severity index uses CT findings to grade pancreatic severity.

Table 41-2 The Acute Physiology and Chronic Health Evaluation II (APACHE II) Classification System

	High Abnormal Range					Low Abnormal Range			
Physiologic Variable	**+4**	**+3**	**+2**	**+1**	**0**	**+1**	**+2**	**+3**	**+4**
Temperature rectal (°C)	41 or more	39–40.9		38.5–38.9	36.0–38.4	34–35.9	32–33.9	30–31.9	29.9 or less
Mean arterial pressure = (2 × diastolic + systolic)/3	160 or more	130–159	110–129		70–109		50–69		49 or less
Heart rate (ventricular response)	180 or more	140–179	110–139		70–109		55–69	40–54	39 or less
Respiratory rate (nonventilated or ventilated)	50 or more	35–49		25–34	12–24	10–11	6–9		<5
Oxygenation	500 or more	350–499	200–349		<200				
A-aDO_2 or Pao_2 (mm Hg); FiO_2 > 0.5; record A–aDo_2; FiO_2 < 0.5, record only PaO_2					>70	61–70		55–60	<55
Arterial pH (if no arterial blood gases [ABGs] record serum HCO_3 below)*	7.7 or more	7.6–7.69		7.5–7.59	7.33–7.49		7.25–7.32	7.15–7.24	<7.15
Serum sodium	180 or more	160–179	155–159	150–154	130–139		120–129	111–119	110 or less
Serum potassium	7 or more	6–6.9		5.6–5.9	3.5–5.4	3–3.4	2.5–2.9		<2.5
Serum creatinine (mg/dL) (double point for acute renal failure)	3.5 or more	2–3.4	1.5–1.9		0.6–1.4		<0.6		
Hematocrit (%)	60 or more		50–59.9	46–49.9	30–45.9		20–29.9		<20
White blood count	40 or more		20–39.9	15–19.9	3–14.9		1–2.9		<1
Glasgow coma scale (GCS) (score = 15 – actual GCS)†	15 – GCS =								
A total acute physiology score (APS)	Sum of the 12 individual variable points =								
*Serum HCO_3 (venous mmol/L) (not preferred, use if no ABGs)	<52	41–51.9		32–40.9	22–31.9		18–21.9	15–17.9	<15

†Glasgow Coma Scale	(Circle Appropriate Response)	B (Age and Points)		C (Chronic Health Points)	
Eyes open	*Verbal—nonintubated*	Age	Points	If any of the 5 CHE categories is answered with yes, give +5 points for nonoperative or emergency postoperative patients	
4—spontaneously	5—oriented and converses	Under 44 y	0		
3—to verbal command	4—disoriented and talks	45–54 y	2		
2—to painful stimuli	3—inappropriate words	55–64 y	3		
1—no response	2—incomprehensible sounds	65–74 y	5		
Motor response	1—no response	Over 75 y	6		
6—to verbal command	*Verbal—intubated*			Liver	Cirrhosis with portal hypertension or encephalopathy
5—localizes to pain	5—seems able to talk			Cardiovascular	Class IV angina at rest or with minimal self-care activities
4—withdraws to pain	3—questionable ability to talk			Pulmonary	Chronic hypoxemia or hypercapnia or polycythemia or pulmonary hypertension >40 mm Hg
3—decorticate	1—generally unresponsive			Kidney	Chronic peritoneal or hemodialysis
2—decerebrate				Immune	Immune-compromised host
1—no response		Age points =		Chronic health points =	

Credit given to Nick Mendel, Kiev, Ukraine, for producing this document.

APACHE II score **(sum of A + B + C): A** APS points + **B** age points + **C** chronic health points = total APACHE II.

From Triester SL, Kowdley KV: Prognostic factors in acute pancreatitis. J Clin Gastroenterol 34(2):167–176, 2002.

The use of serum markers to prognosticate severity has been tested. The most promising has been quantification of C-reactive protein (CRP). CRP rises in relation to severity, is inexpensive, and readily available. Unfortunately, CRP does not become significantly elevated until 48 hours after inflammation, which limits its use in diagnosis of AP.

Complications

The local and systemic complications of AP are summarized in Box 41-11.

Local Complications

The local effects of pancreatitis include inflammation of the peritoneum around the pancreas and fluid accumulation in the peritoneal cavity. These changes can lead to pancreatic pseudocyst, pancreatic abscess, and acute GI hemorrhage.

Pancreatic pseudocysts occur in up to 15% of all cases of AP. A pseudocyst is a collection of inflammatory debris and pancreatic secretions, enclosed by lined epithelial tissue and free of solid debris, that must be present for 4 weeks or more. The pseudocyst can rupture and hemorrhage or become infected, causing bacterial translocation and sepsis. A pseudocyst is suspected in any patient who has persistent abdominal pain with nausea and vomiting, a prolonged fever, and elevated serum amylase. Surgery may also be indicated for pseudocysts; however, it is usually delayed because most pseudocysts resolve spontaneously. Surgical treatment of the pseudocyst can be done through internal or external drainage or needle aspiration. Acute surgical intervention may be required if the pseudocyst becomes infected or perforates.

A pancreatic abscess is a walled-off collection of purulent material in or around the pancreas that usually occurs 6 weeks or more after the onset of AP. Signs and symptoms of an abdominal abscess or infected pancreatic necrosis include increased white blood cell count, fever, abdominal pain, and vomiting. Pancreatic infection from an abscess, pseudocyst, or necrotic tissue may be present whenever a patient has a temperature greater than 39°C (102.2°F), tachycardia, or leukocytosis or shows other signs of clinical deterioration. Often, infections after the onset of pancreatitis, if untreated, are fatal. Broad-spectrum antibiotics are given to those patients with suspected infection.

GI complications of AP include GIB and bacterial translocation. GIB, the most common GI complication of AP, includes bleeding from peptic ulcers, hemorrhagic gastroduodenitis, stress ulcers, and Mallory-Weiss syndrome. Decreased peristalsis can lead to bacterial translocation.

BOX 41-11 Major Complications of Acute Pancreatitis

Local
- Pancreatic necrosis
- Pancreatic pseudocyst
- Pancreatic abscess

Pulmonary
- Atelectasis
- Acute respiratory distress syndrome
- Pleural effusions

Cardiovascular
- Hypotensive shock
- Septic shock
- Hemorrhagic shock

Renal
- Acute renal failure

Hematological
- Disseminated intravascular coagulation (DIC)

Metabolic
- Hyperglycemia
- Hypertriglyceridemia
- Hypocalcemia
- Metabolic acidosis

Gastrointestinal
- Gastrointestinal bleed

Pulmonary Complications

Enzymes and inflammatory cytokines that reach the pulmonary circulation are thought to cause the many pulmonary complications associated with AP. Leukocytes that reach the pulmonary microcirculation migrate into the interstitium, which results in endothelial permeability and tissue edema. This causes lung congestion and alveolar collapse, and it can lead to adult respiratory distress syndrome. Arterial hypoxemia can occur in patients with mild disease without clinical or radiographic findings to support the pulmonary dysfunction. Arterial blood gas results and pulse oximetry should be followed closely for the first few days to detect this complication. Treatment of hypoxemia includes vigorous pulmonary care (eg, deep breathing and coughing) and frequent position changes. Oxygen therapy can also be used to improve overall oxygenation status. Careful fluid administration is also necessary to prevent fluid overload and pulmonary congestion. Patients with acute respiratory compromise may require mechanical ventilatory support. Abdominal distention and diminished diaphragmatic excursion may also contribute to atelectasis seen in AP.

Cardiovascular Complications

Hemodynamically significant fluid sequestration is characteristic of fulminant pancreatitis. Another major systemic effect of enzyme release into the circulatory system is peripheral vasodilation, which in turn can causes hypotension and shock.

Decreased perfusion to the pancreas itself can result in the release of myocardial depressant factor (MDF). MDF decreases heart contractility and affects cardiac output. Perfusion of all body organs can then become compromised. Early and aggressive fluid resuscitation is thought to prevent the release of MDF. Trypsin activation causes abnormalities in blood coagulation and clot lysis. This promotes the development of disseminated intravascular coagulation (DIC) with its associated bleeding (see Chapter 49).

Renal Complications

Acute renal failure is thought to be a consequence of hypovolemia and decreased renal perfusion. Death during the first 2 weeks of AP usually results from pulmonary or renal complications.

Metabolic Complications

Metabolic complications of AP include hypocalcemia and hyperlipidemia, which are thought to be related to areas of fat necrosis around the inflamed pancreas. Hyperglycemia may occur as a result of damage to the cells of the islets of Langerhans; metabolic acidosis can result from hypoperfusion and activation of anaerobic metabolism.

Management

Medical Management

Conventional care of the patient with AP focuses on fluid and electrolyte replacement to maintain or replenish vascular volume and electrolyte balance, pain management, resting the pancreas in an effort to prevent the release of pancreatic secretions, and maintaining the patient's nutritional status (Box 41-12). Close observation and clinical judgment are the basis for therapy and management.

Fluid and Electrolyte Replacement

Most patients with AP require the infusion of IV fluids to replace fluid lost through third spacing into the retroperitoneal space or peritoneal cavity, and intravascular volume depletion as a result of inflammatory mediators and local inflammation caused by pancreatic enzyme exudates. Patients with severe AP may need up to 5 to 10 L of fluid replacement within 24 hours during their initial days of hospitalization. The goal is to administer enough fluid to obtain a circulating volume sufficient to maintain organ and tissue perfusion and prevent end-stage shock. Hypovolemia and shock are major causes of death early in the disease process when aggressive fluid resuscitation fails to reverse the shock process.

Colloid and crystalloid solutions, such as albumin and lactated Ringer's solution, are used for volume replacement. Patients with acute hemorrhagic pancreatitis may also need PRBCs to restore blood volume. Fluid replacement is evaluated by monitoring intake and output and daily weights. Patients with more severe disease may require hemodynamic monitoring with measurement of pulmonary artery occlusion pressure or CVP. Patients with severe disease whose hypotension fails to respond to fluid therapy may need medications to support blood pressure. The drug of choice is dopamine at low doses to maintain renal perfusion while supporting blood pressure.

Urinary output is a sensitive measure of the adequacy of fluid replacement, and it should be maintained at greater than 30 mL/h or 0.6 mL/kg/h. Blood pressure and heart rate are also sensitive measures of volume status.

Patients with severe hypocalcemia are placed on seizure precautions with respiratory support equipment on hand. The nurse is responsible for monitoring calcium levels, administering replacement solutions, and evaluating the patient's response to any calcium supplementation. Calcium replacements should be infused through a central line because peripheral infiltration can cause tissue necrosis. The patient also needs to be monitored for calcium toxicity; symptoms include lethargy, nausea, shortening of the QT interval, and decreased excitability of nerves and muscles. Hypomagnesemia may also be present, so magnesium may need to be replaced as well. Serum magnesium levels usually need to be corrected before calcium levels can return to normal. Potassium may need to be replaced early in the treatment regimen because it is lost through vomiting and sequestration of potassium-rich pancreatic juices.

Hyperglycemia is related to impaired secretion of insulin, an increased release of glucagon, or increased stress response. In some cases, hyperglycemia can be associated with dehydration or other electrolyte imbalances. Sliding-scale regular insulin may be ordered; it needs to be administered very cautiously because glucagon levels are only transiently elevated in AP. Successful fluid replacement is marked by return of alert mental status, urine output, cardiac output, stable hemodynamic values, and a normal serum lactate level.

Pain Management

Pain control is a nursing priority for patients with AP, not only because of the extreme discomfort but also because pain increases pancreatic enzyme secretion. Pain is related to the degree of pancreatic inflammation, can be severe and constant, and can last for many days.

Adequate pain control with the use of IV narcotics, preferably delivered by patient-controlled analgesia, is essential in the treatment of AP. Meperidine (Demerol) has traditionally been the analgesic of choice because of the potential for sphincter of Oddi spasm that can accompany opioid use. However, meperidine is not always effective, and other analgesics (including opioids) should not be withheld. Fentanyl citrate (Sublimaze) and hydromorphone (Dilaudid) have been used successfully to control the pain of AP.

Analgesia should be routinely administered at least every 3 to 4 hours to prevent uncontrollable abdominal pain. Use of a pain rating scale is recommended for evaluating the patient's response to medication. Be alert to the patient's respiratory status because narcotics can induce respiratory depression. A nasogastric tube attached to low intermittent suction can help ease pain considerably, although the use of a nasogastric tube is controversial in patients without vomiting. Patient positioning can also relieve some of the discomfort.

Resting the Pancreas

In some patients with AP, nasogastric suction is used to decompress the stomach and decrease stimulation of secretin. Secretin, which stimulates production of pancreatic secretions, is released in response to acid in the duodenum. Nausea, vomiting, and abdominal pain may decrease when a nasogastric tube is placed and connected to suction early in treatment. A nasogastric tube is also necessary in patients with severe gastric distention or a paralytic

BOX 41-12 COLLABORATIVE CARE GUIDE for the Patient With Pancreatitis

Outcomes	Interventions
Oxygenation/Ventilation	
Arterial blood gases (ABGs) are maintained within normal limits.	• Assist patient to turn, deep breathe, cough, and use incentive spirometer every 4 h and PRN. Provide chest physiotherapy. • Assess for hypoventilation, rapid and shallow breathing, and respiratory distress. • Monitor pulse oximetry, end-tidal CO_2, and ABGs. • Administer analgesics if splinting is reducing effective ventilation. • Provide supplemental oxygen as needed.
The patient's lungs are clear. The patient has no evidence of atelectasis, pneumonia, or acute respiratory distress syndrome.	• Auscultate breath sounds every 2–4 h and PRN. • Suction only when rhonchi are present or secretions are visible in endotracheal tube. • Hyperoxygenate and hyperventilate before and after each suction pass.
Circulation/Perfusion	
Blood pressure, heart rate, and hemodynamic parameters are within normal limits.	• Monitor vital signs every 1–2 h. • Monitor pulmonary artery pressures and right atrial pressure every 1 h and cardiac output, systemic vascular resistance, and peripheral vascular resistance every 6–12 h if pulmonary artery catheter is in place. • Maintain patent IV access. • Administer intravascular volume as indicated by real or relative hypovolemia, and evaluate response.
Serum lactate will be within normal limits.	• Monitor lactate daily until it is within normal limits. • Administer RBCs, positive inotropic agents, colloid infusion as ordered to increase oxygen delivery.
Patient will not experience bleeding related to acute gastrointestinal hemorrhage, coagulopathies, or DIC.	• Monitor PT, PTT, complete blood count daily or PRN. • Assess for signs of bleeding. Observe for Cullen's or Grey Turner's signs. • Administer blood products as indicated.
Fluids/Electrolytes	
Patient is euvolemic.	• Maintain patent IV access. • Monitor daily weights. • Monitor intake and output. • Measure abdominal girth every 8 h at the same location on the abdomen.
No evidence of electrolyte imbalance or renal dysfunction.	• Monitor electrolytes daily and PRN. • Assess for signs of lethargy, tremors, tetany, and dysrhythmias. • Replace electrolytes as ordered. • Monitor BUN, creatinine, serum osmolality, and urine electrolytes daily.
Mobility/Safety	
No evidence of complications related to bed rest and immobility.	• Initiate deep venous thrombosis prophylaxis. • Reposition frequently. • Ambulate to chair when acute phase has passed and hemodynamic stability and hemostasis is achieved.
Patient achieves or maintains ability to conduct activities of daily living and mobilize self.	• Consult physical therapist. • Conduct range-of-motion and strengthening exercises.
No evidence of infection, WBC within normal limits.	• Monitor for systemic inflammatory response syndrome (SIRS). Criteria: increased WBC count, increased temperature, tachypnea, tachycardia. • Use strict aseptic technique during procedures. • Maintain invasive catheter tube sterility. • Change invasive catheters, culture blood, line tips, fluids, etc., according to hospital protocol.
Skin Integrity	
Skin will remain intact.	• Assess skin every 8 h and each time patient is repositioned. • Turn patient every 2 h. • Consider pressure relief/reduction mattress.

(*continued on page 938*)

BOX 41–12 COLLABORATIVE CARE GUIDE for the Patient With Pancreatitis (continued)

Outcomes	Interventions
Nutrition	
Caloric and nutrient intake meets metabolic requirements per calculation (eg, basal energy expenditure).	• Provide parenteral feeding. • Maintain NPO status. • Consult dietitian or nutritional support service. • Adhere to fat or lipid restriction. • Provide small, frequent feedings.
Evidence of metabolic dysfunction is minimal.	• Monitor albumin, prealbumin, transferrin, cholesterol, triglycerides, and glucose levels.
Comfort/Pain Control	
Patient will have minimal pain, <5 on pain scale.	• Assess pain and discomfort using objective pain scale every 4 h PRN and after administration of pain medication. • Administer analgesics and monitor patient response. • Use nonpharmacological pain management techniques (eg, music, distraction, touch) as adjunct to analgesics.
Patient will have minimal nausea.	• Maintain nasogastric tube patency. • Monitor nausea and vomiting. • Administer antiemetic as ordered.
Psychosocial	
Patient demonstrates decreased anxiety.	• Listen to patient's worries and fears. • Assess patient's response to anxiety. • Support effective coping behaviors. • Teach alternative behaviors for those that are not helpful. • Help patient increase sense of control by providing information and explanation. • Allow choices when possible. • Provide as much predictability in routine as possible.
Teaching/Discharge Planning	
Patient/significant others understand procedures and tests needed for treatment. Significant others understand the severity of the illness, ask appropriate questions, anticipate potential complications.	• Prepare patient/significant others for procedures such as paracentesis, pulmonary artery catheter insertion, or laboratory studies. • Explain the widespread effects of pancreatitis and the potential for complications such as sepsis or acute respiratory distress syndrome. • Encourage significant others to ask questions related to pathophysiology, monitoring, treatments, etc. • Instruct patient and family in discharge regimen that may include wound care, medications, and dietary limitations.

ileus. Patients with AP should be placed on NPO status until the abdominal pain subsides and serum amylase levels have returned to normal. Starting oral intake sooner can cause the abdominal pain to return and can induce further inflammation of the pancreas by stimulating the autodigestive disease process.

Nutritional Support

For those patients with AP who are on prolonged NPO status with nasogastric suction because of paralytic ileus, persistent abdominal pain, or pancreatic complications, nutritional support is recommended. TPN has been traditionally used because it was believed that stimulation of the pancreas by solid or liquid nutrients caused pancreatic stimulation and adversely affected the course of AP. Increasing evidence suggests that the use of enteral nutrition delivered past the ligament of Treitz to the distal duodenum or jejunum is safe. In addition, enteral nutrition may reduce infectious complications by maintaining intestinal barrier function and avoiding some of the complications of parental nutrition. Lipid administration is avoided to prevent increasing triglyceride levels, which can exacerbate the inflammatory process. In the patient with mild AP, oral fluids can usually be restarted within 3 to 7 days, with solid food introduced slowly and as tolerated. Supplementation with TPN is appropriate if oral and enteral nutrition cannot provide enough calories to prevent catabolism.

Prolonged NPO status is often difficult for patients. Frequent mouth care and proper positioning of the nasogastric tube are important to maintain skin integrity and maximize patient comfort. Bed rest is prescribed to decrease the patient's basal metabolic rate; this, in turn, decreases the stimulation of pancreatic secretions.

Surgical Management

Surgery for AP is indicated if massive pancreatic necrosis is present in a patient with a worsening clinical status. A pancreatic resection for acute necrotizing pancreatitis can be performed to prevent systemic complications of the disease process. In this procedure, dead or infected pancreatic tissue is surgically removed. In some cases, the entire pancreas is removed. Broad-spectrum antibiotics are given to patients who require surgical débridement of necrotic tissue.

▲ Hepatitis

Etiology

Diffuse inflammation of the liver, otherwise known as hepatitis, is commonly a consequence of a viral infection, but can be secondary to bacterial, fungal, or parasitic infection; a result of toxic exposure; a side effect of a prescribed medication; or a consequence of an immunologic disorder (Box 41-13). Acute hepatitis lasts less than 6 months; it either resolves completely with return of normal liver function or progresses to chronic hepatitis, then cirrhosis, and possibly liver failure. Chronic hepatitis is an inflammatory process that lasts longer than 6 months and may also progress to cirrhosis and possibly liver failure.

Noninfectious Hepatitis

Noninfectious hepatitis can be caused by excessive alcohol consumption, autoimmune disorders, metabolic or vascular disorders (including right-sided heart failure), acute biliary obstruction, and many individual drugs and drug classes (depending on the amount ingested and length of exposure). Examples include but are not limited to acetaminophen (both intentional and unintentional overdosing), isoniazid, HMG-CoA reductase inhibitors, anticonvulsants, antimicrobials, alpha-methyldopa, amiodarone, and estrogens.[29] Although only a minority of chronic alcohol abusers develop the syndrome of alcoholic hepatitis, severe cases carry a significant mortality rate, particularly in the elderly.[30]

Other liver toxins include poisonous mushrooms (*Amanita phalloides*), ecstasy (methylenedioxymethamphetamine), and some herbal medicines (ginseng, comfrey tea, pennyroyal oil, and *Teucrium polium*).[29] Autoimmune hepatitis, a condition in which the patient's own immune system attacks the liver, causes inflammation and hepatocyte injury or death. Autoimmune hepatitis can be mistaken for an acute viral hepatitis if the patient presents with severe symptoms.

Infectious Hepatitis

Viral hepatitis is a highly contagious inflammatory condition. Just like noninfectious hepatitis, infectious hepatitis can be acute, or it can be chronic if infection lasts longer than 6 months. Viral infections of the liver parenchyma have been classified according to their specific infecting agent and corresponding serology markers. Hepatitis A, B, C, D, and E are summarized in Table 41-3. Other viral causes of hepatitis include herpes simplex virus, Epstein-Barr virus, cytomegalovirus, adenovirus, coxsackievirus B, and varicella-zoster virus. Typically, patients with viral hepatitis present with nonspecific, flu-like symptoms, such as malaise, nausea, vomiting, diarrhea, loss of appetite, midepigastric abdominal discomfort, and low-grade fever. In patients with hepatitis B virus (HBV), symptoms may be more severe.

BOX 41-13 Selected Causes of Hepatic Inflammation

Infectious Diseases
- Viral hepatitis (A, B, C, D, E)
- Epstein-Barr virus
- Cytomegalovirus
- Herpes simplex virus
- Coxsackievirus B
- Toxoplasmosis
- Adenovirus
- Varicella-zoster virus

Drugs and Toxins
- Alcohol
- Acetaminophen
- Isoniazid
- Salicylates
- Anticonvulsants
- Antimicrobials
- HMG-CoA reductase inhibitors
- α-Methyldopa
- Amiodarone
- Estrogens
- *Amanita phalloides* mushrooms
- Ecstasy (methylenedioxymethamphetamine)
- Herbal medicines (ginseng, comfrey tea, pennyroyal oil, *Teucrium polium*)

Autoimmune Diseases
- Autoimmune hepatitis
- Primary biliary cirrhosis
- Primary sclerosing cholangitis

Congenital Diseases
- Hemochromatosis (iron overload)
- Wilson's disease (copper deposition)
- α_1-Antitrypsin deficiency

Miscellaneous Causes
- Nonalcoholic fatty liver
- Fatty liver of pregnancy
- Severe right-sided congestive heart failure
- Budd-Chiari syndrome (vascular obstruction)

Hepatitis A

In the United States, the Centers for Disease Control and Prevention (CDC) have reported a significant decline in the incidence of acute hepatitis A virus (HAV); the incidence of acute liver failure from HAV is considered low at an estimated 2,000 to 3,000 total cases per year, with a higher mortality rate in patients either older than 50 or younger than 5 years of age.[31,32] However, sporadic outbreaks continue to be reported due to consumption of contaminated

Table 41-3 Summary of Types of Hepatitis

	Hepatitis A	Hepatitis B	Hepatitis C	Hepatitis D	Hepatitis E
Incubation (days)	15–45	30–180	15–160	30–180	14–60
Onset	Acute	Insidious	Insidious	Acute or insidious	Acute
Transmission	Fecal/oral Contaminated food, water	Blood Sexual Perinatal Percutaneous	Blood May be sexual	Blood Sexual (comorbid infection with HBV)	Fecal/oral Contaminated food, water
Severity	Mild	Often severe	Moderate	May be very severe	Virulent, especially in pregnant women
Prognosis	Generally good	Worse with age, debility	Moderate	Fair, worse with chronic disease	Good, unless pregnant
Diagnosis					
Acute	Anti-HAV IgM	HBsAG Anti-HBc (IgM) HBeAg	HCV ELISA Anti-HCV RIBA HCV RNA	HDV Ag	Clinical
Chronic	—	Anti-HBc (IgG)	Anti-HCV	Anti-HDV	—
Prophylaxis (adults)	Immune globulin	Hepatitis B vaccine Immune globulin	?Immune globulin	None available	None available
Carrier	No	Yes	Yes	Yes	No

food or direct person-to-person contact.[32] HAV is caused by an RNA enterovirus transmitted through the oral–fecal route, predominantly by ingestion of contaminated water or raw or partially cooked shellfish. In most patients, symptoms of HAV infection are either relatively mild or nonexistent, although older patients are at greater risk for more severe symptoms. The incubation period ranges from 15 to 45 days after exposure. HAV infection causes only acute liver disease; recovery is usually complete and does not lead to chronic hepatitis or cirrhosis. Blood tests usually reveal elevations in the aminotransferases (ALT and AST), bilirubin concentrations, and alkaline phosphatase level. In severe cases, the PT may be prolonged. Diagnosis can be made with serology antibody testing. Anti-HAV immunoglobulin G (IgG) antibodies provide immunity and can be found in people who have had a previous HAV infection, but these are not helpful in diagnosing an acute infection. Instead, a positive anti-HAV IgM serology marker indicates HAV infection within the preceding 6 months. HAV infection does not induce a carrier state.

Early in the course of the disease, there is an incubation period during which the patient is asymptomatic but highly contagious, particularly with high HAV levels in the stool. After symptoms are apparent, the hepatitis infection can be misdiagnosed because many of the symptoms are similar to those of the flu. Some patients seek medical attention because they become jaundiced. The two most common physical examination findings are jaundice and hepatomegaly. Acute symptoms can progress or disappear once jaundice is present. By the time symptoms occur, the virus is no longer shed in the stool, and the patient is usually not infectious. Recovery is signaled by liver function test (LFT) results returning to normal.

After exposure to HAV, passive immunization can be achieved through the use of immune serum globulin. Most preparations of immune serum globulin contain adequate quantities of anti-HAV and should be given within 2 weeks of exposure. The immune serum globulin may not entirely abort an infection, but it significantly ameliorates the symptoms. It is usually given to intimate contacts of patients with HAV. There are also two U.S. Food and Drug Administration (FDA)–approved vaccines: Havrix and Vaqta. Vaccination is encouraged for all high-risk groups and all children aged 12 to 23 months and older.[33]

Hepatitis B

HBV is a DNA virus of the Hepadnaviridae family that replicates by reverse transcription. Infection causes both acute and chronic hepatitis, and the incubation period is 30 to 180 days, with the average at 12 weeks. HBV is spread by contact with blood or blood products. The antigen has been identified in body secretions, such as semen, mucus, and saliva; sexual exposure to a person with HBV is the most common mode of transmission.[32] It appears that a break in the skin or the mucous membrane is necessary for the transmission to occur. HBV is also transmitted parenterally through blood transfusions, occupational needlestick injuries, and the use of contaminated needles (eg, illicit drug use). Maternal perinatal transmission can also occur.

The diagnostic criteria of HBV include serologic markers, biochemical markers of liver disease (including elevated liver enzyme levels), and histologic changes in the liver. Incorrect interpretation of HBV serologic markers is common. Familiarity with the serology testing is important for the nurse who is assisting in the diagnostic evaluation of a suspected case of viral hepatitis to prevent inappropriate laboratory testing and patient discomfort. Hepatitis B surface antigen (HBsAg) is a protein that coats the outer surface of the HBV and is produced in

great excess during viral replication. HBsAg is the single most important test to detect infection with HBV. A positive result indicates that a patient is infected with HBV. If the presence of HBsAg is associated with an acute illness and a marked rise in the aminotransferases as well as the presence of hepatitis B core IgM antibody (IgM anti-HBc), the patient has acute HBV. If HBsAg disappears from the blood within 6 months, there is resolution of infection, and the patient does not advance to chronic disease. Patients with acute HBV infection who overcome their infection and eradicate the virus develop antibodies against hepatitis B surface antigen (anti-HBsAg). In some laboratories, these results may be reported as hepatitis B surface antibody (HBsAb) instead of anti-HBs. Regardless of the nomenclature, these people are protected against future HBV infection. A person who receives the HBV vaccine also develops HBsAb.

Clinical signs and symptoms of HBV infection during the acute phase are the same as those of HAV infection. Arthralgia, high fever, and rash are hallmark signs of an acute HBV infection. Acute HBV infection resolves in most adults; however, it can become chronic, particularly in those individuals with immune deficiencies. Those individuals who develop chronic HBV infection will continue to have high levels of HBsAg and can be infectious to others; individuals with a high viral load will also have an increased risk of chronic liver disease (eg, cirrhosis) and hepatocellular carcinoma.[32] Cirrhosis should be suspected if the patient develops hypersplenism, hypoalbuminemia (in the absence of nephropathy), thrombocytopenia, and prolongation of serum PT.[34] Although less than 1% of cases of those infected with HBV will progress to fulminant liver failure (typically occurs within 4 weeks of the onset of symptoms), it is associated with encephalopathy, multiorgan failure, and a mortality rate of 75% if not treated with liver transplantation.[35]

Antihepatitis B core (anti-HBc), also called hepatitis B core antibody (HBcAb), also appears early in the course of infection and may persist for many years, but it is helpful in evaluating an acute versus chronic infection because anti-HBc can be further divided into two subtypes. Anti-HBc (IgM) is the initial response to infection and lasts for 6 to 18 months after recovery from infection. Therefore, high titers of anti-HBc (IgM) indicate acute infection, and low titers indicate chronic liver infection. Anti-HBc (IgG) is the second subtype and is positive in patients who are either chronically infected or previously infected, persisting in the bloodstream for a lifetime. Once an infection by HBV is established with HBsAg and anti-HBc, further serological testing can be ordered. For instance, the presence of the hepatitis Be antigen (HBeAg) indicates active viral replication and helps in diagnosing disease severity, prognosis, and treatment options. Those patients who have a positive HBeAg are considered highly infectious.

HBV exposure is associated with high risk. After accidental exposure, such as an inadvertent needlestick, passive immunoprophylaxis can be achieved by using high anti-HBs titer hepatitis B immune globulin (HBIG). This is a pooled serum containing high titers of the anti-HBIG. It is recommended that HBIG be given within 48 hours of postexposure as inoculations to high-risk patients, to close contacts of patients with active HBV (within 2 weeks after sexual or personal contact with infected body fluids), and to those traveling to endemic areas who do not have time to go through the normal three-dose vaccination series.[35] Many new HBV nucleoside analog treatments are under review. The following medications are currently approved by the FDA for treating chronic HBV infection and include standard interferon alpha (INF-α 2b), pegylated interferon alpha (INF-α 2a), and four oral antiviral agents (ie, lamivudine, adefovir, entecavir, and telbivudine).[36]

Fortunately, vaccines exist for active immunization against HBV (ie, Recombivax-HB or Engerix-B). Vaccination, administered prophylactically over a 6-month period, provides active immunization against HBV. It is highly recommended for health care personnel at risk for infection with HBV. It is also recommended for people who have had intimate contacts with people already infected with HBV. Precautions to protect against exposure to blood-borne pathogens must be followed. In the United States, children are universally vaccinated against HBV. Other combination vaccine preparations include Twinrix (HBV and HAV), Comvax (HBV and *Haemophilus influenza* type b), and Pediarix (HBV, diphtheria, tetanus, pertussis, and polio viruses).[35]

Hepatitis C

Hepatitis C virus (HCV) has surpassed alcoholism to become the leading cause of liver cirrhosis and end-stage liver disease requiring liver transplantation in the United States; it accounts for 40% of cases of end-stage cirrhosis and 60% of hepatocellular carcinoma.[32,37] As diagnostic tests improved, HCV, formerly called non-A, non-B hepatitis, was identified in 1989. It is a single-stranded RNA virus related to the Flaviviridae family. There are 11 HCV genotypes (genotypes 1 to 11), many subtypes (a, b, c, and so forth), and about 100 different strains (1, 2, 3, and so forth).[37] These variations in genotype led to variations in clinical course, problems with vaccine development, treatment response, and, in many cases, duration of treatment. Genotypes 1 and 3 are widely distributed globally, with genotypes 1a and 1b accounting for 60% of all HCV infections.[37] Genotype 1a is predominately located in North America and Northern Europe and is generally associated with a poorer response to treatment compared to genotypes 2 and 3.[37]

HCV, a blood-borne virus, can cause both acute and chronic hepatitis. Of those people who develop acute HCV, chronicity can occur in as many 85%, and 5% to 20% reportedly develop cirrhosis over approximately 20 to 25 years.[32] People with HCV-related cirrhosis are at risk for developing end-stage liver disease (a risk of approximately 30% over 10 years) as well as hepatocellular carcinoma (a risk of approximately 1% to 2% per year).[32]

Before 1992, when testing for HCV was mandated, many people acquired HCV through blood transfusions. The main risk factors in the United States include shared contaminated needles from illicit drug use and occupational needlestick exposure. There are indications that the virus might also be transmitted through perinatal, sexual, and household contacts (eg, sharing of razors and toothbrushes), although these modes of transmission are not as common. It has also

been suggested that acupuncture, body piercing, tattooing, and even commercial barbering carry a risk for HCV transmission.[38] Incubation of HCV is 15 to 160 days, with an average of 7 weeks. Within 6 months of infection, patients may produce anti–hepatitis C antibodies, but these do not confer immunity.

Although most patients are asymptomatic, the most commonly reported symptoms of HCV infection include fatigue, anorexia, weight loss, and abdominal pain. Diagnostic evaluation for HCV infection includes an HCV enzyme-linked immunosorbent assay (ELISA; ordered when the aminotransferase levels are elevated and for screening patients on hemodialysis), an anti-HCV recombinant strip immunoblot assay (RIBA; ordered to confirm a positive HCV test or if a patient presents with symptoms of hepatitis), or an HCV RNA test (ordered when HCV RIBA findings are indeterminate, but there remains a high index of suspicion for HCV). HCV RNA, which tests for the presence of the virus RNA in the blood (rather than antibodies against the virus), is the gold standard for detecting HCV. HCV RNA levels are used to gauge response to treatment, but they are not serially checked because viral load has no correlation to the degree or rate of liver injury progression.[39]

The goal of treatment is virus eradication (ie, sustained virologic response, or SVR) and prevention or delay of cirrhosis, end-stage liver disease, and hepatocellular carcinoma. Current combination therapy consists of pegylated INF-α 2a or 2b plus ribavirin. Infection is considered eradicated when there is a sustained viral response (SVR), which is defined as the absence of HCV RNA (aviremia) at the end of treatment and then 6 months after treatment.[40] Genotypes 1 and 4 are considered the most difficult strains to treat; they require a longer duration of treatment (48 weeks) and have a lower posttreatment SVR rate (40%) compared with genotypes 2 and 3. Genotypes 2 and 3 require a shorter duration of treatment (24 weeks) and have a higher SVR rate of 79% to 82%.[40] Unfortunately, not all patients with an early virologic response to treatment become HCV RNA undetectable, and continuing treatment in these patients cannot lead to an SVR. These differences in treatment duration and outcomes highlight the importance of genotyping to determine who may benefit from combination therapy and optimal treatment duration. Currently, no vaccine is available to prevent HCV, and immune globulin does not afford protection to people who have been exposed.

Hepatitis D

Hepatitis D (delta virus or HDV) is an incomplete RNA virus that depends on HBV envelope proteins to reproduce; it cannot exist or be spread in the absence of HBV. HDV infection may occur as a superinfection in the patient who has chronic HBV, or it may occur simultaneously with an acute HBV infection. In the United States, HDV and HBV coinfection is almost exclusively observed in IV drug users.[40] HDV can progress to fulminant hepatitis and chronic disease. In the United States, HDV infection occurs primarily among people receiving multiple transfusions and those abusing illicit IV drugs. Early in the disease, the hepatitis D antigen (HDV Ag) is present in the blood. Later in the disease, antibodies to the HDV are present (anti-HDV).

Because this disease coexists with HBV, patients with HDV have symptoms similar to those of acute or chronic HBV, but symptoms may be more pronounced. Quantitative real-time polymerase chain reaction (PCR) assays for HDV RNA are monitored at 3 and 6 months. Current treatment includes standard INF, and recent research suggests that pegylated INF-α 2b may improve SVR. The addition of ribavirin has not been shown to provide any additional benefit.[41] Vaccination against HBV also protects against HDV infection.[40]

Hepatitis E

Hepatitis E virus (HEV) is a single-stranded RNA virus similar to HAV. Although rare and sporadic in industrialized countries, it is the most common epidemic, water-borne form of hepatitis in developing countries.[42] It is transmitted by the oral–fecal route from contaminated water and food. In some cases, the source of infection has been traced to contact with infected animals or animal products, notably swine.[43] The incubation period is 14 to 60 days, and it has an overall low mortality rate among the general population. Symptoms typically present with a prodrome of malaise and fever, followed by anorexia, nausea, vomiting, abdominal pain, hepatomegaly, and jaundice. Serum aminotransferase levels will be significantly elevated, but the infection is typically self-limited. For reasons that are still unclear, pregnant women have a more severe illness, with a mortality rate of 20% for women in their third trimester.[42,44] Because the incidence of HEV infection is rare in the United States, the nurse should pay careful attention if a patient presents with symptoms of hepatitis who has recently traveled or lived in endemic areas. A recently developed vaccine against HEV has been developed and is still under investigation.[45] Unfortunately, previous HEV infection is not necessarily protective.

Hepatitis F and Hepatitis G

It was formerly believed that a virus isolated from rare blood samples was a newly identified virus, labeled hepatitis F. However, further investigation has found this virus to be a variant of HCV, and additional research has failed to demonstrate the existence of a new virus. Little is known about the most recently identified virus, hepatitis G (HGV), which has been identified with PCR testing. It is a single-stranded enveloped RNA virus similar to HCV. As with HBV and HCV, HGV is transmitted by blood and body fluids; infection is diagnosed by seroconversion of HGV RNA in blood or liver tissue. No vaccine has been developed against HGV, and current treatment is supportive and focused on symptom management. Infection is considered benign and is rarely associated with elevated LFTs.

Pathophysiology

To improve patient outcomes, nurses must have a solid knowledge base regarding the underlying pathophysiology, assessment, and management of acute and chronic liver disease. Hepatocytes, the functional cells of the liver, perform many essential functions, including the metabolism of nutrients (eg, glucose, proteins, lipids, vitamins) and the detoxification of medications, alcohol, ammonia, toxins, and hormones. In addition, hepatocytes are responsible for synthesis of clotting factors, conjugation and secretion of bilirubin, and synthesis of bile salts. Abnormal liver function is usually not apparent

unless a significant acute insult occurs or chronic liver disease is fairly advanced. Liver failure occurs when there is a loss of 60% of the hepatocytes, and symptoms are usually detectable after 75% or more of the hepatocytes are injured or killed. Liver function testing and evaluation begin with a complete history and physical examination. Interpretation of liver serum enzymes, synthetic function, and cholestasis (or excretory function) tests are important for the nurse to understand and are discussed later in this chapter.

Acute liver disease, typically caused by viral or chemical insults, occurs suddenly and resolves, becomes chronic, or results in a patient's death. Chronic liver disease leading to cirrhosis, typically more insidious in nature, is the 12th leading cause of death in the United States.[46] Disease processes in the liver can affect the hepatocytes, the blood vessels, and the Kupffer cells, which are responsible for uptake and subsequent degradation of foreign and potentially harmful substances in the body. If the injury is mild and reversible, hepatocytes may regenerate, and liver function may return to normal. However, if the injury is more severe or sustained, regeneration may be incomplete, or the healing process may cause fibrosis. Fibrotic changes alter the liver architecture and can lead to cirrhosis and impediment of blood flow through the liver. An acute insult to the liver can progress to fulminant liver failure, which is defined as hepatic encephalopathy (HE) occurring within 8 weeks of jaundice. HE is a state of abnormal mental functioning as a result of the inability of the liver to remove ammonia and other toxins from the blood. If liver function does not return and liver transplantation is unavailable, fulminant liver failure can progress to cerebral edema, coma, and death from brain herniation.

Assessment

History

Questions regarding the patient's alcohol consumption and illicit drug use, use of prescription and over-the-counter medications, use of herbal supplements, surgical and transfusion history, occupational or travel exposure history, and sexual history may be helpful in determining the diagnosis, nursing plan of care, and teaching needs of the patient. In chronic hepatitis, most patients are asymptomatic except for mildly elevated liver enzymes. Constitutional symptoms vary widely but typically include malaise, fatigue, low-grade fever, nausea, vomiting, and sometimes diarrhea.

Physical Examination

When cirrhosis and portal hypertension resulting from chronic hepatitis are present, jaundice (yellow staining of the skin and mucous membranes as a result of bilirubin pigments) may be noted. Hepatomegaly (enlargement of the liver) may result in right upper quadrant tenderness and is a result of portal hypertension or congestion in the liver from altered blood flow resulting from cirrhosis. The liver edge is often firm and nodular. In advanced cirrhosis, although the left lobe may be enlarged, overall liver size is often decreased and difficult to palpate. Dullness of percussion over the liver span can provide serial observations of resolution of hepatitis or progression of cirrhosis. Splenomegaly as a result of portal hypertension and sequestration of fluid in the spleen may result in left upper quadrant tenderness. Muscle wasting and abdominal ascites may develop as a result of malnutrition, portal hypertension, and hypoalbuminemia from the liver's impaired ability to synthesize proteins. Peripheral edema may result from hypoalbuminemia, sodium retention, and ascites obstructing blood return from the lower extremities. Vitamin deficiencies may result in glossitis of the tongue and cheilosis of the lips. Bruising and bleeding tendencies may develop as a result of impaired production of clotting factors and sequestration of platelets in the spleen. Other manifestations include telangiectasis or spider nevi (usually of the upper half of the body). These lesions consist of a pulsating arteriole from which smaller vessels radiate. Palmar erythema, or redness of the palms, is a result of increased blood flow from hyperdynamic cardiac dysfunction associated with hepatitis (with ascites). In men, there may be a loss of body hair, testicular atrophy, and gynecomastia. These changes are thought to be related to altered hormone metabolism and estrogen excess in the liver.

Physical examination may also reveal abdominal wall vein dilation around the umbilicus, known as caput medusae. This is the result of portal hypertension and congestion and collateral vessel development (Fig. 41-4). This congestion may be auscultated as an arterial bruit (systolic phase) or a venous hum (both systolic and diastolic phases) over the liver and epigastrium. Encephalopathy, ascites, and peripheral edema, reflective of advanced disease, may be present. Other observable assessment findings include frothy, dark amber urine and clay-colored stools as a result of alterations in bilirubin excretion. Common signs and symptoms of noninfectious and

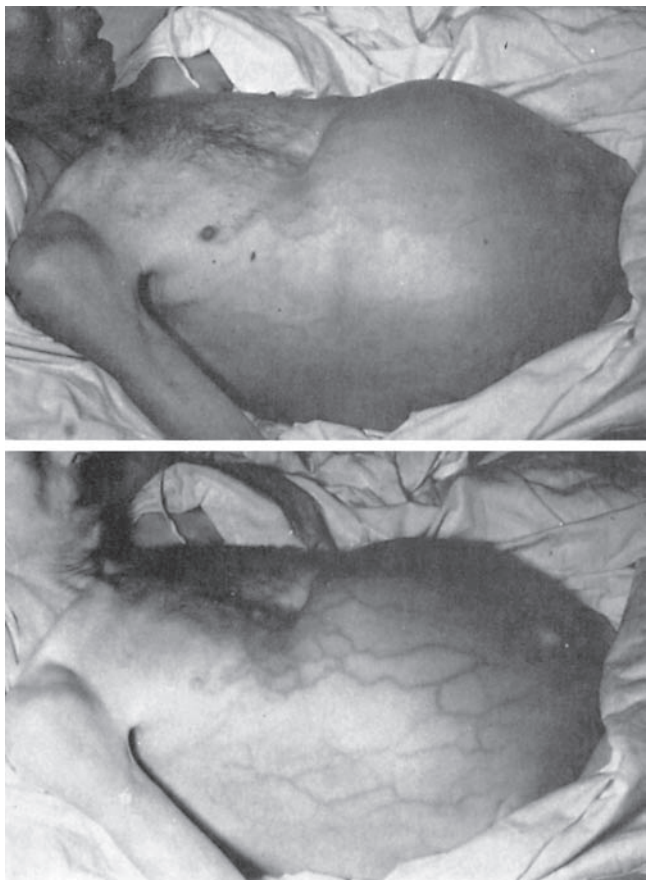


FIGURE 41-4 ▲ Collateral abdominal veins on the anterior abdominal wall in a patient with alcoholic liver disease as recorded by black and white photography (**top**) and infrared photography (**bottom**). (From Schiff L: Diseases of the Liver. Philadelphia, PA: JB Lippincott, 1982.)

infectious hepatitis are summarized in Table 41-4. Patients with signs and symptoms of hepatic decompensation (eg, portal hypertension, ascites, encephalopathy, and coagulopathy) should be hospitalized, evaluated, and treated more expeditiously than those patients who demonstrate adequate hepatic compensation and stability.

Table 41-4 Common Signs and Symptoms of Hepatitis

Signs and Symptoms	Cause
Constitutional	
Fever, chills	Immune response to viral infection
Generalized weakness, malnutrition	Inability to metabolize nutrients
Gastrointestinal	
Right upper quadrant pain	Hepatomegaly
Left upper quadrant pain	Splenomegaly
Loss of appetite	Ascites, fatigue
Abdominal distention	Ascites
Nausea, vomiting/hematemesis	Portal hypertension
Clay-colored feces	Inability to excrete conjugated bilirubin
Diarrhea	Impaired fat metabolism
Melena, hematochezia	Portal hypertension
Pulmonary	
Shortness of breath Increased work of breathing Decreased oxygen saturation Decreased partial pressure of oxygen	Ascites, decreased lung and diaphragmatic expansion
Cardiac	
Increased heart rate	Hypotension, sequestration of fluid in the liver and spleen, third spacing in the peripheral extremities from decreased protein metabolism/low albumin levels
Decreased blood pressure	
Dysrhythmias	Electrolyte disturbances
Peripheral edema	Impaired protein metabolism
Neurological	
Headache Depression/irritability Asterixis	Impaired metabolism of ammonia and other circulating toxins
Genitourinary	
Decreased urinary output	Decreased circulating volume and impaired glomerular filtration rate
Frothy, dark amber urine	Excretion of conjugated bilirubin (water-soluble bile)
Integumentary	
Jaundice	Impaired excretion of bile
Pruritus, dry skin	Impaired excretion of bile
Bruising, ecchymosis	Impaired ability to synthesize clotting factors
Spider nevi, caput medusae	Portal hypertension
Palmar erythema	Portal hypertension
Hair loss	Impaired metabolism of circulating hormones
Endocrine	
Hypoglycemia	Impaired glucose metabolism and storage
Increased weight	Ascites, third spacing of fluid
Gynecomastia, testicular atrophy (in men)	Inability to metabolize hormones (eg, estrogens)
Immune	
Infection, spontaneous bacterial peritonitis	Impaired Kupffer cell function, splenomegaly

Laboratory Studies

TESTS FOR EVALUATING HEPATOCELLULAR INJURY. The clinical significance of any liver chemistry must be evaluated in the context of the patient's history and clinical situation. Liver function test is a commonly used but inaccurate term. Some laboratory tests do measure liver synthetic function, and these include albumin, PT, and total bilirubin. However, other laboratory tests are markers of hepatocellular injury and include AST, previously known as serum glutamic oxaloacetic transaminase, and ALT, previously known as serum glutamic pyruvic transaminase. See Table 39-6 on page 883.

ALT and AST are enzymes present inside the hepatocytes. When hepatocytes are injured or die, they release AST and ALT into the serum. Therefore, the presence of these enzymes in the blood signals the presence of hepatocyte injury. However, AST and ALT lack sensitivity (for a particular diagnosis) in evaluating chronic liver injury for two reasons. First, AST and ALT are also found (to a lesser degree) in the skeletal muscle, and as such, elevations may be related to a skeletal muscle injury or overexertion. This is particularly true for AST because ALT is almost exclusively present in hepatocytes and is the most specific test for hepatocellular damage. Second, it is thought that dying hepatocytes synthesize less AST and ALT enzymes than healthy ones. Therefore, despite inflammation detected on a liver biopsy, patients with chronic hepatitis may have relatively normal levels of AST and ALT.

Despite these difficulties in laboratory interpretation, elevations of AST and ALT are often helpful in evaluating acute liver injury, response to treatment, and monitoring those at risk for liver disease because of medical interventions. Elevations of these enzymes suggest hepatocyte death, and the degree of elevation roughly approximates the amount of liver cell death. AST and ALT elevate at relatively equal levels, but an exception occurs in alcoholic hepatitis, in which AST levels tend to be higher than ALT levels. Although the AST/ALT ratio is not diagnostic, a ratio greater than 2:1 suggests alcohol-induced injury.[34] This is thought to be due to the depletion of vitamin B_6 (pyridoxine) in patients with chronic alcoholism; ALT synthesis is more strongly inhibited by pyridoxine deficiency than AST synthesis. In chronic hepatitis, AST and ALT levels are usually less than 10 times normal. However, in acute viral, toxin-induced, or ischemic hepatitis, these elevations may be greater than 1,000 units/L. In addition, alcoholic hepatitis causes smaller elevations (<300 units/L). Unfortunately, AST and ALT levels have low prognostic value.[34]

TESTS FOR EVALUATING LIVER SYNTHETIC FUNCTION. As mentioned earlier, albumin, total protein, and PT are measures of actual liver synthetic function. Because proteins are synthesized by the liver, albumin and other proteins are an index of liver function. Albumin is the predominant protein in the serum; patients with advanced liver disease and cirrhosis tend to have low serum concentrations (hypoalbuminemia). Because albumin is responsible for colloid osmotic pressure, low concentrations lead to leakage of intravascular fluids into interstitial spaces and peripheral edema. Because albumin levels are also influenced by poor nutrition and renal disease, care must be taken when interpreting laboratory test results.

The PT is a measure of the liver's capacity to synthesize clotting factors. The liver synthesizes blood clotting factors II, V, VII, IX, and X. An elevation in PT values is not seen until more than 80% of hepatocyte function is lost. However, because of the short half-life of factor VII, a PT is helpful in evaluating acute liver failure. Evaluation for vitamin K deficiency is performed because malabsorption or poor nutritional intake must be excluded in a patient who is hypoprothrombinemic. Failure of improvement in a PT level after vitamin K supplementation (5 to 10 mg orally for 3 days) may indicate intrinsic liver disease.

TESTS FOR EVALUATING CHOLESTASIS (EXCRETORY FUNCTION). Tests for cholestasis (lack of bile flow) help determine what is happening in the bile ducts. Obstruction of bile flow may be extrahepatic (eg, gallstones, postsurgical stricture, or malignancy) or intrahepatic (eg, poor hepatocyte function or damage to the small septal or intralobular bile ducts). Present in the biliary epithelium, elevated alkaline phosphatase and gamma-glutamyltransferase levels reflect damage to the bile ducts or obstruction of bile flow.

An elevated serum bilirubin level is roughly proportional to the amount of liver dysfunction or disease severity. Bilirubin is the major source of hemoglobin metabolism from the destruction of adult RBCs. The unconjugated (indirect) form of bilirubin is not water soluble and is bound to albumin as a means of transport to the liver for conjugation and subsequent excretion in the bile. In the hepatocytes, unconjugated bilirubin is combined with glucuronic acid to make it water soluble (or conjugated) for excretion into the bile and feces. Cholestasis causes reflux of conjugated bilirubin into the blood (a condition called conjugated hyperbilirubinemia), so the conjugated bilirubin is instead excreted through the kidneys. The urine becomes frothy and very dark amber in color from bilirubin pigments. The nurse may be asked to perform a dipstick test on the urine for bilirubin to confirm this clinical suspicion. Unconjugated hyperbilirubinemia results from a poor nutritional state (eg, decreased albumin available for transport of bilirubin to the liver) or hepatocyte dysfunction in the conjugation process. Jaundice is usually present when the serum bilirubin level is greater than 2.5 mg/dL.

Management

The primary treatment of acute hepatitis of any type is primarily supportive. Measures include providing rest and adequate nutrition and preventing further liver injury by avoiding hepatotoxic medications and substances. Hospitalization is rarely required but is needed in cases of disease complicated by hemodynamic instability, failure to maintain adequate nutrition and fluid intake, encephalopathy, blood coagulopathies, and renal failure.

In situations of hemodynamic instability, monitoring of blood pressure, heart rate, cardiac dysrhythmias, and urine output is essential. IV fluids will most likely be needed. It is important to avoid lactated Ringer's solutions because of the inability of the impaired liver to metabolize lactate, which could induce or exacerbate a metabolic acidosis. Frequent monitoring of hepatic enzymes and synthetic function is requested to evaluate disease progression and response to treatment interventions. Electrolyte, nutrient, and vitamin abnormalities from disease progression, malnutrition, and nausea and vomiting require repletion.

The nurse may have to assist in invasive treatments or procedures, such as placement of a Sengstaken-Blakemore tube for control of bleeding esophageal varices, paracentesis for ascites, and liver biopsy. In the event of fluid volume overload, diuretics, albumin, and protein supplements may be prescribed. Accurate intake and output, daily weight, and abdominal girth measurements may alert the nurse to significant volume shifts and potential hemodynamic or respiratory issues.

Maintaining adequate nutrition is a priority. Small, frequent meals and antiemetics are administered as needed. A high-calorie, low-protein diet is recommended to prevent complications associated with impaired protein and ammonia metabolism associated with acute HE. However, a low-protein diet is used only in the short term because seriously ill patients actually have increased protein requirements to build and maintain muscle mass and to assist in healing and repair. Parenteral nutrition is needed only if oral intake is impaired by intractable nausea and vomiting. Patients with severe fatigue require frequent rest and spacing of activities.

Because of the risk for coagulopathy, the nurse must monitor for bleeding gums, epistaxis, ecchymosis, petechiae, hematemesis, hematuria, and melena. Vitamin K may be prescribed to help reduce the effects of bleeding tendencies, and a PT may be ordered to monitor the efficacy of treatment.

Avoidance of alcohol, narcotics, barbiturates, and medications that are metabolized by the liver is recommended. Careful observation and documentation of patient responses (eg, mental status, level of consciousness) to medications and treatment regimens is recommended. Because of the liver's inability to metabolize or detoxify many foods, drugs, and toxins, the nurse may be asked to administer frequent medications, such as lactulose, neomycin sulfate, and metronidazole, to treat HE. Lactulose is a laxative that acidifies the colon to prevent the absorption of ammonia. The dose of lactulose is titrated so that the patient has two to three soft stools per day without diarrhea. Neomycin and metronidazole act as antibiotics to clear the colon of bacteria that produce ammonia.

If severe pruritus from jaundice is present, a bile salt sequestering agent (eg, cholestyramine), a topical emollient, or both can be used to help alleviate this symptom. Mittens may need to be used to prevent excessive scratching and subsequent skin breakdown in a confused patient.

Patient teaching for the patient with hepatitis includes measures to prevent infection and transmission, dietary limitations and alcohol avoidance, and the necessity for follow-up care. The patient is advised to monitor activity tolerance and fatigue. If signs and symptoms persist and liver enzymes remain elevated for greater than 6 months, the patient will progress to chronic disease. This is more common in HBV and HCV infection and is confirmed by liver biopsy.

▲ Complications of Liver Disease

Complications of advanced liver disease include cirrhosis, HE, hepatorenal syndrome (HRS), spontaneous bacterial peritonitis (SBP), and hepatocellular carcinoma.

Cirrhosis

Etiology

As noted earlier, chronic HCV infection and alcohol abuse are the most common causes of liver cirrhosis. However, cirrhosis of the liver can result from a number of other diseases, which include but are not limited to nonalcoholic steatohepatitis, hereditary hemochromatosis, Wilson's disease, and alpha$_1$-antitrypsin deficiency.

Pathophysiology

Cirrhosis, which develops over time, can cause severe alterations in the structural architecture of the liver and function of the hepatocytes. These changes are characterized by inflammation and liver cell necrosis, which can be focal or diffuse. Necrosis is followed by regeneration of liver tissue but not in a normal fashion. Fibrous tissue and regenerative nodules develop over time, which distorts the normal architecture of the liver lobule and alters blood flow. These fibrotic changes are irreversible, resulting in chronic liver dysfunction and eventual liver failure. Fatty deposits in the parenchymal cells may be seen initially. The cause of the fatty changes is unclear, but it may be a response to alterations in enzymatic function responsible for normal fat metabolism. Eventually, all of the liver's metabolic processes are altered.

Inflammation, fibrotic changes, and increased intrahepatic vascular resistance cause compression of the liver lobule, leading to increased resistance or obstruction of normal blood flow through the liver, which is normally a low-pressure system. This portal hypertension results in significant venous congestion and dilation (Fig. 41-5). Subsequently, nutrient-rich blood from the GI tract is shunted away from the liver, the first site of metabolism for many nutrients, drugs, and toxins. Pressure builds up in the systemic venous circulation, causing congestion where the portal and systemic venous systems connect: the esophagus, stomach, and rectum. These vascular changes result in varicose veins, or varices. Esophageal varices and gastric varices are of particular concern in the care of these patients because they are extremely friable. Rupture from these varices can result in massive internal bleeding that may be life-threatening. Portal hypertension also promotes increased collateral circulation and allows blood to flow from the intestines directly to the vena cava. This congestion is often seen as a collection of prominent vessels on the surface of the abdomen and is known as caput medusae. Splenomegaly results from the sequestration of trapped blood from portal hypertension. Of particular concern is the trapping of platelets, which can be seen clinically by bleeding tendencies and a thrombocytopenia on laboratory evaluation. Frank hematemesis or bleeding from esophageal and gastric varices can cause melena. Hemorrhoidal varices, or hemorrhoids, can also result from portal hypertension. Finally, portal hypertension may result in abdominal fluid accumulation, known as ascites. As liver disease progresses and cirrhosis develops, mild to moderate high-output cardiac dysfunction may occur. This hyperdynamic dysfunction is characterized by splanchnic and systemic vasodilation, an afterload effect

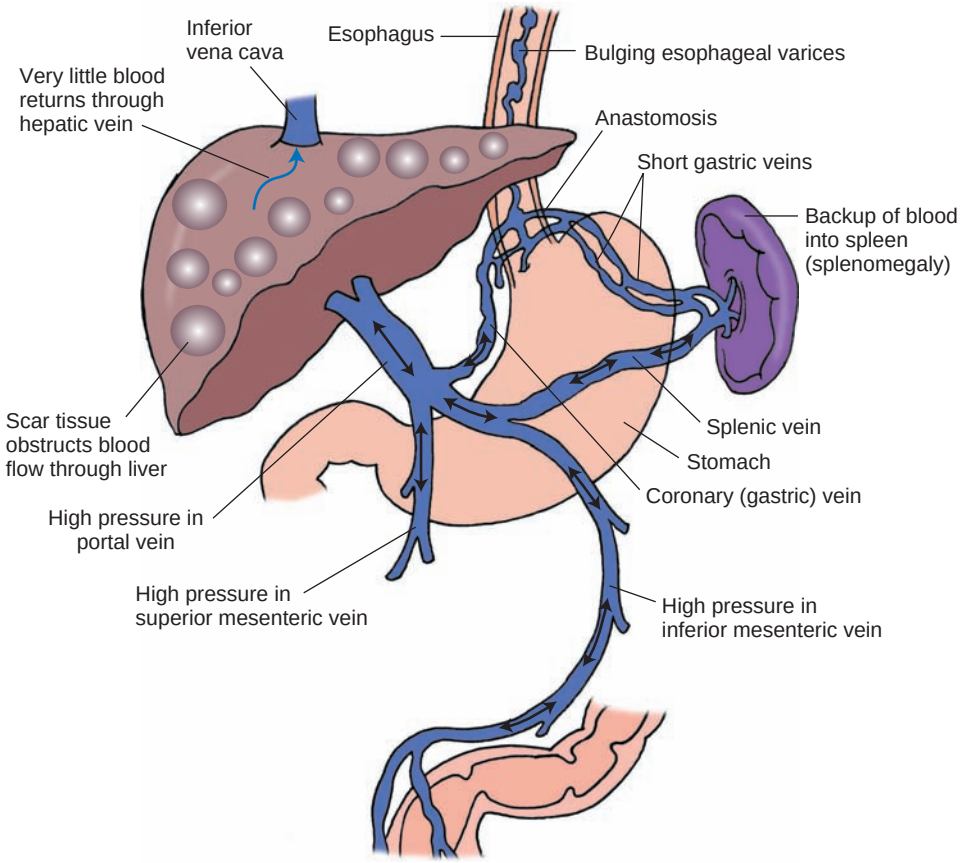


FIGURE 41-5 ▲ Esophageal varices develop from increased portal pressure. In an attempt to return blood to the systemic circulation, collateral veins develop to bypass increased portal resistance. These collateral vessels become tortuous and distended and are called varices.

that decreases cardiac work and elevates cardiac output. Clinically, it is seen as hypotension, tachycardia, and cardiac flow murmurs. As hepatic cirrhosis progresses, these clinical findings become more pronounced. Figure 41-6 illustrates clinical effects of cirrhosis.

Assessment

In some patients, cirrhosis may be subclinical. However, history and physical examination findings may reveal clues to altered liver function. For example, altered carbohydrate metabolism can result in unstable blood glucose levels. Altered fat metabolism can cause fatigue and decreased activity tolerance. Altered protein metabolism results in a decreased synthesis of albumin. Albumin is necessary for colloid osmotic pressure, which holds fluid in the intravascular space. A decrease leads to interstitial tissue edema and decreased plasma volume. Globulin, another protein, is essential for normal blood clotting. This, coupled with a decreased synthesis of many blood clotting factors and decreased metabolism of vitamins and iron, predisposes the patient to hematological complications that range from bruising to hemorrhage. A low-grade DIC also may develop. Portal hypertension, ascites, and lower extremity edema cause hypotension. Initially, the patient may have flushed skin and bounding pulses from the vasodilation in the portal venous system, which leads to a hyperdynamic state with peripheral circulation vasodilation and hypotension. Table 41-5 summarizes laboratory findings in patients with cirrhosis and impending liver failure.

Management

Management goals include preventing additional stress on liver function and early recognition and treatment of complications. Liver functions under stress include nutritional metabolism, clearing medication and metabolic waste products, and formation of clotting factors. Interventions include monitoring nutritional markers and providing nutrition; monitoring fluid balance, urinary output, electrolyte and chemistry studies, drug type, and dose requirements; monitoring bleeding times, platelet function, and hematocrit; and detecting signs of bleeding (Box 41-14). Bowel cleansing regimens may be ordered. The early recognition of complications includes detecting signs of impending liver failure: changes in neurological and mental status, increasing ascites, and HRS.

The critically ill patient in liver failure is often in some state of unconsciousness, with jaundiced skin and sclera. Coagulation times are prolonged, so bleeding is apt to occur from many sources. There is a risk for sores and skin breakdown because of the patient's debilitated state.

Maintaining fluid and electrolyte balance requires ongoing nursing assessment. Imbalance can result from replacement therapy, malnutrition, gastric suction, diuretics, vomiting, diaphoresis, ascites, diarrhea, inadequate fluid intake, and elevated aldosterone levels. The patient may complain of headache, weakness, numbness and tingling of extremities, muscle twitching, thirst, nausea, or muscle cramps and may become confused. The nurse is asked to monitor weight and CVP trends to help determine fluid

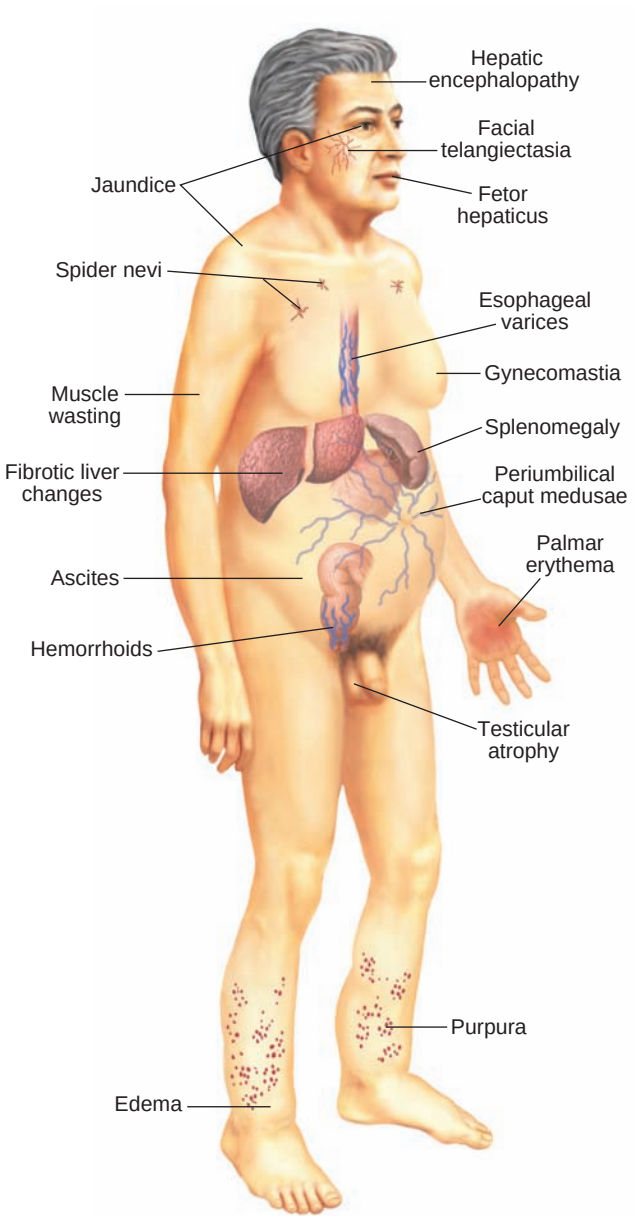


FIGURE 41-6 ▲ Clinical effects of cirrhosis of the liver. (From Porth CM: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2009, p 968.)

retention and vascular loading. Other assessment clues to monitor include any increase or decrease in urinary output, cardiac dysrhythmias, changes in mental status or level of consciousness, prolonged vomiting or frequent liquid stools, muscle tremors, spasms, edema, or poor skin turgor.

Impaired handling of salt and water by the kidney and other abnormalities in fluid homeostasis predispose the patient to ascites, an accumulation of fluid in the peritoneum. This complication can be problematic because it can restrict movement of the diaphragm, impairing the patient's breathing pattern. Therefore, monitoring respiratory status by the nurse is crucial. Ascites is managed through bed rest, a low-sodium diet of no more than 2,000 mg/d, fluid restriction, and diuretic therapy.[47] It has been demonstrated that ascites absorption has an upper limit of 700 to 900 mL/d during diuresis therapy. If diuresis exceeds this limit, it may be at the expense of the intravascular volume and may potentiate hemodynamic instability. Diuresis with spironolactone, an aldosterone antagonist, is first-line diuretic therapy for ascites, although combination therapy with furosemide is more effective.[47] Monitoring for electrolyte imbalance, particularly hypokalemia, is essential. In addition to strict intake and output balance and daily weights, abdominal girth should be measured daily.

Paracentesis is also used to treat ascites in patients unresponsive to salt restriction and maximal diuretic therapy.[47] In this procedure, ascitic fluid is withdrawn from the abdomen through percutaneous needle aspiration. As much as 4 to 6 L/d of ascitic fluid can be withdrawn, and close monitoring of vital signs is important during this procedure because a sudden loss of intravascular pressure may precipitate hypotension, decreased renal perfusion, and tachycardia. Volume expanders are recommended if 5 L or more of ascitic fluid are withdrawn during a single paracentesis procedure (eg, replacement of 5 g of albumin per liter of removed ascites fluid).[47] As with any invasive procedure, there is an increased risk for infection, particularly with repeated large-volume paracentesis procedures (eg, refractory ascites). Refractory ascites results from continued deterioration of liver function and increased portal pressure, increased circulating vasoconstrictors, and decrease in renal blood flow. Refractory ascites marks a sentinel deterioration in the patient's disease trajectory. Refractory ascites requires repeated paracentesis, with decreasing intervals of time between procedures. Unfortunately, paracentesis does not improve the overall poor prognosis, and all patients with refractory ascites should be referred for consideration for a liver transplantation.

A venous–peritoneal (VP) shunt is used to relieve ascites that is resistant to other therapies. The LeVeen shunt (Fig. 41-7) is inserted by placing the distal end of a tube in the abdominal cavity and tunneling the other end into a central vein (eg, the superior vena cava). This perforated intra-abdominal tube allows for ascitic fluid to flow into the central vein. Complications related to placement and use include sepsis, peritonitis, DIC, thrombi formation, and variceal hemorrhage. It is not recommended for patients with infected ascites, encephalopathy, or renal failure. Although the VP shunt controls ascites better than paracentesis, occlusion rates within the first year of placement are high. Because of the aforementioned complications, these shunts are rarely placed in current hepatology practice.

A nonsurgical approach to managing ascites and acute variceal hemorrhage is the TIPS, illustrated in Figure 41-8. The purpose of a TIPS is to decompress the portal venous system and therefore prevent rebleeding from varices or stop or reduce the formation of ascites.[47] TIPS has been associated with improved survival rates, improved renal function via improved flow, and it has even allowed some patients to stop hemodialysis.[47] Absolute contraindications to a TIPS procedure include congestive heart failure, severe tricuspid regurgitation, multiple hepatic cysts, uncontrolled systemic infection or sepsis, unrelieved biliary obstruction, and severe pulmonary hypertension (mean pressures more than 45 mm Hg; these patients are not candidates for liver transplantation). Using an angiographic catheter, a guide wire with a dilating balloon is inserted into the internal jugular vein and

Table 41-5 Laboratory Studies for Hepatic Injury and Function

Parameter	Normal	Increased	Decreased
Hepatocellular Injury			
ALT AST	5–35 IU/L 5–40 IU/L	Acute viral hepatitis (ALT more than AST) Biliary tract obstruction Alcoholic hepatitis (AST more than ALT) Ischemia or hypoxia (“shock liver”) Drug toxicity Right-sided heart failure Liver cancer	Vitamin B deficiency
Liver Synthetic Function			
Albumin	3.4–4.7 g/dL	Dehydration, shock	Chronic liver disease, malnutrition, malabsorption
Total protein	6.0–8.0 g/dL		
PT INR	11–15 s 0.8–1.2 s	Liver disease Vitamin K deficiency Anticoagulants	N/A
Cholestasis or Excretory Function			
Total bilirubin Conjugated (direct) Unconjugated (indirect) Alkaline phosphatase GGT	0.2–1.3 mg/dL 0.1–0.3 mg/dL 0.2–0.7 mg/dL 30–115 IU/L 9–85 units/L	Viral hepatitis Alcoholic hepatitis Obstructive jaundice Primary biliary cirrhosis	N/A

N/A, not applicable, PT, prothrombin time; ALT, alanine transaminase; AST, aspartate aminotransferase; GTT, gamma-glutamyl transpeptidase; INR, international normalized ratio.

is advanced through the liver parenchyma to connect the portal vein, where most of the blood flowing to the liver enters, to the hepatic vein, which empties blood into the inferior vena cava. A stent is then placed to create a conduit between the hepatic and portal vein, which decreases portal pressure. Complications include shunt occlusion, shunt stenosis, and HE. HE increases after a TIPS procedure because this portacaval shunt diverts portal blood flow away

BOX 41-14 COLLABORATIVE CARE GUIDE for the Patient With Cirrhosis and Impending Liver Failure

Outcomes	Interventions
Oxygenation/Ventilation	
The patient’s ABGs will be within normal limits.	• Monitor pulse oximetry and ABG values, respiratory rate and pattern, and ability to clear secretions. • Validate significant changes in pulse oximetry with co-oximetry arterial saturation measurement.
The patient has no evidence of pulmonary edema or atelectasis. Breath sounds are clear bilaterally.	• Assist patient to turn, cough, deep breathe, and use incentive spirometer every 2 h. • Provide chest percussion with postural drainage if indicated every 4 h. • Monitor effect of ascites on respiratory effort and lung compliance. • Position patient on side and with head of bed elevated to improve diaphragmatic movement.
Circulation/Perfusion	
Patient will achieve or maintain stable blood pressure and oxygen delivery. Serum lactate will be within normal limits.	• Monitor vital signs, including cardiac output, systemic vascular resistance, oxygen delivery, and oxygen consumption. • Monitor lactate daily until it is within normal limits. • Administer RBCs, positive inotropic agents, colloid infusion as ordered to increase oxygen delivery.
Patient will not experience bleeding related to coagulopathies, varices, hepatorenal syndrome.	• Monitor PT, PTT, complete blood count daily. • Assess for signs of bleeding (eg, blood in gastric contents, stools, or urine); observe for petechiae, bruising. • Administer blood products as indicated. • Assist with insertion and manage the esophageal tamponade balloon tube. • Perform gastric lavage as needed.

(*continued on page 950*)

BOX 41–14 *COLLABORATIVE CARE GUIDE for the Patient With Cirrhosis and Impending Liver Failure (continued)*

Outcomes	Interventions
Fluids/Electrolytes	
Patient is euvolemic. Patient will not gain weight due to fluid retention.	• Daily weights • Monitor intake and output. • Monitor electrolyte values. • Measure abdominal girth daily at the same location on the abdomen. • Monitor signs of volume overload: Cardiac gallop Pulmonary crackles Shortness of breath Jugular vein distention Peripheral edema • Administer diuretics as ordered.
Mobility/Safety	
Patient is alert and oriented. Ammonia level is within normal limits. Patient achieves or maintains ability to conduct activities of daily living and mobilize self. No evidence of infection, WBC within normal limits.	• Assess serum ammonia level. • Administer lactulose as ordered. • Monitor level of consciousness, orientation, thought processing. • Assess asterixis. • Take precautions to prevent falls. • Consult physical therapist. • Conduct range-of-motion and strengthening exercises. • Monitor SIRS criteria: increased WBC, increased temperature, tachypnea, tachycardia. • Use aseptic technique during procedures and monitor others. • Maintain invasive catheter tube sterility. • Change invasive catheters, culture blood, line tips, or fluids, provide site care, etc., according to hospital protocol.
Skin Integrity	
Skin will remain intact.	• Assess skin every 8 h and each time patient is repositioned. • Turn patient every 2 h. Assist or teach patient to shift weight or reposition. • Consider pressure relief/reduction mattress.
Nutrition	
Caloric and nutrient intake meet metabolic requirements per calculation (eg, basal energy expenditure). Evidence of metabolic dysfunction is minimal.	• Provide nutrition by oral, enteral, or parenteral feeding. • Adhere to sodium, protein, fat, or fluid restrictions as necessary. • Consult dietitian or nutritional support service to evaluate nutritional needs and restrictions. • Provide small, frequent feedings. • Monitor albumin, prealbumin, transferrin, BUN, cholesterol, triglycerides, bilirubin, aspartate transaminase, alanine transaminase. • Administer cleansing enemas and cathartics if ordered.
Comfort/Pain Control	
Patient will have minimal pain. Patient will have minimal pruritus.	• Assess pain and discomfort from ascites, bleeding, pruritus. • Administer analgesics cautiously and monitor patient response. • Bathe with cool water, blot dry. • Lubricate skin. • Administer antipruritic medication; apply to skin PRN as ordered.
Psychosocial	
Patient demonstrates decreased anxiety.	• Assess patient's response to illness. Provide time to listen. • Assess effect of critical care environment on the patient. • Minimize sensory overload. • Provide adequate time for uninterrupted sleep. • Encourage flexible visiting hours for family. • Plan for consistent care giver.

(*continued on page 951*)

BOX 41-14 COLLABORATIVE CARE GUIDE for the Patient With Cirrhosis and Impending Liver Failure (continued)

Outcomes	Interventions
Teaching/Discharge Planning	
Patient/significant others understand procedures and tests needed for treatment of hepatic dysfunction.	• Prepare patient/significant others for procedures such as paracentesis or laboratory studies. • Teach patient and family information regarding sodium, protein, and fluid restrictions. Give written instructions.
Patient/significant others are prepared for home care.	• Teach signs and symptoms of progressing hepatic failure (eg, change in mentation, skin coloration, ascites). • Teach signs and symptoms of occult bleeding and respiratory infection. • Teach home medication regimen. • Teach comfort measures.

from the liver parenchyma. Although TIPS is fairly successful in treating ascites, recent meta-analyses concluded that it can also be associated with the development of increased encephalopathy.[48]

Hepatic Encephalopathy

Patients with severe liver disease can progress to HE, a reversible decrease in neurologic function caused by liver disease. In general, clinical manifestations of HE can be subtle, with changes in memory, personality, concentration, and reaction times. HE can progress to more apparent neurologic cognitive changes, irritability or agitation, reversal of day and night schedules, somnolence, and eventually terminal coma if left untreated. These changes can be graded into stages (subclinical to stupor or coma) by evaluating both intellectual and neuromuscular function. In addition, they may fit into one of three clinical patterns. Type A is related to acute liver failure. Type B occurs in the setting of normal liver histology and the presence of a hepatic vascular bypass (eg, TIPS). Type C is due to cirrhosis and represents most cases in the ICU; it can be divided further into acute and chronic subcategories. Asterixis (a flapping tremor, usually of the hands) is a very early sign of HE. To test for this, the nurse should ask the patient to hold an arm and hand out, with fingers spread, as if stopping traffic and look for involuntary hand "flapping." Other signs are hyperreflexia and muscle rigidity.

The cause of the HE is thought to be related to the accumulation of toxic agents absorbed from the intestinal tract. These substances accumulate because the liver has lost the ability to metabolize and detoxify these substances. Elevated serum ammonia, a byproduct of protein and amino acid metabolism, is one of the suspected neurotoxins. Normally, ammonia is metabolized into urea before entering the systemic circulation, and the urea is then excreted. If the liver is unable to perform this detoxification or if a good portion of the portal blood is shunted around the liver from portal hypertension, the circulating level of ammonia rises. If ammonia and the other toxic agents can be reduced through effective therapy, the encephalopathy gradually clears. Although arterial ammonia levels are more reliable than venous samples, they are often more difficult to obtain and more painful for the patient. In addition, symptoms of HE may lag behind ammonia level elevations, and improvements in HE symptoms may occur before any improvement in ammonia levels.

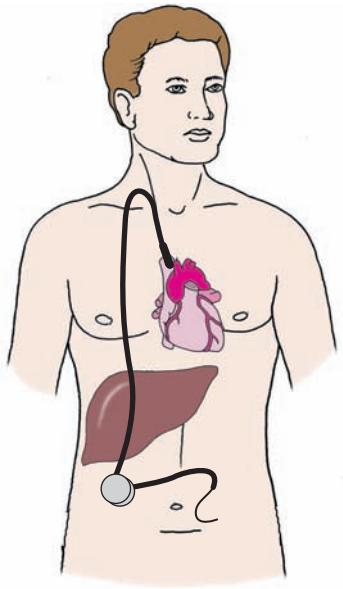


FIGURE 41-7 ▲ The distal end of the LeVeen shunt is tunneled into a central vein. The shunt allows ascites fluid to drain from the abdominal cavity.

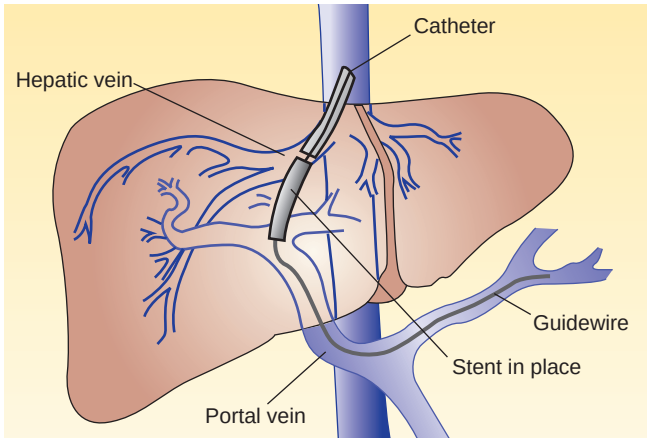


FIGURE 41-8 ▲ Transjugular intrahepatic portosystemic shunt. A stent is inserted through a catheter to the portal vein to divert blood flow and reduce portal hypertension. (From Smeltzer SC, Bare BG, Hinkle JL, et al: Brunner and Suddarth's Textbook of Medical–Surgical Nursing, 11th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, p 1297.)

Unfortunately, bypassing the liver with a shunt may result in decreased clearance and increased accumulation of toxins. HE can develop quite rapidly in patients with portosystemic shunts. People with portal hypertension can hemorrhage from esophageal varices or other sites in the GI tract. The hemorrhage produces a significant nitrogenous load to the intestinal tract in the form of blood, from which bacterial deamination produces the ammonia. Protein intake is limited to 20 to 40 g/d for the treatment of acute HE. Lactulose is used to facilitate bowel movements and clearance of nitrogenous products. Neomycin or metronidazole may be given to clear the gut of bacteria that promote nitrogenous production. Lactulose decreases the colonic pH to prevent the absorption of ammonia. Nursing measures to protect the patient with mental status changes from harm are a priority.

Hepatorenal Syndrome

HRS is the most frequently fatal complication of cirrhosis.[47,49] It is defined as the development of renal failure in patients with severe liver disease (acute or chronic) in the absence of any other identifiable cause of renal pathology.[47,49] Patterns of HRS have been defined as type 1 (acute), 2 (chronic), 3, and 4. The onset of type 1 is rapid, with a creatinine value of more than 2.5 mg/dL or a 50% reduction in initial 24-hour creatinine clearance and a decrease in glomerular filtration rate to less than 20 mL/min within 2 weeks. Type 1 is often observed in acute liver failure or alcoholic hepatitis, or following acute decompensation of a patient with a history of cirrhosis. Patients often appear jaundiced and have a significant coagulopathy; mortality from type 1 HRS is 80% (at 2 weeks) and usually results from a combination of liver and renal failure or variceal bleeding.[49] Type 2 HRS usually occurs in patients with diuretic-resistant ascites. Onset is more insidious, with deterioration in renal failure over months, but it is also associated with a poor prognosis. In both type 1 and 2 HRS, failure of the kidneys is the result of the extreme systemic vasodilation (from the portal hypertension of liver failure), which decreases the effective circulating blood volume. This leads to a compensatory increase in cardiac output and maximal renal vasoconstriction, which reduces renal perfusion and subsequent renal failure. Type 3 categorization defines patients with coexistent intrinsic renal dysfunction and advanced liver disease, whereas type 4 includes patients with acute liver failure.

Ascites, jaundice, hypotension, and oliguria are clinical findings in HRS; laboratory findings typically include azotemia, elevated serum creatinine, urine sodium less than 10 mEq/L, and hyponatremia. Management goals include therapies to support liver and kidney functions. Historically, liver transplantation had been the treatment of choice. However, because elevated pretransplantation serum creatinine levels have been demonstrated as a poor posttransplantation prognostic factor, it is now being suggested that both a liver and kidney transplantation would improve patient survival.[50,51]

Spontaneous Bacterial Peritonitis

Patients with liver disease may be more susceptible to infection because the hepatic Kupffer cells, which are responsible for uptake and subsequent degradation of foreign and potentially harmful substances in the body, do not function as efficiently. SBP occurs when there is a large accumulation of ascites without an identifiable intra-abdominal source of infection (eg, absence of recognizable intestinal perforation). Ascitic fluid contains low concentrations of albumin, which is thought to normally provide some protection against bacteria. Subsequent leakage of bacteria through the abdominal wall or from invasive procedures (eg, endoscopy, nasogastric tube, IV line, or indwelling bladder catheter placement) is thought to precipitate SBP.

Patients with SBP may complain of fever, chills, generalized abdominal pain, or tenderness with palpation (but rarely with rebound tenderness). However, symptoms may be minimal, with only subtle worsening of jaundice or encephalopathic trends.

SBP leads to renal impairment in approximately 10% to 30% of patients with cirrhosis,[49] and an estimated 28% of patients with SBP will develop HRS.[52] The two most common causative organisms are Gram-negative bacteria, *Escherichia coli*, or *Klebsiella*.[53] Subsequently, if SBP is suspected, the ascitic fluid should be evaluated for cell count, differential, and culture. While ascitic fluid bacterial cultures are pending, the diagnosis of SBP is highly likely if ascitic fluid leukocyte count is elevated at more than 500 cells/L (with a proportion of polymorphonuclear leukocytes of more than 50%). The patient should be treated with broad-spectrum antibiotic coverage until the results of these tests are returned. SBP must be differentiated from peritonitis secondary to an abscess or perforation because the latter needs immediate surgical treatment.

Common nursing diagnoses for the patient with a GI disorder are listed in Box 41-15.

BOX 41-15 EXAMPLES OF NURSING DIAGNOSES

For the Patient With a Gastrointestinal Disorder

- Imbalanced Nutrition: Less Than Body Requirements related to altered pancreatic or liver function, impaired digestion from inadequate bile or pancreatic enzyme production, poor eating habits, excessive alcohol intake, nausea, vomiting, or anorexia
- Excess Fluid Volume related to extravascular ascites, portal hypertension, and hypoalbuminemia
- Risk for Deficient Fluid Volume related to overly aggressive diuresis, GIB and coagulopathies, and peritoneal sequestration of intra-abdominal fluids
- Risk for Electrolyte Imbalance related to anorexia, nausea, and vomiting
- Risk for Aspiration related to delayed gastric emptying, intestinal obstruction, ileus, or GIB
- Impaired Gas Exchange related to decreased diaphragmatic excursion secondary to abdominal distention and potential for aspiration
- Decreased Cardiac Output related to hepatic portal hypertension or blood loss from GIB
- Acute Pain related to nasogastric tube irritation, pruritus related to accumulation of bilirubin pigment and salts, inflammation of the pancreas and surrounding tissues, and local peritonitis
- Readiness for Enhanced Self-Health Management, Ineffective Family Therapeutic Regimen Management related to insufficient knowledge of disease process, treatments, contraindications, dietary management, and follow up care

▲ Clinical Applicability Challenges

CASE STUDY

C.K is a 28-year-old female graduate student from China who presents to the emergency department with complaints of hematemesis. She reports dizziness and has had three episodes of vomiting with a large amount of bright red blood present in the emesis. She notes a 3-month history of abdominal distention and bulging flanks. Although the details of her medical history are unclear, she reports a history of "hepatitis." She admits to previous experimental intravenous drug use while attending college, currently drinks approximately three glasses of wine per day, and denies any tobacco history. She reports that she was told her liver function test values were elevated when she last saw her physician in China prior to her immigration 10 years ago. On physical examination her liver span is 6 to 8 cm, and her spleen is palpable. There is scleral icterus. Vital signs are blood pressure, 90/50 mm Hg and pulse rate, 122 beats/min.

Laboratory findings on admission:
Hematocrit (Hct), 22%
Prothrombin time, 20 s
Alanine aminotransferase, 122 units/L
Aspartate aminotransferase 96 units/L
Alkaline phosphatase 76 units/L
Total bilirubin 4.5 mg/dL

1. What is the likely etiology of C.K.'s hematemesis and distention?
2. What other laboratory tests would you expect will be ordered?
3. Discuss potential nursing interventions for C.K.

References

1. Cappell MS, Friedel D: Initial management of acute upper gastrointestinal bleeding: From initial evaluation up to gastrointestinal endoscopy. Med Clin North Am 92:492–509, 2008
2. Cappell MS, Freidel D: Acute nonvariceal upper gastrointestinal bleeding: Endoscopic diagnosis and therapy. Med Clin North Am 92:511–550, 2008
3. Varma MK, Allen AW, Sawyer MAJ: Gastrointestinal bleeding, upper. 2008. Retrieved from: http://emedicine.medscape.com/article/417980
4. Chui PWY, Ng EKW: Predicting poor outcome from acute upper gastrointestinal hemorrhage. Gastroenterol Clin North Am 38:215–230, 2009
5. Kovacs TOG: Management of upper gastrointestinal bleeding. Curr Gastroenterol Rep 10:535–542, 2008
6. Albeldawi M, Qadeer MA, Vargo JJ: Managing acute upper GI bleeding, preventing recurrences. Cleve Clin J Med 77(2):131–142, 2010
7. Kovacs TOG, Jensen DM: The short-term medical management of nonvariceal upper gastrointestinal bleeding. Drugs 68(16):2105–2111, 2008
8. Villanueva C, Balanzo J: Variceal bleeding: pharmacological treatment and prophylactic strategies. Drugs 68(16):2303–2324, 2008
9. Garcia-Tsao G, Bosch J: Management of varices and variceal hemorrhage in cirrhosis. N Engl J Med 362(9):823–832, 2010
10. Smith MM: Variceal hemorrhage from varices associated with alcoholic liver disease. Am J Nurs 110(2):32–39, 2010
11. Toubia N, Sanyal AJ: Portal hypertension and variceal hemorrhage. Med Clin North Am 92:551–574, 2008
12. Barkun AN, Bardou M, Kuipers EJ, et al: International consensus recommendations on the management of patients with nonvariceal upper gastrointestinal bleeding. Ann Intern Med 152:101–113, 2010
13. Leontiadis GI, Howden CW: The role of proton pump inhibitors in the management of upper gastrointestinal bleeding. Gastroenterol Clin North Am 38:199–213, 2009
14. Ali T, Harty RF: Stress-induced ulcer bleeding in critically ill patients. Gastroenterol Clin North Am 38:245–265, 2009
15. Quenot JP, Thiery N, Barbar S: When should stress ulcer prophylaxis be used in the ICU? Curr Opin Crit Care 15:139–143, 2009
16. Sass DA, Chopra KB: Portal hypertension and variceal hemorrhage. Med Clin North Am 93:837–853, 2009
17. Lewis M: Bleeding colonic diverticula. J Clin Gastroenterol 42(10):1156–1158, 2008
18. Weldon DT, Burke SJ, Sun S, et al: Interventional management of lower gastrointestinal bleeding. Eur Radiol 18:857–867, 2008
19. Wilkins T, Baird C, Pearson AN, et al: Diverticular bleeding. Am Family Phys 80(9):977–983, 2009
20. Diaz JJ, Bokhari F, Mowery NT, et al: Guidelines for the management of small bowel obstruction. J Trauma 64:1651–1664, 2008
21. Cappell MS, Batke M: Mechanical obstruction of the small bowel and colon. Med Clin North Am 92:575–597, 2008
22. Trevino C: Small bowel obstruction. AACN Adv Crit Care 21(2): 187–194, 2010
23. American Society for Gastrointestinal Endoscopy Standards of Practice Committee: The role of endoscopy in the management of patients with known and suspected colonic obstruction and pseudo-obstruction. Gastrointest Endosc 71(4):669–679, 2010
24. Batke M, Cappell MS: A dynamic ileus and acute colonic pseudo-obstruction. Med Clin North Am 92:649–670, 2008
25. Stewart D, Waxman K: Management of postoperative ileus. Dis Mon April 2010:204–214, 2010.
26. Andris A: Pancreatitis: Understanding the disease and implications for care. AACN Adv Crit Care 21(2):195–204, 2010
27. Cappell MS: Acute pancreatitis: Etiology, clinical presentation, diagnosis and therapy. Med Clin North Am 92:889–923
28. Lindberg DA: Acute pancreatitis and hypertriglyceridemia. Gastroenterol Nurs 32(2):75–82, 2009
29. Fontana RJ Acute liver failure including acetaminophen overdose. Med Clin North Am 92:761–794, 2008
30. Seitz HK, Stickel F: Alcoholic liver disease in the elderly. Clin Geriatr Med 23(4):905–921, 2007
31. Taylor RM, Davern T, Munoz S, et al: Fulminant hepatitis A virus infection in the United States: Incidence, prognosis, and outcomes. Hepatology 44(6):1589–1597, 2006

32. Gluud LL, Gluud C: Meta-analysis on viral hepatitis. Infect Dis Clin North Am 23:315–350, 2009
33. Centers for Disease Control (CDC): Prevention of hepatitis A through active or passive immunization: Recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR Recomm Rep 55(RR-7):1–23, 2006
34. Lefton HB, Rosa A, Cohen M: Diagnosis and epidemiology of cirrhosis. Med Clin North Am 93:787–799, 2009
35. Shiffman ML: Management of acute hepatitis B. Clin Liver Dis 14: 75–91, 2010
36. Singh SP: Comparison of entecavir and telbivudine in management of chronic Hepatitis B. Hepat B Annu 5(1):134–145, 2008
37. Te HS, Jensen DM: Epidemiology of Hepatitis B and C viruses: A global overview. Clin Liver Dis 14:1–21, 2010
38. Ghany MG, Strader DB, Thomas DL, et al: Diagnosis, Management and Treatment of Hepatitis C: An Update. AASLD Practice Guidelines. Hepatology 49(4):1335–1374, 2009
39. Gardeneir D, ALfandre D: Primary care of the patient with chronic hepatitis C. J Nurse Pract 2(8):517–524, 2006
40. Shiffman ML: Optimizing current therapy for chronic hepatitis C virus: Peginterferon and ribavirin dosing and the utility of growth factors. Clin Liver Dis 12:487–505, 2008
41. Castelnau C, LeGal F, Ripault MP, et al: Efficacy of peginterferon alpha-2b in chronic hepatitis delta: Relevance of quantitative RT-PCR for follow-up. Hepatology 44:728–735, 2006
42. Cappell MS: Hepatic disorders severely affected by pregnancy: Medical and obstetric management. Med Clin North Am 92:739–760, 2008
43. Tan J, Lok ASF: Update on viral hepatitis. Curr Opin Gastroenterol 23:263–267, 2007
44. Patra S, Kumar A, Trivedi SS, et al: Maternal and fetal outcomes in pregnant women with acute hepatitis E virus infection. Annu Intern Med 147(1):28–33, 2007
45. Krawczynski K: Hepatitis E vaccine: Ready for prime time? N Engl J Med 356(9):949–951, 2007
46. Kochanek MA, Murphy SL, Tejada-Vera B: Deaths: Final data for 2007. National Vital Statistic Reports 58(19), 2010. Retrieved November 15, from http://www.cdc.gov/NCHS/data/nvsr/nvsr58/nvsr58_19.pdf
47. Fisher EM, Brown DK: Hepatorenal syndrome. AACN Adv Crit Care 21(2):165–184, 2010
48. Salerno F, Cammà C, Enea M, et al: Transjugular intrahepatic portosystemic shunt for refractory ascites: A meta-analysis of individual patient data. Gastroenterology 133(3):825–834, 2007
49. Munoz SJ: The hepatorenal syndrome. Med Clin North Am 92:813–837, 2008
50. Ruiz R, Kunitake H, Wilkinson AH, et al: Long term analysis of combined liver and kidney transplantation at a single center. Arch Surg 141(8):735–741, 2006
51. Ruiz R, Barri YM, Jennings LW, et al: Hepatorenal syndrome: A proposal for kidney a after liver transplantation (KALT). Liver Transpl 13(6):838–843, 2007
52. Fasolato S, Angeli P, Dallagnese L, et al: Renal failure and bacterial infections in patients with cirrhosis: Epidemiology and clinical features. Hepatology 45:223–229, 2007
53. Lee JM, Han KH, Ahn SH: Ascites and spontaneous bacterial peritonitis: An Asian perspective. J Gastroenterol Hepatol 24:1494–1503, 2009

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

PART 10

ENDOCRINE SYSTEM

42

Anatomy and Physiology of the Endocrine System

Jane Kapustin

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Describe the production, action, and regulation of antidiuretic hormone, growth hormone, and the thyroid hormones.
2. Discuss how activated vitamin D, parathyroid hormone, and calcitonin each influence calcium concentrations in the blood.
3. Explain the production, action, and regulation of insulin.
4. Compare and contrast the pathophysiology of types 1 and 2 diabetes.
5. Describe the roles of counter-regulatory hormones, gut hormones, and glucagon on the regulation of blood glucose.
6. Explain how glucocorticoids are secreted.
7. Discuss the significant effects of glucocorticoid medications.
8. Summarize the renin–angiotensin mechanism for regulating mineralocorticoid secretion.

Communication between systems in the body is accomplished in three ways. One method of communication is the nervous system. A second method is the cellular secretion of chemicals that are released into the interstitial fluid. Examples of this method of communication include the chemicals that trigger a local inflammatory response, such as histamine, complement, and prostaglandins. The third method of communication is the cellular secretion of chemicals that are circulated through the bloodstream. This communication is known more commonly as the endocrine system (Fig. 42-1, Table 42-1). The secretions of endocrine cells are termed hormones. Hormones are molecules synthesized and secreted by specialized cells and released into blood vessels to exert biochemical effects on target cells distant from the site of origin. They control metabolism, transport of substances across the cell membrane, fluid and electrolyte balance, growth and development, adaptation, and reproduction.

Hormone action is specific and depends on linkage with a specialized hormone receptor on the target cell. This hormone–receptor complex is responsible for a series of biological responses. Hormones are either stimulatory or inhibitory. Either their actions are very organ specific, such as prolactin (which only affects the mammary glands), or their effects are generalized, such as insulin (which affects most cellular functions of the body).

Hormone production is maintained by a feedback loop mechanism involving the hypothalamic–pituitary axis system (Fig. 42-2, p. 958). Release of a specific hormone is made possible when the circulating level of that hormone is low (positive feedback). Conversely, when the circulating level of a hormone is high, the release of more hormone is inhibited (negative feedback) until a lower level is reached. This system is regulated by specialized sensors in the hypothalamus that continuously monitor hormone assays to maintain self-regulated homeostasis. Theoretically, when

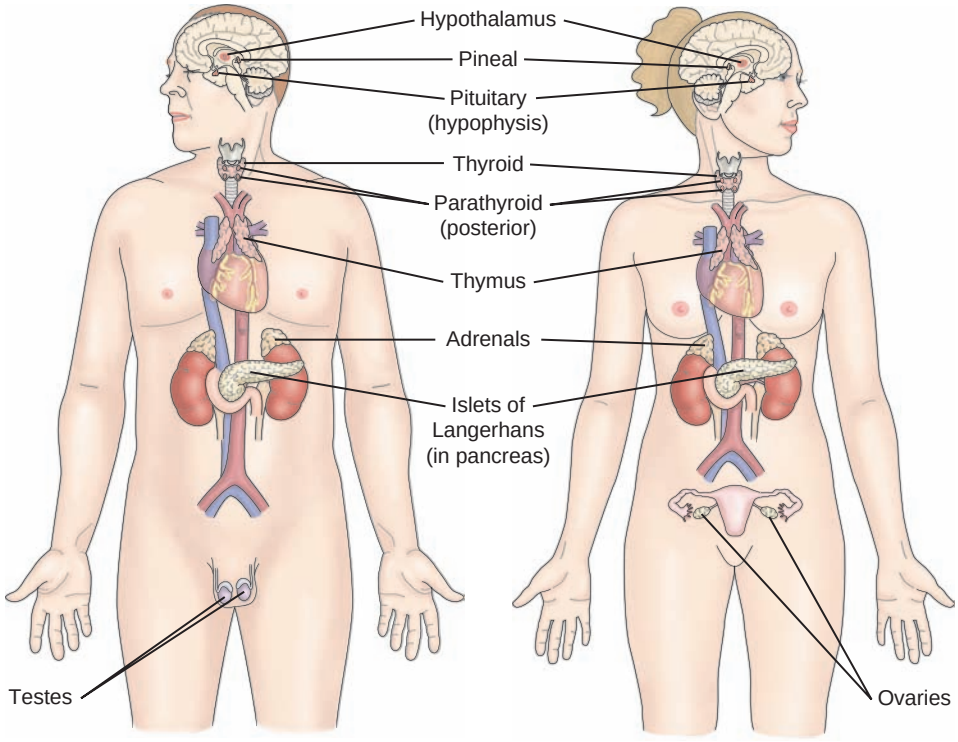


FIGURE 42-1 ▲ The endocrine system. (From Smeltzer SC, Bare BG, Hinkle JL, Cheever KH: Brunner & Suddarth's Textbook of Medical–Surgical Nursing, 12th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 1246.)

Table 42-1 Endocrine System in Summary

Endocrine Gland and Hormone	Principal Site of Action	Principal Processes Affected
Pituitary Gland		
Anterior Lobe		
Growth hormone (GH, somatotropin)	General	Growth of bones, muscles, and other organs
Thyroid-stimulating hormone	Thyroid	Growth and secretory activity of thyroid gland
Adrenocorticotropic hormone	Adrenal cortex	Growth and secretory activity of adrenal cortex
Follicle-stimulating hormone	Ovaries	Development of follicles and secretion of estrogen
	Testes	Development of seminiferous tubules, spermatogenesis
Luteinizing hormone or interstitial cell–stimulating hormone	Ovaries	Ovulation, formation of corpus luteum, secretion of progesterone
Prolactin (luteotropic hormone)	Testes	Secretion of testosterone
Melanocyte-stimulating hormone	Mammary glands and ovaries	Secretion of milk; maintenance of corpus luteum
β-Lipotropin	Skin	Pigmentation
Posterior Lobe		
Antidiuretic hormone (vasopressin)	Kidney	Reabsorption of water; water balance
	Arterioles	Blood pressure
Oxytocin	Uterus	Contraction
	Breast	Expression of milk
Pineal Gland		
Melatonin	Gonads	Sexual maturation
Thyroid Gland		
Thyroxine (T_4) and triiodothyronine (T_3)	General	Metabolic rate; growth and development; intermediate metabolism
Calcitonin	Bone	Inhibits bone resorption; lowers blood level of calcium

(continued on page 957)

Table 42-1 Endocrine System in Summary (continued)

Endocrine Gland and Hormone	Principal Site of Action	Principal Processes Affected
Parathyroid Glands		
Parathyroid hormone (PTH)	Bone, kidney, intestine	Promotes bone resorption; increases absorption of calcium; raises blood calcium level
Adrenal Glands		
Cortex		
Mineralocorticoids (eg, aldosterone)	Kidney	Reabsorption of sodium; elimination of potassium
Glucocorticoids (eg, cortisol)	General	Metabolism of carbohydrate, protein, and fat; response to stress; anti-inflammatory
Sex hormones	General	Preadolescent growth spurt
Medulla		
Epinephrine	Cardiac muscle, smooth muscle, glands	Emergency functions: same as stimulation of sympathetic nervous system
Norepinephrine	Organs innervated by sympathetic nervous system	Chemical transmitter substance; increases peripheral resistance
Islet Cells of Pancreas		
Insulin	General	Lowers blood glucose; utilization and storage of carbohydrate; decreases gluconeogenesis
Glucagon	Liver	Raises blood glucose; glycogenolysis
Somatostatin	General	Lowers blood glucose by interfering with release of GH and glucagon
Testes		
Testosterone	General	Development of secondary sex characteristics
	Reproductive organs	Development and maintenance; normal function
Ovaries		
Estrogens	General	Development of secondary sex characteristics
	Mammary glands	Development of duct system
	Reproductive organs	Maturation and normal cyclic function
Progesterone	Mammary glands	Development of secretory tissue
	Uterus	Preparation for implantation; maintenance of pregnancy
Gastrointestinal Tract		
Gastrin	Stomach	Production of gastric juice
Enterogastrone	Stomach	Inhibits secretion and motility
Secretin	Liver and pancreas	Production of bile; production of watery pancreatic juice (rich in $NaHCO_3$)
Pancreozymin	Pancreas	Production of pancreatic juice, rich in enzymes
Cholecystokinin	Gallbladder	Contraction and emptying

functioning properly, this system prevents the overproduction of hormones.

The effects of aging can influence the endocrine system as well (Box 42-1). As humans age, target organ sensitivity decreases. The target organs demonstrate the effects of aging by either increasing in pigmentation or shrinking in size. This, in effect, decreases hormone receptor binding. This phenomenon explains why older patients, women in particular, are at higher risk for development of hypothyroidism: aging can decrease production of triiodothyronine (T_3) and thyroxine (T_4) and can lead to thyroid gland atrophy.

Endocrine dysfunction can be identified as belonging to one of five major categories:

- Subnormal hormone production as a result of gland destruction or malformation
- Hormone excess
- Production of abnormal hormone resulting from gene mutation
- Hormone receptor disorders resulting from autoimmune processes
- Disorders of hormone transport or metabolism, resulting in increased levels of "free" hormones in the blood

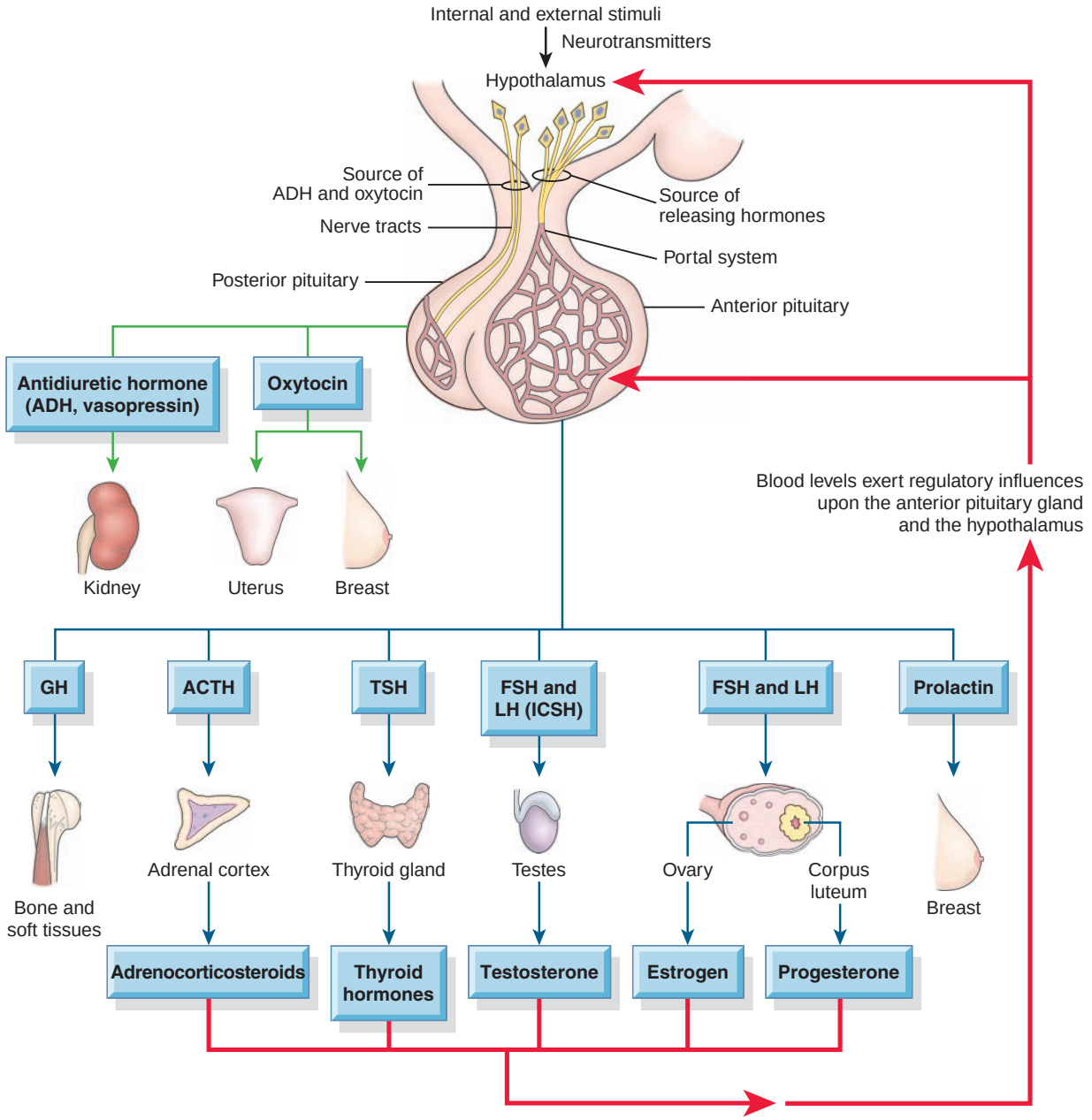


FIGURE 42-2 ▲ The feedback loop mechanism that controls hormone production. Sensors in the hypothalamus monitor hormone levels and initiate or suppress production accordingly. ACTH, adrenocorticotropic hormone; ADH, antidiuretic hormone; CNS, central nervous system; FSH, follicle-stimulating hormone; LH, luteinizing hormone; TSH, thyroid-stimulating hormone. (From Smeltzer SC, Bare BG, Hinkle JL, et al: Brunner & Suddarth's Textbook of Medical–Surgical Nursing, 12th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 1250.)

BOX 42-1 CONSIDERATIONS FOR THE OLDER PATIENT

Physiological Changes in the Endocrine System That Occur With Aging

- Production of thyroid hormone, cortisol, and aldosterone decreases with age.
- Levels of somatostatin, triiodothyronine (T_3), thyroxine (T_4), thyroid-stimulating hormone, aldosterone, renin, calcitonin, and vasopressin decrease with age, as does glucose tolerance.
- Levels of norepinephrine, parathyroid hormone, atrial natriuretic peptide, insulin, and glucagon increase with age.

▲ The Hypothalamus and Pituitary Gland

The key to understanding the physiology of the hormones of the pituitary gland lies in visualizing the anatomy of the gland and its blood supply. The hypothalamus and pituitary share two connecting pathways: a rich vascular network, which connects the hypothalamus with the anterior pituitary, and nerve fibers, which link the hypothalamus with the posterior pituitary. Together, these two glands form a unit that controls the thyroid gland, the adrenal glands, and the gonads, and exerts control over the growth and metabolism of the organism.

Because of the control the pituitary gland exerts over all body functions, it is often referred to as the master gland. It has two distinct regions: the anterior (front) lobe and the posterior (back) lobe. Located at the base of the skull in the sphenoid bone at a site referred to as the sella turcica, this well-protected gland is difficult to reach surgically because it is so deeply embedded in the skull. Despite being well protected, the pituitary is still susceptible to injury as a result of head or facial trauma, edema, or surgical complications. Because it is so vascular, the pituitary is extremely vulnerable to injury from ischemia and infarction.

The hypothalamus is a small area at the base of the brain connected to the posterior pituitary (also known as the neurohypophysis) by the pituitary stalk. This stalk is a direct outgrowth of the neuroectoderm of the base of the brain that drops during development of the gland into the bony sella turcica. This is in direct contrast to the anterior pituitary (adenohypophysis), which arises from the buccal endothelium and develops separately in the same bony structure. Besides being embryogenically separate, the blood supplies of the anterior and posterior pituitary differ. The hypothalamus has specialized nerve cells that are in constant communication with the third ventricle of the brain where continuous sampling of serum osmolality can occur. Information about the osmolality influences the release or inhibition of hormones. It is through this relationship that the hypothalamus influences the release of chemical and neural signals to maintain homeostasis. Because the hypothalamus controls the releasing or inhibiting hormones that influence the pituitary, it assumes the function of the coordinating center of the brain for endocrine, behavioral, and autonomic nervous system function. The hypothalamus is responsible for communicating emotion, pain, body temperature, and other neural input to the endocrine system.

The anterior pituitary hormones are controlled by releasing factors that are secreted from the hypothalamus and are called hypophysiotropic hormones. The hypophysiotropic hormones are secreted into the primary capillary plexus near the median eminence that supplies blood to the anterior pituitary (Fig. 42-3). This blood may also travel in a retrograde fashion and may be responsible for one level of feedback

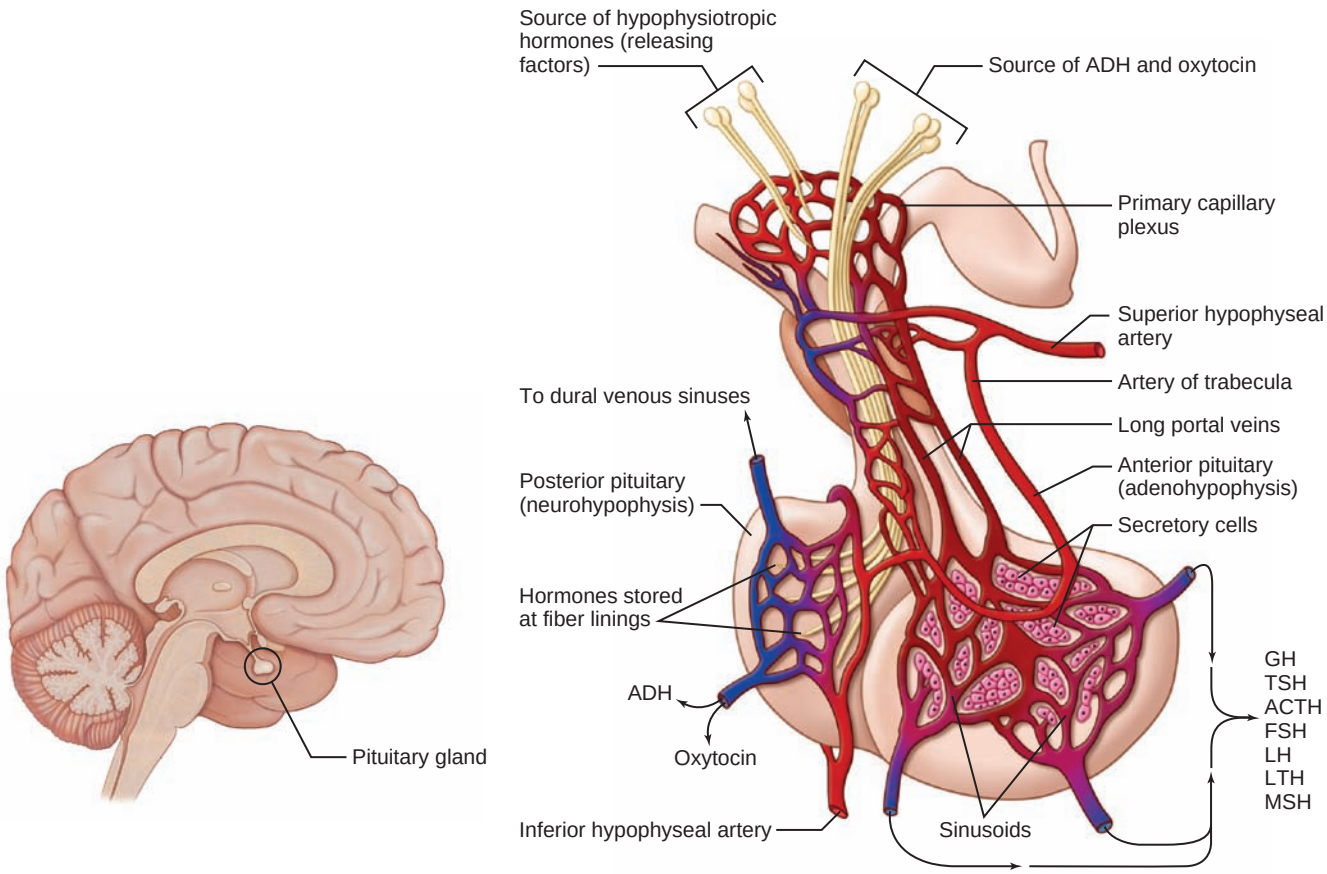


FIGURE 42-3 ▲ **A, B:** Highly diagrammatic and schematic representation of hypophyseal nerve fiber tracts and the portal system in the hypothalamus and pituitary gland. Releasing factors (hypophysiotropic hormones) produced by cell bodies in the hypothalamus trickle down axons to the proximal part of the stalk, where they enter the primary capillary plexus and are transported through portal vessels to sinusoids in the anterior lobe of the pituitary gland (ie, the adenohypophysis) for control of secretions. Antidiuretic hormone (ADH) and oxytocin, produced by other cell bodies in the hypothalamus, trickle down axons for storage in the posterior lobe of the pituitary gland (ie, the neurohypophysis) until they are needed. ACTH, adrenocorticotropic hormone; FSH, follicle-stimulating hormone; GH, growth hormone; LH, luteinizing hormone; LTH, lactogenic hormone; MSH, melanocyte-stimulating hormone; TSH, thyroid-stimulating hormone.

control of the anterior pituitary and hypophysiotropic hormones. A given hypophysiotropic hormone regulates the secretion of one or two anterior pituitary hormones. Both growth hormone (GH, somatotropin) and prolactin are dually controlled by a stimulatory and an inhibitory hypophysiotropic hormone. The posterior pituitary is a direct neural extension of the hypothalamus, and the controlling factors reside in the neural chiasma of the hypothalamus; they are secreted by those cells into the posterior pituitary (see Fig. 42-3). In addition to controlling the pituitary gland through releasing factors, the hypothalamus controls other endocrine roles through releasing factors that control appetite, thirst, emotions, sleep–wake cycles, and cognition.

Such hypothalamic regulation of pituitary functioning can be disrupted by hypothalamic lesions. This can lead to oversecretion or undersecretion of one or more hormones released from the anterior or posterior pituitary. The hypothalamus also receives input from various higher and lower brain centers. These neural connections, together with the influence of the hypothalamus on the pituitary, provide the biological basis for the construction of conceptual models that describe how stress, emotions, environmental stimuli, and perceptions affect endocrine functions.

Posterior Pituitary (Neurohypophysis) Hormones

The posterior pituitary (neurohypophysis) makes up 20% of the gland. The two major hormones of the posterior pituitary gland are antidiuretic hormone (ADH, vasopressin) and oxytocin (see Table 42-1, p. 957). Because oxytocin does not have a role in critical care, it is not discussed here.

The two major actions of ADH are to concentrate the urine (by permitting only water reabsorption from the hypotonic tubular fluid in the distal nephron) and to constrict smooth muscles in the arterial wall. ADH binds to specific receptors in the distal renal tubules to increase their permeability to water. This results in increased water reabsorption but without electrolyte reabsorption. This reabsorbed water increases the volume and decreases the osmolality of the extracellular fluid (ECF). At the same time, it decreases the volume and increases the concentration of the urine excreted. Without ADH, the distal convoluted tubule would be impermeable to water. In the presence of ADH, the tubule and collecting duct are permeable to water, which diffuses from the hypotonic tubular fluid to the hypertonic tissue surrounding the tubules. This concentrates the tubular fluid and ultimately the urine.

The term *vasopressin* originated from the observation that large, supraphysiological dosages of ADH act on arteriole smooth muscle to elevate blood pressure. Although this pressor action of ADH does not appear to play a role in the normal homeostasis of blood pressure, it does counteract a fall in blood pressure that results from hemorrhagic or other drastic hypovolemic states and can be used pharmacologically for that purpose.

There are three major stimuli for the regulation of ADH secretion. The first is plasma osmolality, which is monitored by osmoreceptors in the anterior hypothalamus. An increase above the normal osmolality of plasma (290 mOsm/kg) results in neural stimuli from these receptors to the ADH-secreting cells, increasing ADH secretion. This increases water retention, thereby diluting the ECF and lowering the plasma osmolality back to normal. Similarly, a fall in plasma osmolality triggers a decrease or cessation in ADH secretion. This allows more water excretion, thereby raising the ECF osmolality. ADH secretion can be altered by changes in osmolality of less than 1%. This osmoreceptor-mediated reflex arc functions to maintain osmotic homeostasis of the ECF.

The second stimulus consists of changes in ECF volume. Stretch receptors in the low-pressure portion of the cardiovascular system (eg, the vena cava, the right atrium of the heart, and the pulmonary vessels) monitor blood volume. Stimuli from these receptors are conducted by afferent fibers to the hypothalamus (by way of the brainstem). A decrease in blood volume stimulates ADH secretion. The resultant increase in water retention elevates the blood volume. An increase in blood volume stops ADH secretion. This halts water retention, thereby restoring the normal volume of the ECF compartment. This mechanism alters ADH secretion in response to changes in body position. Movement from the recumbent to the upright position causes a temporary decrease in the stimulation of volume receptors because blood pools in the legs. This results in an increase in ADH secretion. Recumbency increases venous return from the legs. The increased volume triggers a decrease in ADH secretion, thereby increasing the volume of urine excreted.

The third stimulus, changes in arterial blood pressure, also can regulate ADH secretion. The hypothalamus receives information from pressure receptors located in the carotid sinuses and aorta. A fall in arterial pressure increases ADH secretion. The water retention thereby produced increases the plasma volume and pressure. A rise in arterial pressure produces the opposite effect. This mechanism is most important in compensating for large changes in arterial blood pressure (eg, impending or actual shock).

Various other stimuli have been shown to influence ADH secretion. Increased ADH secretion can be prompted by angiotensin II, pain, increased serum osmolality, hypovolemia, nausea and emesis, hypoglycemia, stress, acute infections, malignancies, nonmalignant pulmonary conditions, and trauma to the hypothalamic–hypophyseal system. Secretion of ADH is inhibited by decreased serum osmolality, hypervolemia, water intoxication, cold, trauma to the hypothalamic–hypophyseal system, carbon dioxide inhalation, and alcohol ingestion. Many drugs affect ADH secretion (Box 42-2).

Anterior Pituitary (Adenohypophysis) Hormones

This anterior lobe of the pituitary gland contains five morphologically different types of cells that secrete polypeptide hormones:

- Somatotrophs, which secrete GH (somatotropin)
- Mammotrophs, which secrete prolactin (luteotropic hormone, or LTH)
- Thyrotrophs, which secrete thyroid-stimulating hormone (TSH)
- Corticotrophs, which secrete adrenocorticotropic hormone (ACTH), β-lipotropin, β-endorphin, and melanocyte-stimulating hormone (MSH)
- Gonadotrophs, which secrete luteinizing hormone (LH) and follicle-stimulating hormone (FSH)

BOX 42-2 Drugs that Influence Antidiuretic Hormone (ADH) Secretion

Drugs That Stimulate ADH Secretion
- Diuretics
- Barbiturates
- Glucocorticoids
- Tricyclic antidepressants
- Carbamazepine
- Chlorpropamide
- Anesthetics
- Acetaminophen

Drugs That Inhibit ADH Secretion
- Alcohol
- Phenytoin
- Narcotics
- Lithium
- Demeclocycline
- Norepinephrine
- Chlorpromazine

Each type of cell is separately regulated by hypophysiotropic hormones (Fig. 42-4).

LTH, LH, and FSH are not significant in the critical care arena and are not discussed in this chapter. TSH, which stimulates cells of the thyroid gland to produce and secrete the two thyroid hormones, is discussed later in this chapter, and GH is described in the following paragraph.

The production and secretion of GH occurs in the anterior pituitary in response to GH-releasing hormone produced in the hypothalamus. Growth-inhibiting hormone inhibits the secretion of GH. GH acts both directly on target cells and indirectly by stimulating the liver and other as-yet-unidentified tissues to secrete various growth factors termed somatomedins. These growth factors are structurally similar to insulin. Direct actions of GH include increasing the breakdown of fats (lipolysis) in adipose cells and releasing the fatty acids produced by lipolysis into the bloodstream (this is termed its ketogenic effect); increasing hepatic glycolysis and thereby increasing plasma glucose levels; increasing the sensitivity of insulin-producing cells to certain stimuli; increasing the cellular uptake of amino acids; and stimulating erythropoiesis.

▲ The Thyroid and Parathyroid Glands

The thyroid gland is a bilobed, richly vascularized structure. The lobes lie lateral to the trachea just beneath the larynx and are connected by a bridge of thyroid tissue, the isthmus that runs across the anterior surface of the trachea (Fig. 42-5). Microscopically, the thyroid is composed primarily of spheroid follicles, each of which stores a colloid material in its center. The follicles produce, store, and secrete the two major thyroid hormones: T_3 and T_4. If the gland is actively secreting, the follicles are small and contain little colloid. Inactive thyroid tissue contains large follicles, each of which possesses a large quantity of stored colloid. Parafollicular cells (C cells), which produce the hormone calcitonin, are scattered between the follicles of the thyroid gland.

Each lobe of the thyroid gland typically contains two parathyroid glands: one in its superior pole and one in its inferior pole. Individual variation exists with respect to the number and distribution of parathyroid glands. Some people have more or fewer than four. Others have parathyroid tissue in the mediastinum. The parathyroid glands produce parathyroid hormone (PTH) that is active with maintaining calcium balance.

Thyroid Hormones

The follicular cells absorb tyrosine (an amino acid) and iodide from the plasma and secrete them into the central colloid portion of the follicle, where they are used in the synthesis of T_3 and T_4. (The subscript refers to the number of iodide molecules that each substance contains.) Two iodide molecules are attached, first one and then the other, to each tyrosine molecule. Two such doubly iodinated tyrosines are combined to form T_4. T_3, which is much more biologically active than T_4, is the predominant form of thyroid hormone

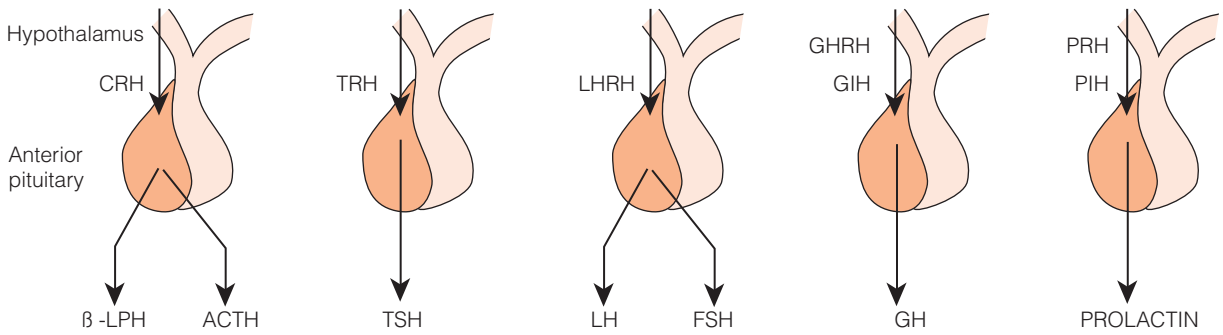


FIGURE 42-4 ▲ Effects of the hypophysiotropic hormones on the secretion of anterior pituitary hormones. The hypophysiotropic hormones are corticotropin-releasing hormone (CRH), thyrotropin-releasing hormone (TRH), luteinizing hormone–releasing hormone (LHRH), growth hormone–releasing hormone (GHRH), growth-inhibiting hormone (GIH), prolactin-inhibiting hormone (PIH); and prolactin-releasing hormone (PRH). CRH prompts the release of β-lipotropin (β-LPH) and adrenocorticotropic hormone (ACTH). TRH prompts the release of thyroid-stimulating hormone (TSH). LHRH prompts the release of luteinizing hormone (LH) and follicle-stimulating hormone (FSH). GHRH and GIH promote and inhibit the secretion of GH, respectively. PRH and PIH promote and inhibit the secretion of prolactin, respectively.

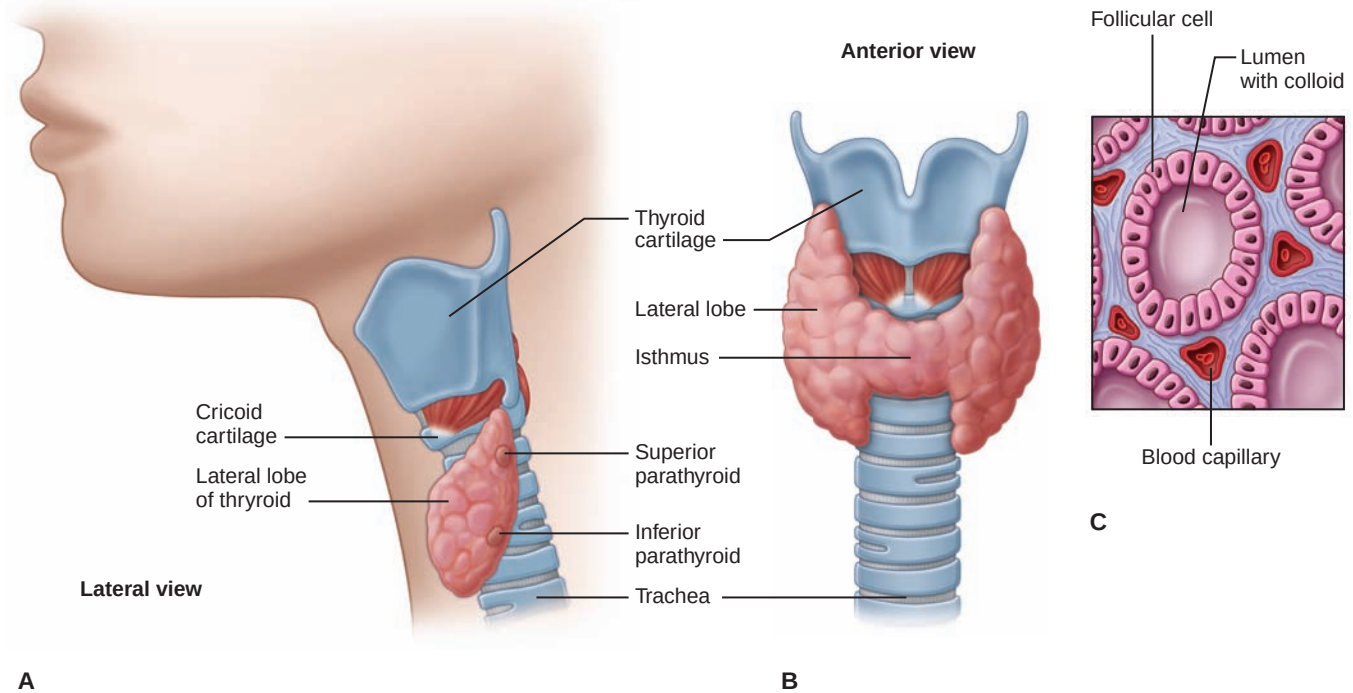


FIGURE 42-5 ▲ **A-C:** The thyroid gland. (Adapted from Porth CM, Matfin G: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 1031.)

produced and is formed by the combination of a doubly iodinated tyrosine with a singly iodinated one. Because of the role of iodine in the manufacture of thyroid hormones, storage and release of small amounts of radioactive iodine by the thyroid can be used to measure the activity of this gland. Because the thyroid gland is virtually the only tissue of the body that absorbs and stores iodine, larger amounts of radioactive iodine can be used to destroy portions of the thyroid gland as a treatment for hyperthyroidism.

T_3 and T_4 are stored in the colloid until they are needed. When they are to be secreted, the follicular cells transport them from the colloid to the plasma. Less than 1% of the secreted T_3 and T_4 remains free and physiologically active in the plasma. The remainder is bound to plasma proteins. The plasma proteins involved in transporting T_3 and T_4 are manufactured in the liver. Consequently, liver damage that decreases the plasma levels of these proteins can produce a condition resembling thyroid hormone excess (ie, hyperthyroidism). Plasma levels of these proteins can also be depressed by glucocorticoids, androgens, and L-asparaginase (an antineoplastic drug). They are elevated during pregnancy, and by estrogens, opiates, clofibrate, and major tranquilizers. Thyroid hormones are deiodinated and catabolized by the liver, kidneys, and various other tissues. A small amount of degraded hormone is added to the bile secreted by the liver and is excreted in the stool.

T_3 directly crosses target cell membranes, whereas T_4 is changed into T_3 by target cell membranes before crossing. The conversion of T_4 to T_3 occurs in the peripheral tissues of the liver and kidneys and accounts for 80% of the T_3 available for metabolic activity. T_4 is 100% produced in the thyroid.

The actions of thyroid hormones are widespread and apparently arise from their stimulation of the basal metabolic rate of most tissues (excluding tissues of the brain, anterior pituitary, spleen, lymph nodes, testes, and lung). Thyroid hormone increases the number of β_1- and β_2-adrenergic receptors in various tissues and the affinity of these receptors for catecholamines; thus, an increased heart rate and sweating often occur in hyperthyroidism. Thyroid hormones increase the catabolism of skeletal muscle proteins to such a degree that pronounced muscle weakness results from prolonged hyperthyroidism (thyrotoxic myopathy). Thyroid hormones increase the rate of carbohydrate absorption from the small intestine and decrease circulating levels of cholesterol.

Thyroid hormones are essential for the normal growth and development of many body systems, notably the skeletal and nervous systems. These hormones stimulate the secretion of GH and potentiate its effect on various tissues. Thyroid hormones are also necessary for normal levels of neuronal functioning. Thyroid insufficiency leads to slowed reflexes, slowed mentation, and decreased level of consciousness (through decreased levels of reticular activating system activity). Hyperthyroidism lowers synaptic thresholds in the central nervous system (CNS), causing hyperreflexia and a fine muscle tremor. The pervasive effects of thyroid hormones on the nervous system are best illustrated by cretinism, a condition resulting from congenital thyroid insufficiency.

The secretion of T_3 and T_4 by the thyroid gland is primarily regulated by the secretion of TSH from the anterior pituitary. In turn, TSH secretion is regulated by a hypothalamic neurosecretory material termed thyrotropin-releasing hormone (TRH). After receiving stimuli from TRH from the hypothalamus, the thyroid secretes TSH to stimulate the manufacture and secretion of T_3 and T_4. A negative feedback regulatory loop exists whereby increased levels of free (unbound) T_3 and T_4 suppress TSH secretion. Decreased

plasma TSH results in decreased thyroid function, which causes a fall in free plasma T_3 and T_4. Low T_3 and T_4 levels stimulate TSH secretion. If a TSH-induced increase in thyroid activity does not raise the plasma levels of free T_3 and T_4, the continued high levels of TSH eventually cause an increase in the size of the thyroid gland (nontoxic goiter). In this case, an enlarged thyroid is not associated with overproduction of hormone. This feedback loop maintains homeostasis of the daily secretion of TSH and thyroid hormones.

Calcitonin and Parathyroid Hormone

Calcitonin, which is secreted by the parafollicular cells of the thyroid gland, and PTH, which is produced and secreted by the parathyroid glands, exert a major influence on calcium metabolism in conjunction with 1,25-dihydroxycholecalciferol, which is produced by the action of the liver and the kidneys on vitamin D. Ultraviolet light changes 7-dehydrocholesterol provitamins in the skin to a group of compounds, collectively called vitamin D. One of these, D_3, can also be obtained from vitamin D–enriched and other foods. The liver converts D_3 to 25-hydroxycholecalciferol, which is then altered by kidney cells to a more active form, 1,25-dihydroxycholecalciferol. (The hypocalcemia seen in chronic renal disease results from an activated vitamin D deficiency.) Activated vitamin D acts on intracellular enzymes of the intestinal mucosal cells to increase calcium absorption. To a lesser extent, it also increases the active transport of calcium out of osteoblasts into the bloodstream. Both of these actions elevate plasma calcium levels. In vitamin deficiency states, the effect of decreased intestinal absorption outweighs any decrease in the mobilization of calcium from bone to produce an overall hypocalcia and poor mineralization of bone.

Vitamin D is synthesized in the skin, absorbed in the small intestine, and transported into the plasma bound to vitamin D–binding proteins. The metabolism of vitamin D is strictly regulated by phosphate concentration in the kidney and by PTH. Thus, the effect of a decrease in dietary phosphate or serum phosphate is to increase levels of 1,25-dihydroxycholecalciferol.

Parathyroid Hormone

PTH is a polypeptide produced and secreted by the chief cells of the parathyroid glands. This hormone is stored in secretory granules and released in response to a decrease in ionized calcium concentrations. It is cleaved into active form in the kidneys and liver. Plasma calcium and phosphate levels operate in a negative feedback loop to influence the activity of the renal enzyme system, which catalyzes the conversion of metabolically inactive vitamin D to the metabolically active form. High plasma calcium levels decrease this activation process, whereas low levels increase it. The formation of activated vitamin D is also facilitated by PTH and decreased by metabolic acidosis and hypoinsulinemia (diabetes mellitus).

PTH is transported free (unbound) in the plasma, has a half-life of less than 20 minutes, and is metabolically degraded by cells in the liver. A decrease in calcium concentration increases PTH secretion. PTH acts on two target tissues: bone cells and kidney tubules. In bone, it stimulates osteoclast activity and inhibits osteoblast activity. This results in bone reabsorption with consequent mobilization of calcium and phosphate from the bony matrix into the bloodstream. In the kidney, PTH increases the reabsorption of calcium by distal tubule cells and decreases the reabsorption of phosphate by proximal tubule cells. The effect of these multiple actions is elevation of plasma calcium levels and lowering of plasma phosphate levels.

Plasma calcium levels alter PTH secretion through a negative feedback loop. Secretion is inhibited by high plasma calcium levels and stimulated by low blood levels of calcium. The activated vitamin D deficiency–induced hypocalcemia, which occurs in chronic renal failure, typically produces a secondary hyperparathyroidism. Secretion of PTH by the parathyroid gland is also stimulated by hypomagnesemia, adrenergic agonists, and prostaglandins.

Calcitonin

This polypeptide hormone is produced by the parafollicular cells (C cells) of the thyroid gland. It can also be secreted by nonthyroidal tissue (eg, tissue of the lung, intestine, pituitary, and bladder). Calcitonin is transported unbound in the plasma. It has a half-life of 5 minutes and is predominantly metabolized in the kidney. Calcitonin lowers plasma calcium and phosphate levels by inhibiting osteoclastic bone reabsorption and increasing urinary phosphate and calcium excretion. Calcitonin levels are elevated during pregnancy and lactation, suggesting that calcitonin may help to protect the mother's skeleton from excess calcium loss during these periods of calcium drain.

Calcitonin does not function in the normal daily homeostasis of plasma calcium levels. It appears to serve more of an emergency function in that it is secreted only if the plasma calcium level exceeds 9.3 mg/dL. At high blood calcium levels, calcitonin secretion is stimulated by increased levels of plasma calcium. Calcitonin is also released by the action of gastrin, glucagon, and secretion of gastrointestinal hormones.

Table 42-2 summarizes the hormones secreted by the thyroid and parathyroid glands.

Table 42-2 Hormones of the Thyroid and Parathyroid Glands and Their Actions

Gland	Hormone	Action
Thyroid gland	Thyroxine (T_4)	Controls basic metabolic rate
	Triiodothyronine (T_3)	Induces growth and development
		Inhibits bone resorption
	Calcitonin	Inhibits calcium reabsorption in gastrointestinal tract
		Increases calcium excretion from kidney
Parathyroid gland	PTH	Promotes bone resorption
		Increases calcium reabsorption
		Increases calcium blood levels

▲ The Endocrine Pancreas

The pancreas lies transversely under the stomach between the duodenum and spleen. Because of its posterior position, it is essentially hidden and is not palpable. The pancreas has both endocrine and exocrine functions, which are under the control of different groups of cells. The organ is made up of two tissue types: the acini, the exocrine portion, and the islets of Langerhans, the endocrine portion. The acini secrete digestive enzymes into the duodenum, whereas the islets of Langerhans secrete hormones into the blood.

The islets of Langerhans secrete the peptide hormones involved in blood glucose regulation. The name "islets of Langerhans" refers to the more than 1 million ovoid islands (clusters) of cells that are scattered throughout the pancreas, predominantly in the tail. Because of this distribution of islet cells, acute attacks of pancreatitis, which usually spare the tail, tend to spare the islets. Episodes of chronic recurrent pancreatitis typically involve the entire pancreas. Consequently, chronic episodes can cause islet cell destruction and diabetes mellitus.

Each cell cluster is richly supplied with capillaries, into which its hormones are secreted. The islets are composed of four types of cells: α cells, which secrete glucagon; β cells, which secrete insulin; δ cells, which secrete somatostatin; and F cells, which secrete pancreatic polypeptide. The hormones secreted by the pancreas are summarized in Table 42-3.

Insulin

Insulin, an anabolic hormone, is regulated by a number of stimulatory and inhibitory factors. It is responsible for the control of blood glucose concentrations and storage of carbohydrate, proteins, and fats. Insulin facilitates the use of glucose as the main source of energy for most body tissues. Insulin is the only hormone with the ability to directly lower the blood glucose level. Also, insulin facilitates an increase in the cellular transport of glucose, amino acids, and fatty acids across cell membranes and modulates intracellular metabolic synthesis of nucleic acids. Cell membranes require a glucose transporter to carry glucose into the cell at a faster rate than diffusion. GLUT-4 is the glucose transporter for skeletal muscle and adipose tissue, and GLUT-2 carries glucose into β cells and liver tissue.

The precursor of insulin, proinsulin, is manufactured in the β cells of the islets of Langerhans. Proinsulin can be thought of as a "necklace" of amino acid beads and is stored as secretory granules in another cell structure. Proinsulin can be found in the plasma as a result of certain islet tumors (insulinoma) or overstimulation of the β cells. Connecting peptide (C-peptide) is a biologically inactive chain and is secreted into the bloodstream along with insulin. Because there is a 1:1 ratio between C-peptide and insulin, plasma C-peptide levels can be used to measure endogenous insulin secretion or degree of β-cell activity. Clinically, C-peptide levels can assist with distinguishing between types 1 and 2 diabetes (C-peptide is low in type 1 diabetes, reflecting autodestruction of β cells and no further production of insulin).[1]

The actions of insulin are summarized in Box 42-3. In addition to facilitating glucose uptake by muscle and adipose cells, insulin facilitates glucose uptake by connective tissue, leukocytes, mammary glands, the lens of the eye, the aorta, the pituitary gland, and α islet cells. In general, insulin enables glucose to be readily available for aerobic oxidation in muscle, adipose, and connective tissue cells. Facilitation of the preferential use of glucose as cellular fuel means that the cells do not need to oxidize fatty or amino acids. Instead, these can be conserved. Protein synthesis and fat storage are increased in liver, muscle, and adipose tissue. Breakdown of fats and proteins is decreased. Hepatic gluconeogenesis also is decreased or halted, and glycogen synthesis is increased.

Insulin acts only on a few types of tissues. However, the membranes of nearly all types of body cells possess insulin receptors. Binding of insulin to the insulin receptors initiates the physiological action of insulin on the cell. Plasma insulin has a half-life of approximately 5 minutes. About 80% of all circulating insulin is catabolized by liver and kidney cells.

Insulin secretion is influenced by a variety of factors as listed in Box 42-4. Monosaccharides are the primary regulatory mechanism for insulin secretion. Elevated plasma levels

Table 42-3 Hormones of the Pancreas and Their Actions

Hormone	Cell	Stimulant	Response
Insulin	β	Glucose	Decreased glucose level Increased fat storage Increased protein synthesis Increased glucogenesis
Glucagon	α	Decreased glucose level, exercise	Increased glucose level Increased gluconeogenesis Increased glycogenolysis
Somatostatin	δ	Hyperglycemia	Increased glucose Increased glycogen
Pancreatic polypeptide	F	Acute hypoglycemia	Increased gallbladder contraction Increased pancreatic enzymes

BOX 42-3 Major Actions of Insulin on Adipose and Muscle Cells

Muscle Cells	Adipose Cells
Increased glucose entry	Increased glucose entry
Increased K^+ uptake	Increased K+ uptake
Increased glycogen synthesis	Increased fatty acid entry and synthesis
Increased amino acid entry	Increased fat deposition
Increased protein synthesis	Increased conversion of glucose to fatty acids
Decreased protein catabolism	Inhibition of lipolysis
Increased ketone entry into cells	

BOX 42-4 Factors Affecting Insulin Secretion

Stimulators	Inhibitors
Glucose	Somatostatin
Mannose	2-Deoxyglucose
Amino acids (leucine, arginine, others)	Mannoheptulose
Intestinal hormones (gastric inhibitory peptide, gastrin, secretin, cholecystokinin, glucagon, others)	α-Adrenergic-stimulating agents (norepinephrine, epinephrine)
β-Keto acids	β-Adrenergic-blocking agents (propranolol)
Acetylcholine	Diazoxide
Glucagon	Thiazide diuretics
Cyclic adenosine monophosphate (AMP) and various cyclic AMP–generating substances	Phenytoin
β-Adrenergic-stimulating agents	Alloxan
Theophylline	Microtubule inhibitors
Sulfonylureas	Insulin

of glucose act in a negative feedback loop to increase the secretion of insulin. Lower levels of glucose decrease insulin output. Glucagon, β-adrenergic agonists, and theophylline increase insulin secretion. β Cells are also stimulated to secrete insulin by tolbutamide and other sulfonylurea derivatives; acetylcholine; impulses from vagal nerve branches to the islets; selected amino acids, such as arginine; and β-ketoacids. The mechanisms of action of these stimuli are as yet unclear. Insulin production is inhibited by α-adrenergic agonists, β-adrenergic blocking agents, diazoxide (Proglycem), thiazide diuretics, phenytoin (Dilantin), alloxan, agents that prevent glucose metabolism (eg, 2-deoxyglucose and mannoheptulose), somatostatin, and insulin itself.

Chronic stimulation of β cells, such as by a high-carbohydrate diet for several weeks, can cause a limited amount of hypertrophy and subsequent increase in the insulin-producing capacity. However, overstimulation produces β-cell exhaustion. Stimulation of these exhausted cells produces β-cell death and depletes the β-cell reserve. β-Cell activity is also decreased by the administration of exogenous insulin. Such decreased activity enables the cells to rest and results in temporary hyperproduction after the withdrawal of exogenous insulin.

Insulin Resistance

Insulin resistance, characteristic of type 2 diabetes, is the main defect seen with the development of hyperglycemia, hyperinsulinemia, and consequent β-cell exhaustion. Insulin resistance is a physiological condition in which a person needs more insulin to lower serum glucose effectively than would normally be required. To compensate for insulin resistance, the pancreas secretes more insulin in an attempt to maintain normal glucose levels. The degree of obesity directly affects the resistance to insulin in most patients with type 2 diabetes. One principal mechanism may be a defect in insulin receptor function because of a genetic mutation of the insulin receptor gene. The quantity and activity of insulin receptors also can be regulated by various factors. Increased amounts of insulin, obesity, acromegaly, excess glucocorticoids, and human immunodeficiency virus therapies can exacerbate insulin resistance by decreasing the receptors' number or activity, or both. Exercise and decreased circulating levels of insulin increase the activity of insulin receptors[2]; therefore, leading a sedentary lifestyle may contribute to insulin resistance.

Insulin resistance plays a role in other metabolic abnormalities including obesity, high levels of triglycerides, low levels of (HDL) lipoproteins, hypertension, and systemic inflammation, macrovascular disease, and abnormal fibrinolysis. These signs and symptoms are called metabolic syndrome, and obesity is the major factor that leads to the development of type 2 diabetes.

Additional conditions play a role in the pathogenesis of type 2 diabetes mellitus: β-cell dysfunction and excess hepatic glucose production that contributes to hyperglycemia. β-Cell dysfunction results when the pancreas is unable to meet the high demands for insulin because of insulin resistance. When type 2 diabetes mellitus is diagnosed, approximately 50% of β-cell function is already lost.

Several theories explain the development of β-cell dysfunction:

- Cell exhaustion results when the pancreas must keep up with the higher demands for insulin. Some functional and morphological changes occur to compensate for the increased demand, but eventually β-cell exhaustion occurs.
- Chronic hyperglycemia leads to the development of glucotoxicity—direct toxicity to the β cells.
- Chronic exposure of β cells to excess free fatty acids damages them, leading to lipotoxicity.
- Apoptosis, or programmed cell death, occurs secondary to chronic glucotoxicity and lipotoxicity. This leads to progressive β–islet cell loss.
- Abnormal deposition of amyloid matter leads to islet cell destruction.[3]

Insulin resistance interferes with normal cellular interactions between insulin, skeletal muscles, and adipose tissues. Insulin binds with cell surface receptors, causing a cascade of intracellular signals; this results in the translocation of glucose transporter cells to cell surfaces and allows entry of glucose into the cell. In addition, insulin may bind normally to receptors but has to work with disrupted signals, resulting in insufficient translocation of glucose transporter molecules. This ultimately leads to excess glucose accumulation.[2]

Insulin resistance triggers β cells to produce more insulin as a compensatory mechanism. Hyperinsulinemia initially meets the additional needs created by excess glucose. However, when β-cell function fails to satisfy these demands, hyperglycemia and type 2 diabetes mellitus ensue. This is termed the dual phenomenon or dual abnormality: insulin resistance and β-cell dysfunction.[3] Data from a landmark trial, the United Kingdom Prospective Diabetes Study, suggest that the process of declining β-cell function occurs for approximately 10 years before the diagnosis of type 2 diabetes is made.[4]

Glucagon

Glucagon, a polypeptide hormone, is manufactured and secreted by the α cells of the islets of Langerhans and is stimulated by pure protein meal ingestion that produces

an aminoacidemia. The half-life of plasma glucagon is 5 to 10 minutes. Glucagon influences enzyme systems in liver, fat, and muscle cells and is degraded mainly by the liver.

The major function of glucagon, which stimulates the synthesis of the gluconeogenic enzyme fructose-1,6-biphosphate, is to elevate blood glucose levels and then to enable this plasma glucose to enter and be used by the cells of the body (eg, the muscle cells) by stimulating the secretion of insulin. In this manner, glucagon prevents hypoglycemia between meals, during exercise, during the first few days of fasting, and after a high-protein meal. Dietary protein stimulates an increase in plasma insulin, which causes a rapid cellular uptake of absorbed dietary carbohydrates.

To elevate blood glucose levels, glucagon stimulates liver cells to perform glycogenolysis and gluconeogenesis. This increases the glucose concentration in liver cells, and because these cells can dephosphorylate intracellular glucose, this glucose can be released from the liver into the bloodstream. The fatty acids and amino acids needed for gluconeogenesis are supplied by the glucagon-stimulated breakdown of fats in adipose cells and the release of fatty acids into the bloodstream. If the supply of fatty acids is insufficient, glucagon also stimulates the breakdown of proteins into amino acids in muscle cells and the release of amino acids into the plasma. These fatty acids and amino acids are then taken up by hepatocytes and used as raw materials in gluconeogenesis. Glucagon also elevates plasma ketone levels by increasing hepatic ketone production, and promotes the secretion of somatostatin and GH.

Although glucagon opposes the effects of insulin on blood glucose levels, it also stimulates the secretion of insulin. This apparent contradiction is actually a logical second step in the biological function of this hormone. It enables the increased plasma glucose to enter and be used by various tissues. An elevated plasma glucose level stimulates insulin secretion, but this takes a while. The direct action of glucagon on β cells simply is faster.

As is the case with β cells, α cells are stimulated by β-adrenergic agonists, theophylline, elevated plasma levels of dietary amino acids (primarily those used in gluconeogenesis), and vagal (cholinergic) stimulation. Glucagon secretion is also prompted by glucocorticoids (eg, cortisol), catecholamines, GH, cholecystokinin (CCK), and gastrin. Exercise, physical stress, and infections also increase α-cell activity. Whereas the effects of exercise on glucagon secretion appear to be mediated by increased β-adrenergic activity, stress and infection probably operate by increasing plasma glucocorticoid levels. Dietary amino acids are believed to enhance glucagon secretion by their effects on CCK or gastrin, or both, because intravenous amino acids exert little or no effect on α cells.

Elevated plasma glucose levels enact a negative feedback loop to retard or halt the output of glucagon; however, plasma insulin must be present for this mechanism to operate. Like β-cell secretion, α-cell secretion is inhibited by adrenergic agonists, phenytoin, and somatostatin. Fatty acids and ketone bodies in the plasma can inhibit glucagon secretion, but this inhibition must be weak because plasma glucagon levels can be quite elevated during diabetic ketoacidosis.

In addition to glucagon, other hormones—cortisol, epinephrine, and GH—have great influence on the regulation of glucose and insulin. These counter-regulatory hormones have a synergistic effect on glucose production as a mechanism to protect the body during stress. They act to inhibit insulin while increasing glucagon, producing an insulin-resistant state, and increasing overall serum glucose levels to produce sufficient energy levels during "fight-or-flight" responses. These hormones elevate serum glucose levels to protect against hypoglycemia and to prepare the body for stress. However, they can also further aggravate a state of hyperglycemia and can lead to dangerous levels of glucose, as seen in diabetic emergencies.[5]

Another group of hormones, incretins, are released by the gut in response to nutrient ingestion. One hormone, glucagon-like peptide-1, is a potent insulin secretagogue released from the L cells in the distal small bowel that will assist with assimilating nutrients. It has a short half-life, inhibits glucagon secretion, delays gastric emptying, and reduces appetite and food intake. When used pharmacologically, incretins can lower blood glucose substantially.[6,7]

Somatostatin

This tetradecapeptide is produced not only by the δ cells of the pancreas but also by the hypothalamus, where it functions as an inhibitor of anterior pituitary GH secretion; neurons of the CNS, where it probably functions as a synaptic neurotransmitter agent; and δ cells in the gastric mucosa, where it inhibits the secretion of gastrin and other lesser known gastrointestinal hormones. Islet cell somatostatin is secreted into the bloodstream and therefore functions as a hormone. Little is known of the metabolism of somatostatin because it is so tightly bound with the actions of GH.

Somatostatin inhibits the release of insulin and glucagon from the pancreas. Pancreatic somatostatin inhibits the activity of all other islet cells. The biological significance of this action is not yet known. The only clinical data of relevance concern δ-cell tumors. These produce a clinical picture that resembles diabetes mellitus but that is reversible with tumor ablation. The secretion of somatostatin from islet cells is increased by glucose, certain amino acids, and CCK. Factors that inhibit islet somatostatin secretion are unknown.

Pancreatic Polypeptide

Not much is known about this islet hormone in humans. It is produced by the endocrine cells found in small clusters of cells located between the cells of the islets of Langerhans and the acinar cells of the pancreas. Its secretion in humans is enhanced by dietary protein, exercise, acute hypoglycemia, and fasting. Somatostatin and elevated plasma glucose levels decrease the secretion of this polypeptide. No definite actions of this hormone have been established for humans, but it appears to have a role in smooth muscle relaxation of the gallbladder.

▲ The Adrenal Glands

The adrenal glands lie at the superior pole of each kidney retroperitoneally. Each gland is composed of an inner core, the medulla, surrounded by an outer layer, the cortex (Fig. 42-6). Although they are structurally related, the medulla and cortex are derived from different embryological tissues and function as separate entities. The hormones produced by the adrenal glands are summarized in Table 42-4.

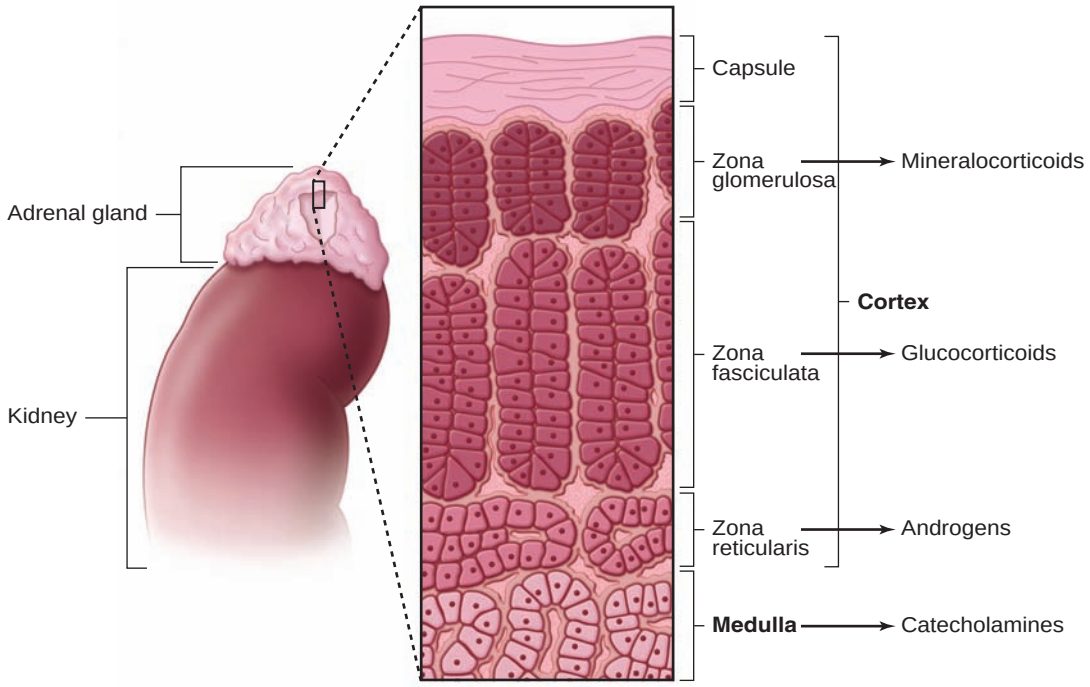


FIGURE 42-6 ▲ The adrenal gland has a cortex and a medulla. (Adapted from Seifter J, Ratner A, Sloane D: Concepts in Medical Physiology. Philadelphia, PA: Lippincott Williams & Wilkins, 2005, p 541.)

Medullary Hormones

The adrenal medulla is basically a modified sympathetic ganglion. The axons of preganglionic sympathetic neurons arrive from the thoracic cord by way of splanchnic nerves. They synapse in the adrenal medulla with modified postganglionic cells that have lost their axons and secrete chemicals directly into the bloodstream. Therefore, the adrenal medulla may appropriately be viewed as an endocrine extension of the autonomic nervous system.

Four chemicals are produced and secreted in the adrenal medulla by two morphologically different cell types:

- Dopamine, a precursor of norepinephrine
- Norepinephrine, the typical product of postganglionic sympathetic neurons
- Epinephrine, a methylated version of norepinephrine
- Opioid peptides (enkephalins).

Table 42-4 Hormones of the Adrenal Gland and Their Actions

Gland	Hormone	Action
Adrenal gland cortex	Mineralocorticoids	Reabsorption of sodium Elimination of potassium
	Glucocorticoids	Responds to stress Decreases inflammation Alters metabolism of protein and fat
Medulla	Epinephrine	Stimulates sympathetic system
	Norepinephrine	Increases peripheral resistance

Not much is known about the opioid peptides. The specific stimulus for their secretion has yet to be identified, and their physiological actions are unknown, as are their metabolism and fate. Dopamine, norepinephrine, and epinephrine are collectively termed catecholamines. They are stored in granules in the medullary cells. The secretion of these chemicals is triggered by stimulation of the preganglionic neurons that innervate the medulla. This causes the neurons to release acetylcholine, which in turn prompts the medullary cells to secrete. The half-life of plasma catecholamines is approximately 2 minutes. These compounds are rapidly degraded by plasma renal and hepatic catechol-*O*-methyltransferase enzymes into vanillylmandelic acid, metanephrine, and normetanephrine, which are excreted in the urine. Measuring urine levels of these compounds is significant if an adrenal tumor, pheochromocytoma, is suspected. In this case, the levels will be high, indicating that a catecholamine secreting tumor is likely.

Predictably, the epinephrine and norepinephrine secreted by the adrenal medulla mimic the effects of a mass discharge from sympathetic neurons. However, apart from this, they produce several metabolic actions. First, they elevate blood glucose levels by activating an enzyme, phosphorylase, which promotes hepatic glycogenolysis. Because liver cells possess the enzyme glucose-6-phosphatase, the glucose produced by this glycogen breakdown is able to diffuse out of hepatocytes and into the bloodstream. These hormones also induce muscle cells to participate in elevating blood glucose levels, although this process is less direct. These hormones can also elevate plasma glucose levels by stimulating the secretion of glucagon and can increase the uptake of glucose into body tissues by stimulating the secretion of insulin. Epinephrine and norepinephrine can also produce the opposite effects by stimulating α-adrenergic receptors on islet cells. Because of differential effects of both hormones on α- and β-adrenergic

receptors, the result is that epinephrine elevates plasma glucose levels much more than does norepinephrine.

A second metabolic effect of catecholamines is promotion of lipolysis in adipose tissue. This elevates plasma-free fatty acid levels and provides an alternative energy source for many body cells. Circulating catecholamines also increase alertness by stimulating the reticular activating system. Last, these hormones produce an increase in the metabolic rate of the body and a cutaneous vasoconstriction, both of which result in an elevation in body temperature. However, the accelerated metabolism requires the presence of the thyroid and adrenal cortex hormones.

Although the physiological action of adrenal medullary dopamine is unknown, exogenous dopamine is useful in combating certain shocks because it has a positive inotropic effect on the heart (by way of β receptors) and produces renal vasodilation and peripheral vasoconstriction. The overall effect of moderate dosages is elevation of systolic blood pressure (without an appreciable increase in diastolic blood pressure) together with retention or restoration of renal output.

Stimulation of the adrenal medulla glands is part of a general sympathetic–adrenal medulla (SAM) response to exercise and to perceived threats to biopsychological integrity and survival. (Cannon called the latter the "fight-or-flight" response.) Hypoglycemia also stimulates increased adrenal medullary secretion. The results of the SAM response enable the body to perform vigorous physical exertion optimally. The heart rate and blood pressure are increased (increasing perfusion), and blood flow is shunted away from the skin and gastrointestinal tract to more vital organs for exertion, such as skeletal muscles, brain, and heart. The reticular activating system is stimulated, fostering alertness. Blood glucose and fatty acid levels are raised, thereby increasing the available energy sources for cells. Pupils are dilated, increasing the field of peripheral vision and the amount of light entering the eyes. Sweat glands are stimulated, cooling the body in advance of and during the time that the body temperature is elevated as the result of the physical exertion. Most of this SAM response is mediated by sympathetic nerve fibers to various body structures; circulating catecholamines play only a minor role. Furthermore, many tissue responses (eg, those of muscle cells) to such sympathetic demands require glucocorticoids to enable the tissues to meet the demands of the SAM response, and the SAM response often accompanies the stress-induced secretion of adrenal steroids discovered by Selye. (This and the endocrine response to physical and psychological stress are discussed in the section on cortical hormones.)

Cortical Hormones

The adrenal cortex is composed of three histologically different layers (see Fig. 42-6).

Its exterior is covered by a capsule. The outermost layer, the zona glomerulosa, lies just beneath the capsule. It produces and secretes primarily mineralocorticoids, such as aldosterone. The inner two layers, the zona fasciculata and zona reticularis, manufacture and secrete glucocorticoids (cortisol and corticosterone) and adrenal androgens and estrogens. If these inner cortical layers are destroyed, they can be regenerated from zona glomerulosa cells.

Figure 42-7 depicts the metabolic pathways for synthesis of all adrenocortical hormones. Each of these metabolic steps is governed by a specific enzyme. Genetic deficiencies in one or more of these enzymes produce syndromes involving the underproduction or overproduction of various cortical hormones. Drugs that inhibit specific enzymes are used clinically

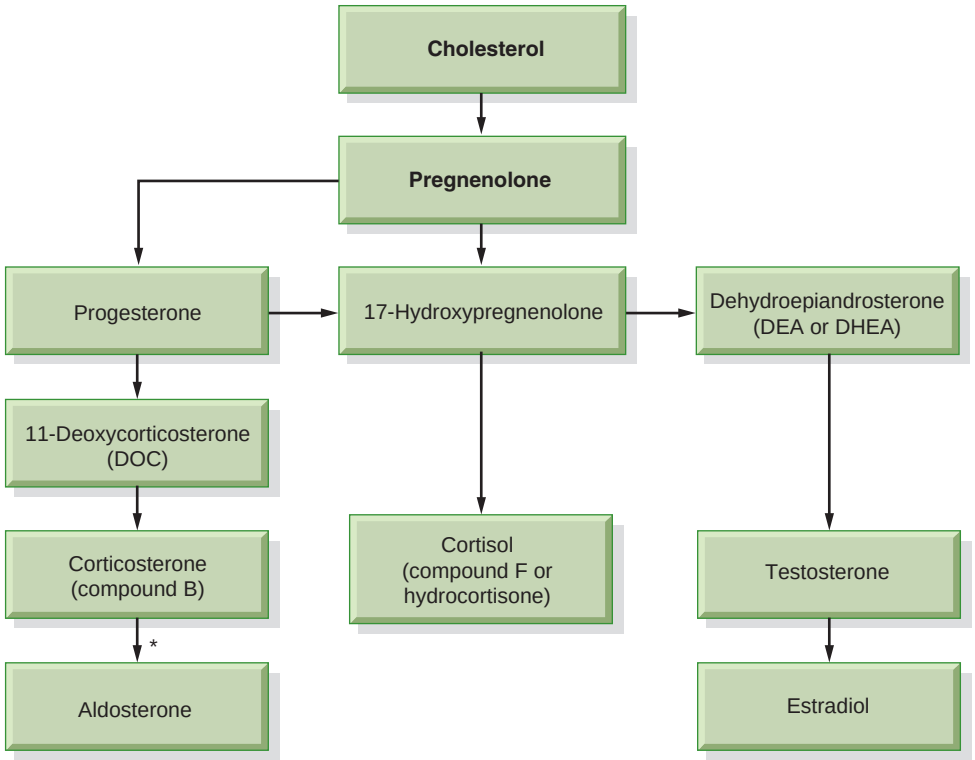


FIGURE 42-7 ▲ Biosynthetic pathways for adrenal cortical hormones. Only cells of the zona glomerulosa can convert corticosterone to aldosterone (*asterisk*). All the other pathways can be carried out by cells in all three layers of the adrenal cortex.

to assess cortical function. One such drug is metyrapone, which inhibits cortisol synthesis.

After secretion, plasma cortisol and, to a lesser extent, corticosterones are bound to a plasma globulin called corticosteroid-binding globulin (CBG), or transcortin. Only the unbound hormones are physiologically active. The bound glucocorticoids serve as a hormone reservoir that is used to replace degraded unbound hormones. The half-lives of plasma corticosterone and cortisol are approximately 50 and 80 minutes, respectively. CBG is manufactured by liver cells. Therefore, decreased hepatic function (eg, cirrhosis) can lead to subnormal quantities of plasma CBG, resulting in excess quantities of circulating unbound, active glucocorticoids, leading to hyperdynamic circulation. Only a small amount of aldosterone is bound to plasma proteins. Its half-life is approximately 20 minutes.

Adrenal steroids are degraded by the liver. Depressed hepatic function can retard the degradation of adrenal steroids, thereby producing a clinical picture of hormone excess. The soluble degraded steroid metabolites are excreted by the kidneys.

Glucocorticoids

As the name glucocorticoid suggests, cortisol and corticosterone influence glucose metabolism. They elevate plasma glucose levels by promoting hepatic gluconeogenesis and glycogenolysis. To facilitate gluconeogenesis, these hormones cause the breakdown of fat and proteins and the release of fatty and amino acids into the bloodstream, which carries them to the liver. Excessive gluconeogenesis can lead to severe hyperglycemia often seen in diabetic patients receiving glucocorticoids.

Glucocorticoids enable tissues to respond to glucagon and catecholamines; they also prevent rapid fatigue of skeletal muscle. Cortisol and corticosterone also act on the kidneys to permit the excretion of a normal water load in one of three ways: glucocorticoids make distal or collecting tubules more permeable to the reabsorption of water independently of sodium reabsorption, they increase the glomerular filtration rate (GFR), or they reduce the output of ADH.

The effects of glucocorticoids on plasma components are mixed. They decrease the number of plasma eosinophils and basophils but increase the number of circulating neutrophils, platelets, and erythrocytes. By suppressing production and increasing destruction, glucocorticoids decrease the number of lymphocytes. They also decrease the size of lymph nodes. A major function of lymphocytes is to provide either humoral immunity (with antibodies) or cell-mediated immunity. Stress-induced elevations in glucocorticoid secretion and the resulting decrease in lymphocytes may explain the decrease in immunocompetence that often occurs in people who are under psychological or physical stress.

Other effects of physiological levels of glucocorticoids include decreasing olfactory and gustatory sensitivity. People with adrenal insufficiency can detect various chemicals (eg, sugar, salt, urea, and potassium chloride) by either taste or smell with a sensitivity that is 40 to 120 times greater than normal.

The effects of pharmacological dosages of glucocorticoids are considered separately from those of normal physiological levels. In pharmacological dosages, glucocorticoids possess immunosuppressive, anti-inflammatory, and antihistaminic activity. Glucocorticoids suppress the immune system by inhibiting the production of interleukin-2 by T4 (helper) lymphocytes. Decreases in interleukin-2 reduce the proliferation of T8 (suppressor, cytotoxic) T cells and B lymphocytes. Glucocorticoids act in several ways to suppress the inflammatory response, including the influx of phagocytes and the activation of complement and kinins.

Conversely, glucocorticoids can be of great benefit in the treatment of certain noninfective inflammatory conditions (eg, rheumatoid arthritis and systemic lupus erythematosus). Glucocorticoids can also be beneficial in treating certain allergies (eg, asthma, hives, and minimal-change glomerular disease) because they prevent the release of histamines from mast cells. Their use as immunosuppressives enables patients to receive organ transplants. In any case, the potentially deleterious side effects of glucocorticoids usually require that they be used only after other treatments (eg, nonsteroidal anti-inflammatory drugs [NSAIDs] or antihistamines) have failed or if the benefits clearly outweigh the risks (eg, in renal disease or with organ transplants). In addition to immunosuppression, glucocorticoids trigger the development of all or part of Cushing's syndrome (eg, diabetes, hypertension, protein wasting, and osteoporosis) and inhibit growth in infants and children. The pharmacological and physiological actions of glucocorticoids are summarized in Table 42-5.

Regulation of glucocorticoid secretion is outlined in Figure 42-8. The secretion of glucocorticoids is triggered by the release of corticotropin-releasing hormone (CRH), a neurosecretory material released by the hypothalamus. CRH stimulates the cells of the anterior pituitary to secrete ACTH. Without the stimulus of ACTH, the cells of the zona fasciculata and zona reticularis do not secrete glucocorticoids. Elevated plasma glucocorticoid levels function in a negative feedback loop to decrease or halt the secretion of CRH and thereby indirectly inhibit the secretion of ACTH as well.

There is a diurnal rhythm to the secretion of CRH that causes a similar rhythm in the output of ACTH and glucocorticoids. The result is that maximal glucocorticoid secretion occurs between 6:00 AM and 8:00 AM in people sleeping from midnight to 8:00 AM in a 24-hour day. Tumors that secrete CRH, ACTH, or glucocorticoids do not demonstrate such a rhythm, a fact that is useful in their diagnosis. The biological clock that regulates this and other diurnal, or circadian, rhythms is located in the hypothalamus, just above the area where the optic nerves cross (optic chiasma).

The beneficial functions of normal levels of glucocorticoids in enabling tissues to respond to glucagon and catecholamines are more than adequate to meet the needs of the SAM mechanism for a short time. If these needs continue, additional stress-induced glucocorticoid secretion is required. Eventually, if the stress continues unameliorated, exhaustion of the adrenal cortex occurs, glucocorticoid levels drop, tissues are no longer able to meet the demands of the SAM mechanism, muscle fatigue occurs, readily available cell energy sources (eg, plasma glucose and fatty acid) are depleted, and vascular collapse and death result.

Mineralocorticoids

Aldosterone and glucocorticoids that have some mineralocorticoid function (eg, 11-deoxycorticosterone) increase

Table 42-5 Actions of Cortisol

Major Influence	Effect on Body
Glucose metabolism	Stimulates gluconeogenesis Decreases glucose use by the tissues
Protein metabolism	Increases breakdown of proteins Increases plasma protein levels
Fat metabolism	Increases mobilization of fatty acids Increases use of fatty acids
Anti-inflammatory action (pharmacological levels)	Stabilizes lysosomal membranes of the inflammatory cells, preventing the release of inflammatory mediators Decreases capillary permeability to prevent inflammatory edema Depresses phagocytosis by white blood cells to reduce the release of inflammatory mediators Suppresses the immune response Causes atrophy of lymphoid tissue Decreases eosinophils Decreases antibody formation Decreases the development of cell-mediated immunity Reduces fever Inhibits fibroblast activity
Psychic effect	May contribute to emotional instability
Permissive effect	Facilitates the response of the tissues to humoral and neural influences, such as that of the catecholamines, during trauma and extreme stress

From Porth CM, Matfin G: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 1040.

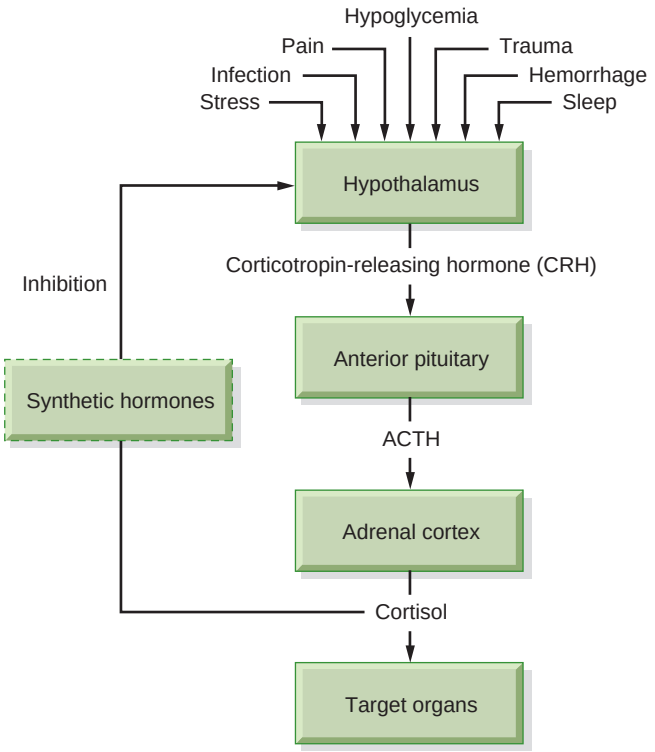


FIGURE 42-8 ▲ The hypothalamic–pituitary–adrenal (HPA) feedback system that regulates glucocorticoid (cortisol) levels. Cortisol release is regulated by adrenocorticotropic hormone (ACTH). Stress exerts its effects on cortisol release through the HPA system and corticotropin-releasing hormone (CRH), which controls the release of ACTH from the anterior pituitary gland. Increased cortisol levels incite negative feedback inhibition of ACTH release. Pharmacological doses of synthetic steroids inhibit ACTH release by way of the hypothalamic CRH.

sodium reabsorption by the cells of the collecting ducts and distal tubules of the nephrons. Because of the cation exchange system in the distal tubule cells, such sodium reabsorption can increase potassium secretion and thereby foster potential hypokalemia. The reabsorption of sodium osmotically causes water reabsorption. This expands the volume of ECF. The increase in blood volume causes an elevation in blood pressure. However, edema does not usually result. Above a certain level of aldosterone-induced sodium reabsorption, the expansion of the ECF compartment can trigger secretion of natriuretic hormone or decreased sodium reabsorption in the proximal tubule. Either of these effects opposes the action of aldosterone and sodium excretion.

The primary mechanism for regulating aldosterone secretion is the renin–angiotensin system (Fig. 42-9). Pituitary ACTH does not stimulate zona glomerulosa cells under normal conditions. Cells of the juxtaglomerular apparatus are wedged between the renal afferent arteriole as it enters the glomerulus and the distal tubule as it passes by this area. The juxtaglomerular apparatus contains baroreceptor cells that monitor the afferent arteriole blood pressure and other cells that monitor the sodium and chloride concentration in the urine in the distal tubule (the lower the concentration, the slower the formation of filtrate, if all other factors are equal). A decrease either in blood pressure or in the concentration of electrolytes stimulates the juxtaglomerular apparatus to secrete the glycoprotein hormone, renin. The major classes of stimuli that trigger renin secretion are decreased renal perfusion (eg, cardiac failure, dehydration, and hemorrhage) and low ECF salt concentrations (eg, from excessive use of diuretics).

Renin converts a circulating plasma globulin into angiotensin I. As the blood passes through the lungs (and to a lesser extent in other parts of the circulatory system), angiotensin I is converted to angiotensin II. This physiologically active chemical acts on the zona glomerulosa to promote aldosterone secretion, which leads to retention of salt and water, and contraction of vascular smooth muscle, thereby stimulating profound vasoconstriction. The result of both actions of angiotensin II is elevation of systemic blood pressure, which, among other things, improves renal perfusion.

The juxtaglomerular apparatus contains β_1 receptors and can be stimulated by sympathetic fibers. Prostaglandins also stimulate the juxtaglomerular apparatus. Sympathetic stimulation through β_1 receptors, renal artery hypotension, and decreased sodium delivery to distal tubules stimulate the secretion of renin. Therefore, the secretion of renin can be pharmacologically decreased by β-blockers (eg, propranolol or atenolol). Prostaglandin inhibitors (aspirin and NSAIDs)

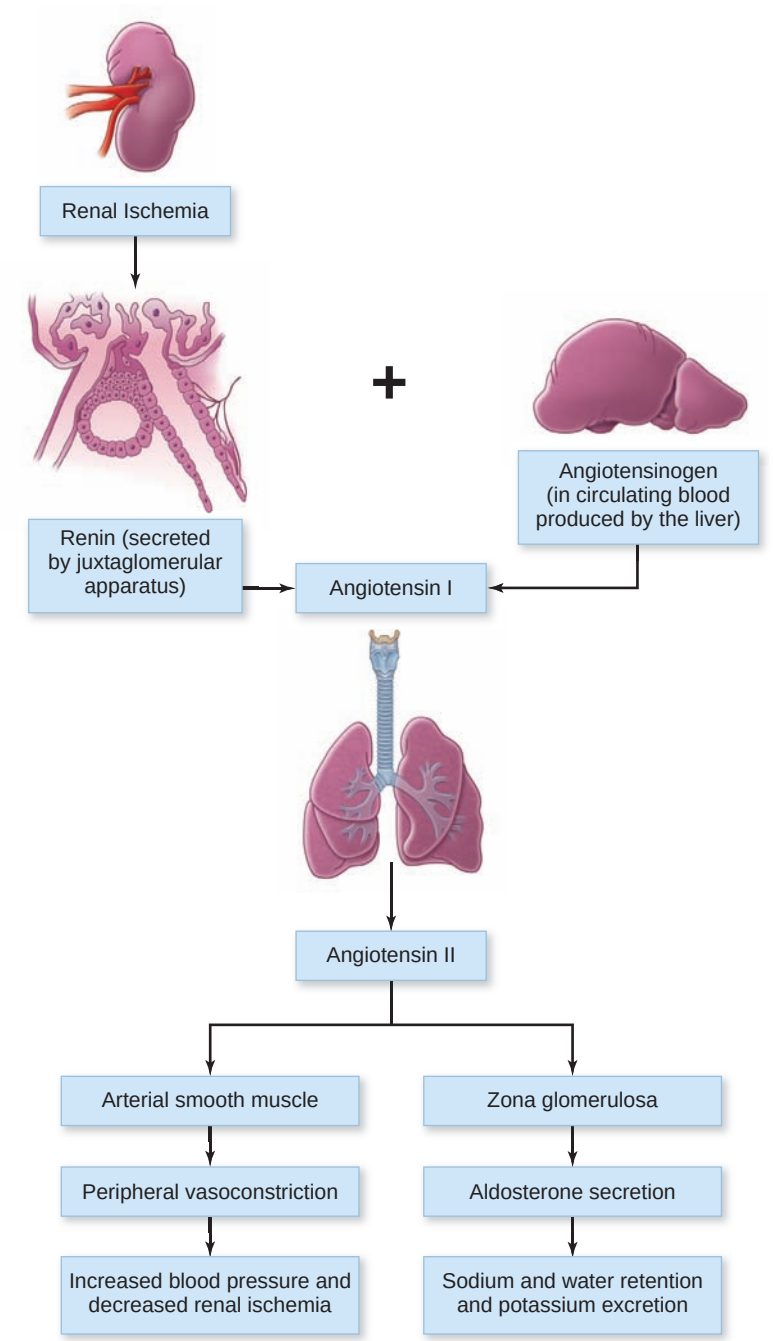


FIGURE 42-9 ▲ The renin–angiotensin system induces aldosterone secretion and vasoconstriction, which in turn elevates the systemic blood pressure.

can exert a similar action. Angiotensin-converting enzyme (ACE) inhibitors (eg, lisinopril) prevent the conversion of angiotensin I to angiotensin II. These effects have made ACE inhibitors and β-blockers useful as antihypertensive agents.

Aldosterone secretion is also stimulated by an increase in plasma potassium levels, but not by increased sodium levels. Another regulating factor for aldosterone secretion is posture. An upright body position increases aldosterone levels by increasing production and decreasing degradation. How this works is unclear, but because of this, aldosterone levels of bedridden patients are slightly subnormal. There also is a poorly understood diurnal rhythm of aldosterone secretion, with highest levels occurring in the early morning hours just before the person awakens.

▲ Atrial Natriuretic Peptide (Natriuretic Hormone)

Atrial natriuretic peptide (ANP) is manufactured by cells in the walls of the atria of the heart. The main stimulus for ANP secretion is atrial stretch. ANP increases renal excretion of salt and water. Some evidence suggests that ANP acts by increasing glomerular filtration. Other evidence indicates that ANP inhibits the membrane active transport mechanism responsible for the reabsorption of sodium by renal tubule cells. Decreased sodium reabsorption decreases the movement of water from the urine in the nephron back into the blood of the peritubular capillaries, thereby increasing

the elimination of water and salt from the body. ANP also inhibits the secretion of renin by the juxtaglomerular apparatus, thereby lowering plasma angiotensin levels. In addition, ANP inhibits the membrane active transport mechanism responsible for pumping sodium out of vascular smooth muscle cells. The consequent rise in intracellular sodium inhibits the entry of calcium ions, thereby lowering the intracellular concentration of calcium ions. The decrease in the intracellular free calcium promotes vasodilation and a lowering of the systemic blood pressure.

ANP is secreted in response to an increase in ECF volume caused by the ingestion of salt and water. The exact stimulus appears to be a stretch of the muscle fibers in the atrial walls, which results from the increased venous return that is caused by the rise in ECF volume. As the natriuresis causes the ECF volume to fall back to normal, the secretion of ANP stops. The capability of ANP to increase the GFR, together with its direct effects on the collecting tubules, results in a profound natriuresis and diuresis.

The metabolic fate of ANP is unknown, but circulating levels of this hormone are elevated in patients with congestive heart failure, cirrhosis, or renal insufficiency and are low in those with nephrotic syndrome or volume depletion. These results suggest liver and kidney regulation.

▲ Clinical Applicability Challenges

SHORT ANSWER QUESTIONS

1. Discuss the effects of hypothalamic and pituitary dysfunction on antidiuretic, thyroid, and adrenal hormone secretion.
2. Describe the negative feedback mechanisms that affect the synthesis and production of thyroxine (T_4) by the thyroid gland.
3. Review the mechanisms by which insulin, glucagon, counter-regulatory hormones, and gut derived hormones regulate blood glucose levels.

References

1. Wahren J, Kallas A, Sima AA: The clinical potential of C-peptide replacement in type 1 diabetes. Diabetes 61(4):761–772, 2012
2. Mathur R: Insulin resistance. eMedicinehealth.com. Available at: http://www.emedicinehealth.com/insulin_resistance/article_em.htm
3. Waller AP, Kohler K, Burns TA, et al: Naturally occurring compensated insulin resistance selectively alters glucose transporters in visceral and subcutaneous adipose tissues without change in AS160 activation. Science Direct 1812(9):1098–1103, 2011.
4. United Kingdom Prospective Diabetes Study (UKPDS) VIII: Study design, progress, and performance. Diabetologia 34:877–890, 1991
5. Kitabchi AE, Impierrez GE, Miles JM, et al: Hyperglycemic crises in adult patients with diabetes. Diabetes Care 32(7):1335–1343, 2009
6. Marini M, Succurro E, Frontoni S, et al: Insulin sensitivity, beta cell function, and incretin effect in individuals with elevated 1-h postload plasma glucose levels. Diabetes Care published ahead of print, February 22, 2012, doi: 10.2337/dc11-2181
7. Stonehouse AH, Darsow T, Maggs DG: Incretin-based therapies. Journal of Diabetes 4(1):55–67, 2012

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

43 Patient Assessment: Endocrine System

Jane Kapustin

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Analyze the relationship between dysfunction of the hypothalamus and the pituitary gland and the signs and symptoms of the resultant disorder.
2. Compare and contrast the signs and symptoms of hypothyroidism and hyperthyroidism.
3. Formulate a plan for collecting history and physical examination data focusing on endocrine function.
4. Differentiate between normal and abnormal findings for an adrenal gland disturbance.
5. Explain laboratory tests used to diagnose acute endocrine disorders.

Endocrine disorders can affect all body systems and are usually caused by the overproduction or underproduction of hormones. This chapter presents an overview of the history, physical examination, and diagnostic studies that help diagnose the following specific endocrine disorders: thyroid crisis; myxedema coma; adrenal crisis; syndrome of inappropriate antidiuretic hormone (SIADH); diabetes insipidus; diabetic ketoacidosis (DKA); hyperglycemic hyperosmolar state (HHS); and hypoglycemia. It builds on the content presented in Chapter 42, which explored the far-reaching effects of the endocrine system on body functions. This chapter also provides a foundation for understanding specific applications described in Chapter 44.

Because the endocrine system affects so many general areas of the body, assessment must include a variety of signs and symptoms. General manifestations of disorders are evident through vital signs, energy level, fluid and electrolyte imbalances, and ability to carry out activities of daily living. Other parameters to be observed include heat or cold intolerance, changes in weight, fat redistribution, changes in sexual functioning, and altered sleep patterns. Box 43-1 summarizes the approach used to assess a patient suspected of having an acute endocrine disorder.

Because the endocrine system exerts control over the entire body, many laboratory tests that have been discussed in other chapters are applicable to the assessment of an acute endocrine disorder. For example, fluid and electrolyte problems accompany many acute endocrine disorders. Therefore, serum sodium, potassium, magnesium, and osmolality are assessed. Blood urea nitrogen and creatinine levels may also help assess renal involvement (see Chapter 29). Arterial blood gases, bicarbonate levels, and anion gap calculation may be necessary to diagnose acidosis. Laboratory studies specific to endocrine gland dysfunction are described in the following sections and summarized in Table 43-1.

Similarly, in the evaluation of endocrine disorders, it is often necessary to evaluate body systems other than the endocrine system using diagnostic studies. For example, electrocardiography and cardiac monitoring may be needed to diagnose cardiac problems, whereas a chest radiograph may be necessary to detect pulmonary problems, such as the pleural effusion that can occur in myxedema coma. Computed tomography (CT), magnetic resonance imaging (MRI), and ultrasound may be used to localize tumors.

▲ The Hypothalamus and Pituitary Gland

Some of the hormones of the hypothalamus and the pituitary have a profound impact on the critically ill patient and are described in detail in this section. They include antidiuretic hormone (ADH), adrenocorticotropic hormone (ACTH), and thyroid-stimulating hormone (TSH). Those hormones that are mainly responsible for normal physiological functioning of the reproductive system are not significant in the care of the critically ill adult and therefore are not covered in this section (oxytocin, follicle-stimulating hormone [FSH], luteinizing hormone [LH], growth hormone [GH], melanophore-stimulating hormone [MSH]).

The pituitary gland hormones are under the control of the hypothalamus. The posterior lobe of the pituitary stores and secretes ADH (vasopressin) in response to serum osmolality. Because the primary function of ADH is to control water excretion by the kidney, attention must be focused on the patient's hydration status (manifests as fluid volume

BOX 43-1 HEALTH HISTORY for Endocrine Assessment

Chief Complaint
Patient's description of the problem

History of the Present Illness
Hypothalamus and pituitary disorders: excessive or inadequate urinary output, excessive thirst, poor skin turgor, cognitive changes, dehydration, or water intoxication
Thyroid disorders: Cold or heat intolerance; edema; cognitive changes, such as slowed mentation, agitation, memory impairment, and stupor; tremulousness; insomnia; fatigue; tachycardia, atrial fibrillation; bradycardia; hypoventilation; constipation; diarrhea; menstrual cycle irregularities; skin problems; husky voice; diplopia, exophthalmos; eye pain, change in vision; depression; hematuria
Parathyroid disorders: apathy, fatigue, weakness, tetany, joint pain
Diabetes mellitus: weight gain or loss, excessive urination, excessive thirst, excessive appetite, blurred vision, dental caries, poor wound healing, chronic vaginitis, neuropathy, nocturia, dehydration, cognitive changes
Adrenal disorders: nausea, vomiting; striae; central obesity with peripheral wasting; moon facies; hirsutism; petechiae, easy bruising; dehydration; fatigue, lethargy

Past Health History
Relevant childhood illnesses and immunizations: history of adenoid or neck/chest radiation, mental retardation, iodine deficiency
Past acute and chronic medical problems: diabetic emergencies, hypertension, high cholesterol, tachydysrhythmias, congestive heart failure, myocardial infarction, Graves' disease, Hashimoto's thyroiditis, head injury, cerebral vascular accident, pancreatitis, unexplained infections
Risk factors: age, heredity, gender, race, tobacco use, alcohol use, elevated cholesterol, obesity, sedentary lifestyle, growth spurt cycles, pregnancy, gestational diabetes, delivery of an infant weighing more than 9 pounds, anemia
Past surgeries: neurosurgical procedures, thyroidectomy, parathyroidectomy, adrenalectomy
Medications: amiodarone, phenytoin, carbamazepine, chlorpropamide, corticosteroids, opioids, lithium, aspirin, iodides, heparin, levothyroxine (Synthroid), neoplastic drugs, estrogen, methadone, androgens, β-blockers, nonsteroidal anti-inflammatory drugs, potassium, diuretics
Allergies and reactions to medication, foods, contrast dye, latex, or other materials
Transfusion history
Family history: thyroid disease, diabetes, lipid disorders, cerebral aneurysms, cancers, autoimmune disorders
Personal and social history: tobacco, alcohol, substance abuse; occupation; living environment; diet, exercise; sleep patterns; cultural beliefs; spiritual/religious beliefs; leisure activities

Review of Other Systems
HEENT: headaches, dizziness, weakness, visual changes
Lymphatics: edema, lymphadenopathy
Genitourinary: sexual dysfunction, infertility, abnormal vaginal bleeding

excess or deficit) and serum and urine osmolality to acquire information about the general functioning of this part of the pituitary.

History and Physical Examination

The nurse obtains important information about the nature of endocrine disorders by conducting a thorough history. Because disorders of the pituitary that could result in critical care admission affect fluid and electrolyte balance, the nurse inquires about general hydration status. Specific parameters are included in the endocrine health history (see Box 43-1).

Physical examination of the patient includes assessment of hydration status. Skin turgor, buccal membrane moisture, vital signs, and weight are assessed. A patient with hypovolemia (as seen in diabetes insipidus) would experience weight loss from excretion of large volumes of dilute urine. Eventually, the patient would experience tachycardia, hypotension, poor skin turgor, dry buccal membranes, and cognitive changes associated with dehydration and hypernatremia. Conversely, a patient with hypervolemia (as seen in SIADH) would display signs of water intoxication, such as edema, scant urinary output, weight gain (1 liter of fluid equals 2.2 pounds of weight), hypertension, moist buccal membranes, good skin turgor, and cognitive changes associated with hyponatremia.

For patients experiencing fluid balance alterations, the nurse needs to maintain strict measuring of intake and output. Urine specific gravity is measured routinely, noting the nature of the urine (color, concentration, and volume). In addition, critically ill patients with fluid imbalance often have advanced monitoring techniques in place, such as central venous pressure or hemodynamic monitoring with a pulmonary artery catheter. Vigilant monitoring of the patient's fluid status needs to be maintained.

Laboratory Studies

Serum Antidiuretic Hormone

The normal serum ADH level is 1 to 13.3 pg/mL. This radioimmunoassay level distinguishes between central diabetes insipidus and SIADH. Elevated serum ADH compared with low serum osmolality and elevated urine osmolality confirms the diagnosis of SIADH. Conversely, reduced levels of ADH with a correspondingly high serum osmolality, hypernatremia, and reduced urine concentration indicate central diabetes insipidus. Table 43-2 compares and contrasts laboratory values for diabetes insipidus and SIADH.

Urine Specific Gravity

Specific gravity reflects the kidneys' ability to dilute and concentrate urine. The range depends on hydration, urine volume, and the amount of solids in the urine. The specific gravity can be measured by using a multiple-test dipstick that has a reagent for specific gravity or by using a refractometer. Low specific gravity (1.001 to 1.010) is seen in diabetes insipidus and is accompanied by copious, dilute urine. Increased specific gravity (1.025 to 1.030) is seen in diabetes mellitus with dehydration; the urine in general is more concentrated with smaller volumes.

Table 43-1 Sampling of Laboratory Studies Used to Assess Acute Endocrine Disorders

Test	Normal Adult Values	Abnormal Values
Total T_4	4–12 mcg/dL	High in hyperthyroidism Low in hypothyroidism
Free T_4	0.8–2.7 ng/mL	High in hyperthyroidism Low in hypothyroidism
Free T_4 index	4.6–12 ng/mL	High in hyperthyroidism Low in hypothyroidism
Free T_3	260–480 pg/dL	Low in hypothyroidism
Thyroid-stimulating hormone	260–480 pg/dL	High in hypothyroidism (primary) Low in hypofunction of anterior pituitary (secondary hypothyroidism)
Cortisol	8 AM 5–23 mcg/dL 4 PM 3–16 mcg/dL	High in Cushing's disease (increased ACTH secretion by pituitary) High in stress, trauma, and surgery Low in hyposecretion of ACTH by pituitary and adrenal insufficiency
Cortisol stimulation	Should increase to 18 mcg/dL	Low or absent in adrenal insufficiency and hypopituitarism
Urine vanillylmandelic acid (VMA) and catecholamines	VMA up to 2–7 mg/24 h Catecholamines: 270 mcg/24 h	High in pheochromocytoma High in hypothyroidism and diabetic acidosis
Urine specific gravity	1.010–1.025 with normal hydration and volume	Low in diabetes insipidus High in diabetes mellitus with dehydration High in SIADH
Urine ketones	Negative	Positive in diabetic ketoacidosis

SIADH, syndrome of inappropriate antidiuretic hormone; T_3, triiodothyronine; T_4, thyroxine.

Serum Osmolality

Serum osmolality ranges from 270 to 300 mOsm/kg and measures the concentration of diluted particles in the bloodstream. Elevated serum osmolality (hemoconcentration) stimulates the release of ADH, which enhances the reabsorption of fluid and sodium at the nephron level. Through this process, extracellular fluid (ECF) volume is restored, and the plasma becomes less concentrated.

Conversely, hemodilution or decreased serum osmolality inhibits ADH, causing excess fluid to be eliminated by the kidneys to maintain homeostasis. Concentration of the plasma is restored.

Urine Osmolality

This test is a more exact measure of urine concentration. It is also a more useful test when performed in conjunction with serum osmolality. It can be used to diagnose kidney function, diabetes insipidus, and psychogenic water drinking. The urine osmolality is increased in Addison's disease, SIADH, dehydration, and renal disease. It is decreased in diabetes insipidus and psychogenic water drinking. The normal range is 300 to 900 mOsm/kg/24 h and 50 to 1,200 mOsm/kg in a random sample.

Water Deprivation Test

Water restriction is a useful test because healthy people respond with a rapid decrease in urine volume when water intake is withheld. However, people with diabetes insipidus have no decrease in urine volume in response to severe water restriction. This signifies that the normal mechanism of ADH release in the face of water restriction and dehydration is dysfunctional. This test is rarely performed in a critical care unit because the patient is too ill and fragile to withstand the rigors of severe dehydration. The preferred test is measurement of serum ADH to diagnose diabetes insipidus.

Antidiuretic Hormone Administration

One final laboratory test used to diagnose diabetes insipidus is ADH administration. Exogenous ADH (vasopressin or Pitressin) given subcutaneously to the person suspected of having diabetes insipidus causes a temporary increase in urine osmolality. For a brief time, the person displays the appropriate response to ADH by conserving water at the kidney level, and urine output slows down in an attempt to restore ECF. This test also helps distinguish between the two types of diabetes insipidus: nephrogenic and central. In nephrogenic diabetes insipidus, the person does not demonstrate a reaction

Table 43-2 Comparison of Laboratory Values in Diabetes Insipidus and Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

Laboratory Test	Diabetes Insipidus	SIADH
Antidiuretic hormone	Decreased	Increased
Serum osmolality	Increased	Decreased
Sodium	Increased	Decreased
Urinary output	Increased	Decreased
Urine specific gravity	Decreased	Increased
Urine osmolality	Decreased	Increased

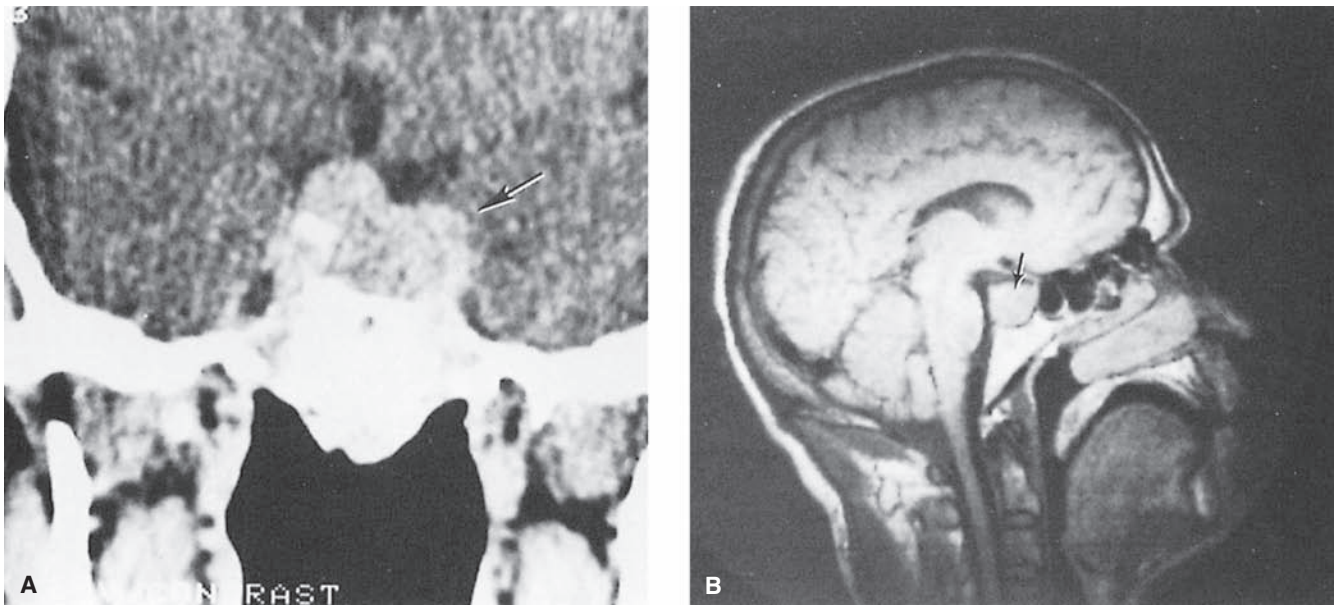


FIGURE 43-1 ▲ **A:** Computed tomography scan showing a suprasellar pituitary tumor (*arrow*) in a thyroid-toxic patient with a thyroid-stimulating hormone (TSH)–secreting pituitary tumor. **B:** T1-weighted magnetic resonance image in the same patient showing a 2- × 2-cm pituitary tumor (*arrow*). T1-weighted images are favorable for demonstrating anatomical detail. (Adapted from Smallridge RC: Thyrotropin-secreting pituitary tumors. Endocrinol Metab Clin North Am 16:3, 1987.)

to exogenous ADH because the kidney receptors in the collecting duct are unresponsive to ADH. People with central diabetes insipidus respond readily to the exogenous ADH.

Diagnostic Studies

Diagnostic imaging studies are frequently used for patients suspected of having pituitary or hypothalamic disorders. CT and MRI are essential in diagnosing primary diseases affecting this area of the brain. Examples of disorders that affect the pituitary–hypothalamic axis are brain tumors, aneurysms, edema from surgical exploration or traumatic injuries, and necrotic lesions. Imaging techniques are used to view the sella turcica and the surrounding structures, including the pituitary within the bony encasement of the middle cranial fossa. Angiography assists with precise viewing of the vascular supply in the area. Figure 43-1 provides examples of MRI and CT scans of a pituitary tumor.

The critically ill patient requires monitoring at all times during these procedures. Quite often, the patient requires sedation to eliminate all patient motion in an effort to ensure clear images. CT is often used with contrast media to highlight specific areas of the brain, and the patient needs to be monitored for adverse allergic reactions if sensitive to iodine, which may be contained in the contrast agent. Institutional policies and procedures need to be followed during diagnostic testing.

▲ The Thyroid Gland

The thyroid hormones are regulated by the hypothalamus and the pituitary gland in a negative feedback system as previously described. Low levels of triiodothyronine (T_3) and thyroxine (T_4) cause the hypothalamus to secrete thyrotropin-releasing hormone (TRH), which then stimulates the anterior pituitary gland to release TSH. TSH stimulates the production and release of the thyroid hormones (Fig. 43-2).

Increased thyroid hormone production results in hyperthyroidism, which can lead to an extreme form of thyrotoxicosis. This is a rare, life-threatening illness necessitating critical

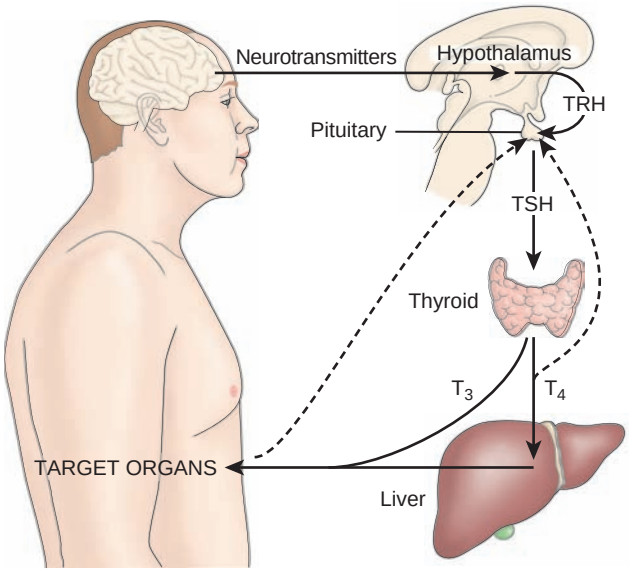


FIGURE 43-2 ▲ The hypothalamic–pituitary–thyroid axis. Thyrotropin-releasing hormone from the hypothalamus stimulates the pituitary gland to secrete TSH. TSH stimulates the thyroid to produce thyroid hormone (T_3 and T_4). High circulating levels of T_3 and T_4 inhibit further TSH secretion and thyroid hormone production through a negative feedback mechanism (*dashed lines*). (From Smeltzer SC, Bare BG, Hinkle JL, et al: Brunner & Suddarth's Textbook of Medical–Surgical Nursing, 12th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 1254.)

Table 43-3 Manifestations of Hypothyroid and Hyperthyroid States

Symptoms of Thyroid Dysfunction		Signs of Thyroid Dysfunction	
Hyperthyroidism	**Hypothyroidism**	**Hyperthyroidism**	**Hypothyroidism**
Nervousness	Fatigue, lethargy	Tachycardia or atrial fibrillation	Bradycardia and, in late stages, hypothermia
Weight loss despite an increased appetite	Modest weight gain with anorexia	Increased systolic and decreased diastolic blood pressures	Decreased systolic and increased diastolic blood pressures
Excessive sweating and heat intolerance	Cold intolerance	Hyperdynamic cardiac pulsations with an accentuated S_1 sound	Dry, coarse skin, and intensity of heart sounds sometimes decreased
Palpitations	Swelling of face, hands, and legs	Warm, smooth, moist skin	Dry, coarse, cool skin, sometimes yellowish from carotene, with nonpitting edema and loss of hair
Frequent bowel movements	Constipation	Tremor and proximal muscle weakness	Impaired memory, mixed hearing loss, somnolence, peripheral neuropathy, carpal tunnel syndrome
Muscular weakness of the proximal type and tremor	Weakness, muscle cramps, arthralgias, paresthesias, impaired memory and hearing	With Graves' disease, eye signs such as stare, lid lag, and exophthalmos	Periorbital puffiness

care admission for management of the patient. Conversely, hypothyroidism can occur, resulting in a severe hypometabolic state. If hypothyroidism is untreated, myxedema coma can develop in the patient, which is most likely to be managed and treated in a critical care unit.

History and Physical Examination

Thyroid hormones affect nearly every cell and tissue in the body. Therefore, manifestations of these disorders are widespread. The typical course of disease progression is insidious, and the nurse needs to take a detailed history to uncover signs and symptoms of either hypothyroidism or hyperthyroidism. History taking focuses on the variety of expected signs and symptoms associated with hypothyroidism and hyperthyroidism. Table 43-3 compares and contrasts the two disorders. Box 43-2 explores the incidence of thyroid disorders in the older patient.

Because of their deep, protected locations in the body, the endocrine glands are in general inaccessible to palpation, percussion, and auscultation. The exception is the thyroid gland, which can be examined physically when it is enlarged. Assessment begins with inspection of the anterior neck area for enlargement, nodules, and symmetry of the gland. The patient is then asked to swallow while the nurse observes the thyroid rising. Next, the thyroid is palpated for size, shape, symmetry, and presence of tenderness (Fig. 43-3). See Box 43-3 for a more detailed description of the steps for palpating the thyroid gland. Thyromegaly (goiter) or thyroid nodules can be detected by palpation. Both lobes of the gland and the isthmus are palpated. Occasionally, a thyroid bruit

BOX 43-2 CONSIDERATIONS FOR THE OLDER PATIENT

Endocrine Disorders

- Expect a higher prevalence of hypothyroidism in the elderly population. Often, the older patient presents with atypical initial symptoms such as depression, apathy, and immobilization.
- Hyperthyroidism in the elderly is much less common; however, the older patient may present with a subclinical picture. Common complaints such as weight loss, fatigue, palpitations and tachycardia, mental confusion, and anxiety are typically attributed to "old age," thus making the disorder harder to detect. Worsening heart failure or unstable angina may result, and often the elderly patient presents with new-onset atrial fibrillation. For these reasons, the highly sensitive thyroid-stimulating hormone (TSH) test should be considered for the older patient with cardiovascular and neurological manifestations.
- The older adult experiences increased insulin resistance and hyperinsulinemia and is, therefore, at higher risk for developing type 2 diabetes.
- Hyperglycemic hyperosmolar state affects the frail elderly population, with the acutely ill older patient at higher risk. Be suspicious of the older patient with diabetes and the new onset of acute illness, such as myocardial infarction, pancreatitis, pneumonia, or other serious infections or illnesses.
- Another expected result of aging is the decrease in secretion of aldosterone and cortisol. This can result in a diminished response to acute illness or trauma. The older patient may have a decreased ability to maintain appropriate fluid and electrolyte balance. In general, older adults display diminished responses to stressors, such as critical illness or trauma.

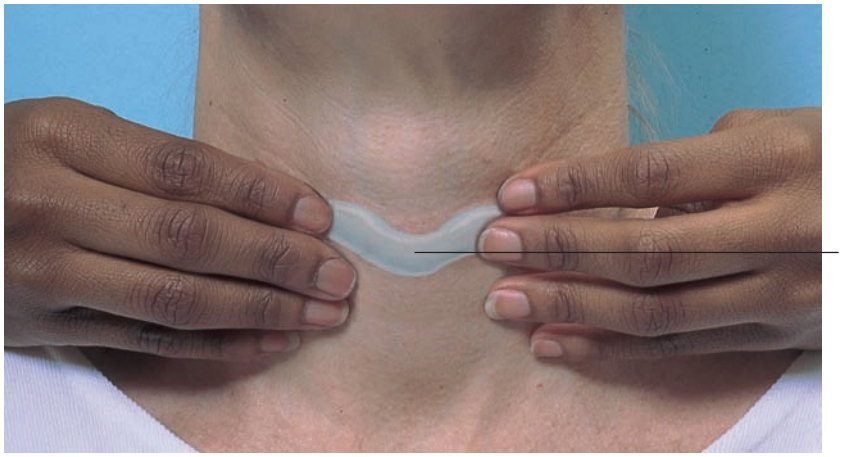


FIGURE 43-3 ▲ The thyroid is examined from behind, with the patient in a sitting position, avoiding hyperextension of the neck. (From Bickley LS, Szilagyi PG: Bates' Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 242.)

can be detected by listening over the gland with the bell of the stethoscope. A bruit is caused by excessive or turbulent blood flow associated with hyperthyroidism and the resultant hypermetabolic state.

Other assessment parameters include noting vital sign changes, skin changes (including edema), neurological changes, and weight changes associated with either disorder. Hypothyroidism is frequently associated with hypotension, bradycardia, hypoventilation, and subnormal temperature. The patient often has dry, flaky skin; edema over the pretibial area; and a deep or husky voice. The patient displays slowed cognitive functioning with slower-than-normal verbal responses, slowed rapid alternating movements, and decreased deep tendon reflexes.

Patients with hyperthyroidism have more neurological manifestations, such as tremor, nervousness, insomnia and restless movements, and hyperactive reflexes. Vital signs are characteristic: hypertension, tachycardia, tachypnea, and hyperthermia. The patient may have a goiter with detectible bruit. Also, the patient may have exophthalmos or proptosis of the eyes. The eyes may unilaterally or bilaterally protrude from the eye sockets, rendering the patient unable to close one or both eyes (Fig. 43-4).

BOX 43-3 Steps for Palpating the Thyroid Gland

- Ask the patient to flex the neck slightly forward to relax the sternomastoid muscles.
- Place the fingers of both hands on the patient's neck so that your index fingers are just below the cricoid cartilage.
- Ask the patient to sip and swallow water as before. Feel for the thyroid isthmus rising up under your finger pads. It is often but not always palpable.
- Displace the trachea to the right with the fingers of the left hand; with the right-hand fingers, palpate laterally for the right lobe of the thyroid in the space between the displaced trachea and the relaxed sternomastoid. Find the lateral margin. In similar fashion, examine the left lobe.
- The lobes are somewhat harder to feel than the isthmus, so practice is needed.
- The anterior surface of a lateral lobe is approximately the size of the distal phalanx of the thumb and feels somewhat rubbery.
- Note the *size*, *shape*, and *consistency* of the gland and identify any *nodules* or *tenderness*.

If the thyroid gland is enlarged, listen over the lateral lobes with a stethoscope to detect a *bruit,* a sound similar to a cardiac murmur but of noncardiac origin.

From Bickley LS, Szilagyi PG: Bates' guide to physical examination and history taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 242.

Laboratory Studies

Thyroid-Stimulating Hormone Test (Thyrotropin Assay)

The TSH test is a highly sensitive test used to diagnose hypothyroidism and hyperthyroidism. The third-generation immunometric assay tests of TSH are 100 times more sensitive than the earlier methods for measuring TSH, and this test is the preferred method for diagnosing and monitoring progression of thyroid disease. Box 43-4 provides a review of common thyroid tests. Table 43-4 lists medications that may interfere with thyroid tests.

The TSH test measures circulating TSH from the anterior pituitary. TSH stimulates the release and distribution of the T_3 and T_4 stored in large amounts in the thyroid gland. Measuring TSH helps determine whether the hypothyroidism is primary (ie, caused by dysfunction of the thyroid gland)

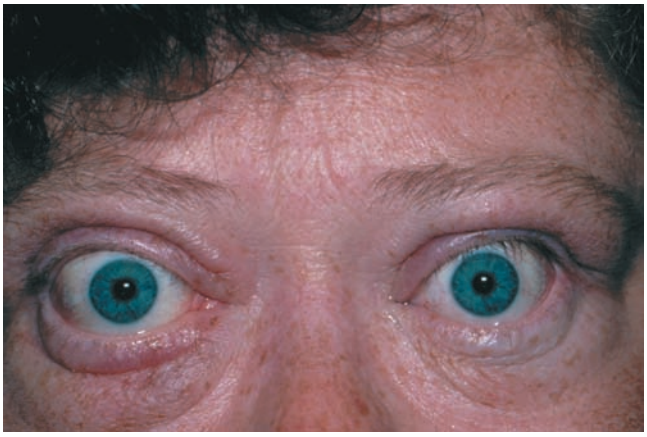


FIGURE 43-4 ▲ A woman with Graves' disease (hyperthyroidism). Note the exophthalmos. (From Goodheart HP: Goodheart's Photoguide of Common Skin Disorders: Diagnosis and Management, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2003, p 391.)

BOX 43-4 Laboratory Evaluation of the Thyroid

Tests That Assess Thyroid Function
- Radioactive iodine uptake

Tests That Assess the Hypothalamic–Pituitary Axis
- Sensitive TSH
- TSH-releasing hormone stimulation test

Tests That Assess Thyroid Hormone Binding Peripherally
- Total thyroxine (T_4) and total triiodothyronine (T_3)
- Free T_4 and free T_3
- In vitro uptake tests (T_3 resin uptake)
- Thyroid hormone–binding ratios (free T_4 index)
- T_4-binding globulin

Diagnostic Studies
- Iodine-131, technetium-99m scans
- Ultrasonography
- Computed tomography
- Magnetic resonance imaging
- Computerized rectilinear thyroid

Miscellaneous Tests
- Thyroid antibodies (thyroid peroxidase, thyroid-stimulating immunoglobulin)
- Thyroglobulin
- Calcitonin
- Basal metabolic rate

or secondary (ie, caused by hypofunction of the anterior pituitary gland). A high TSH level helps diagnose primary hypothyroidism. Measuring the TSH level also helps guide medication titrations for patients requiring exogenous thyroid hormone.

However, the levels of TSH and free T_4 are highly influenced by stress in critically ill patients because of problems with protein levels that are often seen in critical care. Malnutrition, hepatic dysfunction, pregnancy, and drugs affect the TSH and free T_4 levels, and actual thyroid disease is not present. This is termed euthyroid sick syndrome.[1] Therefore, the results of the TSH test need to be analyzed carefully in the critically ill patient. The normal adult value for TSH is 0.4 to 5.4 mIU/L.

Total Thyroxine

Total T_4 measures both the free T_4 and the portion carried by thyroxine-binding globulin (TBG). T_4 is increased in hyperthyroidism and decreased in hypothyroidism. Any factor that affects protein binding affects the results of the total T_4. These factors include pregnancy; estrogen or androgen therapy; and taking oral contraceptives, salicylates, or phenytoin. Normal values depend on the laboratory method used. The normal value is 9.8 to 22.6 mcg/dL in infants; childhood norms are up to 5.6 to 16.6 mcg/dL. Normal adult values range from 4.6 to 12 mcg/dL and are higher during pregnancy. Older adults have lower values because plasma proteins decrease as people age.

Free Thyroxine and Free Thyroxine Index

Free T_4 and free T_4 index measure the free part of T_4, the part that is not bound to protein. Free T_4 is the metabolically active form of the hormone that can be used by tissues. It makes up a small part of the total T_4. The free T_4 test is more useful than the total T_4 test in diagnosing hypofunction and hyperfunction of the thyroid gland because it helps diagnose thyroid function when TBG levels are abnormal. This test can also evaluate thyroid replacement therapy. Radioisotopes can interfere with test results, and heparin can give false high readings. This test can be performed by direct assay or by indirect measurement. The direct assay normal value is 0.8 to 2.7 ng/mL, whereas the free T_4 index is 4.6 to 12 ng/mL.

Free Triiodothyronine

Free T_3 measures the circulating T_3 that exists in the free state in the blood, unbound to protein. This is one measure to evaluate thyroid function. T_3 is about five times more potent than T_4 and is more metabolically active. Decreased values indicate hypothyroidism. Radioisotopes also affect results. Normal adult values are 260 to 480 pg/dL.

Triiodothyronine Resin Uptake Test

The T_3 resin uptake test is an indirect measure of TBG available to bind T_3 and T_4. It is increased with thyrotoxicosis.

Calcitonin

Calcitonin, or thyrocalcitonin, is a hormone secreted by the thyroid. It is secreted in response to high levels of calcium and reduces the calcium level by increasing its deposition in bone.

Thyroid Antibodies

Several autoimmune thyroid diseases produce detectable antibodies. Specifically, Graves' disease, Hashimoto's thyroiditis, and chronic autoimmune thyroid disease cause elevations in antithyroid antibodies, detectable by immunoassay techniques. These conditions can lead to severe hypothyroidism or hyperthyroidism if not treated.

Thyroglobulin

Thyroglobulin can be measured by radioimmunoassay and is elevated in most thyroid disorders. This test has limited diagnostic value because it is nonspecific. It is used clinically to follow the progression of disease in a patient being treated for thyroid cancer.

Diagnostic Studies

Thyroid Scan and Radioactive Iodine Uptake

The radioactive iodine uptake test measures the rate of iodine uptake by the thyroid gland after the administration of iodine-123 tracer (by capsule, solution, or intravenous injection). A scintillation counter then measures gamma rays released from the breakdown of the tracer in the thyroid, producing a visual representation of the radioactivity in the thyroid gland, neck, and mediastinum. Scan time is about 20 minutes. Normally, the radioactive iodine is evenly distributed in the thyroid gland, and the scan shows a normal size, position, and shape.

Table 43-4 Medications That May Interfere With Thyroid Tests

Substance Determined	Drugs Causing Increased Values or False-Positive Values	Drugs Causing Decreased Values or False-Negative Values
Calcitonin (plasma)	Estrogen/progestin, calcium, cholecystokinin, epinephrine, glucagon	Octreotide, phenytoin
Thyroxine (T_4) free (serum)	Amiodarine, aspirin, carbamazepine, danazol, furosemide, levothyroxine, phenytoin, probenecid, propranolol, oral contraceptives, radiographic agents, tamoxifen, thyroxine, valproic acid	Amiodarone, anabolic steroids, anticonvulsants (eg, carbamazepine), asparaginase, clofibrate, corticosteroids, furosemide, isotretinoin, levothyroxine, methadone, methimazole, octreotide, phenobarbital, phenytoin, ranitidine
Free triiodothyronine (T_3) (serum)	Amiodarone, aspirin, carbamazepine, fenoprofen, levothyroxine, phenytoin, ranitidine, thyroxine	Amiodarone, carbamazepine, corticosteroids, methimazole, phenytoin, propranolol, radiographic agents, somatostatin
Free thyroxine index (serum)	Amiodarone, amphetamine, furosemide, levothyroxine, oral contraceptives, Phenobarbital, propranolol	Aspirin, carbamazepine, clomiphene, corticosteroids, co-trimoxazole, ferrous sulfate, iodides, isotretinoin, lovastatin, methimazole, phenobarbital, phenytoin, primidone, radioactive iodine
Thyroglobulin (serum)	Amiodarone	Carbamazepine, neomycin, thyroxine
Thyroid stimulating hormone (serum)	Aminoglutethimide, amphetamine, atenolol, calcitonin, carbamazepine, chlorpromazine, clomiphene, estrogen, ethionamide, ferrous sulfates, furosemide, iodides, lithium, lovastatin, mercaptopurine, metoprolol, morphine, nitroprusside, phenytoin, potassium iodide, prazosin, prednisone, propranolol, radiographic agents, rifampin, sulfonamides, thyrotropin-releasing hormone (TRH)	Amiodarone, anabolic steroids, antithyroid drugs, aspirin, carbamazepine, clofibrate, corticosteroids, danazol, dobutamide, dopamine, fenoldopam, growth releasing hormone, hydrocortisone, interferon, levodopa, levothyroxine, nifedipine, octreotide, phenytoin, pimozide, pyridoxine, somatostatin, thyroxine, troleandomycin
Thyroxine-binding globulin (serum)	Carbamazepine, clofibrate, diethylstilbestrol, estrogens, mestranol, oral contraceptives, perphenazine, phenothiazines, progesterone, tamoxifen, thyroid agents, warfarin	Anabolic steroids, asparaginase, aspirin, chlorpropamide, colestipol, corticosteroids, cortisone, cytostatic therapy, phenytoin, propranolol, sulfonamides
Triiodothyronine (T_3) total (serum)	Amiodarone, amphetamine, clofibrate, estrogens, fenoprofen, fluorouracil, insulin, levothyroxine, mestranol, methadone, opiates, phenothiazines, phenytoin, propylthiouracil, prostaglandins, ranitidine, rifampin, somatotropin, tamoxifen, terbutaline, TRH, valproic acid	Amiodarone, anabolic steroids, androgens, anticonvulsants (eg, phenytoin), asparaginase, aspirin, arenolol, cholestyramine, cimetidine, clomiphene, clomipramine, colestipol, corticosteroids, co-trimoxazole, furosemide, interferon, iodides, isotretinoin, lithium, methimazole, metoprolol, neomycin, netilmicin, oral contraceptives, penicillamine, phenobarbital, phenytoin, potassium iodide, propranolol, propylthiouracil, radiographic agents, reserpine, salicylates (eg, aspirin), somatostatin, sulfonylureas
Triiodothyronine uptake (blood)	Anabolic steroids, androgens, aspirin, colestipol, corticosteroids, cytostatic therapy, dicoumarol, heparin, phenytoin, propranolol, salicylates, sulfonamides, thyroid agents, warfarin	Antiovulatory drugs, antithyroid drugs, carbamazepine, clofibrate, diethylstilbestrol, estrogens, heparin, heroin, mestranol, methadone, oral contraceptives, perphenazine, phenothiazines, progesterones, tamoxifen, thiazide diuretics (eg, hydrochlorothiazide), thyroid agents, warfarin

From Fischbach FT, Dunning MB: A Manual of Laboratory and Diagnostic Tests, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 1239; pp 1253–1254

The thyroid scan may be performed in conjunction with a radioactive iodine uptake study. After the patient takes the radioactive iodine, a count is made over the thyroid gland with a scintillation counter at specific times. These nuclear tests can indicate areas of increased and decreased function and provide data to diagnose hyperthyroidism, hypothyroidism, nodules, ectopic thyroid tissue, and cancer of the thyroid.

Fine-Needle Biopsy

Fine-needle biopsy is the diagnostic tool of choice for detecting malignancy for a thyroid nodule. It is often the initial test for evaluation of any thyroid mass. The test is safe, quick, and accurate, and results are usually available within hours to several days.

Ultrasound

Ultrasound of the thyroid gland uses high-frequency sound waves to produce an image of the gland. Ultrasound is an easy, noninvasive procedure that has no radiation risks and can be performed at the bedside. The test produces good images of structures and can detect masses, nodules, cysts, and enlargements of the gland.

▲ The Parathyroid Gland

The parathyroid gland produces parathyroid hormone (PTH), which maintains blood calcium and phosphorus levels, neuromuscular activity, blood clotting function, and cell membrane permeability. The four parathyroid glands are located just posterior to the thyroid gland and are sometimes damaged during thyroid surgery.

The output of PTH is regulated by the serum level of calcium under a negative feedback system. Overproduction of PTH results in hyperparathyroidism and is characterized by bone decalcification and the development of renal stones containing calcium.

Hypocalcemia, as a result of hypoparathyroidism, manifests neurologically as tetany (general muscular hypertonia, tremor, and spasmodic movements) when calcium levels dip below 5 to 6 mg/dL. The patient may complain of numbness, tingling, and cramps in the extremities. As the hypocalcemia worsens, the patient experiences bronchospasm, laryngeal spasm, carpopedal spasm (flexion of the elbows and wrists with extension of the carpophalangeal joints), dysphagia, photophobia, cardiac dysrhythmias, and seizures.

History and Physical Examination

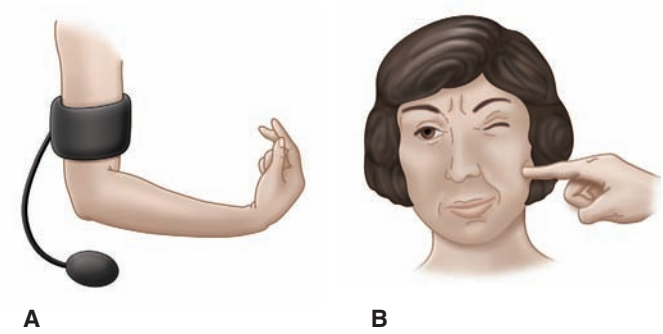
The nurse establishes a history of electrolyte imbalance, specifically related to calcium and phosphorus. Additional information includes a history of a variety of other symptoms listed in the endocrine health assessment (see Box 43-1). The patient may present with kidney stone symptoms, such as severe flank pain, groin pain, frequent urination, hematuria, and nausea and vomiting. The patient may experience joint and bone pains and may sustain pathological fractures, especially of the spine. The nurse remains vigilant for signs of tetany and related complications.

Tetany can be assessed by evaluating the patient for Trousseau's sign or Chvostek's sign (Fig. 43-5). Trousseau's sign is positive when carpopedal spasm is induced by occluding the blood flow to the arm for 3 minutes with the use of a blood pressure cuff. If tapping over the facial nerve just in front of the parotid gland causes twitching of the mouth or eye, the patient has a positive Chvostek's sign.

Laboratory Studies

Normal calcium levels range from 8.6 to 10.3 mg/dL. Most (99%) of body calcium is in the bone, and the remaining 1% is in the ECF. Nearly 50% of serum calcium is ionized or free, whereas the remainder is bound to albumin.

Marked serum calcium elevations (levels >10.3 mg/dL) are the most obvious manifestation of hyperparathyroidism, and common causes include primary hyperparathyroidism, malignancy, sarcoidosis, vitamin D toxicity, hyperthyroidism, and some medications, such as thiazide diuretics and lithium.



A B

FIGURE 43-5 ▲ Tetany is caused by tonic spasm of the intrinsic hand muscles. Evaluate the patient for Trousseau's sign (**A**) or Chvostek's sign (**B**).

Low serum calcium levels are the marker for hypoparathyroidism. Tetany develops at calcium levels of 5 to 6 mg/dL or lower. Common causes of hypocalcemia include hypoalbuminemia, renal failure, hypoparathyroidism, acute pancreatitis, tumor lysis syndrome, severe hypomagnesemia, and multiple citrated blood transfusions.

▲ The Endocrine Pancreas

Disorders of the endocrine pancreas are characterized by chronic hyperglycemia and result in major shifts of fluids and electrolytes as well as in blood glucose levels. The risk for developing diabetes increases with age. The two main types of diabetes are type 1 and type 2, and both forms of diabetes can lead to serious illnesses requiring critical care.

History and Physical Examination

A complete history is multisystem focused because glucose dysfunction affects every system of the body. A good family history is obtained to document the role of familial patterns often seen in type 2 diabetes. The characteristics of patients at risk for developing type 2 diabetes are reviewed in Box 43-5.

BOX 43-5 PATIENT SAFETY

Risk Factors Associated With Developing Type 2 Diabetes

- Family history of diabetes (parents, grandparents, siblings)
- Obesity (body mass index > 27 kg/m^2)
- Race and ethnicity (African American, Native American, Hispanic American, Asian American, Pacific Islander)
- Age greater than 45 years
- History of impaired fasting glucose or impaired glucose tolerance
- Hypertension
- High-density lipoprotein cholesterol less than 35 mg/dL
- Triglyceride level greater than 250 mg/dL
- History of gestational diabetes, the delivery of a baby greater than 9 pounds, or both

For the patient with known diabetes who enters the critical care arena, the nurse focuses on gathering information about the extent of the disease and its duration, the onset of complications, the medications taken for the disease, and other past medical and surgical history. Chronic complications, such as neuropathy, retinopathy, and nephropathy, are explored as well as the coexistence of related medical conditions such as hypertension, hyperlipidemia, obesity, and peripheral vascular disease. Refer to Box 43-1 on page 974 for a health history review of the endocrine system.

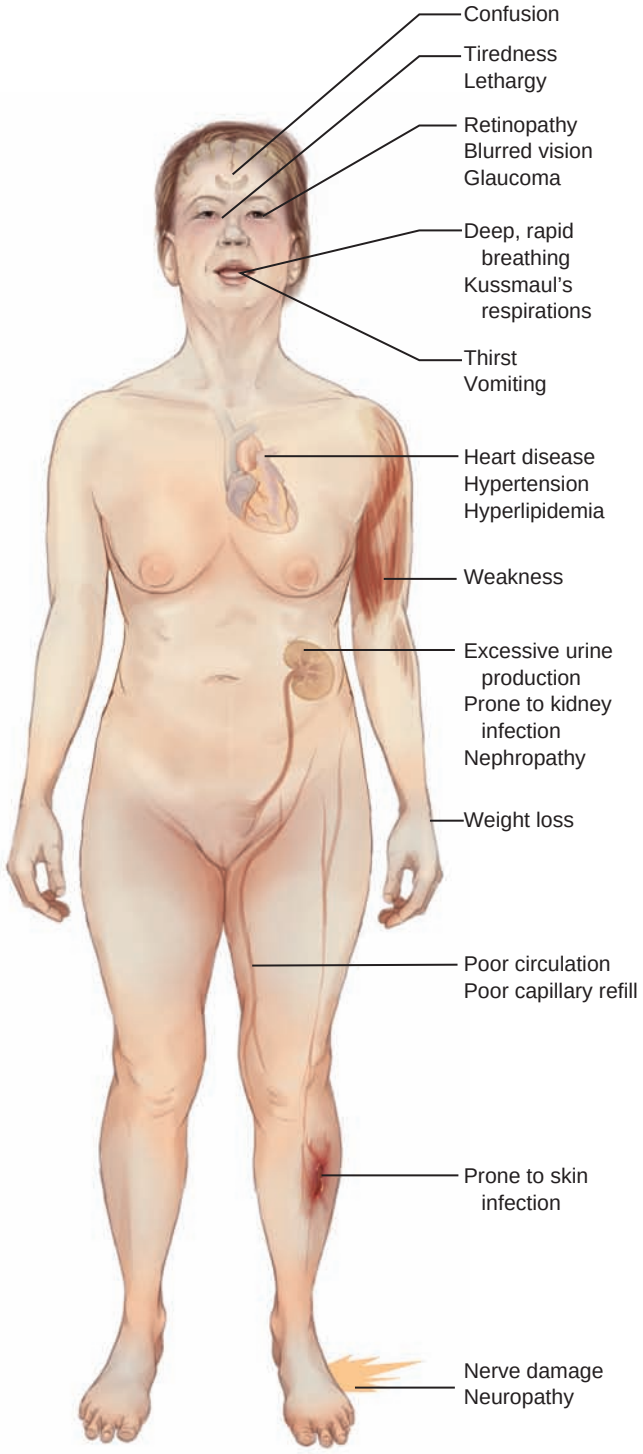


FIGURE 43-6 ▲ Clinical features of diabetes mellitus.

Physical examination focuses on the severe fluid and electrolyte and neurological dysfunction seen with acute diabetes complications such as DKA, HHS, and hypoglycemia. Observation of fluid status and hydration is essential. Skin turgor, buccal membranes, weight, urine specific gravity, and vital signs are assessed. The nurse monitors the patient's neurological status frequently as well as central venous pressures and other advanced monitoring if available. The presence of a fruity odor on the breath (associated with ketonemia) should be noted. In addition, the patient may display Kussmaul's respirations as an attempt to rapidly exhale excess carbon dioxide. This respiratory pattern is characterized by deep, rapid breathing. Figure 43-6 summarizes the physical features seen in the patient with diabetes mellitus.

Laboratory Studies

Fasting Blood Glucose Level and Fingerstick Glucose Analysis

The fasting blood glucose level provides a foundation for managing diabetes mellitus. Very high blood glucose levels can occur in DKA and HHS. In addition, elevated glucose levels can occur in Cushing's syndrome, high-stress states, pancreatitis, and chronic renal and liver disease. Hypoglycemia can occur in Addison's disease, pancreatic tumors, starvation, and hypopituitary problems. The normal value for fasting glucose in adults is 65 to 110 mg/dL. Two-hour postprandial blood glucose testing helps further evaluate carbohydrate metabolism, and the normal value is 65 to 126 mg/dL. The American Diabetes Association (ADA) criteria for the diagnosis of diabetes are given in Box 43-6.

In addition, the ADA recognizes an intermediate group of people who have glucose levels less than 126 mg/dL but nonetheless have glucose levels too high to be considered normal. If their fasting glucose is greater than 100 mg/dL but less than 126 mg/dL, they have the abnormality known as impaired fasting glucose (IFG). If the oral glucose tolerance test (GTT) is performed to diagnose glucose abnormalities, the 2-hour postload level of less than 140 mg/dL is considered normal, a level of 140 to 199 mg/dL is considered impaired glucose tolerance (IGT), and a level greater than 200 mg/dL is provisionally diagnostic of diabetes.[2] Patients with IFG or IGT are now diagnosed with "prediabetes"; they are at high risk for developing diabetes as well as cardiovascular disease. IFG and IGT are associated with metabolic syndrome, which is manifested by increased abdominal obesity, high triglyceride levels, low high-density lipoprotein cholesterol levels, and hypertension.[3]

Numerous drugs can interfere with glucose regulation, including corticosteroids, diuretics, lithium, phenytoin, β-blockers, and estrogen. Hypoglycemic reactions can result from sulfonylureas, insulin, alcohol, β-blockers, angiotensin-converting enzyme inhibitors, and aspirin.

Fingerstick glucose testing can be used at the bedside for immediate feedback regarding the patient's glucose status. In addition, patients can be taught to use fingerstick devices at home to monitor their glucose levels and responses to medication. Standardization of the equipment must be ensured when these devices are used for patient monitoring.

BOX 43-6 Criteria for the Diagnosis of Diabetes Mellitus

1. A1C = 6.5% or greater. The test should be performed in a laboratory using a method that is NGSP (National Glycohemoglobin Standardization Program) certified and standardized to the DCCT assay.*

OR

2. Fasting plasma glucose (FPG) = 126 mg/dL (7.0 mmol/L) or more. Fasting is defined as no caloric intake for at least 8 hours.*

OR

3. Two-hour plasma glucose level 200 mg/dL (11.1 mmol/L) or more during an oral glucose tolerance test. The test should be performed as described by the World Health Organization, using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water.*

OR

4. In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose level of 200 mg/dL (11.1 mmol/L) or more.

*In the absence of unequivocal hyperglycemia, criteria 1 to 3 should be confirmed by repeat testing. DCCT, Diabetes Control and Complications Trial

From American Diabetes Association. Position statement: Diagnosis and Classification of Diabetes Mellitus. Diabetes Care 35(1):S12, 2012.

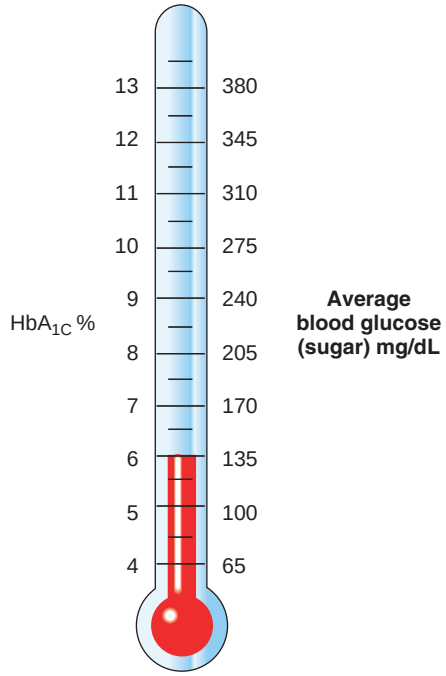


FIGURE 43-7 ▲ Comparison of glycosylated hemoglobin (HbA_{1c}) to average blood glucose level. The normal range is shown in *red*.

In general, point-of-service testing such as this may not be appropriate for the critically ill patient because fingerstick testing requires adequate tissue perfusion for accuracy, and many critically ill patients do not have this required level of perfusion. Testing glucose from more direct sources of blood (ie, veins, venous lines, central lines, arterial lines) may enhance accuracy.

Glycosylated Hemoglobin

Glycosylated hemoglobin (HbA_{1c}) testing offers information about the average amount of serum glucose that is bound to hemoglobin for the 100- to 120-day life span of erythrocytes. This information is now used to diagnose diabetes[4] and to assess data trends for a person who has been previously diagnosed with diabetes. The percentage result (normal: 4% to 7%) reflects an average of 3 months and enhances accuracy because it controls for many variables such as stress, exercise, fasting state, interfering medications, and recent changes in patient compliance. In comparison with the highly variable, "snapshot view" that is provided by a fasting glucose level, HbA_{1c} testing provides insight into the patient's overall status over the previous months.[5] Figure 43-7 compares the HbA_{1C} value to the average blood glucose value.

Fructosamine

Serum fructosamine level measures glycosylation of serum protein albumin. Albumin has a half-life of approximately 2 weeks, as opposed to the half-life of hemoglobin. It is a useful index that reflects chronic glycemic control in patients with diabetes for whom HbA_{1C} may be inaccurate, such as those with anemia or hemoglobin abnormalities (eg, sickle cell disease).[6]

Insulin

This test helps measure abnormal carbohydrate metabolism by measuring the amount of circulating serum insulin in the fasting state. Insulin is released in response to serum glucose levels. When glucose is elevated, insulin levels should increase as well. Abnormally high levels of insulin may help diagnose insulinoma, a tumor of the islets of Langerhans. The normal adult value is 6 to 24 mcU/mL.

A low insulin level helps diagnose diabetes mellitus, especially in the presence of an abnormal GTT. A fasting blood sample is tested. If the insulin test is performed in conjunction with a GTT, blood samples are drawn at that time. Oral contraceptives and recent administration of radioisotopes interfere with results.

C-Peptide Level

C-peptide is a single chain of amino acids connecting A and B chains of insulin in the proinsulin molecule. It has no known physiological function, but because it persists in higher concentrations than insulin, it may be a more accurate reflection of insulin levels. It provides a useful monitor of average β-cell insulin secretion and can be used to distinguish between types 1 and 2 diabetes mellitus. Normal values are 0.5 to 2.0 ng/mL and indicate that the body is still producing some insulin. Low values (or no insulin C-peptide) indicate that the person's pancreas is producing little or no insulin, as in type 1 diabetes.[7]

Glucagon

This hormone, produced in the alpha cells in the islets of Langerhans, controls the production, storage, and release of glucose. Normally, insulin opposes the action of glucagon. This test measures the production and metabolism of glucagon. A deficiency occurs when pancreatic tissue is lost because of chronic pancreatitis or pancreatic tumors. Increased levels occur in diabetes, acute pancreatitis, and catecholamine secretion (such as occurs with infection,

high stress levels, or pheochromocytoma). Chronic renal failure and cirrhosis of the liver can also increase glucagon levels. Normal fasting values are 50 to 200 pg/mL.

Serum Ketones

Measuring serum ketones reveals information about the use of fat metabolism in lieu of carbohydrates as seen in the critically ill person with diabetes. The normal serum ketone level is 2 to 4 mg/dL. Ketonemia (acetone, β-hydroxybutyrate, and acetoacetate) is manifested by Kussmaul's respirations and a fruity, sweet-smelling odor on the exhaled breath. These signs are the result of the patient's attempt to maintain a normal pH during extreme metabolic acidosis. In DKA, metabolic acidosis is primarily the result of the accumulation of acetoacetic acid and β-hydroxybutyric acid, the preferred method for estimating the severity of DKA.[8]

Urine Ketones

Ketones are not normally found in the urine. Ketones in the urine are associated with diabetes and other diseases of altered carbohydrate metabolism. People with diabetes should test for ketones whenever their urine or blood glucose is high. Because ketones appear in the urine before they can be detected in the blood, this test is often used in the emergency department when screening for acidosis. The test is performed by dipping a ketone reagent strip in a fresh urine sample. The presence of ketones in the urine results from lipolysis or fat breakdown in the absence of adequate insulin.

▲ The Adrenal Gland

The adrenal gland is anatomically and functionally divided into two distinct parts—the outer cortex and the inner medulla (see Chapter 42, Fig. 42-6). The two regions secrete different hormones. The cortex produces mineralocorticoids (eg, aldosterone), glucocorticoids (eg, cortisol), and androgens. The medulla secretes catecholamines such as epinephrine, norepinephrine, and dopamine. Disorders of the adrenal gland have widespread effects on the human body because these hormones regulate major body functions, such as fluid and electrolyte balance, sympathetic nervous system responses, inflammation, and metabolism.

The secretion of hormones by the adrenal gland is regulated in a negative feedback system through the hypothalamic–pituitary axis. The hypothalamus releases corticotropin-releasing hormone, which in turn stimulates the release of ACTH from the anterior pituitary. ACTH then stimulates the adrenal cortex to secrete cortisol.

History and Physical Examination

Refer to Box 43-1 for a review of relevant health history questions related to adrenal disorders. Clinical manifestations of adrenal gland dysfunction depend on the nature of the lesion and which hormone is adversely affected. Adrenal medulla lesions may affect the release of catecholamines and cause sudden, severe headache, diaphoresis, palpitations, and other symptoms associated with paroxysmal hypertension. One such lesion is pheochromocytoma, a benign adrenal medulla tumor that mediates this severe outpouring of catecholamines.

Another common pathology affecting the adrenal gland is a pituitary tumor that leads to hypersecretion of ACTH. The resulting disease, Cushing's syndrome, manifests as central obesity, unusual fat deposits, thin extremities, fragile skin, skin discoloration (striae), sleep disturbances, and catabolism (Fig. 43-8). The same clinical picture can result from chronic exogenous steroid use.[9]

Adrenal insufficiency from autoimmune Addison's disease can lead to an adrenal crisis. The patient lacks adequate stimulation of the adrenal gland, or the adrenal gland is rendered ineffective and stops secreting adequate levels of hormone. Consequently, the patient becomes lethargic, dehydrated, and unable to mount any stress response to handle acute illness or trauma.

The critically ill patient often suffers from mild forms of adrenal insufficiency because the patient's normal stores of hormones are used quickly in response to the illness. Many require exogenous steroids to assist with recovery. A summary of the clinical manifestations of adrenal cortical insufficiency and glucocorticoid excess is given in Table 43-5.

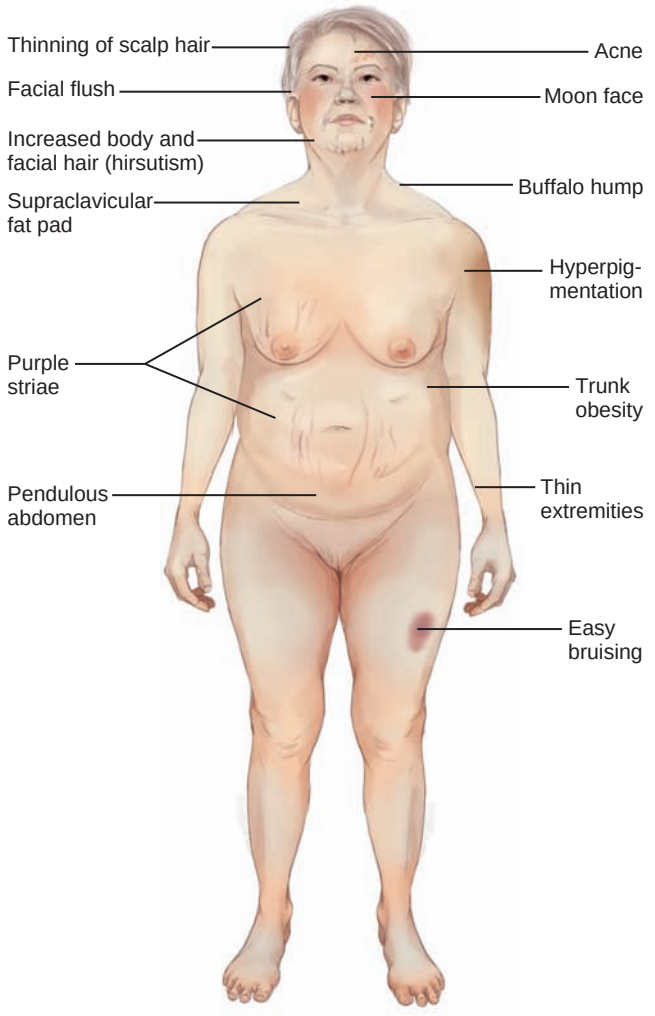


FIGURE 43-8 ▲ Clinical manifestations of Cushing's syndrome.

Table 43-5 Manifestations of Adrenal Cortical Insufficiency and Excess

Parameter	Adrenal Cortical Insufficiency	Glucorticoid Excess
Electrolytes	Hyponatremia* Hyperkalemia*	Hypokalemia
Fluids	Dehydration* (eg, elevated BUN)	Edema
Blood pressure	Hypotension Shock* Orthostatic hypotension	Hypertension
Musculoskeletal	Muscle weakness* Fatigue*	Muscle wasting Fatigue
Hair and skin	Skin pigmentation	Easy bruisability Hirsutism, acne, and striae (abdomen and thighs)
Inflammatory response	Low resistance to trauma, infection and stress	Decrease in eosinophils, lymphocytopenia
Gastrointestinal	Nausea, vomiting* Abdominal pain*	Possible gastrointestinal bleeding
Glucose metabolism	Hypoglycemia*	Impaired glucose tolerance Glycosuria Elevated blood glucose
Emotional	Depression and irritability	Emotional lability to psychosis
Other	Menstrual irregularity Decreased axillary and pubic hair in women	Oligomenorrhea Impotence in the male Centripetal obesity (moon face and buffalo hump)

*Occurs with acute adrenal insufficiency.

BUN, blood urea nitrogen.

Adapted from Porth CM: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009

Laboratory Studies

Cortisol (Hydrocortisone)

This test evaluates the ability of the adrenal cortex to produce the glucocorticoid hormone cortisol. Cortisol is elevated in adrenal hyperfunction and decreased in adrenal hypofunction. Adrenal hyperfunction may be caused by excess secretion of ACTH by the pituitary gland (Cushing's syndrome), high stress, trauma, and surgery. Adrenal hypofunction may be the result of anterior pituitary hyposecretion, hepatitis, and cirrhosis.

Cortisol secretion is diurnal; it is normally higher in the early morning (6:00 AM to 8:00 AM) and lower in the evening (4:00 PM to 6:00 PM). This variation is lost in patients with adrenal hyperfunction and in people under stress. Serum samples are drawn between 6:00 AM and 8:00 AM and between 4:00 PM and 6:00 PM. Normal 8:00 AM values are 5 to 23 fg/dL or 138 to 635 mmol/L. Normal 4:00 PM. values are 3 to 16 fg/dL or 83 to 441 mmol/L.

Cortisol (Dexamethasone) Suppression

When healthy people receive a low dose of dexamethasone (chemically similar to cortisol), ACTH production is suppressed. However, people with adrenal hyperfunction and some with endogenous depression continue to produce ACTH and do not have a diurnal variation of cortisol.

For this test, dexamethasone is given at bedtime. Blood samples are taken the next day at 8:00 AM and 4:00 PM. Medications are discontinued for 24 to 48 hours before this test is started, especially estrogens, phenytoin, and cortisol-related preparations. Radioisotopes should not be given within 1 week of this test. This test is the test of choice to diagnose Cushing's syndrome.[10]

Cortisol Stimulation

This test measures the response of the adrenal glands to an injection of cosyntropin (Cortrosyn, a synthetic ACTH preparation). Blood is drawn for a fasting 8:00 AM cortisol level before cosyntropin is administered, and then blood samples are taken 30 and 60 minutes after it is administered. The adrenal glands normally respond to the cosyntropin by synthesizing and secreting adrenocorticoids. The plasma cortisol level should increase to at least 18 fg/dL. The response to cosyntropin is decreased or absent in people with adrenal insufficiency or hypopituitarism. Long-term steroid therapy affects results. This test may be contraindicated in the presence of infections, inflammatory diseases, and cardiac disease. Cortisol stimulation is the preferred test to diagnose Addison's disease.

Urine and Plasma Catecholamine Levels

Urine vanillylmandelic acid, a metabolite of catecholamines, is rarely used diagnostically today. It is preferred to measure free and fractionated plasma metanephrines, fractionated and total urine metanephrines, and plasma normetanephrines since they yield higher sensitivity levels for pheochromacytoma. Since they have high concentration in the urine and are easy to detect, a 24-hour urine test is performed when a person is suspected of having hypertension due to pheochromocytoma. Elevated levels of catecholamines can be found

in patients with hypothyroidism, DKA, neuroblastomas, and ganglioneuromas.

Urine should not be collected when the patient is fasting. Test results are also affected by many drugs and foods, such as tea, coffee, vanilla, and fruit juice. Therefore, some laboratories restrict certain foods for 2 days before testing and on the day of testing. Certain drugs may also be discontinued for 4 to 7 days before testing. Normal adult values for urine vanillylmandelic acid are 2 to 7 mg/24 h, and for catecholamines, 270 fg/24 h.

Urine 17-Ketosteroids and 17-Hydroxycorticosteroids

These 24-hour urine collection tests reflect adrenal function by measuring the urinary excretion of steroids. They are used infrequently because they have been replaced by serum immunoassays.

Diagnostic Studies

Adrenal Scan

This scan is used to identify the site of certain tumors or sites that produce excessive amounts of catecholamines. The radionuclide iobenguane (^{131}I) is injected intravenously, and scans are performed on days 2, 3, and 4. Sometimes, only 1 day is needed, and other times imaging is needed on days 6 and 7. Normally, tumors and sites of hypersecretion are absent. If ACTH levels are elevated, MRI of the pituitary should be done to seek the source.

▲ Clinical Applicability Challenges

CASE STUDY

Mrs. R., a 61-year-old African American female, arrives in the emergency department after experiencing severe chest pressure, nausea, and diaphoresis at 5:30 AM. She takes a fluid pill for high blood pressure and reports nocturia at least three times. She smokes about a half pack of cigarettes a day (positive history of smoking 1 pack/d for over 40 years) and drinks one to two glasses of wine per week. She is allergic to penicillin (rash). Currently, she works as a secretary part-time and does not exercise much. She has been overweight all of her adult life and has gained about 12 pounds this past year.

The nurse notes that Mrs. R. is fully oriented, obese, with slightly diaphoretic skin and dry buccal membranes. Her vital signs are blood pressure, 165/110 mm Hg; heart rate, 102 beats/min; and respiratory rate, 24 breaths/min. She is afebrile.

Mrs. R.'s laboratory results show a glucose level of 220 mg/dL, serum ketones are mildly positive, and white blood cells are normal. Mrs. R.'s electrocardiogram shows sinus rhythm (rate, 86 to 94 beats/min) with ST elevation in inferior leads. She denies acute chest pain upon arrival as the staff prepares to start an intravenous line.

1. Mrs R. has not been diagnosed with diabetes. Explain how she will be diagnosed using the American Diabetes Association's criteria.
2. Since Mrs. R. was not diagnosed with diabetes previously, discuss several reasons why she may be displaying signs and symptoms of diabetes currently.
3. What specific risk factors for type 2 diabetes mellitus are illustrated in this case study?

References

1. Aytug S, Shapiro LE: Euthyroid sick syndrome. EMedicine Medscape Available at: http://emedicine.medscape.com/article/118651-overview, 2011
2. American Diabetes Association: Position statement: Diagnosis and classification of diabetes mellitus. Diabetes Care 35(1):S11–S63, 2012
3. National Heart, Lung, Blood Institute, National Institutes of Health: Metabolic syndrome. Updated 2011. Available at: http://www.nhlbi.nih.gov/health/health-topics/topics/ms/, 2011
4. American Diabetes Association: Summary of revisions for the 2010 clinical practice recommendations. Diabetes Care 33(1):S53, 2010
5. American Diabetes Association: Standards of medical care in diabetes—2012. Diabetes Care 35(1):S11–S63, 2012
6. American Diabetes Association: Standards of medical care in diabetes—2012. Diabetes Care 33(1):S18, 2012
7. Ko GT, So W-Y, Tong PC, et al: Effect of interactions between C peptide levels and insulin treatment on clinical outcomes among patients with type 2 diabetes mellitus. CMAJ 180(9):907–908, 2009
8. Raghavan V, Bessen HA, Hamdy O, et al: Diabetes ketoacidosis. eMedicine Medscape, Available at: http://emedicine.medscape.com/article/118361-overview, January 2012
9. Adler GK, Ziel FH, Talavera F, et al: Cushing's syndrome. eMedicine Medscape. Available at: http://emedicine.medscape.com/article/117365-overview, 2012
10. Reimondo G, Boviol S, Allasino B, et al: The combined low-dose dexamethasone suppression corticotrophin-releasing hormone test as a tool to rule out Cushing's syndrome. Eur J Endocrinol 159:569–576, 2009

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

44 Common Endocrine Disorders

Jane Kapustin

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Examine the pathophysiological principles that help explain thyrotoxic crises, myxedema coma, adrenal crises, syndrome of inappropriate antidiuretic hormone secretion, diabetes insipidus, diabetic ketoacidosis, hyperglycemic hyperosmolar state, and hypoglycemia.
2. Distinguish key precipitating factors, history, and clinical manifestations of endocrine disorders.
3. Discuss five laboratory studies that are useful in diagnosing acute endocrine disorders.
4. Analyze the similarities and differences in caring for patients with endocrine disorders.
5. Explore the nursing role in assessing, managing, and evaluating a plan of care for patients with endocrine disorders.

Endocrine disorders have multisystem effects. At the same time, acute illness may lead to hypofunction and, less commonly, hyperfunction of the neuroendocrine system. Patients with acute illness who are at risk for endocrine dysfunction may have a preexisting endocrine disorder. Although that disorder may have already been diagnosed, many endocrine dysfunctions are not recognized before acute illness. For this reason, endocrine dysfunction should be considered in the assessment and management of all critically ill patients.

▲ Hypothalamic–Pituitary–Adrenal Function During Critical Illness

Severe illness and stress activate hypothalamic–pituitary–adrenal (HPA) axis, resulting in the release of cortisol from the adrenal cortex. This mechanism is key to providing for positive adaptation to severe stressors and for general cellular and organ homeostasis. The nervous and endocrine systems are both influenced by the responses to stress, and the actions of these systems are intertwined and interdependent. For example, the neurosensory pathways and chemical mediators in the vascular system will detect a potential stressor, and the endocrine and immune systems will be stimulated to provide both an interaction and reaction to deal effectively with it. Table 44-1 defines the hormones that are intricately involved in the response to stress. The stress response is first activated at the level of the central nervous system (CNS). Communication occurs along the many neuronal pathways in the cerebral cortex, limbic system, thalamus, hypothalamus, pituitary gland, and reticular activating system (Fig. 44-1). One area of the brainstem, the locus ceruleus, is responsible for the autonomic nervous system release of norepinephrine, one of the most basic survival responses to stress. This release causes a chain of events that prepares humans for mounting an appropriate reaction to the stressor. In turn, the corticotropin-releasing factor induces the secretion of the adrenocorticotropic hormone (ACTH), which triggers the synthesis and release of cortisol from the adrenal gland.[1]

Acute and chronic stressful events can initiate a significant physiological response in an effort to maintain homeostasis. The "fight-or-flight" response is the initial reaction to a severe stressor, and release of norepinephrine and epinephrine follows. Activation of the HPA axis occurs in response to stress and critical illness, and secretion of cortisol, the primary glucocorticoid hormone, results. Cellular actions of cortisol include stimulation of gluconeogenesis, anti-inflammatory effects of the immune system, maintenance of vascular tone and endothelial integrity, increased sensitivity to pressors, reduction of nitric oxide–mediated vasodilation, and modulation of angiotensinogen synthesis. This hormone plays an important role in survival from major stressful events. Cortisol stimulates the HPA axis during acute and chronic events, such as surgery, sepsis, trauma, burns, and other severe critical illnesses. Initially, elevated cortisol levels are typically detected in the critically ill. If the stressor is prolonged, cortisol levels become depleted. Refer to Table 44-1 and to Chapter 42 for a review of the hormones activated in the neuroendocrine response to stress.

This chapter now presents an overview of the pathophysiology, assessment, management, and complications of patients with acute endocrine disorders. These disorders are

Table 44-1 Hormones Involved in the Neuroendocrine Response to Stress

Hormones Associated With the Stress Response	Source of the Hormone	Physiological Effects
Catecholamines (norepinephrine, epinephrine)	Locus ceruleus, adrenal medulla	Produces a decrease in insulin release and an increase in glucagon release resulting in increased glycogenolysis, gluconeogenesis, lipolysis, proteolysis, and decreased glucose uptake by the peripheral tissues; an increase in heart rate, cardiac contractility, and vascular smooth muscle contraction; and relaxation of bronchial smooth muscle
Corticotropin-releasing factor	Hypothalamus	Stimulates ACTH release from anterior pituitary and increased activity of neurons in locus ceruleus
Adrenocorticotropic hormone (ACTH)	Anterior pituitary	Stimulates the synthesis and release of cortisol
Glucocorticoid hormones (eg, cortisol)	Adrenal cortex	Potentiates the actions of epinephrine and glucagon; inhibits the release and/or actions of the reproductive hormones and thyroid-stimulating hormone; and produces a decrease in immune cells and inflammatory mediators
Mineralocorticoid hormones (eg, aldosterone)	Adrenal cortex	Increases sodium absorption by the kidney
Antidiuretic hormone (ADH, vasopressin)	Hypothalamus, posterior pituitary	Increases water absorption by the kidney; produces vasoconstriction of blood vessels; and stimulates the release of ACTH

From Porth CM: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 202.

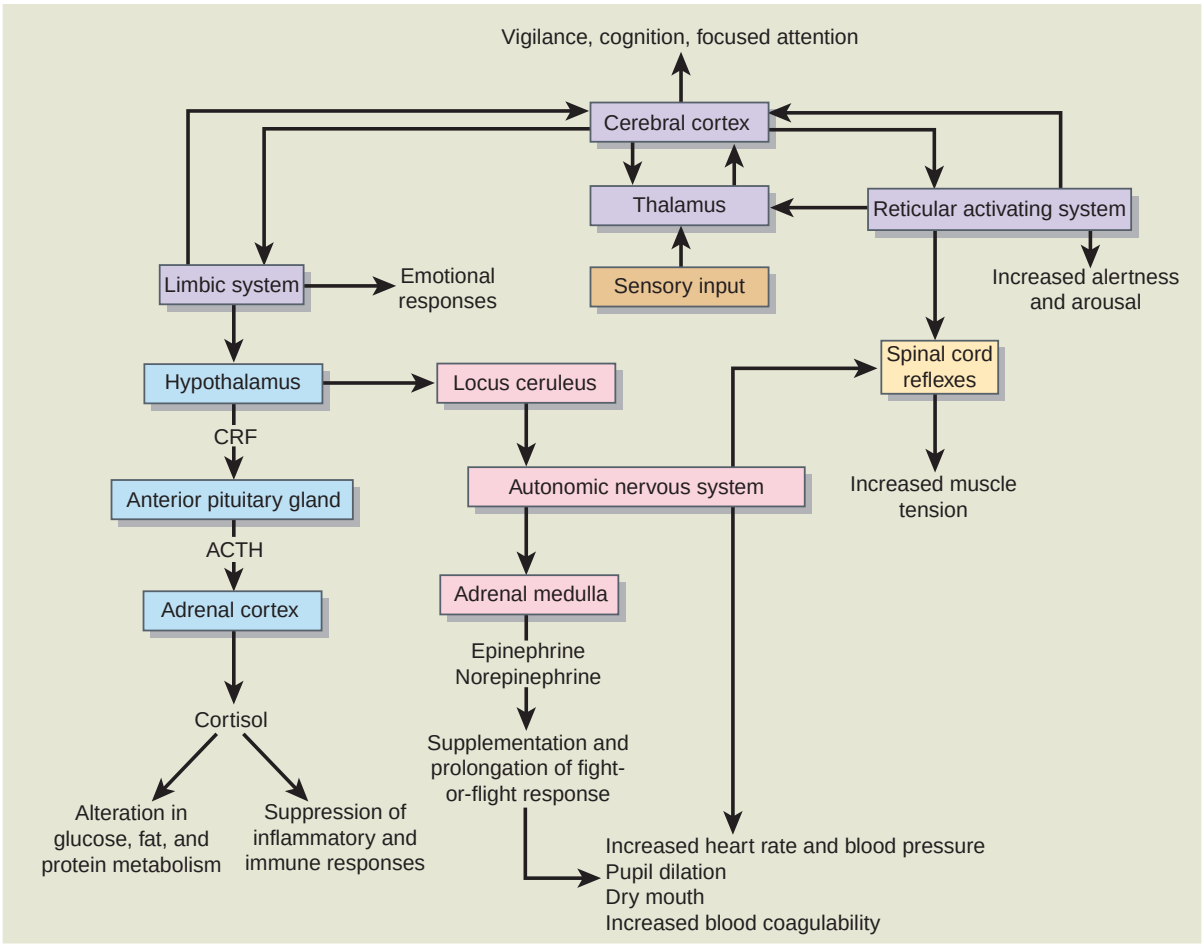


FIGURE 44-1 ▲ Neuroendocrine pathways and physiologic responses to stress. ACTH, adrenocorticotropic hormone; CRF, corticotropin-releasing factor. (From Porth CM: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 203).

SPOTLIGHT ON GENETICS 44-1

FRAGILE X SYNDROME

- Fragile X syndrome occurs in approximately 1 in 4,000 males and 1 in 8,000 females, and is a genetic condition that causes a range of developmental problems including learning disabilities and cognitive impairment.
- Mutations in the *FMR1* gene cause fragile X syndrome. The *FMR1* gene provides instructions for making a protein called fragile X mental retardation 1 protein, whose function is not fully understood.
- Nearly all cases of fragile X syndrome are caused by a mutation in which a DNA segment, known as the CGG triplet repeat, is expanded within the *FMR1* gene. Normally, this DNA segment is repeated from 5 to about 40 times. In people with fragile X syndrome, however, the CGG segment is repeated more than 200 times. The abnormally expanded CGG segment turns off (silences) the *FMR1* gene, which prevents the gene from producing fragile X mental retardation 1 protein.
- Genetic testing is available to diagnose fragile X syndrome.

Genetic Home Reference-http://ghr.nlm.nih.gov—Accessed July 14, 2011
Cornish KM, Gray KM, Rinehart NJ. Fragile X syndrome and associated disorders. Adv Child Dev Behav 39:(C):211–235, 2010.

thyroid dysfunctions, adrenal gland dysfunctions, antidiuretic hormone (ADH) dysfunctions, and emergencies in patients with diabetes. Spotlight on Genetics 44-1 discusses the genetic disorder, Fragile X Syndrome.

▲ Thyroid Dysfunction

Thyroid dysfunction is a common clinical problem in the United States. Women are 5 to 10 times more likely than men to present with thyroid disease. The most common thyroid conditions are hyperthyroidism, hypothyroidism, and thyroid nodule. Clinical presentations may be quite subtle; therefore, patients with endocrine manifestations must be regarded with a high index of suspicion. Extremes of these two conditions are discussed in greater detail in this chapter. Figure 44-2 compares the signs and symptoms of hyperthyroidism and hypothyroidism.

Thyrotoxic Crisis

Thyrotoxic crisis is a severe form of hyperthyroidism often associated with physiological or psychological stress. When the thyroid state worsens critically, it is called thyrotoxic

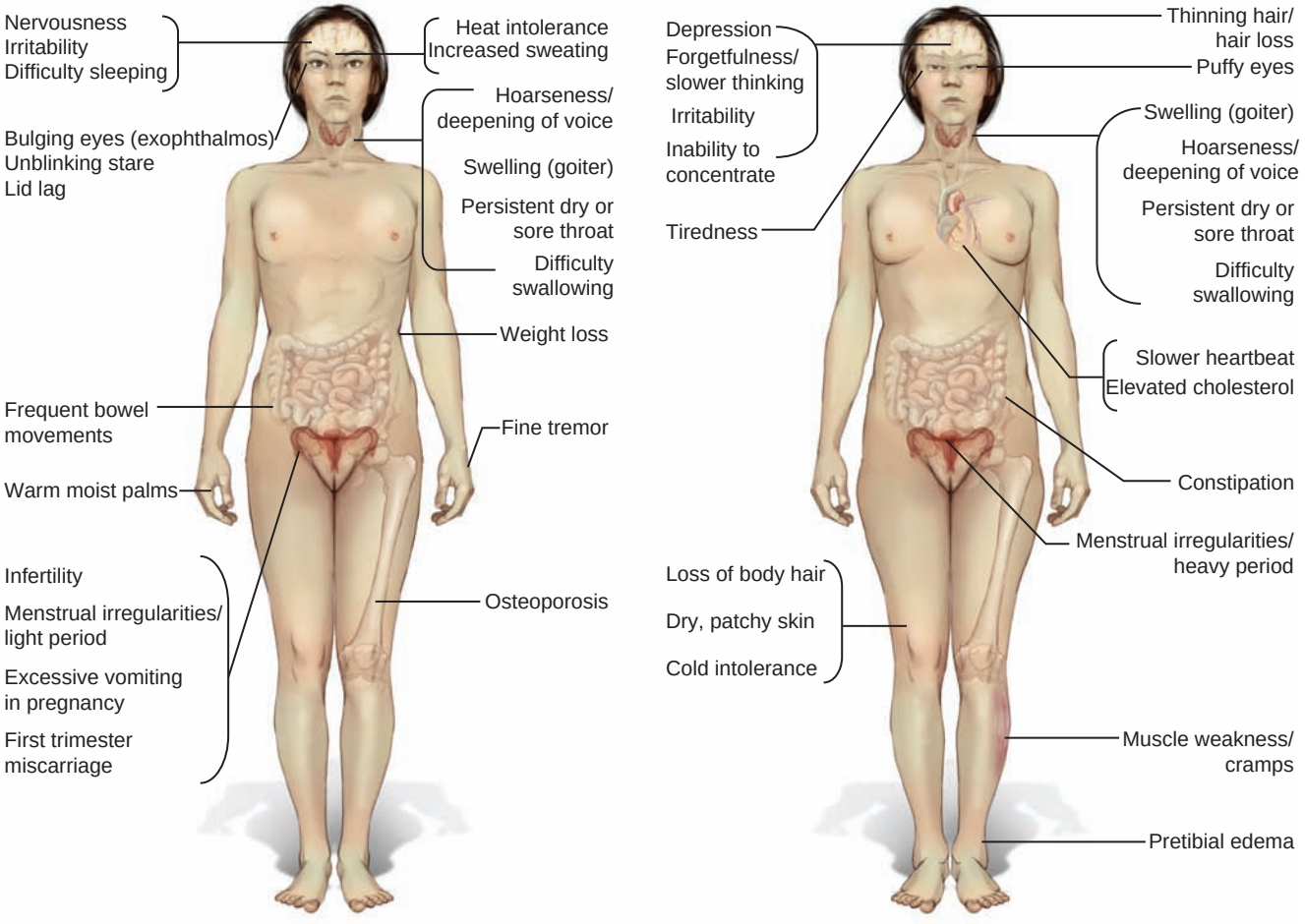


FIGURE 44-2 ▲ Clinical manifestations of hyperthyroidism **(A)** and hypothyroidism **(B)**.

BOX 44-1 Conditions Associated With Hyperthyroidism or Thyrotoxicosis

Endocrine Disorders
- Graves' disease
- Nodular goiter
- Toxic multinodular adenoma
- Radiation-induced thyroiditis
- Subacute thyroiditis

Drugs
- Iatrogenic thyroid replacement
- Accidental or purposeful ingestion of thyroid medication
- Contrast media dye
- Amiodarone
- β-Blockers

Tumors
- Metastatic thyroid cancer
- Hypophyseal tumors
- Hypothalamus tumor
- Hydatidiform mole

crisis. Rapid deterioration and death can occur if the condition is untreated. These patients must be admitted to the intensive care unit for supportive measures, antithyroid medications, steroids, and continuous nursing care. Consultation with an endocrinologist and cardiologist is essential. Even without preexisting coronary artery disease, untreated thyrotoxic crisis can cause angina pectoris and myocardial infarction, heart failure, cardiovascular collapse, coma, and death. The condition may develop spontaneously, but it occurs most frequently in people who have undiagnosed or partially treated severe hyperthyroidism.

By definition, hyperthyroidism is a condition in which the actions of the thyroid hormones result in greater-than-normal responses. Specific diseases that can cause hyperthyroidism include Graves' disease, exogenous administration of levothyroxine, thyroiditis, toxic nodular goiter, toxic multinodular goiter, and thyroid cancer. Certain drugs, such as contrast material for radiographic procedures or amiodarone (an antiarrhythmic drug), may precipitate the thyrotoxic state because of their high iodine content. The conditions associated with hyperthyroidism are summarized in Box 44-1.

Pathophysiology

The cause of thyrotoxic crisis, often referred to as thyroid storm, is poorly understood. Physiological mechanisms that are thought to induce thyrotoxic crises include the sudden release of large quantities of thyroid hormone, low tissue tolerance to triiodothyronine (T_3) and thyroxine (T_4), adrenergic hyperactivity, and excessive lipolysis and fatty acid production. The abrupt release of large quantities of thyroid hormone is thought to produce the hypermetabolic manifestations seen during thyrotoxic crises. The many different endocrine, reproductive, gastrointestinal, integumentary, and ocular manifestations are caused by increased circulating levels of thyroid hormone and by stimulation of the sympathetic nervous system.

Adrenergic hyperactivity is considered a possible link to thyrotoxic crisis. Although thyroid hormone and catecholamines potentiate each other, catecholamine levels during thyrotoxic crisis are usually within the normal range. It is uncertain whether the effects of hypersecretion of thyroid hormone or increased catecholamine levels cause heightened sensitivity and thyroid overfunction. Thyroid–catecholamine interactions result in an increased rate of chemical reactions, increased nutrient and oxygen consumption, increased heat production, alterations in fluid and electrolyte balance, and a catabolic state.

Another mechanism that may contribute to thyrotoxic crisis is excessive lipolysis and fatty acid production. With excessive lipolysis, increased fatty acids are oxidized and produce an overabundance of thermal energy that is difficult to dissipate through vasodilation.

Assessment

HISTORY AND PHYSICAL EXAMINATION. Accurate identification of the precipitating factor for thyrotoxic crisis allows for proper treatment to be initiated. Precipitating factors for people with recognized and unrecognized existing thyroid disease are listed in Box 44-2. Hyperthyroidism's most common form, Graves' disease, is an autoimmune condition caused by thyroid-stimulating immunoglobulins. It is not always apparent that the patient is suffering from

BOX 44-2 PATIENT SAFETY

Risk Factors for Development of Thyroid Crisis

In the Presence of a Known Preexisting Condition

Precipitating factors
- Infection
- Trauma
- Stress
- Coexistent medical illness (eg, myocardial infarction, pulmonary disease)
- Pregnancy
- Exposure to cold

Medications
- Chronic steroid therapy
- β-blockers
- Narcotics, anesthetics
- Alcohol, tricyclic antidepressants
- Glucocorticoid therapy
- Insulin therapy
- Thiazide diuretics
- Phenytoin
- Chemotherapy agents
- Nonsteroidal anti-inflammatory agents (NSAIDs)

In the Presence of an Unknown Preexisting Condition

Precipitating factors
- Pituitary tumors
- Radiation therapy of the head and neck
- Autoimmune disease
- Neurosurgical procedures
- Metastatic malignancies (eg, lung, breast)
- Surgery
- Long-term illness
- Shock
- Postpartum status
- Trauma

BOX 44-3 PATIENT SAFETY

Possible Indications of Thyroid Emergencies

Thyroid Storm	Myxedema Coma
Tachycardia	Bradycardia
Hyperthermia	Hypothermia
Tachypnea	Hypoventilation
Diaphoresis	
Hypercalcemia	Hyponatremia
Hyperglycemia	Hypoglycemia
Metabolic acidosis	Respiratory and metabolic acidosis
Diarrhea	
Cardiovascular collapse	Cardiovascular collapse
Cardiogenic shock	Decreased vascular tone
Hypovolemia	
Cardiac dysrhythmias	
Irritability	
Depressed level of consciousness (LOC)	Depressed LOC
Emotional lability	Seizures, coma
Psychosis	
Tremors, restlessness	Hyporeflexia
Weight loss	Weight gain

this particular disease. Therefore, subtle clues need to be explored, such as the patient's exposure to iodine, prior or current use of thyroid hormone, anterior neck pain, thyroid enlargement, exophthalmos (ie, protrusion of one or both eyes) or other eye symptoms, pregnancy, a history of goiter, and a family history of thyroid disease.

Signs and symptoms of hyperthyroidism affect all body systems and include sweating, heat intolerance, nervousness, tremors, palpitations, tachycardia, hyperkinesis, and increased bowel sounds. Extremes of these manifestations, specifically a temperature greater than 104°F (40°C) in the absence of an infection, tachycardia, and CNS dysfunction, may be present in hyperthyroidism. CNS abnormalities include agitation, restlessness, delirium, seizures, or coma. Signs of thyroid emergencies are listed in Box 44-3.

As discussed in Box 44-4, older patients may not have the classic signs and symptoms of thyrotoxic crisis, causing this condition to be overlooked. However, they frequently have suggestive signs and symptoms. In these circumstances, the nurse asks older patients if they have heart disease and what medications they take. This can be important in determining whether there is underlying thyroid disease because β-blocker medication may mask cardiovascular clues.

BOX 44-4 CONSIDERATIONS FOR THE OLDER PATIENT

Hyperthyroidism

Elderly patients with hyperthyroidism often present with atypical signs and symptoms of the disorder. Apathetic hyperthyroidism, as seen in the older patient, manifests with a single symptom such as depression, atrial fibrillation, heart failure, or muscle weakness. Thus, the elderly patient may present with palpitations, shortness of breath, tremor, and nervousness, but many other symptoms, as seen in younger patients, are masked. Much time can transpire until the patient deteriorates into full-blown thyroid storm.

LABORATORY STUDIES. Laboratory studies may show elevated total T_4, free T_3, and free T_4 levels. The thyroid-stimulating hormone (TSH) level is extremely low (usually <0.1 mcg/mL) in hyperthyroidism. TSH is suppressed because the levels of circulating hormones, T_3 and T_4, are also elevated. Recall that TSH is secreted when thyroid hormone levels are low.

Serum electrolytes, liver function tests, and complete blood counts, although not diagnostic, may help uncover abnormalities that require treatment. They may also help identify the precipitating cause. Electrolyte imbalances from dehydration, excessive bone resorption, and increased insulin degradation often occur. The serum calcium level is often elevated, whereas potassium and magnesium levels are decreased, and liver function test values are increased. Hyperglycemia resulting from insulin resistance and breakdown of stored glucose often occurs.

DIAGNOSTIC STUDIES. Diagnostic tests include the radioactive iodine uptake test, whose result is usually increased. Electrocardiography (ECG) and cardiac monitoring may show atrial fibrillation, supraventricular tachycardia, sinus bradycardia, heart block, conduction disturbances, and ventricular dysrhythmias, all reflective of the hypermetabolic state and the synergized catecholamines.

Management

Management goals for thyrotoxic crises are fourfold: (1) treating the precipitating factor or factors, (2) controlling excessive thyroid hormone release, (3) inhibiting the thyroid hormone biosynthesis, and (4) treating the peripheral effects of thyroid hormone.[1]

Antithyroid drugs (Table 44-2) are used to control thyroid release or biosynthesis. Propylthiouracil (PTU) is the preferred agent during pregnancy, although it can only be given orally. PTU is preferred because it blocks the conversion of T_4 to T_3 in peripheral tissues and binds iodine to prevent synthesis of the hormone. If the oral route is not possible, methimazole can be given rectally. PTU has been associated with severe hepatic side effects and close monitoring is warranted to avoid irreversible damage.

Iodine solutions, such as sodium iodide IV or potassium iodide (SSKI) or Lugol's solution orally, are given to block the release of thyroid hormone. These agents should not be given until 1 hour after the administration of antithyroid medications. Lithium is the choice for patients who are iodine sensitive. Glucocorticoids may be ordered because they also inhibit thyroid hormone release.

Emergency removal of excess circulating hormone replacement therapy can be accomplished by instituting plasmapheresis, dialysis, or hemoperfusion adsorption. Cholestyramine may be used to assist with oral absorption of excess hormone.

Blocking the catecholamine effects that may result in cardiovascular decompensation secondary to decreased stroke volume and reduced cardiac output may be instituted. β-Blockers, specifically propranolol, are used to treat the symptoms of the hyperthyroidism rather than the primary thyroid disease. This therapy may be ordered to restore cardiac function by decreasing the catecholamine-mediated symptoms. The response to β-blockers is carefully monitored because intrinsic cardiac disease may worsen as a result of the negative inotropic effects.[2] Digoxin, diltiazem (Cardizem),

Table 44-2 Drugs Used to Treat Hyperthyroidism

Drug	Dose	Action	Nursing Considerations
Propylthiouracil (PTU)	Loading: 800–1,200 mg Maintenance: 100–400 mg every 4–6 h orally (PO)	Blocks synthesis of hormones (conversion of T_3 to T_4)	Monitor cardiac parameters. Observe for conversion to hypothyroidism. Must be given by mouth. Watch for rash, nausea, vomiting, agranulocytosis, lupus syndrome, hepatitis.
Methimazole	10–20 mg every 6–8 h PO	Blocks synthesis of thyroid hormone	More toxic than PTU. Watch for rash and other symptoms as for PTU.
Sodium iodide	1 g every 12 h (intravenous [IV])	Suppresses release of thyroid hormone	Given 1 h after PTU or methimazole. Watch for edema, hemorrhage, gastrointestinal upset.
Potassium iodide	2–5 gtts every 8 h PO	Suppresses release of thyroid hormone	Discontinue for rash. Watch for signs of toxic iodinism.
SSKI	5–10 gtts every 8 h PO	Suppresses release of thyroid hormone	Mix with juice or milk. Give by straw to prevent staining of teeth.
Dexamethasone	2 mg every 6 h IV	Suppresses thyroid hormone release	Monitor intake and output. Monitor glucose. May cause hypertension, nausea, vomiting, anorexia, infection.
β-Blocker (eg, propranolol)	1–3 mg every 1–4 h IV	β-Adrenergic blocking agent	Monitor cardiac status. Hold for bradycardia or decreased cardiac output. Use with caution in patients with heart failure.

diuretics, or a combination of these agents may also be used to treat congestive heart failure or supraventricular tachydysrhythmias. Oxygen is delivered to address the additional metabolic requirements. The goal of therapy is to decrease myocardial oxygen consumption, decrease the heart rate (ideally to below 100 beats/min), and increase cardiac output.

Corticosteroids may be used to help treat coexisting adrenal insufficiency and thyroid storm. Intravenous (IV) dexamethasone or hydrocortisone can be given to assist with blunting excess thyroid hormone release in this emergency state.

Management also focuses on monitoring multisystem effects from the hypermetabolism of thyrotoxic crisis and the response to treatment. Cardiovascular function, fluid and electrolyte balance, and neurological status require close attention. It is necessary to assess blood pressure, heart rate and rhythm, respiratory rate, and extra heart sounds every hour.

The nurse evaluates fluid status and laboratory values. Hourly monitoring of body temperature is warranted because the patient is at risk for hyperthermia. Antipyretic agents, particularly acetaminophen, are recommended for fever control; aspirin is not appropriate because it increases free T_3 and T_4 levels. Tepid baths or a cooling blanket may be necessary. It is important to avoid cooling to the point of shivering and piloerection because this may have a rebound effect of raising body temperature. IV fluids are necessary to replace the fluids lost from excessive hyperthermia, tachypnea, diaphoresis, and diarrhea that often accompany thyrotoxic crisis.

The nurse assesses neurological status at least hourly. Seizure precautions and safety measures prevent injury. If the patient's level of consciousness (LOC) decreases, it is important to assess airway patency and safety issues. Maintaining a calm environment is necessary to help manage the extreme agitation and restlessness seen in the patient with thyrotoxic crisis.

Energy and nutritional needs are heightened because of the hypermetabolism. Interventions include administering glucose-containing solutions, nutritional support, vitamin supplementation, and sedation if needed. The nurse monitors the patient's glycemic status because the administration of corticosteroids and excess glucose-rich nutrients may lead to hyperglycemia in some patients. Box 44-5 lists examples of nursing diagnoses for the patient in thyrotoxic crisis.

Effective therapy can be expected to result in clinical improvement within 24 to 48 hours. The nurse monitors the patient's mental status carefully and also checks for stabilization of vital signs and normalization of body temperature. Patient follow-up to prevent another episode is

BOX 44-5 EXAMPLES OF NURSING DIAGNOSES

For the Patient in Thyroid Crisis

- Deficient Fluid Volume related to hypermetabolic state
- Hyperthermia related to hypermetabolic state
- Decreased Cardiac Output related to hypermetabolic state and heart failure
- Risk for Ineffective Cerebral Tissue Perfusion
- Risk for Injury related to altered mental status

necessary and may involve lifelong medication or suppressive therapy with thyroid ablation.

Myxedema Coma

Hypothyroidism is a common disorder with a broad clinical spectrum—patients may be asymptomatic, or they may be severely ill with myxedema coma. Hypothyroidism is more common among women, and the incidence increases with age. Approximately 10% to 15% of elderly patients have elevated TSH associated with hypothyroidism, and routine screening of high-risk populations is often done in primary care settings.[3]

Myxedema coma is a rare, life-threatening emergency brought on by extreme hypothyroidism. It usually is seen in older patients during winter months after certain precipitating factors, such as stress, exposure to extreme cold temperatures, or trauma. In addition to coma, complications of myxedema coma include pericardial and pleural effusions, megacolon with paralytic ileus, and seizures. Death can result if severe hypoxia and hypercapnia are not reversed.

Pathophysiology

Deficient production of thyroid hormone results in the clinical state termed hypothyroidism. Hypothyroidism, a chronic disease, is 10 times more common in women than in men. It occurs in all age groups but most commonly in those older than 50 years. It is more common than hyperthyroidism.

Hypothyroidism can be primary or secondary. Primary causes include congenital defects, loss of thyroid tissue after treatment for hyperthyroidism, defective hormone synthesis from an autoimmune process, and antithyroid drug administration or iodine deficiency. Secondary causes include peripheral resistance to thyroid hormone, pituitary infarction, and hypothalamic disorders. Transient hypothyroidism can occur after withdrawal of prolonged T_4 or T_3 treatment. The common causes of hypothyroidism are summarized in Box 44-6.

Hypothyroidism usually affects all body systems. A low basal metabolic rate and decreased energy metabolism and heat production are characteristic. The patient with chronic hypothyroidism may have myxedema, an alteration in the composition of the dermis and other tissues. The connective fibers are separated by an increased amount of protein and mucopolysaccharides. This binds water, producing nonpitting, boggy edema, especially around the eyes, hands, and feet; it is also responsible for thickening of the tongue and the laryngeal and pharyngeal mucous membranes, resulting in slurred speech and hoarseness.

BOX 44-6 Causes of Hypothyroidism

- Destruction of the thyroid gland (eg, surgery, radioactive iodine, external radiation to the neck)
- Infiltrative disease (eg, sarcoidosis, amyloidosis, lymphoma)
- Autoimmune disease (eg, Hashimoto's disease, post-Graves' disease)
- Thyroiditis (eg, viral, silent, postpartum)
- Drug induced (eg, iodides, lithium, amiodarone)
- Hereditary hypothyroidism
- Thyrotropin-releasing hormone deficiency
- Thyroid-stimulating hormone deficiency

Assessment

HISTORY AND PHYSICAL EXAMINATION. Signs and symptoms of hypothyroidism include fatigue, weakness, decreased bowel sounds, decreased appetite, weight gain, and ECG changes. Myxedema coma is a rare manifestation of hypothyroidism, characterized by severe depression of the sensorium, hypothermia, hypoventilation, hypoxemia, hyponatremia, hypoglycemia, hyporeflexia, hypotension, and bradycardia. Patients with myxedema coma do not shiver, although body temperatures below 80°F (26.6°C) have been reported. The diagnosis of myxedema coma depends on recognizing the clinical symptoms and identifying the underlying precipitating factor. The most common precipitating factor is pulmonary infection; other factors include trauma, stress, infections, drugs (eg, narcotics or barbiturates), surgery, and metabolic disturbances (see Box 44-3).

LABORATORY STUDIES. A decrease in T_4 and free T_4 levels is most common, whereas sodium is usually decreased, and potassium is increased. TSH is markedly elevated in severe hypothyroidism. Arterial blood gas (ABG) findings usually show a severe hypercapnia with decreased arterial oxygen tension (PaO_2) and increased arterial carbon dioxide tension ($PaCO_2$).

DIAGNOSTIC STUDIES. A chest radiograph detects pleural effusion. ECG changes include bradycardia, a prolonged PR interval, and decreased amplitude of the P wave and QRS complex. Heart block may develop.

Management

The most serious complication of hypothyroidism is progression to myxedema coma and death, if the condition is untreated. A multisystem approach must be used in treating this emergency. Mechanical ventilation is used to control hypoventilation, hypercapnia, and respiratory arrest. IV hypertonic normal saline and glucose solutions correct the dilutional hyponatremia and hypoglycemia. Fluid administration plus vasopressor therapy may be necessary to correct hypotension.

Pharmacological therapy includes the administration of thyroid hormone and corticosteroids. There are several approaches to this aspect of medical management. Initial drug therapy includes 300 to 500 mcg T_4 IV to saturate all protein-binding sites and establish a relatively normal T_4 level. Subsequent doses may include 75 to 100 mcg daily. IV or oral T_3 is an alternative order. Guidelines for T_3 replacement are 25 mcg IV every 8 hours for the first 24 to 48 hours. Oral T_3 doses every 8 hours are also ordered. Hormone replacement should occur slowly, with continuous monitoring of the patient during treatment to avoid sudden increased metabolic demand and resultant myocardial infarction. Methodical fluid replacement and rewarming of the patient also help to avoid complications.

Additional interventions include treating abdominal distention and fecal impaction and managing hypothermia by gradually rewarming the patient using blankets and socks. Mechanical devices are not used. The nurse monitors

the patient for neurological status and changes in LOC and implements seizure precautions. Care of the comatose patient includes preventing complications related to aspiration, immobility, skin breakdown, and infection. Monitoring of cardiovascular and respiratory function is necessary. Fluid administration must also be monitored because of a risk for fluid overload. An important aspect of care is to detect early signs of complications. As the patient recovers, interventions focus on patient self-care and education.

Patient follow-up includes a thorough investigation of how the severe hypothyroidism occurred and how it can best be avoided in the future. Patient teaching, family follow-up, medical alert activation, and involvement of community supports may be necessary for this complex patient.

▲ Adrenal Gland Dysfunction

Adrenal Crisis

Pathophysiology

Adrenal insufficiency, also known as hypoadrenalism or hypocorticism, is a rare but life-threatening dysfunction of the adrenal cortex. Adrenal hormone insufficiency may be either primary (ie, directly involving the adrenal gland) or secondary (ie, due to hypothalamic–pituitary disease).

Primary adrenal insufficiency is termed Addison's disease. The most common cause of primary hypoadrenalism in the industrialized West is autoimmune adrenalitis. Autoimmune antibody formation leads to the gradual destruction of the adrenal gland, resulting in adrenal insufficiency. The second leading cause of primary adrenal insufficiency is destruction of the gland secondary to *Mycobacterium tuberculosis* infection. Worldwide, tuberculosis remains the most common cause of primary adrenal insufficiency. Other causes include bilateral hemorrhage of the glands secondary to bacterial infection with sepsis and shock, metastatic malignancies, acquired immunodeficiency syndrome (AIDS), fungal infections, surgical adrenalectomy, and sarcoidosis.

The most common cause of secondary adrenal insufficiency is iatrogenic, resulting from abrupt withdrawal of exogenous ACTH or as a complication of cortisol therapy. Suppressed ACTH secretion as a result of exogenous cortisol therapy disrupts the body's natural feedback loop that controls cortisol secretion, rendering the patient in an acute state of adrenal insufficiency. Other causes of secondary adrenal insufficiency include metastatic carcinomas of the lung or breast, pituitary infarction, surgery or irradiation, and CNS disturbances, such as basilar skull fractures or infections.

Acute adrenal insufficiency or adrenal crisis occurs when there is a change in the chronic condition or massive adrenal hemorrhage. In addition to the chronic disease, severe infection, septic shock, trauma, surgical procedure, or some extra stress occurs, precipitating acute adrenal crisis in the patient. The patient is therefore unable to meet the requirements for normal metabolic function or increased metabolic needs as necessary for stress or illness. Any stressed, critically ill patient can develop adrenal insufficiency as a result of suddenly imposed extraneous stressors. As the patient struggles to survive, he or she quickly depletes cortisol stores and may require exogenous replacement.[4]

Assessment

HISTORY AND PHYSICAL EXAMINATION. Symptoms of adrenal insufficiency are the same for primary and secondary disease. Because adrenal insufficiency affects both glucocorticoids and mineralocorticoids, many body functions are affected, including glucose metabolism, fluid and electrolyte balance, cognitive state, and cardiopulmonary status. Weakness, fatigue, anorexia, nausea, vomiting, diarrhea, and abdominal pain may be initial clues to adrenal crisis. These findings are nonspecific until linked with the history of a chronic condition requiring past or present corticosteroid use.[5] Specifically, use of more than 20 mg of hydrocortisone or its equivalent, taken for longer than 7 to 10 days, has the potential for suppressing the HPA axis.

Hyperpigmentation on areas of the elbows, knees, hands, or buccal mucosa is seen in primary adrenal insufficiency. The presence of hyperpigmentation, secondary to the deposition of melanin in the skin, strengthens the clinical picture of adrenal crisis. The most common physical changes include signs of severe dehydration, such as weight loss and orthostatic hypotension. Dehydration occurs secondary to the nephrons' insufficient ability to reabsorb sodium and water. Signs and symptoms of an impending adrenal crisis are summarized in Box 44-7.

LABORATORY STUDIES. Laboratory values in acute conditions of glucocorticoid and mineralocorticoid deficiency show hyponatremia, hyperkalemia, decreased serum bicarbonate levels, and elevated blood urea nitrogen (BUN). Metabolic acidosis may occur because of dehydration. Hypoglycemia is usually present. Other abnormal laboratory findings include anemia and lymphocytosis with eosinophilia. In primary adrenal insufficiency, the patient presents with chronically elevated ACTH levels. ACTH levels are normal or decreased in the patient with secondary adrenal insufficiency.

BOX 44-7 PATIENT SAFETY

Indications of Impending Adrenal Crisis

Aldosterone Deficiency
- Hyperkalemia
- Hyponatremia
- Hypovolemia
- Elevated (blood urea nitrogen [BUN])

Cortisol Deficiency
- Hypoglycemia
- Decreased gastric motility
- Decreased vascular tone
- Hypercalcemia

Generalized Signs and Symptoms
- Anorexia
- Nausea and vomiting
- Abdominal cramping
- Diarrhea
- Tachycardia
- Orthostatic hypotension
- Headache, lethargy
- Fatigue, weakness
- Hyperkalemic electrocardiographic changes
- Hyperpigmentation

Serum cortisol levels and cortisol stimulation (ACTH stimulation) tests are also used to confirm the diagnosis. Cortisol levels below 15 mcg/dL are indicative of adrenal dysfunction. In primary adrenal insufficiency, repeated injections of ACTH (or Cortrosyn) do not cause a rise in cortisol levels because the adrenal gland is dysfunctional. In secondary adrenal insufficiency, ACTH injections cause a normal but delayed response.

DIAGNOSTIC STUDIES. A computed tomography (CT) scan of the adrenal glands and the head may be done to detect tumors or other pathology of the adrenal and pituitary gland.

Management

The immediate goal of therapy is to administer the needed hormones and restore fluid and electrolyte balance. Hydrocortisone, 100 mg IV, is administered immediately, followed by 100 mg every 6 to 8 hours. Fluid resuscitation is also started immediately with normal saline and 5% dextrose solutions. The rate of fluid and electrolyte replacement is dictated by the degree of volume depletion, serum electrolyte levels, and clinical response to therapy. Associated medical or surgical problems may indicate the need for invasive blood pressure and hemodynamic monitoring.

Another management goal is to prevent complications. This includes monitoring signs and symptoms of electrolyte imbalance (hyponatremia and hypercalcemia) and respiratory and cardiovascular function. The nurse looks for changes in blood pressure, heart rate and rhythm, skin color and temperature, capillary refill time, and central venous pressure (CVP). There is a risk for orthostatic hypotension, bradycardia, and dysrhythmias. The nurse also monitors neuromuscular signs, such as weakness, twitching, hyperreflexia, and paresthesia.

Emotional support, a simple explanation, and a quiet environment are effective in assisting the patient emotionally through the physiological crisis. Once the acute crisis is over, patient education is a goal of care. Patient education is necessary because the ultimate prognosis depends on the patient's ability to understand and follow through with self-care. Self-care includes knowing the medication regimen, stress factors and their effect on the disease, and the signs of impending crisis; wearing a medical identification tag or bracelet, or carrying a wallet card; and taking medication as prescribed.

Pheochromocytoma

Pheochromocytoma is a rare catecholamine-secreting tumor that arises from chromaffin cells in the adrenal gland. Because of the excessive catecholamine secretion, pheochromocytoma may precipitate life-threatening hypertension or cardiac dysrhythmias when norepinephrine or epinephrine is released in larger quantities. The trigger for the release of catecholamines is unknown, but the high levels can lead to severe hypertension, atrial fibrillation, ventricular fibrillation, myocardial infarction, or cerebral infarction.

Pheochromocytomas can occur in people of all ages and ethnicities, and the peak incidence is between the third and fifth decades of life. The classic symptomatic triad includes headaches, palpitation, and sweating. When associated with severe, paroxysmal hypertension, the triad is found to be over 90% sensitive and specific for pheochromocytoma. Typically the symptoms worsen with time and become more severe as the tumor grows.

Diagnosis is based on the suspicion of pheochromocytoma for the patient who presents with paroxysmal hypertension and other associated symptoms. Choice laboratory studies include measurement of fractionated plasma and urine metanephrines and normetanephrines. Vanillylmandelic acid is rarely measured because of its limited ability to detect a true positive result (low sensitivity). Diagnosis is confirmed with imaging studies such as abdominal magnetic resonance imaging or CT. Medical care includes surgical resection of the tumor and careful control of the hypertension. Medications that are required preoperatively to control blood pressure and to prevent hypertensive crisis include α-blockers and β-blockers. Usually, the hypertension is no longer a problem postoperatively.

▲ Antidiuretic Hormone Dysfunction

Two disorders involve ADH dysfunction. One, syndrome of inappropriate antidiuretic hormone secretion (SIADH), is an excess of ADH. The second, diabetes insipidus, involves a deficiency of ADH. Both of these disorders can produce severe fluid and electrolyte imbalances and adverse neurological changes. Recall that ADH is synthesized in the hypothalamus and stored in the posterior pituitary. It is released when stimulated by specific conditions and causes the renal tubules to reabsorb more water and sodium.

Syndrome of Inappropriate Antidiuretic Hormone Secretion

Pathophysiology

In SIADH, there may be either increased secretion or increased production of ADH. The increase in ADH occurs despite normal initial osmolality. As a result, increased ADH production causes an increase in total body water. SIADH is considered whenever the patient experiences hypotonic hyponatremia with elevated urine osmolality, the hallmark of the disorder. In SIADH, no edema or hypovolemia is associated with the hyponatremia.

The secretion of ADH is considered "inappropriate" in that it continues despite the decreased osmolality of the plasma. The normal feedback system regulating the release and inhibition of ADH fails, and ADH secretion continues. The circulating ADH acts on the renal tubules, causing reabsorption of water that is inconsistent with the body's needs. Other reasons for the continued secretion of ADH are also lacking. There is no hypokalemia and edema; cardiac, renal, and adrenal function are normal; and there is normal or expanded plasma and extracellular fluid (ECF) volumes.

Occasionally, the cause of SIADH is a pituitary tumor, but more commonly it occurs as the result of a bronchogenic (oat cell) or pancreatic carcinoma. These tumors actually secrete ADH but are independent of normal physiological controls. Other possible causes of SIADH include head injuries; other endocrine disorders; pulmonary diseases, such as pneumonia and lung abscesses; CNS infections or tumors; and drugs. Box 44-8 outlines the most common causes of SIADH.

BOX 44-8 Common Causes of Syndrome of Inappropriate Antidiuretic Hormone Secretion

Malignancies
- Bronchogenic carcinoma
- Pancreatic adenocarcinoma
- Prostate or thymus cancer
- Leukemia

Central Nervous System Causes
- Head injury
- Hemorrhage (subdural hematoma, subarachnoid hemorrhage)
- Brain abscess
- Infection, abscess, meningitis
- Hydrocephalus

Pulmonary Causes
- Mechanical ventilation
- Chronic obstructive pulmonary disease
- Respiratory failure
- Lung abscess, infection, pneumonia

Medications
- Nicotine
- Opiates, morphine
- Chlorpropamide, hypoglycemics, insulin
- Antineoplastics
- Tricyclic antidepressants, SSRIs
- Anesthetics
- Clofibrate
- Diuretics

Other Causes
- Human immunodeficiency virus, acquired immunodeficiency syndrome
- Senile atrophy
- Pain
- Fear
- Myocardial infraction
- Idiopathic

Assessment

HISTORY AND PHYSICAL EXAMINATION. SIADH is characterized by water retention and eventually water intoxication secondary to sustained ADH effect. The hyponatremia in SIADH has two components, an early dilutional component caused by increased intravascular water, and a later, clinically undetectable component, caused by the increased urinary sodium excretion.

The signs and symptoms produced by SIADH are predominantly neurological and gastrointestinal. The most common signs and symptoms are personality changes, headache, decreased mentation, lethargy, abdominal cramps, nausea, vomiting, diarrhea, anorexia, decreased tendon reflexes, disorientation, confusion, and finally, seizures and coma. Many patients remain asymptomatic until the sodium level drops well below 125 mEq/L. Subtle neurological findings such as altered mental state, slight confusion, anorexia, inability to concentrate, and complaints of weakness may be the earliest indication of impending problems.

Hyponatremia is the clinical focus and probable cause of hospital admission. When the serum sodium falls to less than 120 to 125 mEq/L, more pronounced symptoms associated with cerebral edema, such as headache, nausea and vomiting, restlessness, muscular irritability, and seizures often result. When the condition develops acutely (ie, within 24 hours), a mortality rate of 50% has been reported. Children and the elderly are more susceptible to hyponatremia because of their lower body water content.

Physical evidence of hyponatremia includes dyspnea, jugular venous distention, restlessness, hypothermia, weight gain, mild to no edema, reduced and concentrated urine, anorexia, abdominal cramps, and disorientation. Often, the nurse is the first to identify these early, subtle signs.

LABORATORY STUDIES. The main laboratory abnormalities in SIADH are a plasma hyponatremia and hypoosmolality. The urine simultaneously is hyperosmolar, and there is a high excretion of urinary sodium. The urine specific gravity is high (concentrated), usually greater than 1.025, and the overall urinary output is lower (<30 mL/h). Other laboratory findings include low BUN, creatinine, and uric acid levels; hypocalcemia and hypokalemia; and decreased hemoglobin and hematocrit values. The diagnosis can be confirmed by radioimmunoassay of plasma ADH, which is inappropriately elevated relative to plasma osmolality. Table 44-3 presents a comparison of laboratory values in SIADH and diabetes insipidus.

Management

There are three goals in SIADH management: (1) treating the underlying disease, (2) alleviating excessive water retention, and (3) providing the comprehensive care needed when the patient has a depressed LOC.

Table 44-3 Laboratory Values: Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) and Diabetes Insipidus

Value	Normal	SIADH	Diabetes Insipidus
Serum ADH	1–5 pg/mL	Increased	Decreased
Serum osmolality	285–300 mOsm/kg	<285 mOsm/kg	>300 mOsm/kg
Serum sodium	133–145 mEq/L	<33 mEq/L	>145 mEq/L
Urine osmolality	300–1,400 mOsm/kg	>300 mOsm/kg	<300 mOsm/kg
Urine specific gravity	1.005–1.030	>1.030	<1.005
Urine output	1.0–1.5 L/24 h	Below normal	30–40 L/24 h
Fluid intake	1.0–1.5 L/24 h	Goal: <600–800 mL/24 h (restricted fluid intake)	>50 L/24 h

Treatment of the underlying cause of SIADH may or may not be possible, depending on the pathological process. Surgical resection, radiation, or chemotherapy may alleviate some of the water retention caused by some cancers. No drug completely inhibits the release of ADH from the pituitary gland or a tumor. When the cause of the SIADH is unknown, the treatment consists of fluid restriction.

The first step in managing SIADH is to restrict fluid intake. In mild cases, fluid restriction is sufficient. It slows renal blood flow and glomerular filtration, enhancing proximal tubular reabsorption of salt and water; increases aldosterone secretion; and enhances distal tubule sodium reabsorption. As a general guideline, water intake should not exceed urinary output until the serum sodium concentration normalizes and symptoms abate. Fluid restriction is usually successful for the patient with sodium levels between 125 and 135 mEq/L.

In severely symptomatic patients with acute hyponatremia, administration of 3% hypertonic saline solution and furosemide is used to correct hyponatremia in an emergency situation. Infusion of hypertonic saline solution at 0.1 mg/kg/min prevents rapid volume overload and pulmonary edema. Usually, 300 to 500 mL given IV over 4 to 6 hours is appropriate.

One major complication to avoid is central pontine myelinolysis. This may occur when correction of hyponatremia by hypertonic saline infusion is too rapid. Rapid correction of hyponatremia may lead to brain dehydration, cerebral bleeding, demyelination, neurological injury, or death. Initial signs and symptoms include seizures, movement disorders, akinetic mutism, quadriparesis, and unresponsiveness. The best plan is to replace sodium at a rate no faster than 1 to 2 mEq/L/h to avoid the syndrome.

Other medications effectively interfere with the ADH–renal tubule interaction. Conivaptan (Vaprisol) is an inhibitor of ADH that can be administered IV for hospitalized patients with euvolemic hyponatremia. It blocks vasopressin receptors in the renal collecting ducts to decrease water reabsorption. Patients receive an initial IV infusion of 20 mg over 30 minutes followed by an infusion of 20 to 40 mg/d for 4 days. Demeclocycline, an antibiotic, has been effective because it interferes with the normal ADH effect in kidney tubules. The usual dosage is 600 to 1,200 mg/d for 3 to 4 days. Other medications that block the effects of ADH at the tubules include phenytoin (Dilantin), lithium, and fludrocortisone (Florinef).

The nurse monitors fluid and electrolyte balance, especially the serum sodium level, In addition, it is necessary to evaluate intake and output, including hourly urine amounts, and observe for signs of fluid overload. Output should exceed intake. Adequate intake of dietary protein and salt need to be encouraged.

The nurse evaluates the patient's neurological status. Rapid changes in sodium levels can result in neurological deterioration. When the serum sodium is less than 125 mEq/L, there is a significant risk for neurological symptoms, including disorientation and decreasing consciousness. Seizure precautions may be necessary. Complications of SIADH include neurological deterioration leading to seizures, coma, and death.

Patients may find it difficult to limit their fluid intake. Mealtimes may also be difficult because menus are aimed at meeting nutritional needs without increasing fluid intake. Providing good oral care and offering substitutions for fluids (eg, Toothettes, lemon-glycerin swabs) may be helpful for the persistently thirsty patient. Providing information and emotional support and acknowledging the deprivation may help patients through this period.

Diabetes Insipidus

Pathophysiology

Diabetes insipidus is a disease characterized by water imbalance resulting from inadequate ADH or resistance to ADH, leading to water diuresis and dehydration. Polyuria is the hallmark of the disorder, and the kidneys can excrete great quantities of dilute urine, at times up to 20 L/d. Normally, the posterior pituitary releases ADH, which then acts on the distal renal tubules to promote reabsorption of water. When there is an absence of or deficit in ADH, the kidneys lose their ability to reabsorb water and control fluid output (see Chapter 42).

Diabetes insipidus may manifest in two forms: central or nephrogenic. Central diabetes insipidus is the more common condition that results in ADH deficiency and responds favorably to exogenous vasopressin administration. This type of diabetes insipidus is the disease most often encountered in the critical care environment. Nephrogenic diabetes insipidus is a rare genetic disorder that results from the failure of the kidney to respond to ADH. Only central diabetes insipidus is discussed in this chapter.

Diabetes insipidus can be transient, temporary, partial, or permanent, depending on the initial cause and circumstances surrounding the patient illness or injury. The osmolality sensors of the hypothalamus control ADH release from the posterior pituitary. As the osmolality increases, the osmoreceptors are stimulated, releasing more ADH. In the kidneys, ADH causes more water and sodium to be absorbed, restoring adequate fluid balance. In the absence of ADH, the renal tubules and collecting ducts are impermeable to water. Consequently, large volumes of dilute urine are excreted. Serum osmolality and sodium rise, and the patient continues to become progressively more dehydrated. The thirst sensation may or may not be affected, depending on the patient's LOC. For patients with an impaired thirst mechanism, dehydration and hypovolemic shock will result more quickly if the condition is not corrected.

Diabetes insipidus can develop after any event that causes edema or direct damage to the neurohypophysis. After surgery, diabetes insipidus may occur when regions of the brain around the hypothalamus and pituitary are affected. It can occur after head injuries, gunshot wounds, and lesions that disrupt blood supply to the area. Damage to the sphenoid bone, maxillofacial injuries, hypothalamic tumors, and nasopharyngeal tumors that invade the base of the skull may also lead to the development of diabetes insipidus. Direct trauma or ischemic events involving the hypothalamus, such as hemorrhage, infection, or neoplasm, may result in diabetes insipidus. Also, diseases or drugs that affect the renal collecting tubules may lead to diabetes insipidus. There is also a psychogenic polydipsia, in which excessive water is consumed, resulting in excess output.

After trauma or surgery, diabetes insipidus can be transient until initial edema subsides. The neurohypophysis is very sensitive to extraneous pressure; consequently, these

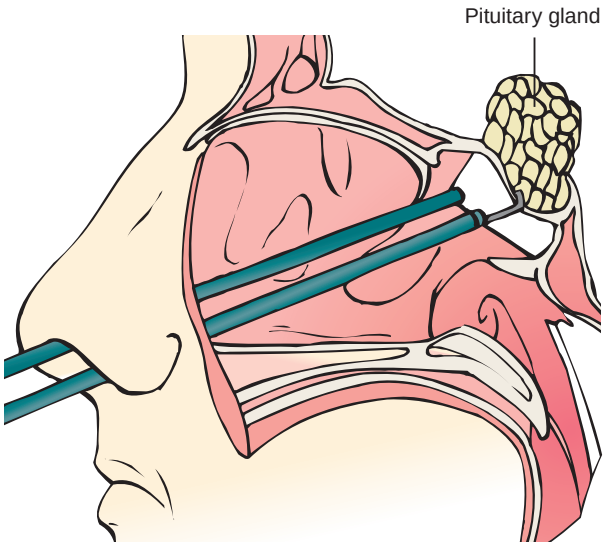


FIGURE 44-3 ▲ The transsphenoidal approach to a pituitary tumor can lead to transient diabetes insipidus. The neurohypophysis is very sensitive to extraneous pressure; consequently, these structures may be unable to produce, secrete, or release antidiuretic hormone as needed. The resultant diabetes insipidus resolves as the edema (from the surgery) resolves.

structures may be unable to produce, secrete, or release ADH as needed. The patient displays temporary signs of diabetes insipidus. As the edema abates, ADH secretion resumes its normal course, and the diabetes insipidus eventually is corrected. In some cases of severe trauma or hemorrhage, the structures may be completely damaged, and the patient may permanently develop diabetes insipidus.

The classic example of the transient type of disorder is illustrated by the patient undergoing a transsphenoidal approach for a hypophysectomy to remove a pituitary tumor. In most cases, the patient experiences temporary problems related to an inability to synthesize, store, or release ADH due to edema of the hypothalamus and pituitary. This patient requires close monitoring for the development of diabetes insipidus and may need treatment. Figure 44-3 illustrates a transsphenoidal approach to a pituitary tumor.

Assessment

Polyuria, polydipsia, and dehydration are the hallmarks of diabetes insipidus. Patients can excrete from 3 to 20 L of urine/d. When patients are alert, they experience excessive thirst and excessive urinary output. They try to increase their fluid intake, but this can cause exhaustion and eventually result in dehydration. On the other hand, when people are not alert enough to detect thirst and increase their fluid intake, they can quickly become hypovolemic because of the fluid loss. If left untreated, this can lead to death.

Signs of dehydration include dry skin, dry mucous membranes, confusion, sunken eyeballs, constipation, poor skin turgor, lethargy, muscle weakness, muscle pain, and pallor. Vital signs are adversely affected, with severe tachycardia, hypotension, low CVP, and a possible rise in body temperature. Weight loss may be apparent.

Recognizing diabetes insipidus may be more difficult when patients are recovering from surgery because steroids and cerebral dehydrating agents used before and during surgery promote diuresis for the first postoperative day or so. If awake, the patient complains of progressive thirst if diabetes insipidus is present. Urine output increases and persists regardless of the amount of fluid intake. Urine specific gravity falls or remains at about 1.001 to 1.005. Urine is copious, clear, and almost colorless. Plasma osmolality increases, often to levels greater than 300 mOsm/kg. Urine osmolality decreases to 50 to 100 mOsm/kg. The urine sodium concentration is below normal, whereas the serum sodium concentration is elevated (see Chapter 43, Table 43-2, p. 975). The water deprivation test is also helpful to diagnose diabetes insipidus. These tests, combined with the constellation of signs and symptoms, lead to the diagnosis. Table 44-3 on page 997 presents laboratory values for patients with diabetes insipidus.

Management

The objective of therapy is to prevent dehydration and electrolyte imbalance, while treating the underlying cause and preventing complications. Hypotonic IV solutions, such as 0.45% sodium chloride solution, are administered to match the urine output. The volume of replacement fluids depends on the degree of dehydration and the amount needed to reverse hypovolemic shock.

A variety of replacement ADH (vasopressin) therapies are available. Desmopressin acetate is synthetic ADH that can be administered IV, orally, or as a nasal spray. Aqueous vasopressin (Pitressin) may be given as an IV bolus, continuous infusion, or subcutaneously. The medications can be used for temporary or permanent ADH replacement. Permanent hormone replacement requires more patient and family education. In addition, the patient should obtain medical identification to carry at all times. Table 44-4 reviews the commonly administered medications for diabetes insipidus.

Management also focuses on monitoring fluid and electrolyte balance. The nurse detects fluid excesses or deficits by evaluating hourly intake and output, serum and urine electrolytes and osmolality results, and urine specific gravity. In addition, changes in blood pressure, pulse, and respirations, as well as the onset of pulmonary crackles, neck vein distention, peripheral edema, and increasing CVP and pulmonary artery occlusion pressure (PAOP, also known as pulmonary artery wedge pressure) are noted. The nurse observes skin turgor and mucous membranes and changes in alertness and cognition. Drowsiness, confusion, and headache may indicate water intoxication. Body weight is another indicator of fluid status.

Complications

Major complications of diabetes insipidus are cardiovascular collapse and tissue hypoxia. Seizures and encephalopathy can also result from fluid and electrolyte imbalance. Prognosis is excellent as long as the patient receives prompt and aggressive treatment.

Table 44-4 Commonly Administered Drugs for Diabetes Insipidus

Drug	Dosage	Route of Administration	Duration of Drug	Adverse Effects
Desmopressin	5–20 mcg each day	IV, by mouth, nasal spray (cannot be given if nasal passages are blocked)	8–24 h	Headache, chest pain, nausea, diarrhea, edema
Aqueous pitressin	2–4 units every 4–6 h	Intramuscularly, subcutaneously, intranasally	1–8 h	Headache, chest pain, nausea, diarrhea, edema
Pitressin tannate in oil	2.5–5 units	Intramuscularly	36–48 h	Headache, chest pain, nausea, diarrhea, edema
Lysine vasopressin nasal spray	5–20 units three to seven times daily; titrate to output	Intranasally	2–6 h	—
Chlorpropamide (Diabinese)	100–250 mg/d	By mouth	60–72 h	Hypoglycemia, headache, tinnitus, alcohol intolerance, gastrointestinal disturbances, diarrhea
Clofibrate	250–500 mg	By mouth	6–8 h	Gastrointestinal disturbances

▲ Emergencies for Patients With Diabetes Mellitus

Diabetes mellitus is a complex and chronic metabolic disorder characterized by hyperglycemia and defects in insulin secretion. Figure 44-4 illustrates how chronic hyperglycemia is associated with long-term organ dysfunction, particularly of the eyes, kidneys, nerves, heart, and blood vessels. These long-term microvascular and macrovascular complications of retinopathy, neuropathy, nephropathy, and cardiovascular disease are the primary causes of morbidity and mortality in people affected with diabetes.

The incidence of diabetes in the United States has risen dramatically, and diabetes morbidity and mortality are also increasing. Diabetes is one of the most common diseases in the United States, with an estimated 25.8 million adults afflicted, representing 8.3% of the population.[6] Prevalence rates approach 50% in certain population subgroups (Native American, Hispanic American, African American). This rate is strongly related to the epidemic of obesity and the socioeconomic inequalities that plague the United States.

The pathogenic processes associated with diabetes mellitus range from autoimmune destruction of the islet beta cells of the pancreas (type 1 diabetes mellitus) to insulin resistance (type 2 diabetes mellitus). The derangements of carbohydrate, protein, and fat metabolism all result from deficient action of insulin on target tissues. The main effect is hyperglycemia. Hyperglycemia is manifested as polyuria, polydipsia, polyphagia, weight loss, and blurred vision. Acute, critical illnesses associated with diabetes are hyperglycemia with ketoacidosis and nonketotic hyperosmolar state.

Often almost half of patients with type 2 diabetes mellitus are not diagnosed until complications have already developed, and many suffer acute syndromes requiring emergency department evaluation, intensive care management, or both.

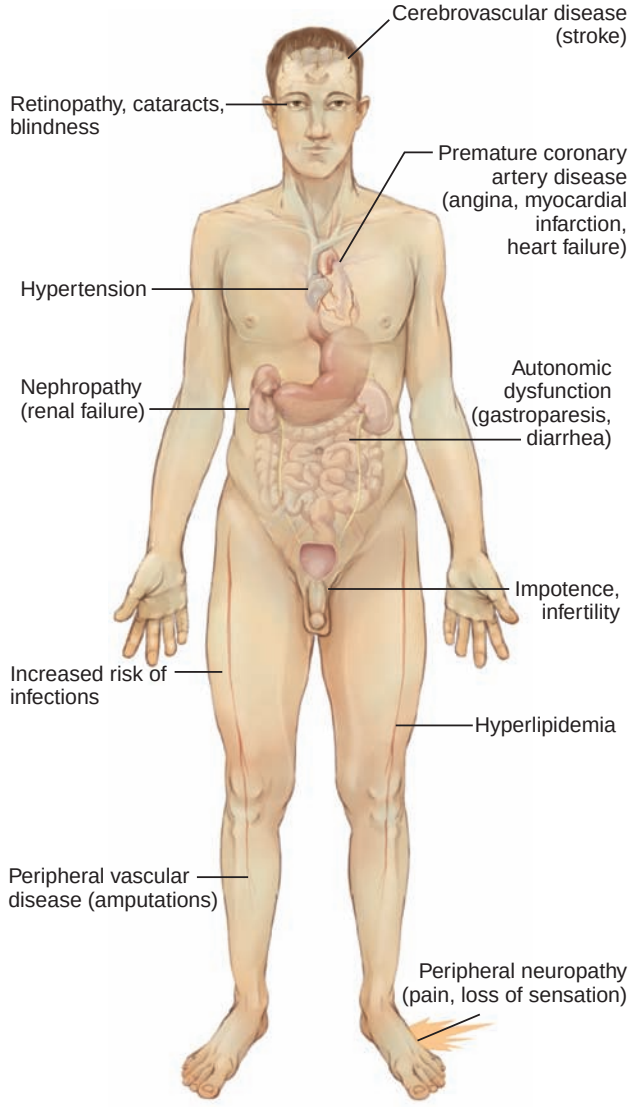


FIGURE 44-4 ▲ The complications of diabetes can be widespread.

Table 44-5 Comparison of Type I and Type 2 Diabetes Mellitus

	Type 1 Diabetes	Type 2 Diabetes
Etiology	Autoimmune destruction of islet cells	Insulin resistance
Incidence	5%–10%	90%–95%
Age of onset	Usually before 35 y	Usually after 35 y
Speed of onset	Usually rapid	Usually gradual
Nutritional state	Usually thin	Usually overweight, obese
Endogenous insulin	Absent	Low or high, rarely absent
Symptoms	Polyuria, polydipsia, polyphagia, weight loss	Same, plus blurred vision, fatigue
Ketosis	Frequently present with poor control	Infrequent
Treatment goal	Exogenous insulin management	Weight loss, exercise, improved insulin resistance
Treatment	Exogenous insulin, diet control, exercise, weight maintenance	Oral agents, diet control, exercise, weight loss

The critical care nurse must be vigilant in identifying high-risk patients. Box 43-6 on page 983 contains the American Diabetes Association's Position Statement on the diagnostic parameters for diabetes.

Most patients with diabetes can be classified into two main groups, those with type 1 diabetes mellitus and those with type 2 diabetes mellitus (Table 44-5). The cause of type 1 diabetes is an absolute deficiency of insulin secretion. This insulin secretion impairment results from autoimmune destruction of the beta cells of the pancreas. Markers of immune destruction include islet cell autoantibodies (ICAs), glutamic acid decarboxylase (GAD-65) antibodies, islet cell antibodies (ICA512-1A-2), and insulin antibodies (IAAs). The best predictor for future development of type 1 diabetes is the expression of multiple autoantibodies.[6] The rate of islet cell destruction is variable, and it occurs more rapidly in younger patients and more slowly in older patients. Some children and adolescents present with ketoacidosis as the first manifestation of the disease. As the islet cell destruction occurs, the patient is rendered insulin dependent for survival.

Type 1 diabetes is primarily a disease of the young, with a peak incidence at ages 10 to 12 years for females and 12 to 14 years for males. Although onset occurs mainly during childhood or at puberty and most patients receive a diagnosis before age 20 years, the disease can occur at any age. Type 1 diabetes accounts for approximately 5% to 10% of all cases of diabetes. A genetic predisposition for type 1 diabetes may exist because it appears that genetically predisposed patients contract the disorder after an environmental factor (viruses, congenital rubella, enteroviruses) triggers the autoimmune destruction of the islet cells, leading to insulin deficiency.[7,8] These patients are rarely obese. Type 1 diabetes is associated with other autoimmune diseases, such as Graves' disease, Addison's disease, and autoimmune polyendocrine syndromes.

Type 2 diabetes mellitus manifests as insulin resistance with relative, rather than absolute, insulin deficiency. Most of the patients with this form of diabetes do not require insulin, at least initially. The specific cause of insulin resistance is multifactorial; however, these patients do not suffer from autoimmune destruction of the islet cells of the pancreas. Most patients are overweight or obese, and excess adiposity itself can lead to insulin resistance. Ketoacidosis seldom occurs in this form of diabetes because the patient still secretes just enough insulin to avoid critical illness. When the patient does sustain severe complications associated with hyperglycemia, usually he or she has concomitant illness such as myocardial infarction, infection, or trauma. Because of the high incidence of insulin resistance and relative insulin deficiency, hyperglycemic hyperosmolar state (HHS) usually develops in patients with type 2 diabetes when they become critically ill.

Type 2 diabetes can go undiagnosed for many years because this disease progresses slowly. However, the patient is at high risk for developing macrovascular and microvascular complications. Quite often, these patients have normal to higher-than-normal insulin levels because of the ensuing insulin resistance that they develop. Insulin resistance develops, and their circulating insulin is insufficient to prevent hyperglycemia. Insulin resistance is best treated with weight loss, exercise, and pharmacological management. The medications to treat type 2 diabetes range from insulin secretagogues and medications that affect insulin sensitivity to insulin. Refer to Table 44-6 and Figure 44-5 for a review of the commonly used oral medications for type 2 diabetes and their mechanisms of action.

Eventually, many people with diabetes require insulin as their disease progresses. The risk for developing type 2 diabetes increases with age, obesity, sedentary lifestyle, and family history of type 2 diabetes. Its incidence varies with ethnicity but is increasingly more common in African Americans, Hispanics, Native Americans, South Pacific Islanders, and Asian Americans. Epidemiological and genetic studies suggest a strong genetic basis for developing type 2 diabetes; however, candidate genes that account for the majority of cases have not been identified. Type 2 diabetes is a multifactorial disorder with genetic and environmental implications.[6]

Results from two landmark trials involving people with types 1 and 2 diabetes mellitus, the Diabetes Control and Complications Trial (DCCT)[9] and the United Kingdom Prospective Diabetes Study (UKPDS),[10] have profoundly affected the current management of diabetes. These two trials have demonstrated that very tight glycemic control is necessary to avoid the costly and life-threatening complications resulting from poorly controlled diabetes. This approach extends to the management of diabetic emergencies in the critical care arena as well, and another landmark trial conducted in 2001 demonstrated significant improvement in patient outcomes when glycemic control was maintained at 80 to 110 mg/dL. The evidence that was produced by these trials significantly changed the management of diabetes among the critically ill and created a need for continuous

Table 44-6 Oral Drugs Used to Treat Diabetes Mellitus

Drug	Example	Action	Duration of Action	Nursing Considerations
First-generation sulfonylureas	Tolinase, Diabinese	Stimulates pancreatic insulin secretion	12–60 h	Side effects include hypoglycemia, gastrointestinal disturbances, and rash Contraindicated in pregnancy Seldom used
Second-generation sulfonylureas	Glyburide, Glucotrol, Micronase, Amaryl	Stimulates pancreatic insulin secretion	10–24 h	Side effects include hypoglycemia, gastrointestinal disturbances, and rash Safer in elderly patients
Biguanides	Metformin (Glucophage)	Reduces hepatic glucose production, increases insulin sensitivity	8 h	Lactic acidosis is a serious side effect (stop when using contrast medium for x-rays); other side effects include gastrointestinal disturbances (eg, flatulence, diarrhea, nausea) Use with caution in patients with renal disease Improves insulin resistance Promotes weight loss
Thiazolidinediones	Actos	Enhances insulin's effects at receptor sites	12–24 h	Will not increase level of circulating insulin Side effects include edema, weight gain, and anemia Monitor liver function tests Improves lipid profile Extreme caution with heart failure
α-Glucosidase inhibitors	Precose, Miglitol	Inhibits metabolism of carbohydrates in intestines	8 h	Take with meals Side effects include gastrointestinal symptoms (eg, flatulence, abdominal pain, diarrhea, nausea) Use with caution in patients with renal disease
Meglitinides	Prandin	Stimulates β-cell insulin release		Side effects include hypoglycemia, upper respiratory infection, headache, and diarrhea Use with caution in patients with hepatic or renal disease
Amino acid derivatives (insulin secretagogue)	Starlix	Stimulates β-cell insulin release		Side effects include hypoglycemia, gastrointestinal disturbances (eg, nausea), upper respiratory tract symptoms, and dizziness Use with caution in patients with hepatic disease
Incretion mimetics				
Glucagon-like peptide-1 (GLP-1) analogues		Decreases postprandial glucose rise and slows gastric emptying, promotes satiety	8–12 h	Given subcutaneously May produce weight loss
Exenatide	Byetta Bydureon (weekly injected exanatide)			
Liraglutide	Victoza			
Amylin analogue	Symlin	As above		
Dipeptidyl peptidase-4 inhibitor		Increases availability of GLP-1 above actions	12–24 h	May produce weight loss
Linagliptin	Tradjenta			
Sitagliptin	Januvia			
Saxagliptin	Onglyza			
Combination therapy Glyburide and metformin	Glucovance	As above with each drug	As above with each drug	As above with each drug

(continued on page 1003)

Table 44-6 Oral Drugs Used to Treat Diabetes Mellitus (continued)

Drug	Example	Action	Duration of Action	Nursing Considerations
Metformin and glipizide	Metaglip	As above with each drug	As above with each drug	As above with each drug
Repaglinide and metformin	PrandiMet	As above with each drug	As above with each drug	As above with each drug
Sitagliptin and metformin	Janumet	As above with each drug	As above with each drug	As above with each drug
Pioglitazone and metformin	Actoplus Met	As above with each drug	As above with each drug	As above with each drug
Pioglitazone and glimepiride	Duetact	As above with each drug	As above with each drug	As above with each drug
Rosiglitazone and glimepiride	Avandaryl	As above with each drug	As above with each drug	As above with each drug

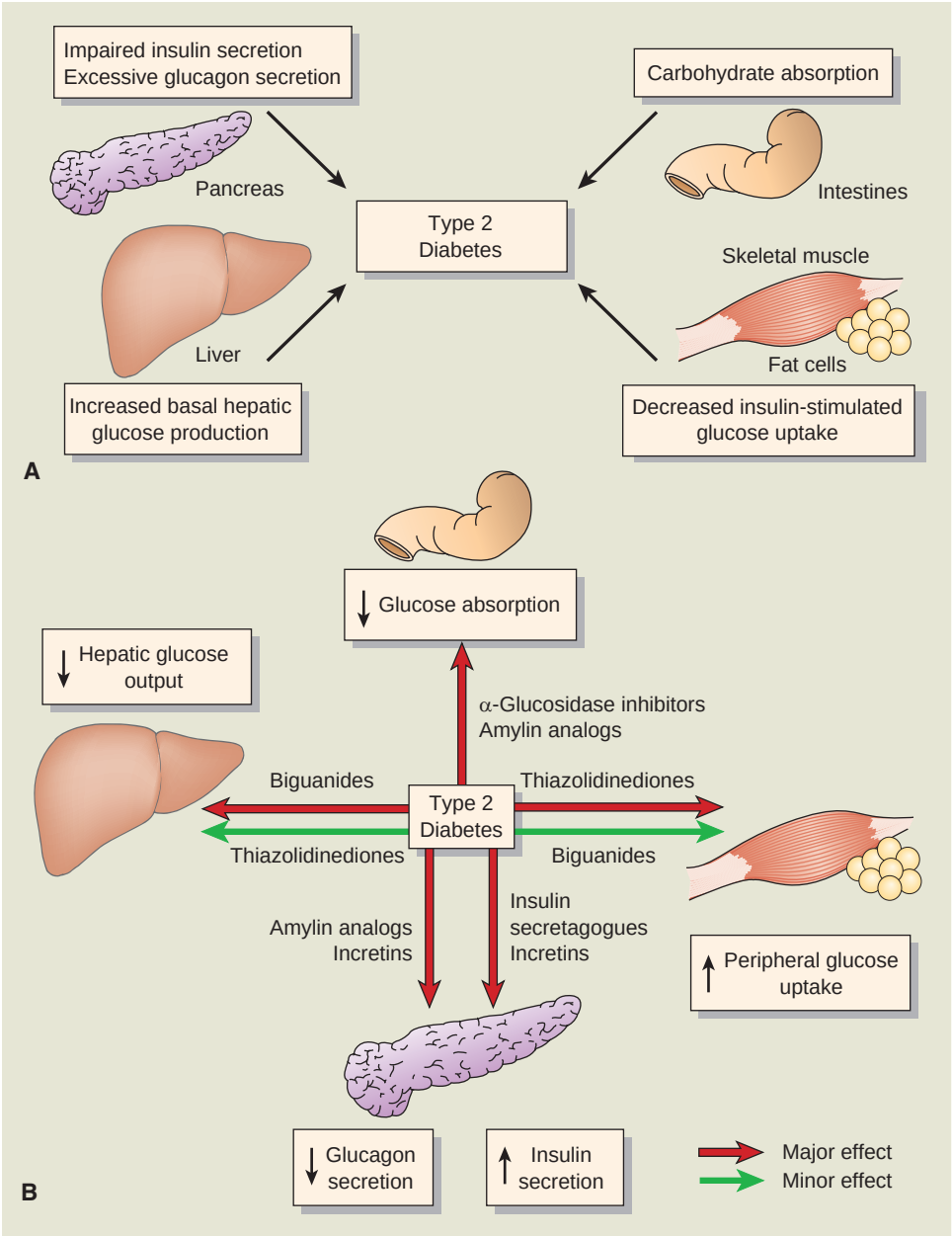


FIGURE 44-5 ▲ A: Factors leading to elevated blood glucose levels in type 2 diabetes mellitus. **B:** Mechanisms of action of the oral hypoglycemic agents used in the treatment of type 2 diabetes mellitus. (From Porth CM: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 1063.)

IV insulin infusions to control the patient's glucose at all times. The critical care nurse needs to monitor the patient very closely to avoid serious complications associated with insulin infusions.

New evidence now exists that questions the need for very strict glycemic control in the hospital setting. In 2008, the Action to Control Cardiovascular Risk in Diabetes[11] trial, aiming to lower Hb A_1C levels below 6% to study effects on cardiovascular risk, was discontinued prematurely after participants in the intensive-therapy group experienced higher mortality rates. The Normoglycemia in Intensive Care Evaluation–Survival Using Glucose Algorithm Regulation (NICE-SUGAR)[12] trial, a large randomized prospective study that assessed the effects of intense glucose lowering on critically ill adults (80 to 108 mg/dL), demonstrated that study participants experienced unacceptably severe hypoglycemic episodes of less than 40 mg/dL and an associated higher risk of mortality than among the control group. The two trials' results have questioned the need for very strict glycemic control in critically ill patients.

Diabetic Ketoacidosis

Pathophysiology

Diabetic ketoacidosis (DKA) is a critical illness that manifests with severe hyperglycemia, metabolic acidosis, and fluid and electrolyte imbalances. DKA results from severe insulin deficiency that leads to the disordered metabolism of proteins, carbohydrates, and fats. The concomitant elevation of counter-regulatory hormones such as growth hormone (GH), cortisol, epinephrine, and glucagon exacerbates the condition, leading to further hyperglycemia and hyperosmolality, ketoacidosis, and volume depletion. Figure 44-6 outlines these mechanisms and their interrelationships.

DKA continues to be an important cause of morbidity and mortality among people with diabetes. DKA is responsible for more than 100,000 hospital admissions per year in the United States. Most patients with DKA have type 1 diabetes mellitus; however, it is possible for patients with type 2 diabetes to manifest DKA during catabolic stress associated with severe critical illness. DKA is associated with less than 2% mortality rate overall, but it is a common cause of death among children and adolescents with type 1 diabetes mellitus.[13] Treatment can cost more than one of every four health care dollars spent on direct medical care for adults with type 1 diabetes mellitus, resulting in up to 2.4 billion U.S. dollars annually.[13] The cause of death is rarely a direct result of the metabolic acidosis or from hyperglycemia; instead, death is more often related to the underlying illness that precipitated the metabolic decompensation. Therefore, successful treatment requires prompt attention to the precipitating causes of the hyperglycemic event.

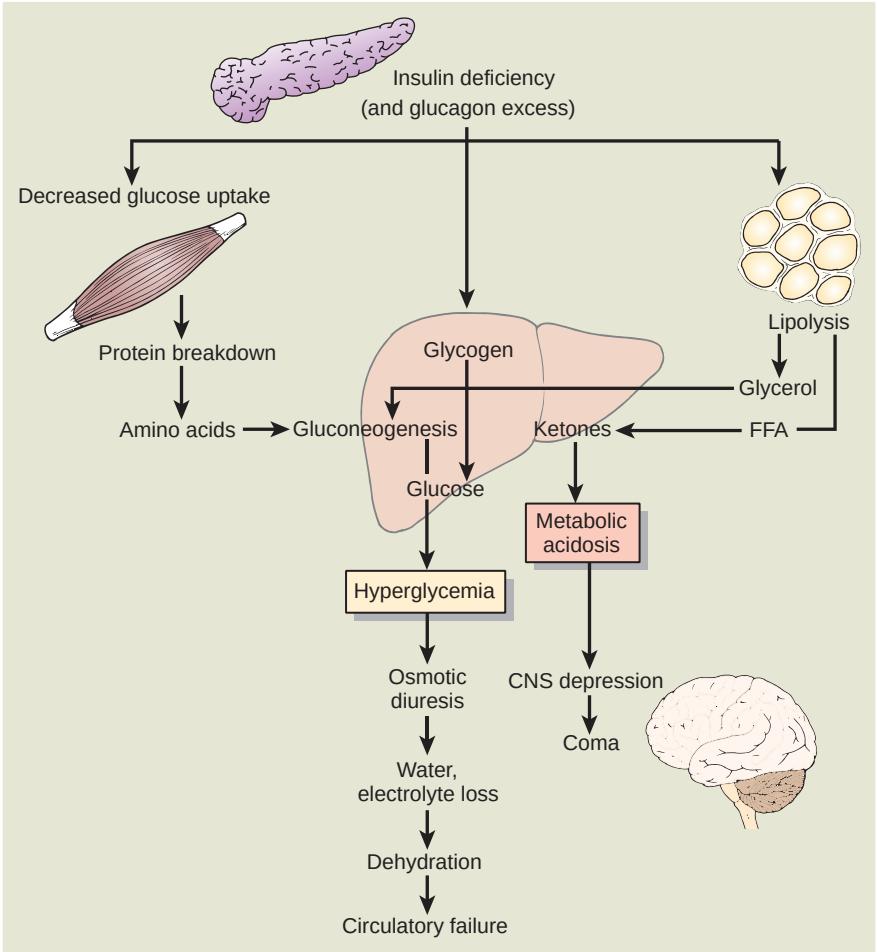


FIGURE 44-6 ▲ Mechanisms of diabetic ketoacidosis (DKA). DKA is associated with very low insulin levels and extremely high levels of glucagon, catecholamines, and other counter-regulatory hormones. Increased levels of glucagon and catecholamines lead to mobilization of substrates for gluconeogenesis and ketogenesis by the liver. Gluconeogenesis in excess of that needed to supply glucose to the brain and other glucose-dependent tissues produces a rise in blood glucose levels. Mobilization of free fatty acids from triglyceride stores in adipose tissue leads to accelerated ketone production and ketosis. (From Porth CM: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 1068)

Three major physiological disturbances exist in DKA: (1) hyperosmolality from hyperglycemia, (2) metabolic acidosis from accumulation of ketoacids, and (3) volume depletion from osmotic diuresis. Each of these three disturbances may be more or less severe in any patient. Furthermore, interactions among these disturbances may occur, aggravating (or possibly partially compensating for) one another.

HYPERGLYCEMIA AND HYPEROSMOLALITY. The first major consequence of DKA is hyperosmolality resulting from hyperglycemia. The hyperglycemia seen in DKA is the result of insulin deficiency and excessive hepatic (gluconeogenesis) and renal (glycogenolysis) glucose production and reduced glucose utilization in peripheral tissues. With insulin deficiency, the plasma glucose level rises. As illustrated in Figure 44-7, the concomitant effects of the counter-regulatory hormones, particularly cortisol and catecholamines, further aggravate hyperglycemia by enhancing gluconeogenesis, insulin resistance, and lipolysis. This leads to hepatic fatty acid oxidation to ketone bodies (β-hydroxybutyrate and acetoacetate), ketonemia, and metabolic acidosis.

The central mechanism that protects against hyperosmolality is excretion of glucose by the kidneys. Glucose is filtered at the kidney glomerulus. With normal circulating blood volume and a normal glucose load, all this glucose is reabsorbed into the bloodstream. However, when the blood glucose level exceeds the normal threshold of about 180 mg/dL, glucose begins to escape into the urine because the reabsorption capacity of the tubules is exceeded. As the glucose load to be filtered increases, glucose is lost rapidly in the urine. Eventually, nearly all of the additional glucose put into the circulation is lost into the urine. The renal "escape valve" serves as a protective device to prevent extreme accumulation of glucose in blood. Indeed, in people with diabetes whose circulating blood volume is well maintained, it is extremely unusual to find blood glucose levels in excess of 500 mg/dL because of the intense glucose diuresis. Conversely, any patient whose blood glucose level is higher than this level has a severely reduced circulating blood volume, renal damage, or both.

Glycosuria is largely responsible for volume depletion. Additionally, high ketone levels cause osmotic diuresis that leads to hypovolemia and decreased glomerular filtration rate. A vicious cycle occurs in a patient whose diabetes is badly out of control and who cannot take in enough sodium and water to compensate for urinary losses. Hyperglycemia leads to volume depletion, which in turn reduces urinary glucose losses and permits the blood glucose level to rise even higher.

This hyperosmolality of body fluids and dehydration probably accounts for the lethargy, stupor, and ultimately, coma that occurs as DKA worsens. Diabetic patients who have ketoacidosis without hyperosmolality are less likely to have changes in consciousness.

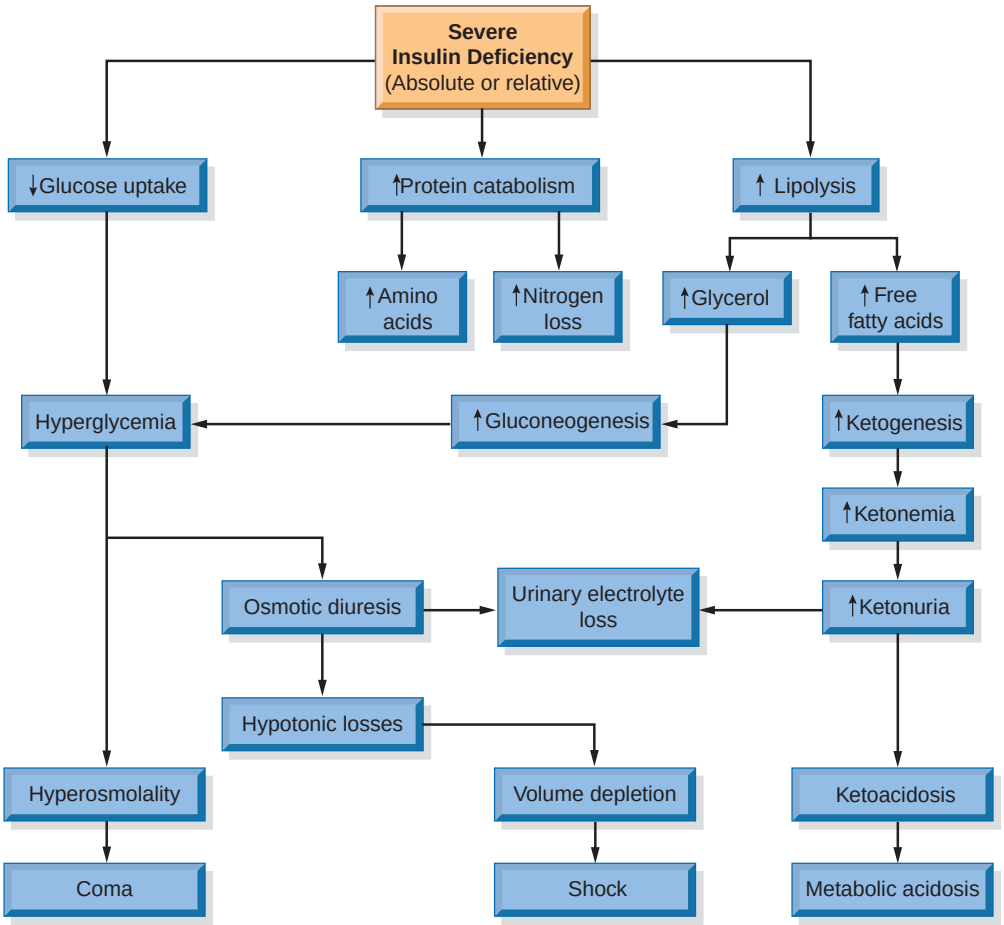


FIGURE 44-7 ▲ The metabolic consequences of severe insulin deficiency and the interrelations of these consequences lead to DKA.

KETOSIS AND ACIDOSIS. The second major consequence of severe insulin deficiency is uncontrolled ketogenesis (see Fig. 44-7). The combination of insulin deficiency and enhanced effects of the counter-regulatory hormones causes the activation of lipase in adipose tissue. Lipase causes the breakdown of triglycerides into glycerol and free fatty acids (FFAs); massive amounts of FFAs are released as precursors of ketoacids. In the liver, they are oxidized to ketone bodies.

As ketoacids enter the ECF, the hydrogen ion is stripped from the molecule and neutralized by combining with the bicarbonate ion buffer, thereby protecting the pH of the ECF and leaving behind ketoacid anion residues. The resulting carbonic acid breaks down into water and carbon dioxide gas, which is exhaled. As ketoacid anions accumulate, they progressively displace bicarbonate from the ECF. The usual laboratory determination of electrolytes does not measure ketoacid concentration directly. However, an excess of total measured cations (sodium plus potassium) over total measured anions (chloride plus bicarbonate) provides a clue to the presence of these so-called unmeasured anions. This excess, referred to as the anion gap, can serve as an indirect measure of the quantity of ketoacids present.

The following formula is used to calculate the anion gap:

$$(\text{Sodium}) - (\text{chloride} + HCO_3)$$

The normal value is less than 15 mEq/L. An abnormal result indicates metabolic acidosis. For example, if sodium = 144 mEq/L, chloride = 92 mEq/L, and bicarbonate = 26 mEq/L, then the anion gap is 26 mEq/L, a value that indicates severe metabolic acidosis. As the ketoacids continue to accumulate, the serum bicarbonate falls and the anion gap increases. If this continues, the pH falls, and the acidosis becomes life-threatening.

Another cause of metabolic acidosis in DKA is the formation of lactic acidosis resulting from poor tissue perfusion and hypovolemia. This further exacerbates the anion gap, decreasing the serum bicarbonate level. Neutrality of body fluids is protected primarily by the bicarbonate buffering system, which determines the pH at all times by the ratio of bicarbonate anion to carbon dioxide in plasma. If bicarbonate anion is lost because of its displacement by ketoacid anions, excess carbon dioxide gas must be driven off at the level of the lung by hyperventilation. This process keeps the ratio at or close to its usual value of 20:1 and maintains the pH close to its physiological value of 7.4. Hyperventilation, which is gradual at first and then rapidly becomes more vigorous and more obvious as the arterial pH drops below 7.2, is a characteristic physical finding in DKA.

This dramatic increase in ventilation, which occurs more by an increase in the depth than in the frequency of breathing, is known as Kussmaul's respirations. It is associated with the classic "fruity" odor of the breath in DKA. The presence of clearcut Kussmaul's respirations is a signal that the ECF pH is at or below 7.2, a relatively severe degree of acidosis.

VOLUME DEPLETION. Ketoacids are excreted in the urine largely as sodium, potassium, and ammonium salts. This contributes to the third pathophysiological problem of DKA: volume depletion and fluid and electrolyte loss as a result of osmotic diuresis. The fluid loss associated with DKA is approximately 6 L.

Although loss of glucose through the kidneys helps protect against the ravages of extreme hyperosmolality, the diabetic patient who develops ketoacidosis pays a price for this glycosuria. Glucose remaining in the glomerular filtrate, after the renal tubules have reabsorbed all they can, forces water to remain in the tubules. This glucose-rich filtrate then flows out of the body, carrying with it water, sodium, potassium, ammonium, phosphate, and other salts. This rapid urine flow and obligate loss of water and electrolytes is known as an osmotic diuresis. Salts of ketone bodies and the urea resulting from rapid protein breakdown and accelerated gluconeogenesis also contribute to the solute load in the renal tubule, further aggravating the diuresis. The average amounts of salts and water lost to the body through osmotic diuresis during the development of DKA have been measured. Overall water loss in a 70-kg adult patient with DKA can be 5 to 8 L, or 15% of total-body water.

The fluid lost to the body is slightly hypotonic; it contains a slight excess of water compared with the volume of salts. This is expected from an osmotic diuresis due to glucose and urea. The fluid losses result from the combination of many different factors, including the intensity and duration of the hyperglycemia and osmotic diuresis; the amount of water and electrolyte replaced orally during this time; the presence of other fluid and electrolyte losses, such as vomiting, diarrhea, or sweating; and the integrity of renal function.

Sodium and water make up the central structure of the ECF, including the vascular volume. When large quantities of sodium and water are lost in the urine, the body perceives it as a serious threat to the maintenance of the circulation. A variety of compensatory mechanisms are called into play to prevent vascular collapse and shock. For example, an increase in pulse rate usually occurs, which helps maintain cardiac output in the face of shrinking intravascular volume.

At least as important, however, is a protective shift in body fluid brought about by the hyperglycemia. Because free glucose is limited almost entirely to the extracellular water, an osmotic pressure gradient is set up across the cell membrane, between the extracellular compartment and the interior of the cells. Therefore, the higher the blood glucose, the more water is drawn out of cells and into the extracellular space. Therefore, as sodium and water are lost into the urine, shrinking the ECF, they are "replaced" (at least as to their osmotic effect) by glucose entering from the liver and by water entering from all cells. This re-expands the ECF.

Although hyperosmolality produces damaging CNS effects and osmotic diuresis, it provides a temporary mechanism for preventing vascular collapse. Despite these compensatory mechanisms, circulatory volume falls as DKA progresses. This leads to decreased glomerular filtration, decreased tissue perfusion, metabolic acidosis, and shock.

As the vascular volume falls, glomerular filtration also falls. This decreasing renal function leads to increasing blood levels of glucose, potassium, urea nitrogen, and creatinine. The excretion of potassium by the kidney occurs through the exchange of potassium for sodium. Therefore, adequate sodium must be present at the exchange site in the kidney for the rate of potassium excretion to keep pace with the need for excretion. If renal perfusion falls, enough sodium may not be available for this exchange. As a result, despite a total-body depletion of potassium, the serum potassium level may rise above normal, even to dangerously high levels.

A second major consequence of diminished vascular volume is a generalized decrease in tissue perfusion. Well before the drop in volume has reached the point at which blood pressure actually falls and full-blown shock occurs, blood is shunted away from many tissues, and the perfusion of nearly all tissues suffers. The resulting decrease in oxygen causes those tissues to shift to some degree of anaerobic glucose metabolism. This results in the increased production of lactic acid. The release of lactic acid into the circulation lowers the bicarbonate further, aggravating the already existing metabolic acidosis. Therefore, in patients with DKA, combined lactic acidosis and ketoacidosis is a common finding.

The loss of phosphate in the urine worsens tissue hypoxia. As body phosphate stores are depleted, circulating plasma phosphate levels fall quite low, depriving the red blood cells of organic phosphate compounds. Under these circumstances, the red blood cells become depleted of certain key phosphate derivatives, increasing the tightness of oxygen binding to the hemoglobin in these cells. Therefore, less oxygen is given up, and tissue hypoxia worsens.

Finally, if vascular volume falls low enough, compensation mechanisms fail, blood pressure drops, and true shock supervenes, changing the situation. A rapidly worsening cycle of acidosis, tissue damage, and deepening shock may then occur, leading ultimately to irreversible vascular collapse and death. The full-blown syndrome of DKA is characterized by major contributions from all three major pathophysiological disruptions, each of which is primarily responsible for one of the major clinical features: coma, shock, and metabolic acidosis. There is evidence to suggest that hyperglycemia crisis is associated with severe inflammatory state due to the elevation of proinflammatory cytokines, C-reactive protein, reactive oxygen species, and lipid peroxidation as well as plasminogen activator inhibitor-1. This partially explains the hypercoagulable state associated with hyperglycemic crisis.[14]

Causes

The most common cause of DKA is infection, occurring in 30% to 50% of cases. Urinary tract infection and pneumonia account for the majority of infections.[13] Other precipitating factors include inadequate insulin therapy, severe illness (cerebrovascular accident [CVA], myocardial infarction, pancreatitis), alcohol abuse, trauma, and drugs. In addition, many people with type 1 diabetes present with DKA on initial diagnosis. Also, many patients with type 1 diabetes suddenly discontinue their insulin and deteriorate; reasons for insulin omission in younger patients include fear of weight gain, fear of hypoglycemia, rebellion against authority, and the stress of chronic disease. In one study of 341 females with type 1 diabetes, psychological problems complicated by disorders were a contributing factor in 20% of cases.[14] Other reasons given for sudden discontinuation of insulin or oral medications include lack of knowledge and poor compliance related to lack of financial resources. Noncompliance with therapy has been implicated as a major precipitating cause of DKA in urban African American and medically indigent patients.[12]

Assessment

Initial laboratory analysis should include an immediate glucose level using a venous sample and glucose meter measurement at the bedside to confirm the diagnosis. While these preliminary data are collected, the nurse inserts an IV line and starts volume replacement. A more considered assessment follows, which begins with details of the history and physical examination, a search for precipitating causes, and more complete laboratory tests. Physical examination and laboratory findings in DKA are summarized in Box 44-9.

BOX 44-9 Signs of Diabetic Ketoacidosis (DKA)

- Hyperventilation
- Kussmaul's respirations and "fruity" breath
- Lethargy, stupor, coma
- Hyperglycemia
- Glycosuria
- Volume depletion
- Hyperosmolality
- Increased anion gap (>7 mEq/L)
- Decreased bicarbonate (<10 mEq/L)
- Decreased pH (<7.4)

HISTORY AND PHYSICAL EXAMINATION. If ketoacidosis is strongly suspected, an effort is made to establish the diagnosis quickly so that life-preserving therapy can be started. Initial data collection includes an abbreviated history from the family or friends of an unconscious patient, a search for a diabetic identification card, and rapid assessment for clinical clues of volume depletion. After asking about the diabetic regimen, medications, and recent changes in health, the clinician should perform a review of systems. Questions concern appetite, weight change, food and fluid intake, thirst, abdominal bloating and discomfort, bowel function, and urinary frequency and amount. During the interview, he or she should observe the patient's cognition and responsiveness.

DKA develops rapidly, and patients may display polydipsia, polyuria, and weight loss several days before ketoacidosis is established. Frequently, abdominal pain and vomiting are presenting symptoms. Approximately 40% to 75% of patients experience abdominal pain that mimics an acute abdomen; the severity of abdominal pain often correlates with more severe metabolic acidosis. Other possible findings include thirst, frequent urination, poor appetite, nausea and vomiting, fatigue, weakness, and drowsiness. The patient may also have symptoms related to urinary tract infection, upper respiratory infection, and chest symptoms because infection is often a precipitating factor.

The physical examination includes blood pressure, heart and respiratory rate, breathing pattern, heart sounds and rhythm, breath sounds, capillary refill, skin color and warmth of extremities, temperature, signs of hydration (eg, skin turgor, mucus pool under tongue), deep tendon reflexes, LOC, and an abdominal examination. Possible findings include hyperventilation, Kussmaul's respirations and fruity breath, dehydration, abdominal distention, dry mucous membranes, flushed skin, poor skin turgor and perfusion, hypotension, tachycardia, and varying degrees of responsiveness from lethargy to coma. Even though infection is often concurrent, the patient may be normothermic due to vasodilation. Severe hyperthermia is a poor prognostic sign.

LABORATORY STUDIES. Laboratory studies include blood glucose, chemistries, osmolality, anion gap, pH, ABGs,

urine acetone, and glucose. Possible findings include hyperosmolality, increased anion gap (>7 mEq/L), decreased bicarbonate (<10 mEq/L), and decreased pH (<7.4). The serum glucose may range from 300 to 800 mg/dL or higher. Sodium, potassium, creatinine, and BUN levels are all elevated. Magnesium and phosphate may also be high. Patients with DKA often present with leukocytosis and the presence of greater than 10% neutrophil bands. The key diagnostic feature of DKA is the presence of serum ketones per the nitroprusside test or by direct measurement of β-hydroxybutyrate.[12]

DIAGNOSTIC STUDIES. Tissue cultures of the throat, urine, or blood may also be performed to determine the presence of infection. A chest radiograph should be obtained to rule out acute infection, and an ECG should be obtained.

Management

The severity of DKA can be categorized as mild, moderate, or severe depending on the level of metabolic acidosis and altered mental state. Treatment goals for the patient with DKA include the following:

- Improve circulatory volume and tissue perfusion
- Correct electrolyte imbalances
- Decrease serum glucose concentration
- Correct ketoacidosis
- Determine precipitating events

Treatment protocols for the adult with DKA are given in Figure 44-8, and a Collaborative Care Guide is given in Box 44-10.

FLUID REPLACEMENT. The immediate threat to life in a critically ill ketoacidotic patient is volume depletion. After establishing an IV line, the nurse rapidly infuses 0.9% (normal) saline solution. The goal is to reverse the severity of the extracellular volume depletion and restore renal perfusion as soon as possible. The first liter may be infused in 1 hour in patients with normal cardiac function; on average, the rate will be equal to 15 to 20 mL/kg body weight per hour. This replaces only a fraction of the extracellular loss in the average patient, which can range from 6 to 10 L.[14]

Fluid replacement continues at roughly 1 L/h until the heart rate, blood pressure, and urine flow indicate that hemodynamic stability is attained. Hypotonic solutions, such as 0.45% (half normal) saline solution, can be administered at a rate of 150 to 250 mL/h after the intravascular volume has been restored, or if the serum sodium level is greater than 155 mg/dL. Other plasma expanders, such as albumin and plasma concentrates, may be necessary if low blood pressure and other clinical signs of vascular collapse do not respond to saline solution alone.

Rapid infusion of saline solution in DKA has possible complications. It can dilute plasma proteins and lower the osmotic pressure of the plasma. This allows fluid to leak out of the vascular space through the capillary walls and contributes to the development of pulmonary edema or cerebral edema, particularly in children and older adults. Therefore, patients must be observed carefully during the first 24 to 36 hours for signs of pulmonary or cerebral edema.

Volume losses continue throughout the first hours of treatment until the glycosuria and osmotic diuresis are controlled. The next step of fluid replacement can be based on an estimate of the patient's total-body fluid loss. About 80% of the fall in blood glucose level during treatment of DKA is due to glucose loss into the urine, rather than the result of insulin-induced changes in glucose production and consumption. Therefore, in the earliest phases of treatment, insulin therapy complements fluid and electrolyte replacement. Lowering of glucose levels will occur more rapidly (as long as 6 hours) than correction of ketoacidosis (as long as 12 hours).

INSULIN THERAPY. Insulin therapy is the cornerstone for managing ketoacidosis for several reasons. It decreases the production of ketones by shutting off the supply of FFAs emerging from adipose tissue. It inhibits hepatic gluconeogenesis, thereby preventing further glucose from being added to the ECF. Simultaneously, hepatic ketogenesis is further reduced. Insulin also restores cellular protein synthesis. This effect occurs more slowly and permits the restoration of normal potassium, magnesium, and phosphate stores in tissues. Insulin also increases peripheral glucose utilization.

Careful glycemic control is the goal for managing patients with diabetes in the acute care setting. However, the blood glucose level should not fall too fast or too far. Sudden and rapid lowering of the blood glucose with insulin allows water to move very rapidly back into the cells. This can potentially lead to vascular collapse. Instead, early volume replacement should include sodium and water either before or along with insulin therapy.

It is necessary to give low-dose regular insulin by continuous IV infusion rather than by IV bolus or subcutaneous doses for more severe DKA or HHS cases. Subcutaneous insulin injections given every 1 to 2 hours are an alternative to IV insulin for mild or moderate DKA, and usually rapid-acting analog insulin (lispro, aspart, or glulisine) is used.[12] Box 44-11 on page 1012 summarizes guidelines for insulin administration.

Initially, insulin administration involves giving an IV bolus of regular insulin at 0.15 units/kg body weight followed by a continuous infusion of regular insulin at a dose of 0.1 units/kg/h (5 to 10 units/h). This produces a steady decline in glucose concentrations at a rate of 65 to 125 mg/h. A recent prospective study demonstrates that an hourly insulin infusion of 0.14 units/kg/h without initial bolus is sufficient to lower glucose and suppress hepatic ketone body production.[14]

When the plasma glucose reaches 250 mg/dL, it is necessary to decrease the insulin infusion to 0.5 units/kg/h and add dextrose (5% or 10%) to the IV fluids. To prevent cerebral edema that may occur when the blood–brain barrier is affected by extreme fluid shifts, avoidance of hypoglycemia is warranted at this point. It is vital to start subcutaneous insulin 1 to 2 hours *before* IV insulin is stopped.[13] See Table 44-7 on page 1013 for commonly used insulins.

POTASSIUM AND PHOSPHATE REPLACEMENT. The initial plasma potassium in patients with DKA can range from very low to very high. Therefore, potassium is not given until the laboratory report is available. Beginning IV potassium therapy in the presence of unrecognized hyperkalemia and inadequate renal mechanisms for handling potassium loads can be fatal. Although the ECG can provide clues to the presence of high or low potassium levels, potassium therapy should not be based on the ECG alone.

If the initial serum potassium level is low, IV potassium is usually started right away. This is particularly important because

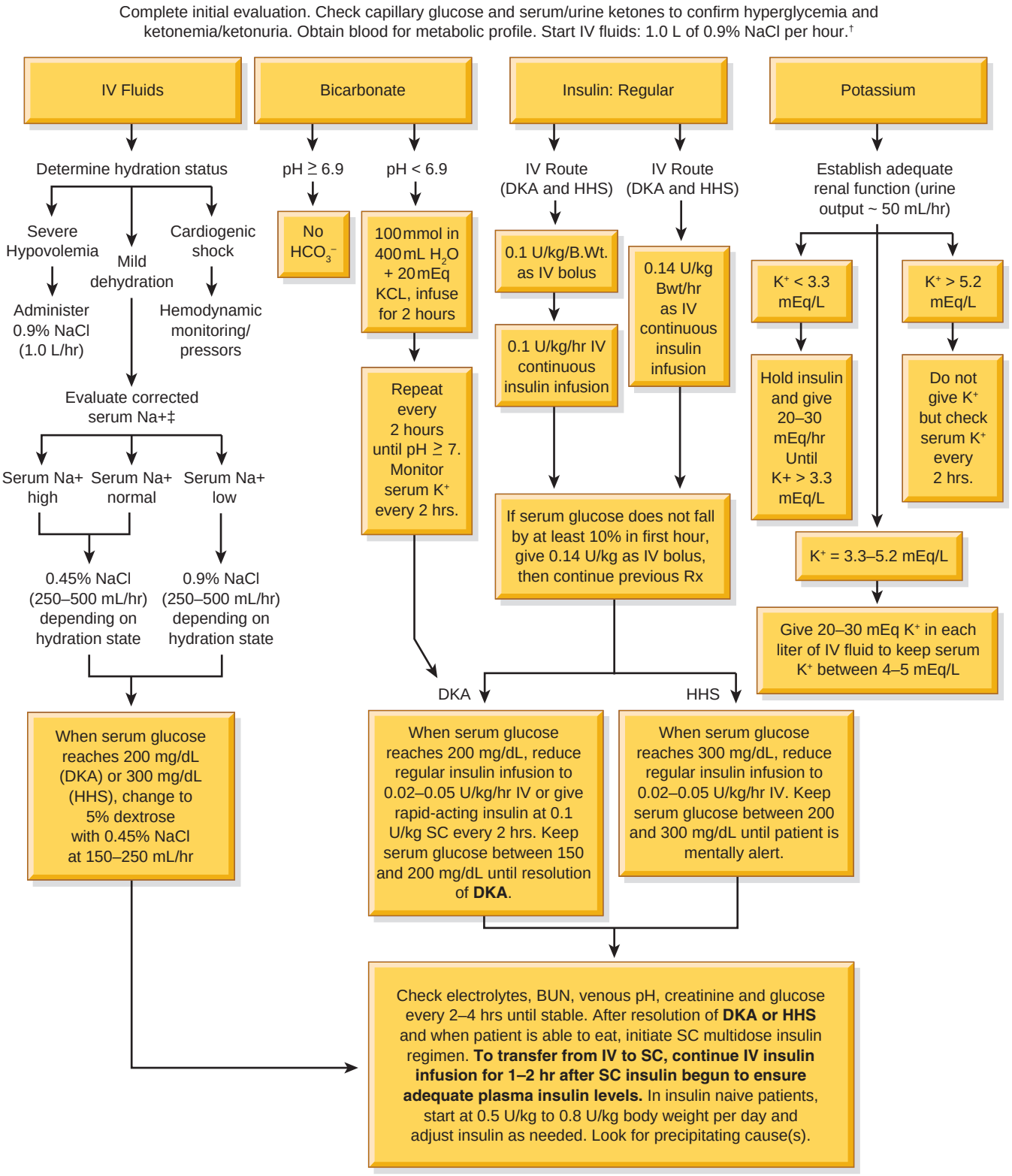


FIGURE 44-8 ▲ Protocol for management of adult patients with DKA or hyperglycemic hyperosmolar state (HHS). DKA diagnostic criteria: blood glucose 250 mg/dL, arterial pH 7.3, bicarbonate 15 mEq/L, and moderate ketonuria or ketonemia. HHS criteria: serum glucose greater than 600 mg/dL, arterial pH greater than 7.3, serum bicarbonate greater than 15 mEq/L, and minimal ketonuria and ketonemia.

†15 to 20 mL/kg/h; ‡serum Na should be corrected for hyperglycemia (for each 100 mg/dL glucose, add 1.6 mEq to sodium value for corrected serum value). Bwt, body weight; IV, intravenous; SC, subcutaneous. (From Kitabchi AE, Umpierrez GE, Miles JM, et al: Hyperglycemic crises in adult patients with diabetes. Diabetes Care 32(7):1335–1343, 2009)

BOX 44–10 COLLABORATIVE CARE GUIDE for the Patient With DKA

Outcomes	Interventions
Oxygenation/Ventilation	
Arterial blood gases are maintained within normal limits.	• Provide chest physiotherapy, turn, deep breath, cough, incentive spirometer every 4 h and PRN.
No evidence of acute respiratory failure.	• Continuously monitor patient's respiratory rate, depth, and pattern. Observe for Kussmaul's respiration, rapid and shallow breathing, and other signs of respiratory distress. • Monitor arterial blood gases, pulse oximetry, and, if intubated, end tidal CO_2. • Provide supplemental oxygen. • Prepare for intubation and mechanical ventilation (see Box 25-16).
The patient's lungs are clear. There is no evidence of atelectasis or pneumonia.	• Auscultate breath sounds every 2 h and PRN. • Take daily chest x-ray. • Provide chest physiotherapy every 4 h. • Mobilize out of bed as soon as patient is stabilized.
Circulation/Perfusion	
Blood pressure and heart rate are within normal limits. If pulmonary artery catheter is in place, hemodynamic parameters are within normal limits.	• Monitor vital signs hourly and PRN. • Assess for dehydration/hypovolemia: tachycardia, decreased central venous pressure (CVP) and pulmonary artery occlusion pressure (PAOP). • Assess for hypervolemia: neck vein distention, pulmonary crackles and edema, increased CVP and PAOP. • Administer vasopressor agents if hypotension is related to vasodilation.
Patient is free of dysrhythmias.	• Monitor electrocardiography (ECG) continuously. • Evaluate and treat the cause of dysrhythmias (eg, acidosis, hypoxia, hypokalemia/ hyperkalemia).
Fluids/Electrolytes	
Evidence of rehydration without complications: • balanced intake and output • normal skin turgor • hemodynamic stability • intact sensorium	• Infuse normal saline or lactated Ringer's solution, then 0.45% normal saline solution. • Monitor serum osmolality, urine output, neurological status, and vital signs closely during rehydration. Observe for complications of DKA (eg, shock, renal failure, decreased LOC, and seizures). • Assess BUN, creatinine, and urine for glucose and ketones.
Normal serum electrolytes and acid–base balance.	• Assess and replace electrolytes, Mg, and PO_4, as indicated. • Closely monitor potassium fluctuations as serum glucose is decreased and acidosis reversed. • Assess arterial pH and bicarbonate level every 2–4 h during rehydration and insulin administration.
Serum glucose returns to normal range.	• Monitor serum glucose every 30–60 min, then every 1–4 h after level is <300 mg/dL. • Administer intravenous (IV) insulin bolus then continuous low-dose infusion. • Infuse D5 half normal saline solution or D5W, after glucose level is <300 mg/dL.
Mobility/Safety	
The patient will be free of injury related to altered sensorium or seizures.	• Place on seizure and falls precautions. • Assess neurological status hourly, then every 2–4 h after initial rehydration phase.
Maintain muscle tone and joint range of motion.	• Provide range-of-motion exercises every 4 h. • Reposition in bed every 2 h. • Mobilize to chair when condition stable. • Consult physical therapist.

(continued on page 1011)

BOX 44–10 *COLLABORATIVE CARE GUIDE for the Patient With DKA (continued)*

Outcomes	Interventions
Skin Integrity	
Skin will remain intact.	• Assess risk for skin breakdown using the Braden Scale (see Fig. 51-7, p. 1164). • Initially assess skin and circulation every 1–2 h for 12 h. • If risk for skin breakdown is low, assess skin every 8 h and each time patient is repositioned. • Turn patient every 2 h. • Consider pressure relief/reduction mattress if at risk for skin breakdown.
Nutrition	
Calorie and nutrient intake meet metabolic requirements per calculation (eg, basal energy expenditure).	• Provide parenteral feeding if patient is fasting (NPO). • Provide clear, then full liquid diet, and assess patient response. • Progress to diabetic diet (ADA). • Consult dietitian or nutritional support service regarding special nutritional needs.
No evidence of metabolic dysfunction.	• Monitor albumin, prealbumin, transferrin, cholesterol, triglycerides, glucose, and protein levels.
Comfort/Pain Control	
Patient will have minimal pain, <5 on pain scale.	• Assess pain and discomfort. If pain present, use objective pain scale every 4 h PRN and following administration of pain medication. • If analgesics are needed, administer cautiously due to risk of respiratory and neurological complications. • Consider nonpharmacological pain management techniques (eg, distraction, touch).
The patient's nausea, vomiting, and abdominal pain or tenderness will resolve.	• Maintain nasogastric tube patency. • Assess bowel sounds every 1–2 h. • Administer antiemetic as ordered. • Provide ice chips and frequent oral hygiene.
Psychosocial	
Patient demonstrates decreased anxiety.	• Provide nonjudgmental atmosphere in which patient can discuss concerns and fears. • Provide patients who are intubated with a method to communicate. • Provide patients with decreased LOC with sensory input. • Provide for adequate rest and sleep.
Teaching/Discharge Planning	
Patient/significant others understand the tests needed for treatment.	• Prepare patient/significant others for procedures such as electroencephalography, ECG, and multiple laboratory studies.
Significant others understand the severity of the illness, ask appropriate questions, and anticipate potential complications.	• Explain the widespread effects of diabetes and the potential for complications of DKA such as seizures, renal failure, or vascular collapse. • Encourage significant others to ask questions related to complications, pathophysiology, monitoring, treatments, etc.
Patient/significant others are prepared for home care.	• Teach patient and family information needed to manage diabetes: diabetic diet, skin care, glucose monitoring, insulin administration, signs and symptoms of hypoglycemia and hyperglycemia, and appropriate actions. • Discuss sick-day management and factors that can precipitate DKA. • Initiate contacts with diabetic support groups, social services, and home health agency.

BOX 44-11 NURSING INTERVENTIONS

For Insulin Administration

- Administer insulin IV to the patient with DKA to minimize the trauma of repeated injections.
- Use only human regular insulin in IV insulin infusions because it is less antigenic than animal (beef, pork) insulins.
- Administer the insulin infusion through an IV infusion pump. Flush tubing well with insulin mixture before infusing to patient to prevent tubing from absorbing too much insulin.
- When the serum glucose level reaches 250 mg/dL, the IV fluids should be changed to a glucose-based solution.
- Changes in blood glucose level and clinical state should indicate a clear cut beneficial response to insulin and fluid replacement. If blood glucose level does not drop and blood pressure and urine output do not stabilize, insulin or fluid replacement may not be adequate.

both insulin and saline solutions drive the potassium even lower, possibly to dangerously low levels at which skeletal muscle paralysis and cardiac arrest may occur. If the initial potassium is normal or high, IV potassium is usually withheld until the level has begun to drop and urine flow is established. Potassium is usually replaced at concentrations of 20 to 40 mEq/L of IV fluid, depending on the serum potassium level. Failure of the potassium level to fall can occur for the following reasons:

- Persistent, uncorrected acidosis (which drives potassium out of cells and into ECF)
- Hyperosmolality
- Intrinsically impaired renal function
- Insufficient circulating volume

Phosphate levels usually also drop during therapy, aggravating any preexisting tendency of red blood cells to bind oxygen more tightly. Therefore, many patients receive phosphate in the middle and later phases of therapy. It is usually combined with potassium replacement in the form of potassium phosphate salts added to the IV infusion. Patients who are receiving IV phosphate therapy should be watched carefully for signs of tetany: tingling around the mouth or in the hands, neuromuscular irritability, carpopedal spasm, or even seizures. Tetany can occur because phosphate lowers the level of circulating calcium.

BICARBONATE REPLACEMENT. Patients with mild or moderate ketoacidosis who are treated with salt, water, and insulin eventually excrete and metabolize the ketone bodies remaining in ECF. As this process continues, more bicarbonate anions are reabsorbed from the renal tubules, and the bicarbonate deficit is slowly repaired. Sometimes the large amounts of chloride administered along with the sodium in IV saline solution can produce a transient hyperchloremia; this delays the full return of the bicarbonate level to normal for several days.

Bicarbonate replacement in patients with DKA remains controversial because evidence-based research has failed to demonstrate benefit in patients with an arterial pH between 6.9 and 7.1.[13] However, most experts recommend bicarbonate replacement with severe acidosis as indicated by an arterial pH of 7.0 or less. It is also necessary to give bicarbonate when there is cardiac decompensation. The bicarbonate deficit can be calculated and replaced IV over several hours to raise the level at least to the 10- to 12-mEq/L range. Sodium bicarbonate should be administered by slow IV infusion over several hours. It is administered as a bolus injection only in the case of cardiac arrest. Sodium bicarbonate administration can cause a rapid reduction in plasma potassium concentration and sodium overload.

REESTABLISHING METABOLIC FUNCTION. Gastric motility is greatly impaired in DKA. Gastric distention with dark, hemopositive fluid and vomiting is common. Abdominal pain, tenderness, and a paralytic ileus may also be due to DKA. The patient may need a nasogastric tube to decompress the stomach. This increases comfort and decreases the risk for aspiration. Patients should not eat or drink in this phase of illness. Ice chips may decrease thirst. Later, when distention lessens and motility returns, oral intake begins in order to provide the complex nutritional requirements for recovery.

Metabolic abnormalities should not be corrected too rapidly, especially in patients in whom DKA has been developing for a long time. The key risks during this phase are worsening stupor or coma, hypotension, and hyperkalemia. Osmotic or pH disequilibrium may occur when blood glucose or bicarbonate has been corrected too rapidly. The patient's mental state may worsen even though the blood chemistries are improving. Rapid reduction of blood glucose without sufficient sodium and water replacement may be responsible for hypotension. However, sepsis, myocardial infarction, and other causes of shock may also cause hypotension. Hyperkalemia usually results from premature potassium infusion, persistent acidosis, and insufficient volume replacement. However, there may be an early occlusion of the arterial supply to a limb. This can cause large amounts of potassium to leak into the circulation. Therefore, limbs are monitored for asymmetrical pallor, coolness, and rubor.

Although patients begin to improve during the initial phase of treatment, recovery usually takes place over approximately 12 days. Reversal of most metabolic abnormalities and replenishment of body stores of many nutrients (eg, magnesium, protein, and phosphate) occur during this time. Once recovery is well underway, it is time to help the patient and family understand how to prevent a recurrence.

Patient Education

Many cases of DKA are preventable with good education. Patients and families who are well informed about diabetes may be more likely to recognize early signs of complications, minimize their development, and seek help if they begin to occur. Although people usually understand the need for insulin injections when they are hungry and eating normally, they may not understand why they need their insulin when they are ill, have no appetite, are not eating, or are vomiting. Box 44-12 outlines appropriate teaching points after an episode of DKA. Box 44-13 presents a "sick day" plan for managing diabetes.

Table 44-7 Types of Insulin

Preparation	Brand	Onset (h)	Peak (h)	Duration (h)	Nursing Points
Very Rapid Acting					
Insulin analog	Humalog (Lispro) NovoLog (Aspart) Glulisine (Apidra)	<0.5	0.5–1.5	3–5	Must take with food (shorter acting than regular)
Short Acting					
Regular (R)	Humulin R Novolin R Velosulin BR	0.5–1	2–3	5–8	Only form available for IV continuous infusion
Intermediate Acting					
NPH	Humulin N Novolin N	1–4	4–12	10–16	
Insulin Regular (R)	Humulin R U-500	0.5–1.0	1.7–4	6–8	5 times the concentration of insulin; for patients on high doses of insulin
Long Acting					
Insulin glargine Insulin detemir	Lantus Levemir	1–2 0.8–2	None 3–9	24 Up to 24	
Combination					
					Mixture of long- and short-acting insulins
NPH and R (70/30)	70/30 (70% NPH, 30% regular) 50/50 (50% NPH, 50% regular)	0.5–3	Dual	12–14	
Humalog Mix (75/25)	75% insulin lispro protamine 25% insulin lispro (Humalog mix 75/25)	0.1–0.25	Dual	10–16 h	
Humalog Mix (50/50)	50% insulin lispro protamine 50% insulin lispro	0.1–0.25	Dual	10–16 h	
NovoLog Mix (70/30)	70% insulin aspart protamine 30% insulin aspart	0.1–0.25	Dual	10–16	

Hyperosmolar Hyperglycemic State

Sometimes a marked hyperglycemia and hyperosmolality without ketoacidosis develop in patients with diabetes; this is characteristic of HHS. Usually, patients with HHS are middle-aged or older (55 to 70 years), have undiagnosed type 2 diabetes, and are frequently residents of nursing homes. HHS is the initial manifestation in 7% to 17% of patients.[14]

HHS has a higher mortality rate than any other complication of diabetes, reflecting the higher morbidity associated with patients affected by this type of diabetic emergency. Often, these elderly, obese patients suffer from other severe medical conditions, such as congestive heart failure or kidney disease. Extremely high levels of glucose, coupled with severe dehydration in a vulnerable, elderly patient with other medical illnesses, explain the higher mortality rate associated with this diabetic complication. HHS is compared with DKA in Table 44-8.

Pathophysiology

It is not known specifically why some people with diabetes develop HHS rather than DKA, although it is speculated that these patients may have just enough insulin to prevent

BOX 44-12 TEACHING GUIDE Self-Management After Ketoacidosis

- A person with diabetes, as any other person, must have insulin, even if no food is being taken in.
- The amount of insulin required when the person with diabetes is not eating is about half the total needed when eating.
- The amount of insulin required when a person with diabetes is fasting must be spread out as an insulin "trickle" rather than as an insulin "burst."
- Illness generally increases the need for insulin so that even if the person with diabetes is not eating, he or she may actually require more than 50% of the usual daily dose.
- Always keep enough insulin on hand for daily injections.
- Know how to reach a health care provider for timely phone advice.
- When you are ill, adjustments for managing your diabetes may need to be made (see Box 44-13).

BOX 44-13 TEACHING GUIDE "Sick Day" Plan for Managing Diabetes

- Take your usual daily dose of insulin or oral hypoglycemic agent.
- Place an early call to your health care provider to inform him or her of your symptoms and actions.
- Monitor your blood glucose every 4 hours or a minimum of four times a day.
- Test your urine for ketones every 4 hours if your blood glucose level is 240 mg/dL or greater.
- Inject small, supplemental doses of short-acting insulin several times daily, if necessary, according to blood glucose test results until glucose levels come under control.
- Consume liberal amounts of fluids, such as water, tea, broth, apple and grape juice, and popsicles.
- Eat easily digested carbohydrates, such as custard, pudding, cream soup, saltine crackers, and toast, if you are unable to eat your normal diet.

ketosis. Pathophysiologically, the mechanisms of disease are the same as for DKA. A reduction in circulating insulin coupled with the effects of counter-regulatory hormones such as cortisol and epinephrine leads to the development of hyperglycemia and the extreme hyperosmolar state. Usually, the patient has coexisting impaired renal excretion of glucose and antecedent renal insufficiency or prerenal azotemia. Because basal insulin levels are unaffected, excessive ketone production does not occur. The acidosis that these patients develop is attributed to lactic acidosis from poor tissue perfusion instead of ketoacidosis.

HHS develops slowly over days to weeks, and patients often experience polydipsia, polyuria, and progressive decline in LOC. Marked dehydration occurs if the patient is unable to maintain an adequate fluid intake. The typical fluid loss associated with HHS is 9 L. As dehydration worsens, the patient develops increasing serum glucose concentrations and serum osmolality. The life-threatening cycle of hyperglycemia, hyperosmolality, osmotic diuresis, and profound dehydration triggers the sympathetic nervous system fight-or-flight response. The counter-regulatory hormones epinephrine and cortisol stimulate gluconeogenesis and increase hepatic

Table 44-8 Comparing Signs and Symptoms of Diabetic Ketoacidosis (DKA) and Hyperglycemic Hyperosmolar State (HHS)

Characteristics	DKA	HHS
Onset	Gradual or sudden, usually <2 d	Gradual, usually >5 d
Previous history of diabetes mellitus	85% (15% have new onset)	60%
Type of diabetes mellitus	Type 1	Type 2
Age of patient	Usually younger than 40 y	Usually older than 60 y
Mortality risk	1%–15%	20%–40%
Drug history	Insulin	Steroids, thiazides, oral agents
Physical signs	Polydipsia, polyuria, dehydration, Kussmaul's respirations, mental status changes, "fruity breath," febrile at times, ketoacidosis, nausea and vomiting	Dehydration; obtundation; hypothermia; toxic appearance; Kussmaul's respirations absent; nonketotic
Glucose level	Mean, 600 mg/dL Range, 250–1,200 mg/dL	Mean, 1,100 mg/dL Range, 400–4,000 mg/dL
Ketones	Present	Absent
Osmolarity	Mean, 320 mOsm/L	Mean, 400 mOsm/L
Arterial pH	Mean, 7.07	Mean, 7.26
Bicarbonate	Markedly low (<10 mEq/L)	Normal or >15 mEq/L
Anion gap	>12 mEq/L	<12 mEq/L, variable

Adapted from Kitabchi AE, Umpierrez GE, Miles JM, et al: Hyperglycemic crises in adult patients with diabetes. Diabetes Care 32(7):1335–1343, 2009.

glucose production. Dehydration worsens and leads to CNS dysfunction. Confusion and lethargy ensue quickly. Hemoconcentration of the blood increases the risk for clot formation, thromboemboli, and infarctions in major organs.

Causes

Infection is a major cause of HHS, occurring in 30% to 60% of patients. Urinary tract infections and pneumonia are the most common associated infections. In some cases, acute illness such as CVA, myocardial infarction, or pancreatitis provokes the release of counter-regulatory hormones resulting in hyperglycemia. HHS may also occur secondary to extreme stress associated with severe medical illness, such as stroke, myocardial infarction, pancreatitis, trauma, sepsis, burns, or pneumonia. Often, HHS results from excessive exposure or carbohydrate intake, such as dietary supplements, total enteral support with tube feedings, or peritoneal dialysis. Elderly people are at particularly high risk, especially those who have impaired cognition and who are in long-term chronic care facilities. Drugs, such as corticosteroids, thiazide diuretics, sedatives, and sympathomimetics, affect carbohydrate metabolism adversely and may lead to glucose impairment.

Assessment

HISTORY AND PHYSICAL EXAMINATION. The nurse assesses the patient for precipitating or associated events. This syndrome can be iatrogenic (eg, induced by certain medications such as steroids, hemodialysis against hyperosmolar glucose solutions, or prolonged IV hypertonic glucose infusions such as those given for total parenteral nutrition). It can also be precipitated by serious medical illnesses such as pneumonia or pancreatitis.

Often family members or long-term care personnel report that the patient has become a bit drowsy, taken in less food and fluid over several days, and slept more until he or she became difficult to awaken. The patient often arrives at the hospital with serious volume depletion and in a stupor or coma. The signs and symptoms of HHS are given in Table 44-8.

The clinical manifestations may take days to weeks to develop, and the patient often displays weakness, polyuria, polydipsia, and impaired mental state ranging from confusion to coma. Dehydration is manifested by tachycardia, hypotension, low cardiac output, poor skin turgor, rapid respirations without Kussmaul's breathing, and warm, flushed skin. The presence of hypothermia is a poor prognostic sign in HHS.

LABORATORY STUDIES. Laboratory values for HHS are similar for those with DKA with four main exceptions:

1. The hyperglycemia in HHS is, by definition, a blood glucose level greater than 600 mg/dL, and this is a significantly higher level than in DKA. Glucose can be in excess of 2,000 mg/dL.
2. Plasma osmolality is higher than in DKA and is reflective of more severe dehydration. In addition to extracellular sodium and water losses, a large additional "free water" deficit exists, probably because patients do not become thirsty, causing them to take in decreasing amounts of fluid. As a result, patients have very high serum levels of sodium and glucose. Serum osmolality is extremely high (>310 to 320 mOsm/kg).
3. Patients may have some degree of ketosis as well but usually are nonketotic. In DKA, the degree of ketosis is much more severe.
4. In HHS, acidosis is not present or is very mild. In HHS, the anion gap attributable to ketoacids usually is less than 7 mEq/L. The patient may present with azotemia, hyperkalemia, and lactic acidosis.

Management

Therapy for HHS is directed at correcting the volume depletion, controlling hyperglycemia, identifying the underlying cause of HHS and treating it. The volume depletion is usually greater in HHS than in DKA. Rapid rehydration is more cautiously carried out because of the fragile state of the patient, who often has comorbidities. Isotonic saline or hypotonic saline solution is administered initially to correct the fluid imbalance, and some patients may require as many as 9 to 12 L of fluid overall. The nurse should be vigilant for the signs of fluid overload during rehydration.

Critically ill patients require hemodynamic monitoring during fluid resuscitation, especially elderly patients with cardiac or renal disease. Careful monitoring of fluid intake, urine output, blood pressure, hemodynamic pressures, pulse, breath sounds, and neurological status is one of the nurse's primary responsibilities. In addition, the patient requires frequent monitoring of laboratory test findings.

Patients receive low doses of insulin along with the fluid replacement. It is necessary to give low-dose insulin by continuous infusion (0.1 mg/kg/h) because these patients are vulnerable to the sudden loss of circulating blood volume that occurs with higher doses of insulin and a rapid blood glucose reduction. As the glucose level returns close to normal (250 to 300 mg/dL), it is appropriate to stop the insulin infusion and add dextrose to the IV fluids to prevent a sudden drop in the blood glucose level. At this point, subcutaneous administration of insulin can proceed.

Investigation of the underlying cause of HHS is warranted, and treatment, if possible, is necessary. For example, management of underlying infection from pneumonia involves aggressive use of antibiotics, chest physiotherapy, turning, and coughing and deep breathing or suctioning as needed to clear the infiltrate. Removal of exogenous sources of glucose (tube feedings, peritoneal dialysis, medications) is appropriate while treating the hyperglycemic state.

Older patients who develop HHS have frequent complications and high mortality rates. They often have difficulty handling the fluid volume shifts that occur during the development and treatment of this disorder. The nurse gives fluid slowly to avoid complications associated with cerebral edema, such as seizures and an altered neurological state. These patients are also at risk for intravascular thrombosis and focal seizures because of the hemoconcentration of the blood and the hyperosmolar state. Use of seizure precautions is necessary at all times. Treatment of acute cerebral edema usually involves infusion of osmotic diuretic such as 20% mannitol. Because these patients usually have a preexisting cardiac or renal history that predisposes them to complications, fluid resuscitation should proceed slowly and carefully.

Critical care management continues until the patient's hyperglycemic state has stabilized, his or her neurological condition and vital signs return to normal, and the precipitating

cause has resolved. Discharge criteria also include having an adequate plan in place for the patient to maintain glycemic control and avoid future hyperglycemic emergencies.

Patient Education

As with patients with DKA, the patient and family experiencing HHS need education. The prevention of many cases of HHS and DKA entails better access to medical care, proper education, and effective communication with a health care provider during an intercurrent illness. Many uninsured or underinsured patients stop insulin therapy for economic reasons. The nurse must assess for this possibility.

For the person with newly diagnosed diabetes, the nurse needs to provide information about the pathophysiology of the disease, the signs and symptoms of complications, and methods of treatment, including medications, diet, and exercise. Information about how to manage "sick days" and other tips to avoid acute complications such as HHS must be part of the educational plan.

The patient may need instruction on home management and glucose testing. Often the critical care staff consults with the diabetes educator as the patient's educational plan is being formulated. The main theme should focus on effective techniques to avoid emergency intervention in the future. It is necessary to make appropriate referrals to a diabetic educator, social worker, dietitian, or a combination of these because diabetes outcomes are optimized when a team approach is used.

Hypoglycemia

Hypoglycemia is a well-recognized complication among patients with type 1 diabetes, and it is the most common diabetes-related emergency.[16] The issue of hypoglycemia is well documented in the landmark DCCT,[9] in which people with diabetes who maintained strict, intensive therapy for their diabetes experienced a threefold greater incidence of severe hypoglycemia than those patients with less strict treatment protocols. The UKPDS[10] demonstrated some increased incidence of hypoglycemia among people with type 2 diabetes, although few severe, life-threatening cases were documented in the study.

Insulin-induced hypoglycemia reactions often occur in the midst of the patient's daily life; this can be, at the very least, embarrassing, and at worst, dangerous. Mild hypoglycemia causes unpleasant symptoms and discomfort; however, severe hypoglycemia can lead to life-threatening complications such as seizures, coma, and even death if not reversed. Even though measurable recovery from hypoglycemia is rapid and complete within minutes after proper treatment, many patients remain emotionally (and possibly physiologically) shaken for hours or even days after insulin reactions. In extreme situations, prolonged or recurrent hypoglycemia, although uncommon, has the potential to cause permanent brain damage and can even be fatal.

Pathophysiology

Minute-to-minute dependence of the brain on glucose supplied by the circulation results from the inability of the brain to burn long-chain FFAs, the lack of glucose stored as glycogen in the adult brain, and the unavailability of ketones. The brain recognizes its energy deficiency when the serum glucose level falls abruptly to about 45 mg/dL. The term neuroglycopenia refers to the degree of hypoglycemia sufficient to cause brain dysfunction resulting in personality changes and intellectual deterioration. The exact level at which symptoms occur varies widely from person to person, however, and it is not uncommon for levels as low as 30 to 35 mg/dL to occur (eg, during glucose tolerance tests) with no symptoms whatsoever among the long-term diabetic population. See Box 44-14 for common signs and symptoms of hypoglycemia.

BOX 44-14 Signs and Symptoms of Hypoglycemia

Signs
- Hypothermia
- Tachypnea
- Tachycardia
- Dysrhythmias
- Hypertension
- Diaphoresis
- Tremulousness
- Hunger
- Nausea
- Belching

Symptoms
- Headache
- Personality changes
- Palpitations
- Blurred vision
- Combativeness, confusion, coma, convulsions

Symptoms result from either the sympathetic nervous system response to hypoglycemia or the neuroglycopenic response. The hypothalamus reacts to the lower glucose levels to mount the adrenergic response, including tachycardia, palpitations, tremors, and anxiety. The goal is to activate the counter-regulatory hormones (glucagon, catecholamines, cortisol, GH) to raise the glucose level and protect vital organs from hypoglycemia. This involves glycogenolysis and gluconeogenesis.

Assessment

Occasional reactions occur in even the most stable insulin-dependent diabetic patient. As long as the reactions are mild, they can usually be tolerated without difficulty and are not cause for alarm or for changes in regimen. Frequently, the precipitating event is clear (eg, a skipped meal or an unusually strenuous bout of exercise). Box 44-15 reviews the common causes of hypoglycemia.

When hypoglycemic reactions are frequent, recurrent, or severe, it is important to identify the cause and prevent further reactions. Otherwise patients may limit their functional activities and may become unwilling or unable to drive. They may overeat in an effort to prevent reactions. Usually the underlying mechanism can be discovered. If not, the patient should be admitted for work up and further evaluation.

HISTORY AND PHYSICAL EXAMINATION. The nurse asks about food intake and exercise because these often contribute to hypoglycemia. Problems with insulin dosage or

BOX 44-15 Common Causes of Hypoglycemia

- Insulin shock
- Insulinoma
- Inborn errors of metabolism
- Stress
- Weight loss
- Postgastrectomy
- Alcohol excess
- Glucocorticoid deficiency
- Fasting hypoglycemia
- Profound malnutrition
- Prolonged exercise
- Severe liver disease
- Severe sepsis
- Drug effects
  - Ethanol
  - Salicylates
  - Quinine
  - Haloperidol
  - Insulin
  - Sulfonylureas
  - Sulfonamides
  - Allopurinol
  - Clofibrate
  - β-Adrenergic agents

administration may be noted. It is necessary to investigate every detail of insulin therapy thoroughly, including insulin purchase and its appearance, species, and units; syringes, injection sites, and injection technique; and especially any recent change in any part of the regimen. The nurse explores for flaws and inconsistencies in reporting. Prescription errors, mismatched syringe and insulin units, use of new injection sites, and other errors may emerge.

The administration or withdrawal of other drugs may be the precipitating event for recurrent insulin reactions. For example, salicylates in large doses can reduce blood glucose and, in combination with insulin, can produce hypoglycemia. Also, the nurse asks about the use of glucocorticoid medications. Because these medications cause insulin resistance, insulin doses are often raised to meet the increased insulin demand. If the steroids are then tapered without reducing the insulin dose, hypoglycemic reactions can occur. Alcohol often causes hypoglycemia. Not only do patients often eat less when they have a few drinks, but also alcohol shuts off gluconeogenesis by interfering with intermediate biochemical steps in the liver. When combined with injected insulin, this frequently leads to hypoglycemia. Oral hypoglycemic agents can also produce severe and long-lasting hypoglycemia. Patients who experience such episodes tend to be older and undernourished with impaired renal or hepatic function. Nevertheless, any patient on oral agents can become hypoglycemic, especially when substances such as salicylates and alcohol potentiate the effects of the oral agents.

Another common mechanism that can cause hypoglycemia is an atypical (eg, early or late) response to insulin therapy. Once the response pattern is defined, it is possible to adjust the insulin regimen and eliminate the insulin reactions. Occasionally, when a stable, reaction-free patient begins to experience hypoglycemic episodes, the clinician should explore the likelihood of insulin sensitivity due to weight loss or the onset of azotemia.

As the blood glucose level falls below normal, the CNS responds in two distinct ways: first, with impairment of higher cerebral functions, and second, soon thereafter, with an "alarm" response in vegetative functions. Patients most commonly describe the symptoms of mild or early insulin reactions as fuzziness in the head, trouble thinking or concentrating, shakiness, light-headedness, or giddiness. These changes occur when the cerebral cortex is deprived of its main energy supply, usually when the blood glucose level has fallen to 50 mg/dL or less or is rapidly declining. The cerebral cortex is apparently the most sensitive to the loss of glucose.

Changes in personality and behavior vary with the person and may not be apparent to them during an insulin reaction. Changes range from silly, manic, inappropriate behavior to withdrawn, sullen, grumpy, irritable, suspicious behavior. There may be difficulties in motor function, such as trouble walking and slurred speech, and patients who are well into insulin reactions may closely resemble people who have been drinking alcohol in excess.

Some patients experience aphasia, vertigo, localized weakness, and even focal seizures with their insulin reactions. Such focal changes usually occur when there is prior damage to a specific area of the cortex, such as a head injury or CVA.

Closely following the cortical changes is a series of autonomic neurological responses. The primary response is discharge from the centers that control adrenergic autonomic impulses. This results in the release of norepinephrine throughout the body and epinephrine from the adrenals. Tachycardia, pallor, sweating, anxiety, and tremor are characteristic signs of hypoglycemia and are important early warning signs for patients who recognize a reaction. Headache can occur, and the stress response can occasionally trigger secondary sequences of symptoms, including angina or pulmonary edema in patients with fragile cardiovascular disease.

As hypoglycemia persists and worsens, consciousness is progressively impaired, leading to stupor, seizure, or coma. This is characteristic of severe hypoglycemia. The autonomic centers controlling fundamental systems, such as respiration and blood pressure, are the most resistant to hypoglycemia and continue to function even when most other cerebral functions are lost.

The more profound the hypoglycemia and the longer it lasts, the greater the chance of transient or even permanent cerebral damage after the blood glucose level is restored. There does not seem to be a clear duration threshold for such damage, but severe hypoglycemia lasting more than 15 to 30 minutes can result in some symptoms that persist for a time after glucose is given. Blood glucose measurement, before the administration of glucose if possible, verifies the diagnosis; but waiting for a glucose test result should be avoided in an emergency situation.

Management

Treatment for insulin reactions is always glucose. If the patient can swallow, the most convenient form is a glucose- or sucrose-containing drink because it probably gets through the stomach and into the absorbing intestine in this form in the shortest possible time. If the patient is too groggy, stuporous, or uncooperative to drink, the glucose is in the form of an IV bolus of 25 g of 50% dextrose given over several minutes. If this route or dosage is unavailable, 1 mg of glucagon given subcutaneously or intramuscularly reverses the symptoms by inducing a rapid breakdown and release of glucose into the bloodstream from hepatic glycogen stores.

The amount of glucose needed to reverse an insulin reaction acutely is not large. In an average-sized adult, less than 15 g (three teaspoons) of glucose can raise the blood glucose from 20 to 120 mg/dL. Glucose in almost any oral form will serve. Typical treatments for hypoglycemia include three glucose tablets, 6 oz of regular cola, 6 oz of orange juice, 4 oz of skim milk, or 6 to 8 Lifesaver candies. Starch, found in crackers and cookies, is broken down to free glucose after passing through the stomach and is absorbed so rapidly that blood glucose rises virtually as fast as with free glucose or sucrose.

Patients frequently express concern about what to do if they do not respond to the initial therapy, and they fear that they might "never wake up" from a nocturnal insulin reaction. The nurse must reassure them that if the first bolus of glucose consumed does not seem to work, the sensible thing to do is to take in more. Insulin reactions are always reversible with enough glucose. The response to oral glucose, of course, takes time, perhaps 5 to 15 minutes, whereas the response to IV glucose should occur within 1 or 2 minutes at most. Failure to respond fully in the appropriate time indicates that not enough glucose has been given, that the diagnosis is incorrect, or that the hypoglycemia has been long and severe enough to produce persistent, although not necessarily permanent, cerebral dysfunction.

Patient Education

The nurse should teach all people with diabetes to report hypoglycemic reactions to their health care provider for adjustments in their medical regimen. If they are taking insulin, they should know when to expect peak effects of the drug so that they can predict high-risk times for hypoglycemia. They should always carry a high-glucose snack with them for emergency use. The nurse should encourage them to carry medical identification at all times.

▲ Clinical Applicability Challenges

CASE STUDY

J., a 17-year-old female, was admitted to the hospital with symptoms of fever, nausea, vomiting and right flank pain for 2 days. She had been diagnosed with diabetes mellitus at 7 years of age and has been taking a maintenance insulin regimen of 18 units of longer-acting insulin glargine (Lantus) at bedtime and a sliding scale dose of aspart (NovoLog) before meals. For the past 2 days, she omitted her insulin because she was not eating anything.

Her vital signs are temperature 102°F; pulse, 120 beats/min; respirations, 32 breaths/min and deep; and blood pressure, 80/52 mm Hg. She is lethargic but fully oriented, complaining of chills and flank pain. Admission laboratory work reveals hematocrit, 48.6%; white blood cells, 36,400/mm^3; glucose, 610 mg/dL; sodium, 138 mEq/L; potassium, 5.7 mEq/L; chloride, 90 mEq/L; bicarbonate, 4 mEq/L; blood urea nitrogen, 43 mg/100 mL; creatinine, 2.2 mg/dL; serum ketones, 4+; and urine glucose and ketones 4+, blood 4+, leukocyte esterase+ and nitrites +. Arterial blood gas values are arterial blood pH, 7.06; PaO_2, 90 mm Hg; $PaCO_2$, 13 mm Hg; and bicarbonate, 2.5 mEq/L. Her contrast-enhanced helical/spiral computed tomography scan confirms right-sided pyelonephritis.

Emergency department physicians diagnose J. with moderate to severe diabetic ketoacidosis with right pyelonephritis. The initial therapy consists of several liters of normal saline solution infused intravenously and 20 units regular insulin by intravenous push, followed by an infusion of insulin at 5 units/h during the first 6 hours. She is immediately started on broad-spectrum antibiotics, antipyretics, and morphine for pain. Her mental status improves as her pain is controlled and she is rehydrated.[17] The flow sheet below summarizes the biochemical changes over the first 15 hours.

Biochemical Flow Sheet Indicating Diabetic Ketoacidosis for J.

Time	Sugar	pH	Na	K	Cl	HCO_3	BUN/ Creatinine
1:00 PM	610	7.06	138	5.7	90	4	40/2.1
3:00 PM	492		137	4.8	101	6	41/1.7
5:15 PM	375	7.25	137	4.1	106	8	45/1.4
10:00 PM	303		139	4.7	114	15	27/1.2
4:00 AM	204		143	4.3	113	22	22/1.1

By the time of discharge 5 days later, J. is eating well, her fever is gone, and she has well-controlled blood glucose levels on her usual doses of insulin glargine and insulin aspart.

1. J. receives the diagnoses of ketosis and acidosis. What are the indicators of ketosis and acidosis?

2. J. experiences volume depletion and electrolyte imbalances. Why does this occur and what are the clues?

3. What are the main teaching points regarding future management of J.'s diabetes in this case?

References

1. Sharma ST, Nieman LK: Cushing's syndrome: All variants, detection, and treatment. Endocrinol Metab Clin North Am. 40(2):379–391, viii–ix, 2011
2. Lee SL, Ananthakrishnan S: Hyperthyroidism. eMedicine Medscape.com. Updated October 27, 2011. Retrieved May 29, 2012. Available at: http://emedicine.medscape.com/article/121865
3. Bharaktiya S, Orlander PR, Woodhouse WR, et al: Hypothyroidism. eMedicine Medscape.com Updated 2010. Retrieved May 23, 2010. Available at: http://emedicine.medscape.com/article/122393-overview
4. Marik PE, Pastores SM, Annane D, et al: Recommendations for the diagnosis and management of corticosteroid insufficiency in critically ill adult patients: Consensus statements from an international task force by the American College of Critical Care Medicine. Crit Care Med 36(6): 1937–1949, 2008
5. Hahner S, Allolio B: Therapeutic management of adrenal insufficiency. Best Pract Res Clin Endocrinol Metab 23(2):167–179, 2009
6. American Diabetes Association: Diabetes statistics, 2010. Retrieved May 27, 2012. Available at: http://www.diabetes.org/diabetes-basics/diabetes-statistics/
7. Hussain AN, Vincent MT: Diabetes mellitus type 1, 2010. eMedicine Endocrinology. Retrieved May 9, 2010. Available at: http://emedicine.medscape.com/article/117739-overview
8. Henderson KE, Baranski TJ, Bickel PE, et al: Endocrinology Subspecialty Consult, 2nd ed. Philadelphia, PA: Wolters Kluwer Health | Lippincott Williams & Wilkins, 2009
9. Diabetes Control and Complications Trial (DCCT) Research Group: The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. N Engl J Med 329:977–986, 1993
10. United Kingdom Prospective Diabetes Study Group: Tight blood pressure control and risk of macrovascular and microvascular complications in type 2 diabetes (UKPDS 38). BMJ 317:703–713, 1998
11. Effects of intensive glucose lowering in type 2 diabetes. The Action to Control Cardiovascular Risk in Diabetes Study Group (AACORD Trial): New Engl J Med 358(24):2545–2559, 2008
12. Intensive versus conventional glucose control in critically ill patients. The NICE-SUGAR Study Investigators (Normoglycemia in Intensive Care Evaluation–Survival Using Glucose Algorithm Regulation): New Engl J Med 360(13):1283–1297, 2009
13. Kitabchi AE, Umpierrez GE, Miles JM, et al: Hyperglycemic crises in adult patients with diabetes. Diabetes Care 32(7):1335–1343, 2009
14. Kitabchi AE, Murphy MB, Spencer J, et al: Is a priming dose of insulin necessary in a low-dose insulin protocol for the treatment of diabetic ketoacidosis? Diabetes Care 31:2081–2085, 2008
15. Rosenbloom AL: The management of diabetic ketoacidosis in children. Diabetes Ther 1(2):103–120, 2010
16. Goh HK, Chew DE, Miranda IG, et al: 24-Hour observational ward management of diabetic patients presenting with hypoglycaemia: A prospective observational study. Emerg Med J 26(10):719–723, 2009
17. Shoff WH, Green-McKenzie J, Edwards C, et al: Pyelonephritis, acute. eMedicine Medscape.com. Updated March 2010. Available at: http://emedicine.medscape.com/article/245559-overview, 2010

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

PART 11

HEMATOLOGICAL AND IMMUNE SYSTEMS

45

Anatomy and Physiology of the Hematological and Immune Systems

Thomasine D. Guberski

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Describe the blood and its components and the function of each component.
2. Delineate the clotting factors and the role each plays in coagulation.
3. Describe the anatomy and physiology of the immune system.
4. Differentiate between innate and adaptive immunity including humoral and cell-mediated immunity.

Because cells for both the hematological and the immune systems originate in bone marrow, the systems are interrelated. As a result, a change in one system can manifest itself in the other system. For example, a decrease in the number of white blood cells results in an immune system that is less able to resist infection. The anatomy and physiology of these two systems are discussed separately in this chapter, but the reader should keep in mind their close relationship.

▲ Hematological System

Veins, venules, capillaries, arterioles, and arteries constitute an intricate network of conduits for the transportation of blood, which carries respiratory gases, nutrients, and waste products to and from body tissue. Patency of the conduits and containment of blood within the vasculature depend on the integrity of the transporting conduits. A delicate balance must be maintained in the vasculature to ensure both its patency and the liquid state of blood so that neither thrombosis nor hemorrhage occurs. This delicate balance is provided by the hemostatic and fibrinolytic systems working in concert.

Blood and Its Functions

Blood is an aqueous solution of colloid and electrolytes that serves as a medium of exchange between body cells (interior environment) and the exterior, or external, environment. It has distinct characteristics, including variable color (arterial blood is bright red; venous blood is dark red), viscosity (blood is three to four times thicker than water), a pH of 7.35 to 7.4, and a volume of approximately 70 to 75 mL/kg of body weight (5 to 6 L). Plasma constitutes approximately 55% of blood volume, whereas cellular elements suspended in the plasma constitute the remaining 45%. The vital functions of blood are as follows:

- Transport of oxygen and absorbed nutrients to cells
- Transport of carbon dioxide and other waste products to the lungs, kidneys, gastrointestinal system, and skin
- Transport of hormones from endocrine glands to target organs and tissues
- Protection of the body from life-threatening microorganisms
- Regulation of acid–base balance
- Protection from blood loss through hemostasis
- Regulation of body temperature by heat transfer

See Chapter 16 for a more detailed discussion of circulation.

Components of Blood

Plasma

Plasma, the liquid portion of the blood contains a wide variety of organic and inorganic components (Table 45-1). The concentration of these components reflects diet, metabolic demand, hormones, and vitamins. Plasma is approximately 90% water and 10% dissolved solutes. The most prevalent solutes by weight are the plasma proteins and clotting factors. Serum is plasma that has had clotting proteins removed.

Plasma proteins play a role in transport, volume regulation, immune function, and coagulation. Most plasma proteins, including albumin and fibrinogen, are synthesized by the liver; however, the immunoglobulins are synthesized by B lymphocytes. Albumin is essential for regulation of the colloidal osmotic pressure, which is critical for movement of water and solutes through the microcirculation. Plasma also is a carrier molecule for normal blood components and exogenous agents, such as drugs. Immunoglobulins (antibodies) are essential for defense against infectious microorganisms (see the Immune System section in this chapter for a further description). Fibrinogen is the clotting factor that forms the fibrin clot, essential to the clotting cascade.

Lipoproteins, which include the plasma lipids, triglycerides, phospholipids, cholesterol, and fatty acids, are carried through the blood as complexes with the plasma proteins. Also contained within the plasma, electrolytes (sodium, potassium, calcium, magnesium, chloride, bicarbonate, phosphate, and sulfate) maintain the pH and osmolality of the blood. Plasma nutrients, such as glucose, and gases, such as oxygen and carbon dioxide, are circulated to and from the tissues. Waste products, a final component of plasma, are carried to the appropriate organ for excretion.

Table 45-1 Organic and Inorganic Components of Arterial Plasma

Constituent	Amount/Concentration	Major Functions
Water	93% of plasma weight	Medium for carrying all other constituents
Electrolytes	Total <1% of plasma weight	Maintain H_2O in extracellular compartment; act as buffers; function in membrane excitability
Na^+	142 mEq/L (142 mM)	
K^+	4 mEq/L (4 mM)	
Ca^{2+}	5 mEq/L (2.5 mM)	
Mg^{2+}	3 mEq/L (1.5 mM)	
Cl^-	103 mEq/L (103 mM)	
HCO_3^-	27 mEq/L (27 mM)	
Phosphate (mostly HPO_4^{2-})	2 mEq/L (1 mM)	
SO_4^{2-}	1 mEq/L (0.5 mM)	
Proteins	7.3 g/dL (2.5 mM)	Provide colloid osmotic pressure of plasma; act as buffers; bind other plasma constituents (eg, lipids, hormones, vitamins, minerals, etc.); clotting factors; enzymes; enzyme precursors; antibodies (immune globulins); hormones; transporters
Albumins	4.5 g/dL	
Globulins	2.5 g/dL	
Fibrinogen	0.3 g/dL	
Transferrin	250 mg/dL	
Ferritin	15–300 mcg/L	
Gases		
CO_2 content	22–32 mmol/L plasma	Byproduct of oxygenation, most CO_2 content from HCO_3 and acts as a buffer
O_2	PaO_2 80 torr or greater (arterial); PvO_2 30–40 torr (venous)	Oxygenation
N_2	0.9 mL/dL	Byproduct of protein catabolism
Nutrients		Provide nutrition and substances for tissue repair
Glucose and other carbohydrates	100 mg/dL (5.6 mM)	
Total amino acids	40 mg/dL (2 mM)	
Total lipids	500 mg/dL (7.5 mM)	
Cholesterol	150–250 mg/dL (4–7 mM)	
Individual vitamins	0.0001–2.5 mg/dL	
Individual trace elements	0.001–0.3 mg/dL	
Iron	50–150 mcg/dL	
Waste products		
Urea (blood urea nitrogen)	7–18 mg/dL (5.7 mM)	End product of protein catabolism
Creatinine (from creatine)	1 mg/dL (0.09 mM)	End product from energy metabolism
Uric acid (from nucleic acids)	5 mg/dL (0.3 mM)	End product of protein metabolism
Bilirubin (from heme)	0.2–1.2 mg/dL (0.003–0.018 mM)	End product of red blood cell destruction
Individual hormones	0.000001–0.05 mg/dL	Functions specific to target tissue

From McCance KL, Heuther SE: Pathophysiology: The Biologic Basis for Disease in Adults and Children, 6th ed. St. Louis, MO: Mosby, 2010, p 953.

Cellular Elements

Summarized in Table 45-2 along with their functions, the cellular elements of the blood are erythrocytes (red blood cells), leukocytes (white blood cells), and platelets. All blood cell types are believed to be derived from a single stem cell known as the pluripotent stem cell, as shown in Figure 45-1. The production of blood cells (hematopoiesis) occurs in the bone marrow and is a two-stage process that involves mitotic division (proliferation) and maturation (differentiation). Each blood cell line results from pluripotential cells that become committed to a specific cell line when they receive specific biochemical signals. These signals occur when one or more populations of circulating cells have decreased to a certain level. Mitosis occurs, and proliferation continues until the needed number of mature daughter cells enters the circulation.

Erythrocytes

There are approximately 5 million erythrocytes per cubic millimeter of blood. They are produced in the red bone marrow found in the sternum, ribs, skull, vertebrae, and bones of the hands, feet, and pelvis. Normal cell formation requires nutrients such as iron, vitamin B_{12}, folic acid, and pyridoxine. Reticulocytes (immature nucleated red blood cells) are released from the bone marrow and circulate for 1 to 2 days while maturing into adult cells. The average life span of an erythrocyte is 115 to 130 days. Dead red blood cells are eliminated mainly by phagocytosis in the liver and spleen.

The erythrocyte is a small, biconcave disk that is reversibly deformable. The flattened, biconcave shape has a surface area: volume ratio that is optimal for the diffusion of gases into and out of the cell. Reversible deformability allows the cell to alter its shape to squeeze through the microcirculation and then return to its normal shape.

Hemoglobin is the iron-containing substance of the erythrocyte. The normal amount of hemoglobin in the body is 12 to 18 g/dL of blood, with the lower level more common in women and the higher level more common in men. Hemoglobin is composed of a red compound called heme (which contains iron and porphyrin) and a simple protein called globin. One iron atom is present for each heme molecule. Total body iron ranges from 2 to 6 g. Two thirds of this is in hemoglobin, whereas the rest is stored in the bone marrow, spleen, and liver. When red blood cells break down, hemoglobin splits

Table 45-2 Cellular Components of the Blood

Cell	Structural Characteristics	Normal Amounts in Circulating Blood	Function	Life Span
Erythrocyte (red blood cell)	Nonnucleated cytoplasmic disk containing hemoglobin	4.2–6.2 million/mm^3	Gas transport to and from tissue cells and lungs	80–120 d
Leukocyte (white blood cell)	Nucleated cell	5,000–10,000/mm^3	Bodily defense mechanisms	See below
Lymphocyte	Mononuclear immunocyte	25%–33% of leukocyte count (leukocyte differential)	Humoral and cell-mediated immunity	Days or years, depending on type
Monocyte and macrophage	Large kidney-shaped mononuclear phagocyte	3%–7% of leukocyte differential	Phagocytosis; mononuclear phagocyte system	Months or years
Eosinophil	Segmented polymorphonuclear granulocyte with granules stainable by eosin dyes	1%–4% of leukocyte differential	Phagocytosis; response to parasites, control of allergic reactions;	8–12 d
Neutrophil	Segmented polymorphonuclear granulocyte with granules stainable by neutral staining	57%–67% of leukocyte differential	Phagocytosis, particularly during early phase of inflammation, bacterial killing	4 d
Basophil	Lobate nuclear granulocyte with granules stainable by basic dyes	0%–0.75% of leukocyte differential	Similar to mast cell, secretes inflammatory mediators (eg, histamine, chemotactic factors for eosinophils and neutrophils), involved with allergic reactions	Few hours to days
Platelet	Irregularly shaped cytoplasmic fragment (not a cell)	140,000–340,000/mm^3	Hemostasis following vascular injury; normal coagulation and clot formation/retraction	8–11 d

From McCance KL, Huether SE: Pathophysiology: The Biologic Basis for Disease in Adults and Children, 5th ed. St. Louis, MO: Mosby, 2010, p 954.

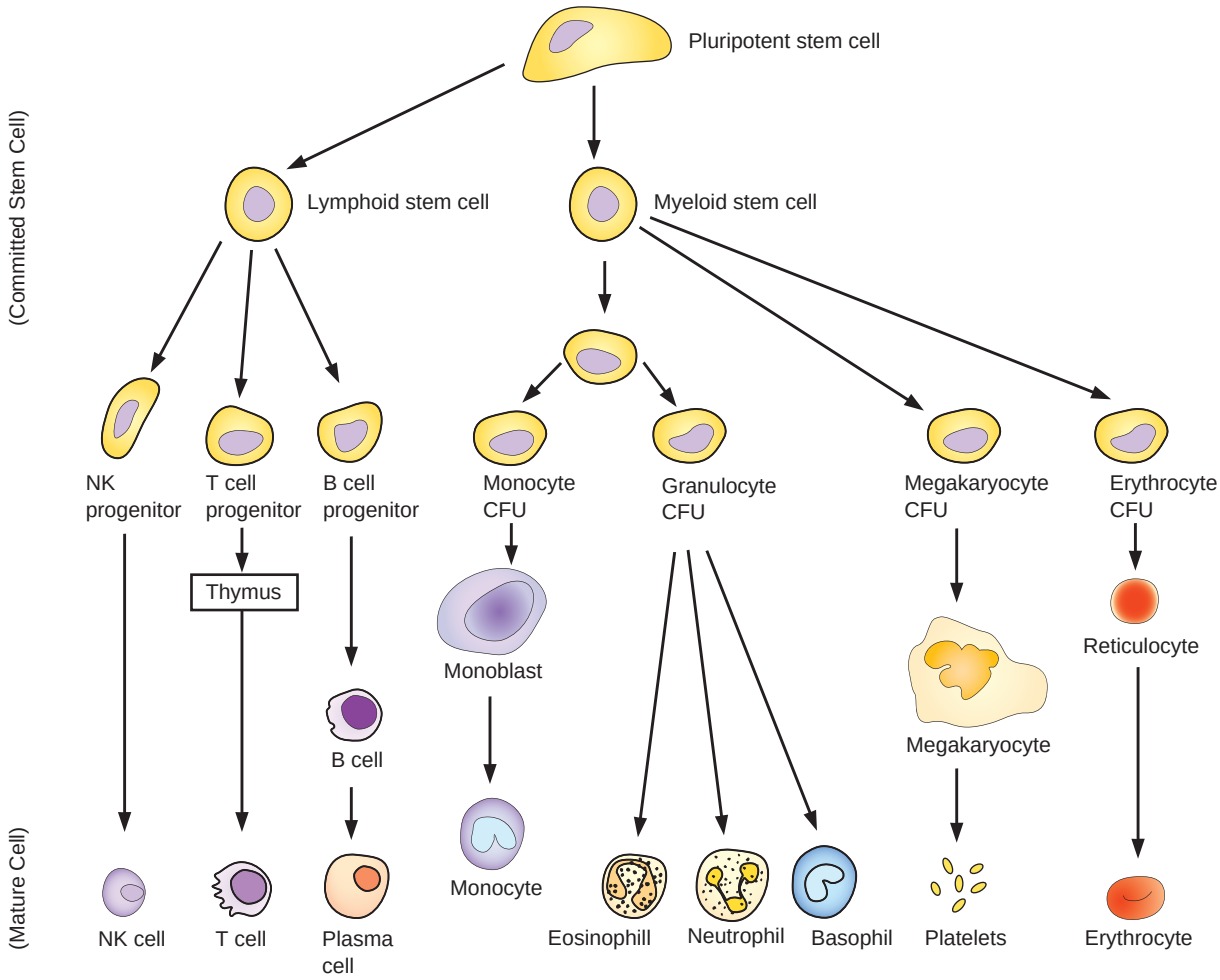


FIGURE 45-1 ▲ Major maturational stages of blood cells. CFU, colony-forming unit; NK, natural killer cell. (From Porth CM: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 259.)

into heme and globin factors. The liver stores the iron portion of heme for production of new hemoglobin, and the remainder is converted into bilirubin, which is excreted in feces and urine after conjugation by the liver. This conjugation process is important to the excretion of bilirubin, which causes jaundice when it accumulates in the tissues (see Chapter 38). Each red blood cell contains 200 to 300 million molecules of hemoglobin, which combines with oxygen to form oxyhemoglobin. Hemoglobin also combines with carbon dioxide. Thus, the blood can carry oxygen to the tissues and carbon dioxide from the tissues to the alveoli of the lungs, where it is expelled into the atmosphere. Hemoglobin can also combine preferentially with carbon monoxide, which displaces oxygen.

Respiration is a major function of erythrocytes. Hemoglobin combines with oxygen in the lungs. The saturation of hemoglobin with oxygen is influenced by the partial pressure of oxygen available in the lungs, the temperature of the blood, the pH of the blood, and the amount of intracellular 2,3-biphosophyglycerate. For example, people who live at sea level and vacation in high altitudes, where the partial pressure of oxygen in the lungs is lower, may experience shortness of breath with activity because less oxygen is available to combine with hemoglobin.

Leukocytes

Leukocytes, or white blood cells, are transported in the circulation but act primarily in the body tissues, defending the body against microorganisms and foreign antigens and removing debris, such as dead or injured host cells. There are approximately 5,000 to 10,000 white blood cells per cubic millimeter of blood. The two major categories of leukocytes are granulocytes and agranulocytes.

Granulocytes make up approximately 70% of all white blood cells and include neutrophils, eosinophils, and basophils. They are produced by the bone marrow from myeloid stem cells, and their function depends on the type of enclosed granule. Polymorphonuclear leukocytes, or neutrophils, fight bacterial and fungal infections and digest foreign particulate matter or break down products from cells through phagocytosis. Neutrophils are present during the early acute phase of an inflammatory reaction. After bacterial invasion or tissue injury, they migrate from the capillaries into the inflamed area, reaching their peak activity in 6 to 12 hours. In the inflamed area, they destroy and ingest microorganisms and other debris. They die in 1 or 2 days, releasing digestive enzymes that dissolve cellular debris and prepare the inflamed site for healing.

Eosinophils are particularly important in detoxifying foreign protein. They ingest antigen–antibody complexes, attack parasites, and are elevated during allergic reactions. Eosinophils have surface receptors for immunoglobulins and histamine.

Basophils contain cytoplasmic granules with vasoactive amines (histamine, bradykinin, and serotonin), which are thought to play a role in the symptoms of acute systemic allergic reactions. Basophils also contain the anticoagulant heparin, histamine, and other vasoactive substances.

Agranulocytes (monocytes, macrophages, and lymphocytes) are leukocytes that do not contain lysosomal granules in their cytoplasm. Agranulocytes also come from the myeloid stem cell. Monocytes (immature macrophages) and macrophages comprise the mononuclear phagocyte system. They are responsible for the phagocytosis of dead leukocytes and erythrocytes in the blood and for processing antigenic material as neutrophils start to decrease in number. Some of the circulating macrophages migrate out of the blood vessels in response to inflammation or infection, whereas others migrate to fixed sites in lymphoid tissues of the liver, spleen, lymph nodes, peritoneum, or gastrointestinal tract, where they may remain active for months or years. Lymphocytes are immunocompetent cells that are involved in producing antibodies and maintaining the immune response. The most important classifications are B and T lymphocytes, which are discussed later in the chapter.

Platelets

Platelets are disk-shaped cytoplasmic fragments formed from bone marrow stem cells, specifically, a giant cell called the megakaryocyte. Platelets maintain capillary integrity, accelerate coagulation, and retract clots. There are approximately 250,000 to 500,000 platelets per cubic millimeter of blood; one third resides in a reserve pool in the spleen. Platelets live about 10 days; when they die, they are removed from the circulation by macrophages, mostly in the spleen.

Blood Coagulation

Hemostatic homeostasis is maintained through three interdependent components: blood vessels, platelets, and blood coagulation factors. In the course of normal wear and tear, the endothelial lining of blood vessels is subject to damage that requires local repair to prevent blood leakage. The body repairs the vessels through a process called coagulation. In this process, damage to, or sloughing of, the endothelium exposes the underlying collagen. This exposed collagen attracts and activates platelets to adhere to it, which begins the formation of platelet plugging. With the attraction of platelets to the exposed collagen, an initial barrier of platelets is formed. These platelets release small amounts of adenosine diphosphate, which attracts additional platelets that stick to each other. Following this process, there is a release of platelet factor 3 from the platelet membrane, which interacts with various blood coagulation proteins and accelerates clotting.

Platelets play two major roles in the clotting process. First, the platelet plug temporarily plugs the leak in the blood vessel. This plug provides the architectural foundation for the building of the fibrin clot. Second, platelets initiate clotting by way of the intrinsic pathway through the release of platelet factor 3.

Coagulation Factors

Coagulation factors are designated by Roman numerals and numbered according to the order in which they were first identified. When the factors are in active form, they are designated by a lowercase "a" (eg, factor XIIa). Box 45-1 lists the factors by Roman numeral and common name.

Coagulation Pathways

Blood coagulation proteins, or coagulation factors, are found in the extrinsic and intrinsic pathways to coagulation. Tissue factor TFVIIa activates the coagulation pathway.[1] There is interplay between the extrinsic and intrinsic pathways and activation of factor VII, found in the extrinsic pathway, can activate intrinsic pathway factor XI. Likewise, several factors in the intrinsic pathway activate factor VII.

Extrinsic Pathway

The extrinsic pathway is a series of chemical reactions that originate outside the injured structure. Critical steps are shown in Figure 45-2. Injury to tissues and blood vessels triggers coagulation and results in the release of factor III (thromboplastin) into the circulation. Factor III, catalyzed by factor VII (proconvertin), activates factor X (Stuart-Prower factor). In the presence of calcium ions, factor V (proaccelerin), and platelet factor 3, factor Xa catalyzes the conversion of factor II (prothrombin) to IIa (thrombin) and factor I (fibrinogen) to the fibrin clot.

The result of the interaction among the blood vessels, platelets, and blood coagulation factors is the formation of factor Xa, which, as noted, converts prothrombin to thrombin and results in fibrin formation. The intrinsic and extrinsic pathways merge at factor Xa into a final common pathway to clot formation. Figure 45-2 diagrams the sequence of clot

BOX 45-1 Coagulation Factors

I	Fibrinogen
II	Prothrombin (thrombin in active form–IIa)
III	Thromboplastin
IV	Calcium
V	Proaccelerin
VI	Unassigned
VII	Proconvertin; prothrombinogen; convertin
VIII	Antihemophiliac factor A (factor VIIIR–von Willebrand)
IX	Antihemophiliac factor B; Christmas factor; platelet cofactor II
X	Stuart-Prower factor; prothrombinase
XI	Plasma thromboplastin antecedent
XII	Hageman factor; glass factor
XIII	Fibrin-stabilizing factor; Laki-Lorand factor

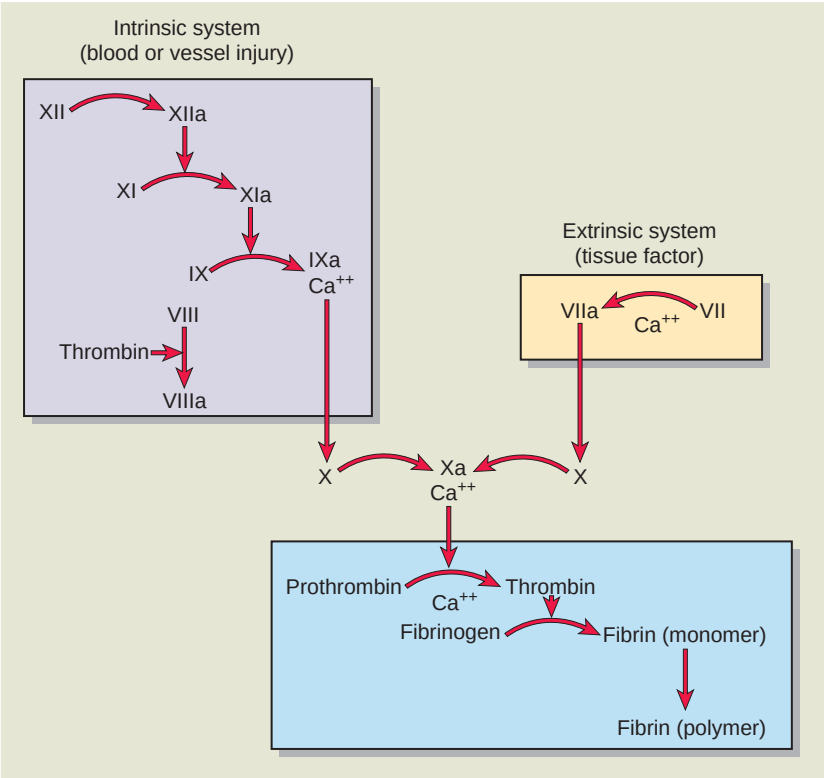


FIGURE 45-2 ▲ The coagulation cascade. (From Porth CM: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 265.)

formation. Note that the activation of factor VIII (antihemophiliac factor A) by thrombin causes the activation of factor X, resulting in a self-perpetuating effect.

Calcium plays an important role along the clotting cascade. Many coagulation factors carry two negative charges. Calcium, with its two positive charges, creates a strong affinity for the factors to bind at the site of clotting.

Intrinsic Pathway

Normally, blood coagulation factors circulate in an inactive state. After an initiating stimulus, changes in the coagulation factors occur immediately. The stimulus, damaged subendothelium, causes molecular alteration in any inactive coagulation factor, known as a proenzyme, converting it to an active form. The product of this enzymatic reaction activates the next coagulation factor in a chainlike reaction, leading to final clot formation. This chain of chemical reactions is termed the intrinsic pathway, which indicates its origin from within the tissue.

The release of platelet factor 3 initiates the activation of the intrinsic pathway by activating factor XII (Hageman factor). It is also a necessary component for complex reactions at the levels of factors V and VIII. The exposed collagen, phospholipids from injured erythrocytes and granulocytes, antigen–antibody complexes, and endotoxins are thought to be other activators of factor XII. These activators convert factor XII to the active enzymatic form XIIa, which acts on the next clotting proenzyme, factor XI (plasma thromboplastin antecedent), converting it to XIa. Factor XIa is responsible for the activation of factor IX (antihemophiliac factor B), which requires calcium ions. The activation of the next factor, factor X, requires factor VIII and platelet factor 3. The conversion of factor II to factor IIa (thrombin) requires factor V, platelet factor 3, and calcium ions.[2] Thrombin acts on fibrinogen, converting it to fibrin. This initial soluble fibrin clot is stabilized by factor XIII in the presence of calcium.

Again, the activation of factor VIII by thrombin creates the activation of factor X, resulting in a self-perpetuating effect. Thrombin enhances the activity of factor VIII so that it interacts more rapidly with factor IXa and thus catalyzes the activation of factor X. At this point, the intrinsic and extrinsic coagulation cascades merge. Thrombin also interacts with platelets, resulting in the release of platelet factor 3, which activates factor XII.

Coagulation Inhibitors

Unchecked activation of the blood clotting factors would cause clots to form on top of the platelet plug, releasing thrombin in the process of clotting, further attracting platelets to the clot site, and causing additional clots to form at the local site of the vessel leak (Fig. 45-3). There would be total vessel occlusion if there were no mechanisms operating to maintain the blood in a fluid state and prevent uncontrolled clotting.

However, there is a well-controlled balance between clot formation and clot inhibition. Through the action of physiological coagulation inhibitors, the blood is maintained in its fluid state, and blood vessels remain patent. These inhibitors—adequate blood flow, mast cells, antithrombin III, the mononuclear phagocyte system, and the fibrinolytic system—work by limiting reactions that promote clotting and by lysing any clots that do form, thereby preventing total occlusion of the vessels. Maintaining adequate blood flow facilitates the quick delivery of dilute-activated clotting factors to the

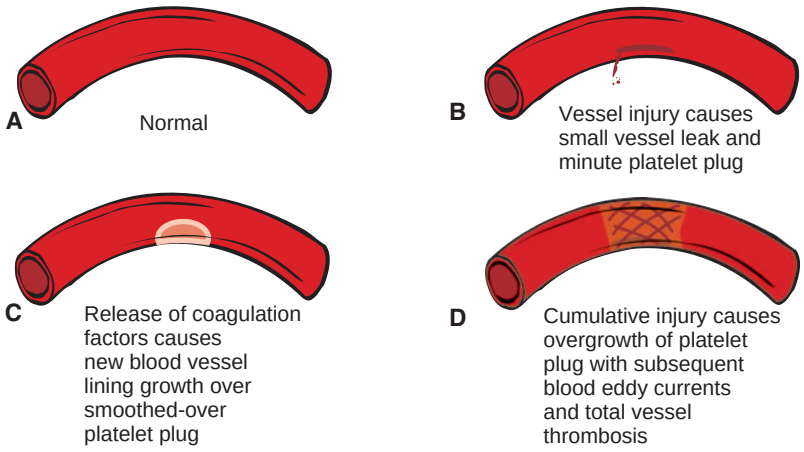


FIGURE 45-3 ▲ A–D: Sequence of thrombus formation in blood vessels.

liver, where they are cleared from circulation. Also, mast cells, which are located in most body tissues, produce heparin (which has a low anticoagulant activity compared with that of commercially produced heparin). Next, the liberation of antithrombin III in response to thrombin inactivates the circulating thrombin and neutralizes activated factors XII, XI, and X. This retards the conversion of fibrinogen to fibrin, thereby stopping sequential activation of clotting factors. Also, the mononuclear phagocyte system, composed of tissue macrophages located throughout the body, inhibits coagulation by clearing activated factors from the blood. Finally, the fibrinolytic system interferes with thrombin at its site of action on fibrinogen. It also involves a chain reaction whereby activation of a series of proenzymes produces lytic enzymes capable of dissolving clots.

The proenzyme plasminogen circulates in the blood. It is believed that the endothelial cells that constitute the endothelial lining of blood vessels release plasminogen activator, converting plasminogen to plasmin. In addition, activated factor XII, thrombin, kallikrein, and substances in the tissues are thought to be involved in the conversion of plasminogen to plasmin. Plasminogen activator levels are transiently elevated in response to exercise, stress, anoxia, and pyrogen. Plasmin, then, is the dissolving, or lytic, enzyme that lyses fibrin and attacks factors V, VIII, IX, and fibrinogen.

Finally, the lysis of fibrinogen and fibrin results in the liberation of fibrin degradation products (FDPs). FDPs inhibit platelet aggregation, exhibit an antithrombotic effect, and interfere with formation of the fibrin clot.

Fibrinolytic System Inhibitors

Similar to coagulation inhibitors, there are inhibitors of the fibrinolytic system. These inhibitors prevent inappropriate lysis of needed clot formation. The mononuclear phagocyte system clears FDPs from the circulation. Also, antiplasmin, a protein circulating in the blood, binds with plasmin and renders it inactive. The level of circulating antiplasmin far outweighs plasmin concentrations, and plasmin is neutralized rapidly.

It is evident that the systems of hemostasis and fibrinolysis, in conjunction with their system inhibitors, function within a narrow margin to ensure the liquidity of the blood and patency of the vasculature. An upset in these systems can result in clinical evidence of thrombosis, hemorrhage, or the catastrophic event of disseminated intravascular coagulation.

▲ Immune System

The immune system is composed of the following organs and cells: spleen, lymph nodes, thymus, bone marrow, appendix, tonsils and adenoids, B and T lymphocytes, eosinophils, basophils, and phagocytes. The organs of the system are connected with one another and with other organs through a network of lymphatic vessels. Immune cells and foreign particles are conveyed through the vessels in lymph fluid.

As noted, the hematological and immune systems are closely related. In addition to both originating in the bone marrow, blood carries components of the immune system throughout the body. The coagulation system helps keep microorganisms at the inflammatory site. The following are functions of a healthy immune system:

- Protection of the body from destruction by foreign agents and microbial pathogens
- Degradation and removal of damaged and dead cells
- Surveillance and destruction of malignant cells

Immune Response

The immune system is the body's internal response to substances recognized as foreign. People have two types of immunity: innate (general) immunity and adaptive (acquired) immunity. Innate immunity is the body's capacity to resist invasion by foreign agents. The innate immune system has an array of toll-like receptors, that exist on cells at the host–environment interface and have direct and early contact with potential pathogenic microorganism.[3] The receptors interact with membrane patterns shed by infectious agents and trigger the innate immune response.[3] In addition, innate immunity makes use of phagocytes and natural killer cells (NKCs) in its inflammatory response to microorganisms. Innate immunity is nonspecific and has no memory.

Adaptive immunity is the specific capacity of a person's immune system to identify a substance as foreign and mount an antibody response. It occurs when a person develops his or her own antibodies in response to exposure to an antigen.

Antibodies function as memory cells, so that subsequent exposures to specific antigens cause a quicker response. Adaptive immunity also occurs when another source supplies a person with antibodies. The maternal-to-fetal transfer of antibodies is an example of one person supplying antibodies to another. Because it may take several days for acquired immunity to generate sufficient activity to protect a person, a major role of the innate immune system is to limit microbial replication until the specific immune response is mobilized.

Basically, the immune system protects the body, or "self," from invasion by "nonself." The concept of immune tolerance infers a nonactive immune system with self while producing immunity to foreign substances.[4] Any foreign substance capable of eliciting a specific immune response is referred to as an antigen. Antigens are most often composed of proteins, but polysaccharides, complex lipids, and nucleic acids also can act as antigenic materials; bacteria, viruses, fungi, parasites, and foreign tissue are all antigens. For instance, transplant rejection occurs when the body recognizes transplanted tissues or organs as foreign. Discrete, immunologically active sites on antigens enable immunoglobulins, lymphocytes, or antibodies to identify target cells, against which destructive forces are directed. Immune responses are not equally potent. The intensity of the system's response is affected by the route of invasion, the dosage of the antigen, and the antigen's degree of foreignness.

Immunological competence refers to the immune system's capacity to identify and reject foreign materials. The system's failure to recognize antigens and mobilize effective defenses results in infection or malignancy. Failure to recognize markers of self can result in autoimmune diseases, such as multiple sclerosis, rheumatoid arthritis, or systemic lupus erythematosus. The system's "battle against imaginary enemies," such as pollen or dust, may result in allergies.

The major histocompatibility complex, essential for recognizing self from nonself, is a group of genes contained in a section of chromosome 6 that encode molecules that mark a cell as self. These genes vary widely in structure from one person to another. Their presence is a major factor in transplant rejection because they determine to which antigens one responds and how strongly. They also allow immune cells to recognize and communicate with one another.

Innate Immunity

Innate immunity is present in all healthy people and forms the first line of defense against illness. Previous exposure to an organism or toxin is not required for activation. Also, mechanisms of innate immunity do not distinguish among microorganisms of different species and do not alter in intensity on re-exposure. Innate immune defenses include physical, chemical, and mechanical barriers; biological defenses; phagocytosis; inflammatory processes; and cytokines, dendritic cells, macrophages and neutrophils.

Physical, Chemical, and Mechanical Barriers

Physical barriers prevent harmful organisms and other substances from gaining entrance into the body or body cavities. These barriers include skin, mucous membranes, the epiglottis, respiratory tract cilia, and sphincters. Chemical barriers such as antibacterial agents, antibodies, and acid solutions create an environment hostile to many pathogens. Lysozymes in tears, lactic acid in vaginal secretions, and hydrochloric acid in gastric secretions all act as chemical barriers. Mechanical barriers help rid the body of potentially harmful substances through some action (eg, lacrimation, intestinal peristalsis, urinary flow).

Biological Defenses

Under normal conditions, large areas of the human body are colonized with microorganisms of low pathogenicity. The skin and mucous membranes of the oropharynx, nasopharynx, intestinal tract, and parts of the genital tract each have their own microflora, referred to as normal flora. These microorganisms influence patterns of colonization by competing with more harmful organisms for essential nutrients and by producing substances that inhibit the growth of other microorganisms. Vitamin D deficiency may increase the risk of infection in epithelial tissues that directly interact with the environment,[5,6] thereby reducing the effectiveness of biological defenses.

Phagocytes and Phagocytosis

Phagocytosis is a process by which injured cells and foreign invaders are ingested by leukocytes, specifically, neutrophils and mononuclear phagocytes (monocytes and macrophages). Both cell types originate from stem cells in the bone marrow and, although structurally different, approach phagocytosis in a similar manner.

Surface receptors on their cell membranes allow them to attach to foreign substances and then engulf, internalize, and destroy these substances using enzymes present in their cellular interior. Both neutrophils and macrophages are attracted to the site of microorganism invasion by chemokines. Neutrophils provide the "first-wave" cellular attack on invading organisms during the acute inflammatory process. The number of neutrophils at the site peaks in 6 to 12 hours after inflammation begins. The second wave of cells is primarily monocytes. Monocytes spend only a short time in the bloodstream before escaping through the capillary membranes into the tissue. Once in the tissue, they swell to much larger sizes to become macrophages, which either attach to certain tissues and destroy bacteria, or wander through the tissue phagocytizing foreign matter. These cells are strategically placed throughout the body tissues, where they can exist for months and even years. Macrophages in different tissues differ in appearance because of environmental variations and are known by different names (ie, Kupffer's cells in the liver, alveolar macrophages in the lungs, histiocytes in the skin and subcutaneous tissue, and microglia in the brain).

Inflammatory Responses

Inflammation is an acute physiological nonspecific response of the body to tissue injury caused by factors such as chemicals, heat, trauma, or microbial invasion. It is the primary process through which the body repairs tissue damage and defends itself against infection. The initial inflammatory response is localized but may lead to systemic consequences such as fever, malaise, and neutrophilia. The inflammatory response contains three stages:

1. The vascular stage involves an immediate but short-term vasoconstriction, followed by vasodilation of arterioles and venules and hyperemia and swelling resulting from

the secretion of histamine, prostaglandins, serotonin, and kinins.
2. The cellular exudate stage is characterized by neutrophilia, secretion of colony-stimulating factors into the interstitial fluid, and formation of exudate, a clear serous fluid with a high protein count. The functions of exudate are to transport leukocytes and antibodies to the inflammatory site, dilute toxins and irritating substances, and transport materials necessary for tissue repair. As the inflammatory process continues, the serous exudate changes to a creamy white fluid containing cellular debris.
3. The tissue repair and replacement stage, in which inflammatory material is removed and connective tissue cells proliferate. Collagen synthesis occurs, resulting in tissue replacement.

The most important result of these processes is accumulation at the site of injury of large numbers of neutrophils and macrophages, which inactivate or destroy invaders, remove debris, and begin the initial tissue repair.

Cytokines

Cytokines are chemical messengers produced by T lymphocytes that function as immune system hormones and play a role in adaptive immunity and in mediating the inflammatory response. They enhance cell growth, promote cell activation, direct cellular traffic, stimulate macrophage function, and destroy antigens. They are also called interleukins (ILs) because they serve as messengers between leukocytes. Interferons and tumor necrosis factor are also cytokines.

IL-1 augments the synthesis of IL-2, IL-3, IL-4, interferon-γ, and IL-2 receptors. It can also activate lymphokine-activated killer cells. IL-2 binds to specific receptors on activated T cells and markedly enhances the cytolytic activity of NKCs, which are a specialized group of lymphoid cells that act directly, without prior sensitization, to lyse a variety of malignant and virus-infected cells. IL-3 and B-cell differentiation factor provide critical signals for the growth and maturation of antigen-primed B cells.

Cytokines can be classified as either lymphokines (secreted by lymphocytes) or monokines (secreted by monocytes or macrophages). Interferons (a type of lymphokine) provide some protection to the body against invasion by viruses until more slowly reacting specific immune responses take over. Interferons are produced when a virus infects a host cell; they affect the transcription and translation of viral genes. In addition, interferons appear to be involved in protecting the body against some forms of cancer. Specifically, these substances have been demonstrated to interfere with cellular division and proliferation of abnormal cells. They also enhance the activity of NKCs.

Adaptive Immunity

If a foreign agent persists despite innate immune responses, the activation of adaptive immune responses occurs. To be most effective, these responses require previous exposure to a foreign agent or organism. The cellular components of these types of responses are capable of distinguishing among microorganisms and can alter their intensity and response time significantly on re-exposure.

Two types of adaptive immune responses have been identified: cell-mediated immunity and humoral immunity. Most foreign substances stimulate both cellular and humoral immune responses; this results in an overlapping of their reactions and maximal protection against damage from the invading substances.

B and T Lymphocytes

B and T lymphocytes originate from stem cells produced in the bone marrow. During fetal development and shortly after birth, primary lymphoid organs are the sites where these cells differentiate and mature into the competent cells responsible for cell-mediated and humoral immune responses. For B lymphocytes, this preprocessing is believed to occur in the bone marrow and, possibly, the fetal liver; for T lymphocytes, it occurs in the thymus gland.

As they develop, both B and T lymphocytes acquire receptors for specific antigens that commit them to a single antigenic specificity for their lifetime. Subsequently, each of these "preprogrammed" B or T lymphocytes (on activation by its specific antigen) is capable of producing tremendous numbers of clones or duplicate lymphocytes. The different types of T cells produced are categorized according to their function, as shown in Table 45-3.

Table 45-3 Types of T Cells and Their Functions

Cell Type	Function
Cytotoxic T cells (T8)	Direct-attack cells capable of killing many microorganisms; predominant effector cell Virus-infected cells, cancer cells, and transplanted cells especially susceptible
Helper-inducer T cells (T4)	Most numerous Play pivotal role in overall regulation of immune response Often called "master conductor" Secrete lymphokines
Suppressor T cells (T8)	Act as negative feedback controllers of T4 cells May also limit ability of immune system to attack body tissues
Memory T cells	Sensitized to antigens during specific immune responses Remain stored in body Capable of initiating far more rapid response by T cells on re-exposure to same antigen

Lymphoid System

After preprocessing in the primary lymphoid organs, B and T lymphocytes migrate to secondary lymphoid tissues, where the interaction with antigens and immune responses actually occurs. Secondary lymphatic tissue is located extensively in the lymph nodes. It is also found in special lymphoid tissue, such as that of the spleen, tonsils, adenoids, appendix, bone marrow, and gastrointestinal tract. This lymphoid tissue is placed advantageously throughout the body to intercept invading organisms or toxins before they can enter the bloodstream and disseminate widely.

Cell-Mediated Immune Response

Cell-mediated immunity provides a response to fungi, parasites, and intracellular bacteria. It also plays a major role in the rejection or acceptance of certain tissue grafts, the stimulation and regulation of antibody production, and defense against various malignant changes. As noted, T lymphocytes contain an antigen receptor that allows for the binding of a specific type of antigen.

Each T cell, when activated by its specific antigen, is capable of producing large numbers of clones. After preprocessing, T cells migrate to lymphoid tissue, where they act as effector cells (directly attacking antigens and malignant cells) and regulators of both the cellular and humoral immune response.

Antigenic stimulation of T lymphocytes initiates the cell-mediated response. This step of the response may be mediated by macrophages that bind to the antigen, facilitating its recognition. The macrophages then produce cytokines, which stimulate T lymphocytes, increase B-lymphocyte proliferation, and activate phagocytes. People with impaired cell-mediated immunity are at high risk for infections with pathogens that replicate within cells such as viruses or parasites.[3]

Humoral Immune Response

The humoral immune response is extracellular; that is, it occurs in blood and tissue fluid. It begins in response to most bacteria, bacterial toxins, and the extracellular phase of viral invasion. Humoral immunity involves two types of serum proteins: immunoglobulins (Igs) and complement. Vitamin D can decrease immunoglobulin production and slows differentiation of B-cell precursors into plasma cells.[5]

Igs are antibody molecules made by B lymphocytes that differentiate to plasma cells and memory cells. The plasma cells then secrete antibodies that bind to antigens; the resulting antigen–antibody complexes are ingested by phagocytes. After the complexes are eliminated, the memory cells remain in circulation and in lymphoid tissue to mature into plasma cells when the antigen is encountered again. Immunoglobulins are specific to antigens and are of several types:

- IgA (two types) concentrates in body fluids, such as tears, saliva, and secretions of respiratory and gastrointestinal tracts; it guards entrances to the body.
- IgM tends to remain in the bloodstream, where it is effective in killing bacteria.
- IgG (four types) is able to enter tissue spaces and works efficiently to coat microorganisms before phagocytosis occurs.
- IgD is found mostly in the membrane of B cells, where it is believed to regulate the cells' activation.
- IgE is normally present in only trace amounts; it is responsible for symptoms of allergy by activating mast cells.

Complement is a nonspecific series of 15 proteins that circulate in an inactive form in the bloodstream. These proteins activate one another in a cascading sequence when the first complement molecule C1 encounters an antigen–antibody complex. The end product of the cascade is a cylinder that lyses the cell membrane of the target cell, allowing fluids and molecules to flow in and out, which kills the target cell.

Complement can be activated in three ways. Classic activation is initiated by the antibody–antigen complex. The alternate pathway and lectin pathways are not initiated by antibodies but start in response to bacteria, fungi, and mannose found on the surfaces of some bacteria (Fig. 45-4).[2] In addition, complement facilitates the interaction of antigens and antibodies and enhances all aspects of the inflammatory process, especially increasing vascular permeability and phagocytosis.

Combined Immune Responses

The specific immune response is complex and involves the interaction of macrophages, complement proteins, and the cellular components of both the cellular and humoral systems (Fig. 45-5). Macrophages initially function to recognize, process, and present the antigen to antigen-specific T lymphocytes in the lymphoid tissues. Helper–inducer T4 cells are subsequently activated with the help of a chemical factor (IL-1) released by the presenting macrophage. The T4 cells proliferate and produce their own chemical substances, known as lymphokines, which in turn stimulate the activation and proliferation of antibody-producing B lymphocytes, cytotoxic T cells, suppressor T cells, and phagocytic macrophages. The production of antibodies leads to the activation of complement proteins. All these components work together to destroy the antigen, either through complex processes involving direct attack or through modulation by chemical processes. Suppressor T cells provide feedback to the T4 helper cells to halt these defense reactions when they are no longer needed, and memory cells reactivate them on re-exposure to the antigen.

Impaired Host Resistance

The various components of the immune system provide a complex network of mechanisms that, when intact, defend the body against foreign microorganisms and malignant cells. However, in some situations, components of the system can fail, resulting in impaired host resistance. Often the state of immunosuppression is chemically induced by drugs or medications, such as corticosteroids and cytotoxic chemotherapeutic agents. People who acquire an infection because of a deficiency in any of their host defenses are referred to as immunocompromised, or immunosuppressed.

The exact effects of, and symptoms related to, defects in host defense vary according to the part of the immune system affected (Table 45-4). General features associated with compromised host resistance include recurrent infections, infections caused by usually harmless agents (opportunistic organisms), chronic infections, skin rashes, diarrhea, growth impairment, and increased susceptibility to certain cancers.

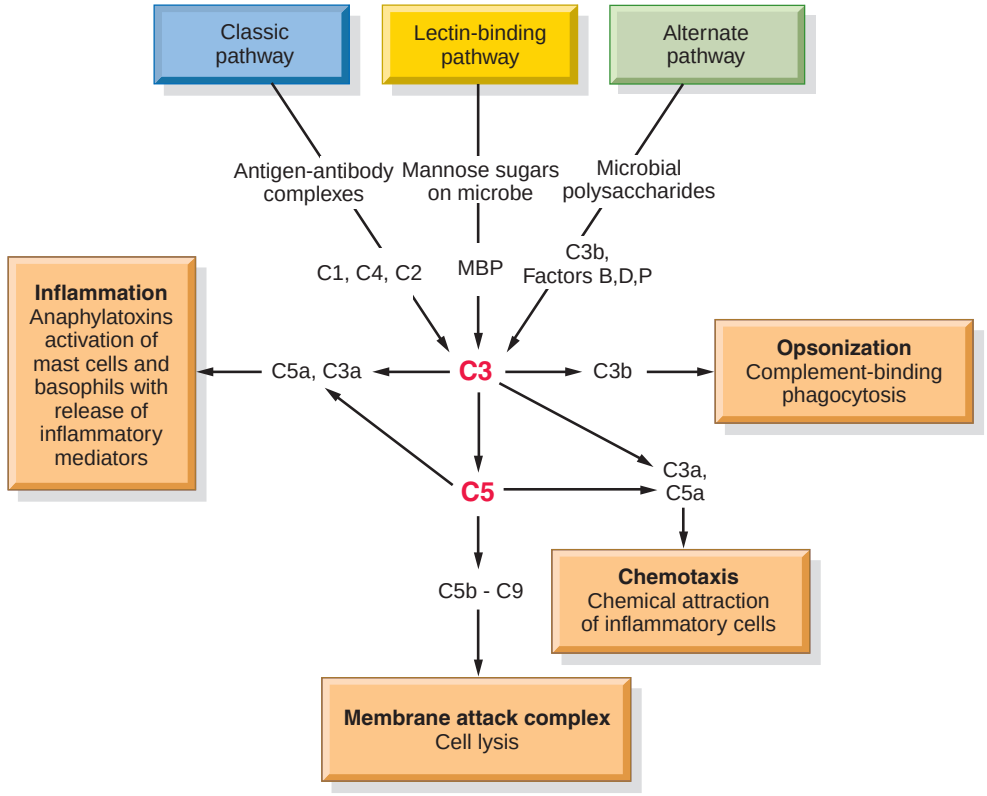


FIGURE 45-4 ▲ Classic, lectin, and alternative complement pathways. (From Porth CM: Pathophysiology: Concepts of Altered Health States, 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2005, p 381.)

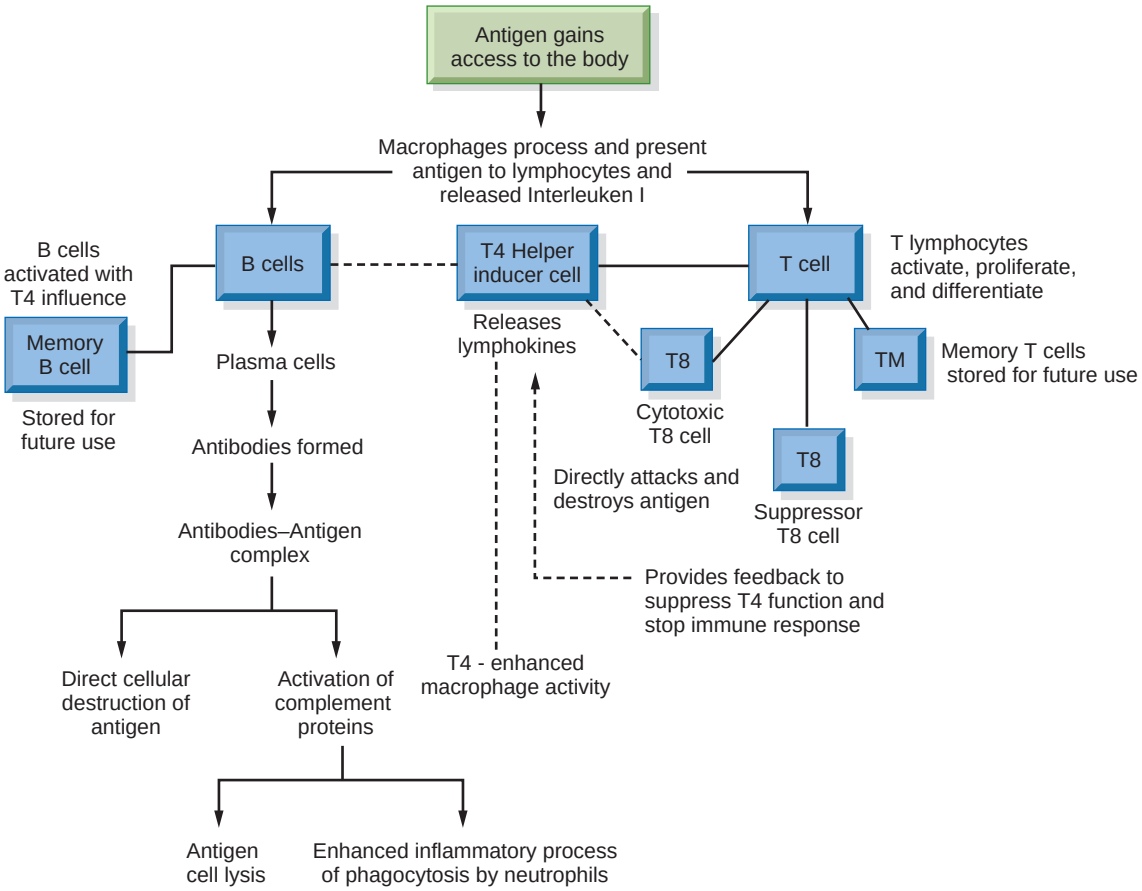


FIGURE 45-5 ▲ A schematic representation of the combined immune responses.

Table 45-4 Risk Factors for Compromised Host Defenses

Host Defect	Diseases, Therapies, and Other Conditions Associated With Host Defects
Impaired phagocyte functioning	Radiation therapy Nutritional deficiencies Diabetes mellitus Acute leukemias Corticosteroids Cytotoxic chemotherapeutic drugs Aplastic anemia Congenital hematological disorders Alcoholism
Complement system deficiencies	Liver disease Systemic lupus erythematosus Sickle cell anemia Splenectomy Congenital deficiencies
Impaired cell-mediated (T lymphocyte) immune response	Radiation therapy Nutritional deficiencies Aging Thymic aplasia AIDS Hodgkin's disease/lymphomas Corticosteroids Antilymphocyte globulin Congenital thymic dysfunctions
Impaired humoral (antibody) immunity	Chronic lymphocytic leukemia Multiple myeloma Congenital hypogammaglobulinemia Protein-losing enteropathies (inflammatory bowel disease)
Interruption of physical/mechanical/ chemical barriers	Traumatic injury Decubitus ulcers/skin defect Invasive medical procedures Vascular disease Skin diseases Nutritional impairments Burns Respiratory intubation Mechanical obstruction of body drainage systems, such as lacrimal and urinary systems Decreased level of consciousness
Impaired mononuclear phagocyte system	Liver disease Splenectomy

▲ Clinical Applicability Challenges

SHORT ANSWER QUESTIONS

1. Individuals have been encouraged to use sunblock to reduce the risk of skin cancer. Discuss the effects on the immune system.
2. A patient is receiving anticoagulant medication. What is the effect on the coagulation inhibitors?
3. Explain the mechanism that causes a blunted white blood cell response in individuals with a viral infection.

References

1. Hoffman R, Benz J, Shattel S, et al (eds): Hematology: Basic Principles and Practice, 5th ed. Philadelphia, PA: Churchill Livingstone, 2008
2. Porth C: Pathophysiology, Concepts of Altered Health Status, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009
3. Iwasaki A, Medzhitov R: Regulation of adaptive immunity by the innate immune system. Science 327:291–295, 2010
4. Goldman L, Auseillo D (eds): Cecil Medicine, 23rd ed. Philadelphia, PA: Saunders, Elsevier, 2008
5. Bikle D: Vitamin D and immune function: Understanding common pathways. Curr Osteoporos Rep 7(2):58–63, 2009
6. vanEtten E, Gysemans C, Mathieu C, et al: Regulation of vitamin D homeostasis: Implications for the immune system. Nutr Rev 66(Suppl 2), S125–S134, 2008

46 Patient Assessment: Hematological and Immune Systems

Kenneth Rempher and Patricia Gonce Morton

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Describe areas of a patient's history and physical assessment pertinent to assessing hematological and immune disorders.
2. Differentiate diagnostic tests used to assess hematological and immune disorders.
3. Synthesize the results of a patient's history, physical examination, and diagnostic tests to identify hematological and immune disorders.
4. Describe key aspects of the assessment of the immunocompromised patient.

Hematological and immune disorders encompass numerous ailments, many of which are life threatening. In general, hematological disorders can be classified as overproduction or underproduction of hematological components or dysfunction of these components. Immune disorders usually are caused by underactivity or overactivity of immune system elements. Immune disorders can be inherited, or they can be acquired through disease or treatments, such as chemotherapy and transplant immunosuppression. The hematological and immune systems are complex and closely interrelated; therefore, disorders or dysfunctions of one system often alter the effectiveness of the other.

▲ Assessment

History

The patient history is essential when evaluating potential hematological or immune disorders. While discussing the chief complaint, a patient may state symptoms that seem vague and unrelated, so a detailed history is critical. It is important to keep in mind the complex physiology of these systems when assessing a patient's health history (see Chapter 45). After obtaining information about the chief complaint and history of the present illness, the nurse inquires about the patient's past health history, family history, and personal and social history (Box 46-1). The patient's immunization history and occupation may provide helpful clues. A review of relevant systems concludes the history. Table 46-1 summarizes conditions and treatments that may predispose patients to hematological and immune disorders (see Chapters 48 and 49). Box 46-2 summarizes special considerations for older patients.

Physical Examination

A thorough physical examination is necessary to identify physical signs that may indicate a hematological or immune system disorder. Table 46-2 on page 1037 summarizes physical findings that may suggest various disorders of these systems. (Many of these disorders are further described in Chapter 49.) The physical examination of the hematologically or immunocompromised patient focuses on four major areas: skin, liver, spleen, and lymph nodes. The entire examination must be thorough to help identify the exact source of the problem. The nurse examines the patient's skin for pallor or jaundice as well as for signs of abnormal bleeding. He or she also evaluates the patient's joints for pain, swelling, and limited range of motion, which may suggest hemarthrosis from coagulopathy or sickle cell anemia. Superficial mucocutaneous bleeding and a dependent distribution of petechiae can indicate thrombocytopenia, whereas clusters of palpable, pruritic petechiae can suggest vasculitis. Extensive superficial purpura, deep hematomas, or hemarthroses may indicate a coagulation disorder. The nurse also notes skin rashes, pruritus, and excoriations. It is necessary to assess the extremities for areas of redness, tenderness, warmth, or swelling, which can indicate thrombophlebitis. Leg and ankle ulcers may be present in patients with sickle cell anemia. The nurse assesses the lips and nail beds for cyanosis; digital clubbing may be present in patients with chronic hypoxemia. For more information on assessment of nails, see Chapter 51.

The nurse also examines the patient's eyes and mouth. Visual changes can indicate hyperviscosity from polycythemia or retinal infarcts from sickle cell anemia. It is important to assess the nares, gums, and mucous membranes of the mouth for signs of bleeding. The oral examination is a good time to ask about bleeding gums while brushing. Pallor of the oral mucosa can be a significant indicator of anemia. Tongue changes can occur in

BOX 46-1 HEALTH HISTORY for Hematological and Immune Assessment

Chief Complaint
- Patient's description of the problem

History of the Present Illness
- Complete analysis of the following signs and symptoms (using the NOPQRST format; see Box 17-1, p. 207)
- Unusual bruising or bleeding, frequent infections, fatigue/malaise, headache, dizziness/gait disturbance, pain, enlarged lymph nodes, fevers, night sweats, weakness, limb pain/limp, seizure, weight loss, abdominal pain, vomiting, heat intolerance, poor wound healing, nevi

Past Health History
- Relevant childhood illnesses and immunizations—mononucleosis, malabsorption, hepatitis, pernicious anemia
- Past acute and chronic medical problems, including treatments and hospitalizations—anemia, cancer, infections, autoimmune hemolytic anemia/Evans syndrome, hemochromatosis, hereditary spherocytosis, iron deficiency anemia, polycythemia, hemophilia, sickle cell disease, thalassemia, idiopathic thrombocytopenia, glucose-6-phosphate dehydrogenase (G6PD) deficiency, aplastic anemia, myelodysplastic syndrome, cirrhosis, HIV, major trauma, sepsis
- Risk factors—recent exposure to benzenes, pesticides, mustard gas, antineoplastic agents
- Past surgeries—splenectomy, cardiothoracic surgery, total gastrectomy
- Past diagnostic tests and interventions—bone marrow aspiration, radiation therapy, chemotherapy, multiple blood transfusions, administration of blood products (cryoprecipitate)
- Medications—chemotherapeutic agents, antibiotics, antihypertensives, diuretics, glucocorticoids, nonsteroidal anti-inflammatory drugs, aspirin, heparin, warfarin, antiplatelet agents
- Allergies and reactions
- Transfusions

Family History
- Health status or cause of death of parents and siblings—cancer, anemia, hereditary spherocytosis, G6PD deficiency, sickle cell anemia, methemoglobinemia, thalassemia, Glanzmann's thrombasthenia, von Willebrand's disease, polycythemia

Personal and Social History
- Tobacco, alcohol, and substance use
- Family composition
- Occupation and work environment—exposure to chemicals: benzenes, mustard gas, cigarette smoke, butadiene, dioxins (pesticides), hexachlorobenzene, ozone, polybrominated biphenyls, polychlorinated biphenyls, phenols, toluene-di-isocyanate vinyl chloride, lead, naphthalene
- Living environment; see above
- Diet (insufficient intake of foods rich in iron and folic acid, including liver, eggs, whole grains, breads, cereals, potatoes, leafy green vegetables, fruits, and legumes). Assess for poor intake of foods rich in vitamin B_{12}, including liver, fish, and fortified cereals.
- Sleep patterns—disruptive sleep patterns modulate changes in immune system activity[1]
- Exercise—regular exercise is associated with optimizing immunity[2]
- Cultural beliefs
- Spiritual or religious beliefs—refusal of blood products, including transfusions is a core belief for some patients, including some Jehovah's Witnesses[3]
- Coping patterns and social support systems
- Leisure activities
- Sexual activity
- Recent travel

Review of Systems
- HEENT: oral infections, gum bleeding, epistaxis, mouth sores, sore throat, smooth tongue texture, jaundiced sclera, conjunctival pallor, retinal hemorrhages
- Cardiac: tachycardia, S_4 sounds
- Respiratory: recent upper or lower respiratory infections, hemoptysis
- Gastrointestinal: blood in emesis, blood in stools, dark tarry stools, unintentional weight loss, splenomegaly, hepatomegaly, splenic bruits, and rubs
- Musculoskeletal: weakness, bone pain, back pain, arthralgia
- Neurological: mental status changes, pain to touch, position and vibratory sensation, tendon reflexes
- Genitourinary: blood in urine, urinary tract infections
- Reproductive: heavy menstruation, vaginal bleeding

patients with an iron deficiency and megaloblastic anemias. Inspection of the throat and palpation of lymph nodes to assess for infection or malignancy is warranted. Figure 46-1 shows palpation of the lymph nodes in the neck.

Tachycardia and tachypnea may be present in patients with anemia or infection. An S_4 heart sound may be heard in persons with severe anemia. Dyspnea on exertion and orthostatic changes in blood pressure can be other symptoms of anemia and not just volume insufficiency. Many patients present with new-onset chest pain made worse by anemia. As the body loses oxygen carrying capacity resulting from loss of hemoglobin, the myocardium becomes stressed and can trigger angina. It is necessary to perform thorough lung auscultation and inspection of sputum, if present, to rule out respiratory infection and hemoptysis. Symptoms of intermittent claudication (see Chapter 19) and angina pectoris (see Chapter 21) indicate problems with oxygen delivery in patients with polycythemia. Polycythemia is an unusual myeloproliferative disease in which there is excessive production of erythrocytes. The increased number of erythrocytes results in increased blood volume, increased viscosity of blood, and clogging of microcirculatory blood vessels, which leads to decreased tissue perfusion. Subsequently, patients with polycythemia commonly experience hypertension as part of the sympathetic response to decreased tissue perfusion.

Pertinent physical assessment findings of the abdominal and pelvic region include lymphadenopathy, splenomegaly, and hepatomegaly, which can indicate a number of hematological or immune conditions. Figure 46-2 shows the technique for palpating the spleen in the supine and side-lying positions. Figure 46-3 on page 1039 shows the degrees of splenomegaly. The nurse also thoroughly assesses for urinary tract infections, vaginal infections (including those with

Table 46-1 Hematological and Immune Disorders Based on Patient History

Patient History	Potential Disorder
Chronic disease (inflammation, infection)	Anemia
Nutritional deficiencies (iron, folate, vitamin B_{12})	Anemia
Nutritional deficiencies (vitamin K, malabsorption)	Coagulopathy
Endocrine (thyroid, pituitary) dysfunction	Anemia
Hypersplenism	Anemia, thrombocytopenia
Acquired immunodeficiency syndrome	Anemia, neutropenia
Malignancy	Pancytopenia
Chemical exposure	Neutropenia, hemolytic anemia
Prosthetic heart valve or vascular graft	Hemolytic anemia
Collagen vascular disorder	TTP
Hypersensitivity reaction	TTP
Viral, bacterial, or fungal infection	TTP
Uremia	Coagulopathy
Chronic alcoholism	Coagulopathy
Liver disease	Coagulopathy, thrombosis
Vasculitis	Thrombosis
Atherosclerosis	Thrombosis
Chronic obstructive pulmonary disease	Polycythemia
Smoking	Polycythemia
Congenital cardiac disease	Polycythemia
Previous Therapies/Medications	
Heparin	Thrombocytopenia
Antibiotics	Agranulocytosis
Carbamazepine	Agranulocytosis
Alkylating agents	Leukemia, lymphoma, pancytopenia
Blood transfusion	Anemia
Aspirin, nonsteroidal anti-inflammatory drugs	Coagulopathy
Warfarin	Coagulopathy
Steroids	Leukocytosis
Various drugs, chemicals, and toxins (see Box 49-1, p. 1135)	Hemolytic anemia
Family History	
Sickle cell anemia	Anemia
Thalassemia	Anemia
Congenital hemolytic anemia	Anemia
Polycythemic disorders	Polycythemia vera
von Willebrand's disease	Bleeding disorder
Hemophilia	Bleeding disorder

TTP, thrombotic thrombocytopenic purpura.

yeast), and perirectal inflammation. In addition, the nurse checks all body secretions and fluids (stool, urine, emesis, or gastric secretions) for the presence of blood.

Neurological abnormalities may be present in patients with hematological conditions. Altered mental status, paresis, aphasia, dysphasia, coma, seizures, paresthesia, and visual problems may be caused by thrombotic thrombocytopenic purpura (TTP; see Chapter 49). An altered level of consciousness, papilledema, vomiting, and bradycardia with widening pulse pressure are signs of increased intracranial pressure, which may be caused by intracranial bleeding in patients with coagulopathy.

BOX 46-2 CONSIDERATIONS FOR THE OLDER PATIENT

Risk Factors for Hematological Disorders

- Decreased iron intake, resulting from poor dentition (difficulty chewing meat) or a fixed income (making sources of iron such as meat or supplements unaffordable), can place the older adult at risk for iron deficiency anemia. Older adults may also experience low-grade gastrointestinal bleeding as a result of nonsteroidal antiinflammatory drug use for the treatment of arthritis, from hemorrhoids and polyps, or from undiagnosed colon cancer. This blood loss may also place them at risk for iron deficiency anemia.
- Poor absorption of vitamin B_{12} (as a result of atrophic gastritis) places the older adult at risk for megaloblastic anemia.
- Declining immune function places the older adult at risk for leukemia, lymphoma, and multiple myeloma.
- Anticoagulation therapy (eg, to treat atrial fibrillation) can result in platelet dysfunction and places the older adult at risk for hemorrhage. This is a particularly significant risk in older adults who are disoriented or have decreased mobility.

▲ Diagnostic Studies and Results Interpretation

Laboratory test results are usually the most sensitive and specific determinants of hematological and immune problems. Specialized testing may be required to ascertain whether the components are functioning properly. Because patients with severe presentations of hematological and immune conditions may be seen in the intensive care unit (ICU), tests to differentiate the conditions and their causes are presented here.

Tests to Evaluate Red Blood Cells

Red blood cells (RBCs) are essential for oxygenating tissues. An overproduction of RBCs results in polycythemia, which is indicated by a high hematocrit level and an increased RBC mass (see Chapter 49). Anemia is a condition marked by a decrease in the RBC mass caused by decreased production of RBCs, increased RBC destruction, a combination of these two conditions, or acute blood loss. All patients being evaluated for anemia should have a complete blood count (CBC) with RBC indices, a reticulocyte count, iron studies, and a peripheral smear analysis. Abnormalities in these test results indicate the need for subsequent testing.

Table 46-2 Findings Indicating Possible Hematological or Immune Disorders

Physical Findings*	Related Information From Patient History	Possible Disorder
Pallor, dyspnea, dizziness, tachycardia, glossitis	Fatigue Headache Pica (compulsive craving for clay, laundry starch, earth, or ice)	Iron deficiency anemia
As above, also smooth tongue, stomatitis, icterus, paresthesias, gait ataxia, mental status changes	Fatigue Headache Premature graying of hair	Megaloblastic anemia
Bleeding (ecchymosis, petechiae, epistaxis, hemorrhage), pallor, dizziness, tachycardia	Fatigue Headache History of frequent infections (eg, upper respiratory, cellulitis, perirectal) Previous viral infection (hepatitis, infectious mononucleosis, HIV, cytomegalovirus) Family history of aplastic anemia	Aplastic anemia
Pallor of conjunctiva, mucous membranes, palms and soles of feet; dyspnea; dizziness; tachy- cardia; bone pain; pain in chest or abdomen; splenomegaly; fever; leg and ankle ulcers; painless hematuria	African American descent Family history of sickle cell anemia Frequent infections Impaired vision Damage to joints Chronic renal failure History of stroke	Sickle cell anemia
Pallor, dyspnea, dizziness, jaundice, splenomegaly, cholelithiasis	Mediterranean descent, also Middle Eastern, South and Southeast Asian, and African	Thalassemia
Splenomegaly, hepatomegaly, facial and conjunctival plethora, hypertension, pruritus, dizziness, headache, thrombosis, thrombophlebitis	Visual disturbances Epigastric distress Cardiovascular insufficiency Bleeding tendency Numbness and burning of toes (from peripheral vascular insufficiency)	Polycythemia
Mouth sores, sore throat, lymphadenopathy, splenomegaly, hepatomegaly, infection (signs of infection may be minimal)	History of recurrent, severe infections Fatigue Recent radiation or chemotherapy treatment	Leukopenia
Infection, bleeding, bone pain, splenomegaly, skin and gum lesions, leukostasis if WBC count is extremely high (headache, confusion, Central nervous system infarctions, acute respiratory insufficiency, pulmonary infarctions)	History of recurrent infections Fatigue Anorexia Weight loss	Acute or chronic leukemia
Bone pain, pallor, weakness, fatigue	History of recurrent infections Renal insufficiency Hypercalcemia (thirst, lethargy, confusion, polyuria, constipation)	Multiple myeloma
Weight loss, fever, night sweats, painless lymphadenopathy, splenomegaly, abdominal pain	Fatigue Anorexia History of infections	Hodgkin's disease or non-Hodgkin's lymphoma
Superficial mucocutaneous bleeding, petechiae on dependent areas of the body, epistaxis, hemoptysis, hematemesis, hematuria, rectal bleeding, vaginal bleeding, intra-abdominal hemorrhage (diffuse abdominal pain, restlessness, anxiety, pallor, rigidity, dusky coloration of abdominal skin, tachycardia, tachypnea, and hypotension), intracranial bleeding (headache, vomiting, decreasing level of consciousness, papilledema, bradycardia)	History of viral or bacterial infection Hypersplenism Malignancies affecting the bone marrow History of immune disorders Alcoholism Pregnancy	Thrombocytopenia

(continued on page 1038)

Table 46-2 Findings Indicating Possible Hematological or Immune Disorders (continued)

Physical Findings*	Related Information From Patient History	Possible Disorder
Confusion, headache, altered mental status, paresis, aphasia, dysphagia, coma, seizures	Paresthesias Visual disturbances	TTP
Superficial purpura, mucocutaneous bleeding, hemorrhage, joint pain and swelling (from bleeding into joints), deep hematomas	History of excessive or recurrent bleeding in patient or family members Alcoholism Hepatitis Liver disease Malnutrition Malabsorption syndromes (affects absorption of vitamin K from gastrointestinal tract)	Coagulation disorder

TTP, thrombotic thrombocytopenic purpura; WBC, white blood cell.

*Findings listed may not always be present.

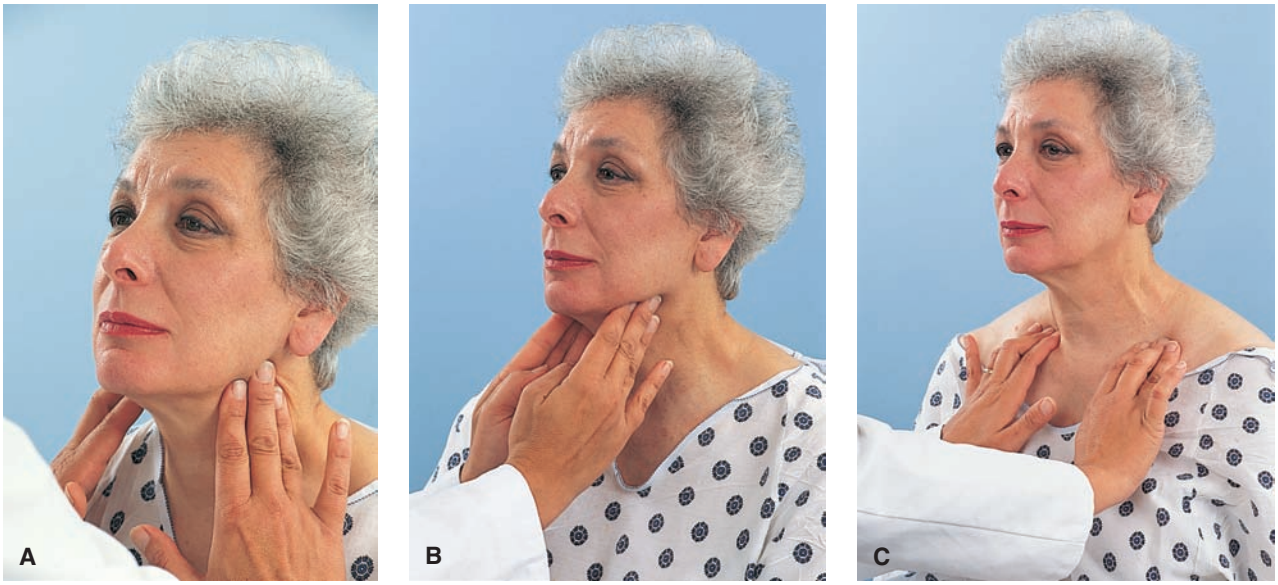


FIGURE 46-1 ▲ **A:** Palpating the tonsillar nodes. **B:** Palpating the submandibular nodes. **C:** Palpating the supraclavicular nodes. (From Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 222.)

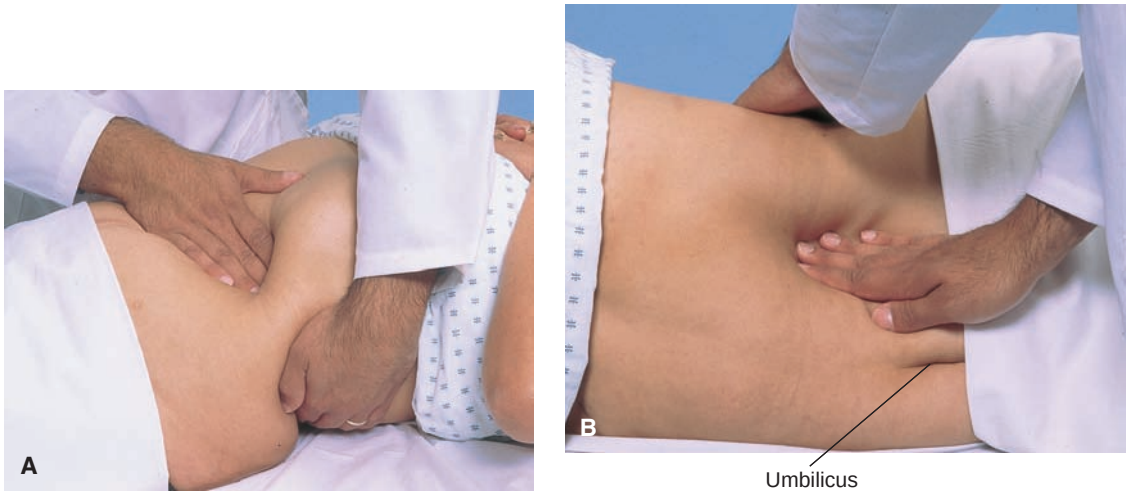


FIGURE 46-2 ▲ **A:** Palpating the spleen in the supine position. **B:** Palpating the spleen in the side-lying position. (From Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, pp 436, 437.)

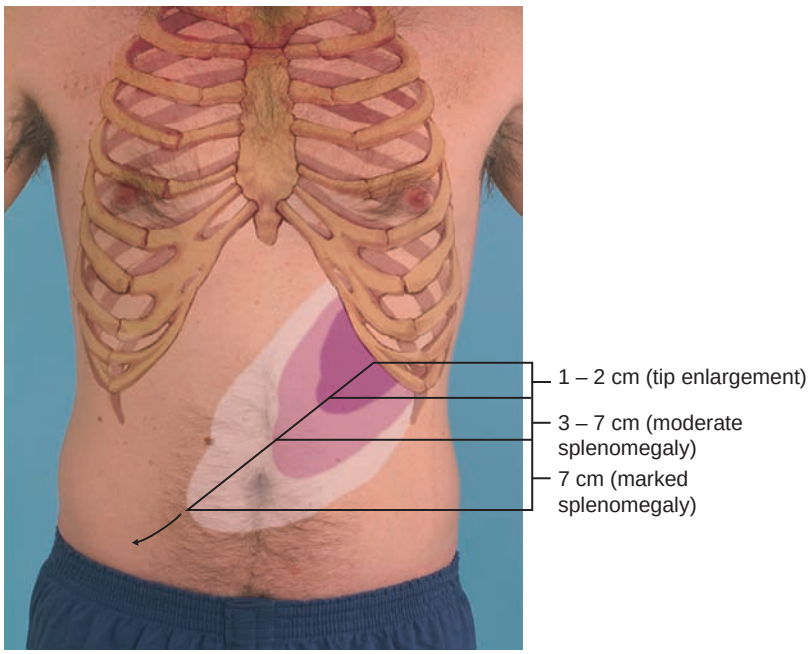


FIGURE 46-3 ▲ Degrees of splenomegaly. (From Rhoads J: Advanced Health Assessment and Diagnostic Reasoning. Philadelphia, PA: Lippincott Williams & Wilkins, 2006, p 283.)

Complete Blood Count

The CBC provides an overall indication of bone marrow production of RBCs, white blood cells (WBCs), and platelets. It also indicates the patient's hemoglobin level, hematocrit value, RBC indices, and WBC differential. (See Chapter 17, Table 17-2 for normal hemoglobin and hematocrit values.) Patients usually extract about 25% of the oxygen from saturated hemoglobin. An increase in oxygen extraction can occur in patients with extreme anemia. As the patient's extraction increases, so does his or her oxygen debt and shock state. Anemia from any cause is a major determinant in tissue hypoxia.

Red Blood Cell Indices

RBC indices are laboratory values that describe RBC structure or function. Table 46-3 presents RBC indices and some of the conditions that can cause abnormal laboratory results.

Peripheral Smear

The peripheral smear can indicate disorders of the structure of RBCs. Table 46-4 lists various abnormalities detected by examining the peripheral smear, along with further testing that may be appropriate.

Mature RBCs do not contain a nucleus. Nucleated RBCs mature in the bone marrow and are not normally present in peripheral blood. They appear in the peripheral smear after profound stimulation, such as that from acute hemorrhage, hypoxemia, hemolytic anemia, or megaloblastic anemia. If these causes are ruled out, the appearance of nucleated RBCs may be caused by infiltrative processes in the bone marrow from malignancy, myelofibrosis, or granuloma. Nucleated RBCs may also be seen in asplenic patients because the spleen normally recognizes and removes these abnormal cells.

Spherocytes and elliptocytes are abnormally shaped RBCs. They usually appear in patients with a hereditary disorder that causes RBC membrane defects. These irregular cells are trapped and destroyed in the spleen, causing hemolytic anemia. Testing for RBC osmotic fragility demonstrates that these cells are more likely to lyse than normal RBCs. Serum lactate dehydrogenase and serum bilirubin levels should be ordered if hemolysis is suspected.

The presence of Rouleaux formations (the RBCs on the peripheral smear resemble a stack of coins) can indicate multiple myeloma.[1] If clinical findings support this suspicion, serum protein electrophoresis and urine analysis for Bence Jones protein are the next steps in determining a diagnosis.

Target cells, sickled cells, and RBC cytoplasmic inclusions on the peripheral smear suggest the need for hemoglobin electrophoresis and analysis of hemoglobin F and A_2 levels. The most common anemias diagnosed in this manner are β-thalassemia and sickle cell anemia.

The presence of schistocytes in a patient with a prosthetic heart valve may indicate mechanical hemolysis. Schistocytes in a patient with fever, thrombocytopenia, renal dysfunction, and neurological abnormalities require immediate interventions for suspected TTP (see Chapter 49).

Tests to Evaluate White Blood Cells

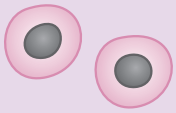
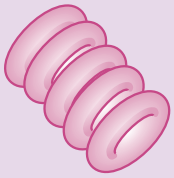
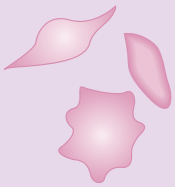
Because WBCs detect and destroy pathogens, an elevated WBC count usually indicates infection and tends to correlate with the severity of the infection.

Table 46-3 Red Blood Cell Indices: Laboratory Abnormalities

Test	Normal Value	Significance	Possible Causes for Abnormal Results
Mean corpuscular volume	82–98 m^3	Indicates the average volume of a red blood cell (RBC) in the blood sample. Low value indicates RBCs are smaller than normal (microcytic). High value indicates RBCs are larger than normal (macrocytic).	Decreased: Anemia (iron deficiency, sickle cell, hemolytic), α- or β-thalassemia, chronic disease, radiation therapy, endocarditis, diverticulitis, warm autoantibodies Increased: Alcoholism, cirrhosis, folate deficiency, vitamin B_{12} deficiency, pancreatitis, chronic lymphocytic leukemia, aplastic anemia
Mean corpuscular hemoglobin	26–34 pg	Indicates the average weight of hemoglobin in each RBC.	Decreased: Anemia (iron deficiency, microcytic, normocytic) Increased: Anemia (macrocytic, pernicious), cold agglutinin conditions, presence of monoclonal blood proteins, heparin sodium, heparin calcium
Mean corpuscular hemoglobin concentration	31%–38%	Indicates the amount of hemoglobin in the RBC compared with its size. Results expressed as hypochromic or normochromic, referring to the concentration of hemoglobin and color of RBCs.	Decreased: Anemia (iron deficiency, chronic, megaloblastic, microcytic, sideroblastic) Increased: Cold agglutinins, hereditary spherocytosis, intravascular hemolysis, heparin calcium, heparin sodium
Red cell distribution width	13.4%–14.6%	Measures the amount of homogeneity in the RBC width in the blood sample. Much variation in RBC width indicates the red cell distribution width is elevated. RBCs of similar size indicates red cell distribution width is low.	Decreased: Defects in iron reutilization Increased: Iron deficiency states
Reticulocyte count	1%–2%	Indicates the amount of immature RBCs that have been recently released from the bone marrow, expressed as a percentage of the total RBCs. Low reticulocyte count in presence of decreased RBCs indicates possible bone marrow dysfunction.	Decreased: Alcoholism, anemia (aplastic, iron deficiency, megaloblastic, pernicious), chronic infection, myxedema, radiation therapy Increased: Hemolytic anemia, hemorrhage, leukemia, malaria, polycythemia, pregnancy, sickle cell anemia, thalassemia, TTP
Serum iron	Adult men 50–160 mcg/dL Adult women 40–150 mcg/dL	Indicates the amount of iron in the serum. Low serum iron needs to be correlated with other testing (ie, ferritin, transferrin, and total iron binding capacity) to determine if iron deficiency anemia is present.	Decreased: Acute blood loss, iron deficiency anemia, gastrectomy, malabsorption, malignancy, rheumatoid arthritis, uremia Increased: Acute hepatitis, aplastic anemia, blood transfusion, hemochromatosis, lead poisoning, pernicious anemia, thalassemia, vitamin B_6 deficiency
Serum ferritin	Adult men 15–200 ng/mL Adult women 40 y or more, 11–122 ng/mL Adult women <40 y 12–263 ng/mL	Correlates well to the size of iron stores in the body. Ferritin is stored in the liver and reticuloendothelial system and released into the serum to meet the body's demand for iron.	Decreased: Hemodialysis, inflammatory bowel disease, iron deficiency anemia, gastrointestinal surgery, pregnancy Increased: Anemia (chronic, hemolytic, megaloblastic, pernicious, sideroblastic), chronic infection, chronic inflammation, chronic renal disease, excess ingestion of iron, hepatic disease, liver disease, malignancy, multiple blood transfusions, rheumatoid arthritis, thalassemia
Total iron-binding capacity	250–400 mg/dL	Indicates the maximum amount of iron that can be bound to transferrin. Useful for differentiating anemia from chronic inflammatory disorders.	Normal: Chronic inflammatory disorders Increased: Iron deficiency anemia
Serum transferrin	200–400 mg/dL	Plasma protein that transports iron by binding iron to serum transferrin receptors. Emerging as more sensitive indicator of iron deficiency and may replace more conventional indices (serum iron and ferritin).	Decreased: Cirrhosis, hemochromatosis, inflammatory states, renal disease, hemorrhage, hepatitis, hypothyroidism, microcytic anemia, pernicious anemia, thalassemia Increased: Iron deficiency states

From Chernecky CC, Berger BJ: Laboratory Tests and Diagnostic Procedures, 5th ed. Philadelphia, PA: WB Saunders, 2008.

Table 46-4 Peripheral Smear Red Blood Cell Abnormalities

Abnormality	Potential Diagnoses	Further Testing
Nucleated RBCs 	Acute hemorrhage, hypoxia, megaloblastic anemia	Vitamin B_{12} and folate levels; assess for bleeding; O_2 saturation, arterial blood gases for hypoxia
Spherocytes, elliptocytes 	Hemolytic anemia from hereditary spherocytosis, hereditary elliptocytosis	Reticulocyte count, serum bilirubin, serum lactate dehydrogenase, direct Coombs', osmotic fragility
Rouleaux formations 	Multiple myeloma	Serum protein electrophoresis, urine for Bence Jones proteins
Target cells, sickle cells, red cell cytoplasmic inclusions 	Sickle cell anemia, thalassemia	Hemoglobin studies (hemoglobin electrophoresis, hemoglobin F and A_2)
Schistocytes 	TTP, mechanical homolysis	Reticulocyte count, serum lactate dehydrogenase, serum bilirubin, coagulation studies, cardiac auscultation

White Blood Cell Count

The WBC count measures circulating leukocytes and should always be assessed in conjunction with the WBC differential and the patient's clinical condition. The WBC differential is relative and describes the percentages of the WBC subtypes (neutrophils, eosinophils, basophils, monocytes, and lymphocytes). WBC subtypes can also be measured using absolute numbers. It is important to consider both the absolute and relative values of the WBC subtypes when assessing the differential. For example, 60% segmented neutrophils may seem within normal limits, but if the total WBCs are 18,000 cells/mm^3, the absolute value (18,000 × 0.60) is 10,800 cells/mm^3, which is well above normal. Table 46-5 indicates normal absolute and differential values for the WBC count.

Conditions other than infection that may elevate the WBC count include use of steroids, trauma, stress, leukemia, hemorrhage, tissue necrosis, and dehydration. Table 46-5 summarizes abnormalities of WBC overproduction and potential physiological causes. In some cases, patients with leukemia have WBC counts greater than 100,000 cells/mm^3 because of excessive bone marrow production of blast cells (immature granulocytes). These patients are at risk for leukostasis, in which blasts aggregate in the capillaries of the brain and lungs. Clinical findings of leukostasis include headache, confusion, central nervous system infarcts, acute respiratory insufficiency, and pulmonary infiltrates.

A low WBC count usually indicates decreased production caused by immunosuppressive therapy or a disorder of bone marrow production due to infiltrative processes or bone marrow failure. Decreases in circulating neutrophils may be caused by decreased production from bone marrow injury, bone marrow infiltration, nutritional deficiencies, or congenital defects of the stem cells in the bone marrow. Other causes of neutropenia are splenic sequestration and destruction, immune-mediated granulocyte destruction, or overwhelming infection. Lymphocytopenia is most commonly caused by malignancy, followed by collagen vascular disease. Acquired immunodeficiency syndrome (AIDS) and AIDS-related complex are other notable causes of lymphocytopenia (see Chapter 48).

There are numerous potential abnormalities in the WBC differential. A left shift refers to an increase in the number of bands (neutrophil precursors), which usually indicates an

Table 46-5 White Blood Cell (WBC) Count and Differential: Laboratory Abnormalities

Test	Normal Value: Relative	Normal Value: Absolute (cells/mm^3)	Significance	Possible Causes of Abnormal Results
WBC count		4,500–10,000	Measures number of WBCs	Infection, inflammation, leukemia, trauma, stress
Differential	All percentages for various types of WBCs must add up to 100%		Describes the percentage of each WBC found in blood	See examples of specific cell type below
Granulocytes	50%–70%		Type of WBC categorized by presence of granules in cytoplasm	See specific granulocyte subtypes below
Segmented neutrophils	3%–5%	2,500–7,000	Mature neutrophil with nuclei segmented into lobes	Increased: bacterial infection, inflammatory disorder, tissue destruction, malignancy, drug-induced hemolysis, diabetic ketoacidosis, myeloproliferative disorders, idiopathic, smoking, obesity Decreased: compromised immune system, depressed bone marrow, heart-lung bypass, hemodialysis, overwhelming infection, tuberculosis, typhoid
Band neutrophils	1%–3%	135–500	Immature neutrophil with nuclei that have smooth edges, unsegmented	Increased: acute stress, active bacterial infection Decreased: compromised immune system
Eosinophils	0.4%–1%	100–300	Also known as acidophils (acid loving); combat infections caused by parasites; play a role in allergic reactions; cause bronchoconstriction in asthma	Increased: parasitic infection, asthma, allergies, dermatoses (hives and eczema), adrenal insufficiency Decreased (note: a low eosinophil count is not a cause for concern); Cushing's disease; administration of glucocorticosteroids; various pharmaceuticals
Basophils	4%–6%	40–100	Similar mechanism to mast cells in allergic response; triggered by immunoglobulin E binding to antigens; releases proinflammatory mediators	Increased: hyperlipidemia, viral infections (smallpox, chickenpox), inflammatory conditions (ulcerative colitis, chronic sinusitis, asthma), Hodgkin's lymphoma, increased estrogen, hypothyroidism, myeloproliferative disorders Decreased: stress, hyperthyroidism, pregnancy
Monocytes	25%–35%	200–600	Monocytes become macrophages after migration into tissue; macrophages perform phagocytosis	Increased: viral infection, parasitic infection, myeloproliferative disorders, inflammatory bowel disease, sarcoidosis, cirrhosis, drug reactions Decreased: administration of glucocorticoids, aplastic anemia, lymphocytic anemia
Lymphocytes		1,700–3,500	Primary source of viral defense and antibody production	Increased: viral infections, pertussis, tuberculosis, acute lymphoblastic leukemia, cytomegalovirus infection, mononucleosis, post transfusion, splenomegaly, hyperthyroidism, connective tissue disorder Decreased: AIDS, bone marrow suppression, aplastic anemia, steroid use, neurological disorders (multiple sclerosis, myasthenia gravis, Guillain-Barré syndrome)

infectious process. The presence of blasts in the peripheral blood is always an aberrant finding and suggests the presence of leukemia or a myeloproliferative disorder.

T- and B-Lymphocyte Tests

As discussed in Chapter 45, lymphocytes are classified as T cells and B cells. T cells are important in the body's ability to distinguish between self and nonself. Monoclonal antibodies against specific lymphocyte surface proteins are used to identify types of circulating lymphocytes and their subset populations, which can be useful in characterizing hematological malignancies and identifying immunological and autoimmune diseases. A specific example of this is assessment of the $CD4^+$ subpopulation of T cells in patients with AIDS; a $CD4^+$ count of less than 400/mm^3 is associated with a poorer prognosis. Table 46-6 lists possible causes of lymphocytosis and lymphopenia.

When an antigen stimulates B cells, they differentiate into plasma cells and produce antibodies. Although plasma cells reside in lymphoid tissue, their antibody production can be evaluated through serum and urine protein electrophoresis. Autoimmune diseases occur when the body produces antibodies directed against its own tissues. These diseases can be organ specific (eg, Graves' disease in the thyroid) or widely disseminated and involve multiple organs, such as systemic lupus erythematosus, which can attack almost every body system. Laboratory testing is aimed at the detection of serum antibodies against various tissues. C-reactive protein, antinuclear antibody, rheumatoid factor, and erythrocyte sedimentation rate are additional tests used in diagnosing autoimmune disorders.[2]

Tests to Evaluate Disorders of Primary Hemostasis

Laboratory testing that evaluates hematological dysfunction should be guided by the information gathered in the history and physical examination. Family history, underlying clinical conditions, and the duration and type of abnormal bleeding can indicate appropriate testing and diagnostic workup. Because hematological and immune disorders are so pervasive in the human body, the nurse must be careful not to disregard innocuous symptoms. The patient's history must be thorough.

Primary bleeding disorders are caused by problems with platelets and small blood vessels that can result in subtle bleeding. For instance, mucocutaneous bleeding, petechiae, and superficial purpura may be early signs of impending critical illness. Decreased platelets or increased capillary fragility can cause the sudden appearance of petechiae, especially in dependent areas, such as the lower extremities.

Platelet Count

The primary phase of hemostasis involves aggregation of platelets at the site of vessel injury. These platelets initiate the coagulation cascade, which results in the deposit of fibrin at the site of injury to stabilize the clot (secondary hemostasis). See Chapter 45, Figure 45-2 for an illustration of the processes that occur during hemostasis.

When evaluating primary hemostasis, one first obtains a platelet count from the CBC. A platelet count of less than 150,000/mm^3 is abnormal, but bleeding from thrombocytopenia alone usually does not happen unless the platelet count falls below 20,000/mm^3.[3] However, prolonged bleeding from surgery or trauma may occur with platelet counts of 40,000 to 50,000/mm^3. Severe, spontaneous hemorrhage may result when platelet counts reach 5,000 to 10,000/mm^3. Causes of thrombocytopenia include decreased bone marrow production, splenic sequestration due to splenomegaly, or peripheral destruction of platelets by the body's own immune system. Disseminated intravascular coagulation (DIC) and TTP (see Chapter 49) are other serious disorders that involve low platelet counts. Drugs are the first suspect in thrombocytopenia (Box 46-3); once the nurse has ruled them out, she or he moves to consider other causes (Box 46-4). Finally, one must consider that

Table 46-6 Possible Causes of Lymphocytosis and Lymphopenia

Finding	Possible Causes
Lymphocytosis	Lymphatic leukemia, Epstein-Barr virus, viral infections of the upper respiratory track, cytomegalovirus, measles, mumps, chickenpox, acute HIV infection, infectious hepatitis, tuberculosis, Crohn's disease
Lymphopenia	Aplastic anemia, Hodgkin's disease, acquired immune deficiency syndrome, congestive heart failure Drugs include chemotherapeutic agents, immunosuppressive medications, radiation therapy

From Fischbach FT, Dunning MB: A Manual of Laboratory and Diagnostic Tests, 8th ed. Philadelphia, PA: Wolters Kluwer: Lippincott Williams & Wilkins, 2009.

BOX 46-3 PATIENT SAFETY

Drugs That Decrease Platelet Production or Function

The drugs listed below may cause complications, interactions, or other undesired effects.

- Aspirin
- Barbiturates
- Cimetidine
- Chemotherapeutic agents
- Chloramphenicol
- Chlorothiazides
- Digitalis
- Digitoxin
- Furosemide
- Glyburide
- Ibuprofen
- Penicillin
- Phenobarbital
- Quinidine
- Streptomycin
- Sulfonylureas
- Tetracycline

From Fischbach FT, Dunning MB: A Manual of Laboratory and Diagnostic Tests, 8th ed. Philadelphia, PA: Wolters Kluwer: Lippincott Williams & Wilkins, 2009.

BOX 46-4 Causes of Platelet Disorders in the Intensive Care Unit

Thrombocytopenia
Heparin (1% to 3%)
Sepsis (>50%)
AIDS (40% to 60%)
Disseminated intravascular coagulation (DIC)
Thrombotic thrombocytopenic purpura (TTP)

Abnormal Platelet Function
Renal insufficiency
Cardiopulmonary bypass
Aspirin
Dextran

The incidence of platelet disorders is indicated in parentheses.

From Marino PL: The ICU Book, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1998, p 710.

some people may produce adequate numbers of platelets but ones that function abnormally.

An elevated platelet count greater than 400,000/mm^3 indicates increased platelet production or decreased platelet destruction. These platelets may function abnormally, causing aberrant bleeding and clotting. A cause of primary thrombocytosis is bone marrow disease. Causes of reactive thrombocytosis include chronic inflammation, infection, malnutrition, acute stress, malignancy, splenectomy, or the postoperative state.

Peripheral Smear

A peripheral blood smear may reveal megathrombocytes (large platelets), which may be present during premature platelet destruction. Also, note that some patients' platelets clump when exposed to ethylenediamine tetraacetic acid (the anticoagulant used in the "purple top" or CBC tube). Examination of the peripheral smear shows this clumping, and a repeat CBC in a heparinized "green top" blood collection tube reveals an accurate platelet count.

Platelet Function Assay

The platelet function assay is a screening method that tests platelet function in adhesion and aggregation quality. This test is helpful in the evaluation of platelet function in patients with menorrhagia, drug-induced platelet dysfunction, and high-risk pregnancy.

Bleeding Time

The bleeding time test assesses the length of time required for a clot to form at the site of vessel injury. A prolonged bleeding time in a patient with a normal platelet count may indicate a disorder of platelet function that requires further testing. Remember that patients can bleed to hemorrhage with a normal platelet count if the platelets are not functioning. A deficiency in factor VIIIR (von Willebrand's disease) results in decreased ability of the platelets to adhere to the injured vessel wall. Uremia from renal failure, drugs (especially aspirin), foods, and spices can also cause abnormal platelet function. Box 46-3 lists some drugs known to decrease platelet production or function. Platelet aggregation studies are performed to detect inherited or acquired disorders in platelet function.

Prothrombin Time and Activated Partial Thromboplastin Time

Screening for coagulation abnormalities includes evaluating the prothrombin time (PT) and the activated partial thromboplastin time (aPTT). Prolongation of either of these tests indicates coagulation factor deficiencies or inhibition. The PT test screens for dysfunction in both the extrinsic portion that includes tissue thromboplastin and factor VII and in the common pathway that includes factors X, V, and II and fibrinogen. Prolongation of PT can result from disorders, such as liver disease, vitamin K deficiency, clotting factor deficiencies, or DIC. Numerous medications, including allopurinol, aspirin, β-lactam antibiotics, chlorpropamide, digoxin, diphenhydramine, and phenytoin sodium, can also cause a prolonged PT. PT is also used to monitor patient response to anticoagulation therapy with warfarin.

In 1983, the World Health Organization introduced the international normalized ratio (INR) to provide a common standard for interpretation of PT. The INR value depends on the sensitivity ratio of the thromboplastin reagent used in the laboratory to the International Reference Preparation. It is now widely accepted practice to assess the level of anticoagulation and warfarin dosing based on the INR ratio.

The aPTT measures how well the coagulation sequences of the intrinsic pathway (factors XII, XI, IX, VIII) and the common pathway (factors X, V, II, and fibrinogen) are functioning. An elevated aPTT could indicate disorders of any coagulation factors except VII and XIII. Clinical conditions associated with an elevated aPTT include DIC, von Willebrand's disease, and liver disease. Drugs that may affect aPTT include chlorpromazine, codeine, phenothiazines, salicylates, and warfarin. The aPTT test is also used to monitor patient response to heparin therapy.

- **Thrombin time:** Thrombin time is a test that measures the clotting time of a sample of plasma to which thrombin has been added. Thrombin is important in converting fibrinogen to fibrin in the final phase of the coagulation cascade. Thrombin time is increased in conditions such as DIC, liver disease, clotting factor deficiencies, shock, and hematological malignancies. Decreased thrombin time occurs with thrombocytosis.
- **Fibrinogen level:** Fibrinogen is converted by thrombin to fibrin, which then combines with platelets to form a stable clot. Patients with DIC, severe liver disease, sepsis, TTP, or trauma have a low fibrinogen level. Elevated fibrinogen levels occur in conditions involving tissue damage and inflammation.
- **Fibrin degradation product level:** Fibrin degradation products (FDPs) accumulate when a large amount of clotting has occurred and has then been broken down. Increased FDPs, along with an elevated PT/aPTT, decreasing platelets, and a low fibrinogen level, indicate possible DIC (see Chapter 49).
- **D-Dimer:** D-Dimer is a more specific test than FDP measurement for detecting an event in which fibrin is being broken down. Its indications include ruling out and monitoring DIC, deep venous thrombosis, venous and arterial thrombotic conditions, and the monitoring of thrombolytic therapy. Recent studies continue to support the often disputed claim that D-dimer is beneficial in the evaluation of acute pulmonary embolism.[4]

Tests to Evaluate Disorders of Secondary Hemostasis

Disorders of secondary hemostasis involve clotting factor deficiencies and are characterized by recurrent oozing of blood and hematoma formation. The onset of these symptoms may be delayed because of the initial plugging of the vessel injury; however, defective clotting mechanisms fail to provide a stable fibrin clot.

When assessing disorders of secondary hemostasis, one must determine whether the disorder is congenital or acquired. A history of excessive or recurrent bleeding in the individual or family members suggests a congenital disorder as the more likely cause. Table 46-7 describes potential complications experienced by people with congenital bleeding disorders. The most common congenital bleeding disorders are von Willebrand's disease, hemophilia A, and hemophilia B. PT and aPTT are ordered for patients with suspected congenital disorders, along with factor VIIIR, VIII, and IX assays. A deficiency in von Willebrand's factor (VIIIR) results in the decreased ability of platelets to adhere to the injured vessel wall and a deficiency of factor VIII. A deficiency of factor VIII causes hemophilia A; a deficiency of factor IX causes hemophilia B. Table 46-8 summarizes laboratory abnormalities that indicate these congenital disorders.

An acute bleeding problem without a prior history of chronic bleeding suggests an acquired disorder. Acquired disorders of hemostasis occur with vitamin K deficiency, severe trauma, hemorrhage, massive transfusion, overwhelming infection, severe liver disease, and DIC. A deficiency of vitamin K decreases synthesis of prothrombin, factor VII, factor IX, and factor X. Liver disease impairs the absorption of vitamin K by decreased production of bile salts necessary for vitamin K absorption from the gut or through obstruction of the biliary system. Dysfunctional hepatocytes in the liver are unable to produce the vitamin K–dependent factors, as well as fibrinogen; factors V, XI, XII, and XIII; and other clotting factors. Some patients with liver disease may demonstrate thrombotic tendencies caused by decreased liver synthesis of anticoagulants, such as protein C, protein S, plasminogen, and antithrombin III.

Laboratory testing for an acquired disorder of hemostasis varies based on the suspected etiology of the disorder. In general, testing includes PT, aPTT, thrombin time, bleeding time, liver enzyme and liver function tests, fibrinogen levels, and FDPs. Table 46-9 summarizes laboratory abnormalities that indicate some of the acquired coagulation disorders.

Table 46-7 Potential Complications in Congenital Bleeding Disorders

Site	Potential Complication
Abdomen	Hypotension, hypovolemic shock (eg, retroperitoneal)
Muscle	Compartment syndrome
Joint	Hemarthrosis with destruction of bone and cartilage in joint capsule
Intracranial	Increased intracranial pressure
Retropharyngeal	Airway obstruction
Gastrointestinal	Anemia, melena
Urinary tract	Hematuria; clots in ureters may occur after factor administration

From Stabler SP: Hemophilia. In Wood ME (ed): Hematology/Oncology Secrets, 3rd ed. Philadelphia, PA: Hanley & Belfus, 2003.

A hypercoagulable state causes an increased tendency for thrombosis. Box 46-5 summarizes some of the risk factors for hypercoagulability. Hereditary thrombotic disease is a group of genetic abnormalities causing defects of coagulation, fibrinolysis, or their regulatory systems. Laboratory abnormalities in hereditary hypercoagulability include deficiencies of antithrombin III, protein C, protein S, plasminogen, tissue plasminogen activator, and dysfibrinogen; however, 65% to 70% of the causes remain unknown. Lupus anticoagulant (LA) is an autoimmune disorder in which patients have an elevated aPTT, yet thrombosis develops in 30% of the patients. LA is confirmed by the presence of anticardiolipin antibodies, positive platelet neutralization procedure, or positive dilute Russell viper's venom test.

Tests to Evaluate Hematological and Immune Disorders

Table 46-10 lists common laboratory tests to assess immune system functioning. Diagnostic tests for human immunodeficiency virus (HIV) are listed in Chapter 48, Table 48-4.

Bone marrow aspiration and biopsy are the most important diagnostic tests for determining bone marrow function. The biopsy provides information about the precursors of the blood's components to determine whether hematological

Table 46-8 Laboratory Abnormalities in Congenital Bleeding Disorders*

	PT	aPTT	vWF	vWF Antigen	VIII	IX	BT†
von Willebrand's	N	↑	↓	↓	↓	N	↑
Hemophilia A	N	↑	N	N	↓	N	↑
Hemophilia B	N	↑	N	N	N	↓	↑

*Other congenital bleeding abnormalities are rare and not mentioned in this text.

†BT depends on the severity of the condition; it may be normal in mild cases.

PT, prothrombin time; aPTT, activated partial thromboplastin time; vWF, von Willebrand's factor; VIII, factor VIII; IX, factor IX; BT, bleeding time; N, normal.

Table 46-9 Laboratory Abnormalities in Acquired Bleeding Disorders

	PT	aPTT	TT	FDP	Plt
Vitamin K deficiency	X	X			
Liver disease: Acute hepatitis, early liver disease	X				
Chronic liver disease	X	X	X	X	X
DIC	X	X	X	X	X
Massive transfusion	X	X	X		X

PT, prothrombin time; aPTT, activated partial thromboplastin time; TT, thrombin time; FDP, fibrin degradation products; Plt, platelets; DIC, disseminated intravascular coagulation; X, elevated laboratory result.

abnormalities are a production defect. Bone marrow examination is useful in detecting infiltrative processes, such as malignancy, which can affect blood cell production. This procedure is also performed to determine response to therapy in patients with hematological malignancies or solid tumor infiltration of the bone marrow.

Tissue biopsy may be performed on skin lesions in which malignancy (eg, cutaneous T-cell lymphoma) or an autoimmune process (eg, pemphigus) is suspected. Lymph node biopsy is required for lymphadenopathy that does not appear to be caused by an infectious process.

Internal lymph nodes of the chest, abdomen, and pelvis can be evaluated by computed tomography (CT) scanning. A CT scan may be used to determine the presence of masses in suspected malignancy, especially lymphoma. Positron emission tomography is used in lymphoma and non-Hodgkin's lymphoma to diagnose and stage cancer, evaluate response to therapy, and assess for recurrence. Liver disease, an important factor in coagulopathy, and splenomegaly may also be evaluated through a CT scan. A skeletal survey (skull, vertebrae, ribs, pelvis, arms, forearms, thighs, and lower legs) is done in patients with suspected multiple myeloma to assess for the typical "punched-out" lytic lesions that occur in this condition.

Intradermal skin testing is used to evaluate cell-mediated immunity. Various antigens are injected just below the skin's surface to check for delayed-type hypersensitivity. Commonly used antigens include mumps, *Candida* species, trichophytin, and tuberculin. If the patient fails to respond to the injected antigens, they are said to have cutaneous anergy. This implies a defect in the patient's cellular immunity. Some causes of cutaneous anergy include AIDS; acute leukemia; chronic lymphocytic leukemia; carcinoma; Hodgkin's disease; non-Hodgkin's lymphoma; congenital immune conditions; bacterial, fungal, or viral infections; immunosuppressive medications; cirrhosis; and malnutrition.

BOX 46-5 *PATIENT SAFETY*

Risk Factors for Hypercoagulability

Physiological
- Pregnancy
- Postpartum
- Venous stasis
- Age more than 40 years
- Immobilization
- Varicose veins
- Previous venous thromboembolism

Pathological
- Malignancy
- Liver disease
- Disseminated intravascular coagulation
- Polycythemia
- Lupus anticoagulant
- Vascular injury
- Sepsis
- Heart failure
- Myocardial infarction
- Inherited abnormalities

Environmental
- Smoking
- Stress
- Heat

Iatrogenic
- Surgery
- Postsurgical
- Oral contraceptives
- Estrogens

▲ Assessment of the Immunocompromised Patient

Immunocompetence refers to the body's ability to protect itself against disease (see Chapter 45). Figure 46-4 illustrates areas to be assessed for immunocompetence.

It is essential that critically ill patients' immunocompetence be assessed at frequent intervals. Physical and psychological stress from overwhelming illness or trauma in the critically ill patient can depress functioning of the immune system. Invasive procedures, indwelling catheters, intravenous lines, mechanical ventilation, nutritional compromise, and the intensive care environment itself can predispose patients to infections and sepsis. As always, handwashing is the best tool available for these patients. Also, patient teaching and aseptic technique are essential to minimize exposure to infectious organisms. The nurse closely monitors potential sites of infection, changes or fluctuations in body temperature, nutritional status, and laboratory findings for indications of compromised immune function or the onset of infection. Septic shock is a life-threatening complication that can develop rapidly in immunocompromised patients. Patient outcomes are greatly improved when septic shock is detected in its early stages and interventions are instituted promptly. Box 46-6 presents signs of early septic shock (also see Chapter 54).

History

Reducing susceptibility to infection is key to the care of patients in the ICU, and the history can identify susceptibility and guide critical care nursing practice. The type of infection often provides the clues regarding the nature of

Table 46-10 Common Laboratory Tests of the Immune System

Laboratory Test	Reference Range	Use
C-reactive protein	Not available; levels <10 mg/L indicate patient no longer has clinically active inflammation; high or increasing levels are consistent with infection and/or inflammation	Evaluation of various inflammatory conditions, including rheumatoid arthritis and systemic lupus erythematosus
Antinuclear antibody	Low titers are negative; elevated titers demonstrate an elevated concentration of antinuclear antibodies	Screening and diagnosis of autoimmune disorders
Human leukocyte antigen (HLA) typing	Reported as phenotype for each of the six HLA loci tested. HLA (protein marker found on most of the body's cells) typing involves either serologic or DNA methods. Antibody screen test is reported as the percentage of panel reactive antibodies (PRAs); percent PRA is number of wells reactive with patient's serum expressed in percent. Cross-match is reported as compatible or incompatible.	Determination of tissue compatibility for organ transplantation; also used to determine paternity and to diagnose HLA-related disorders
Erythrocyte sedimentation rate (ESR)	1–13 mm/h for males; 1–20 mm/h for females	Evaluation of inflammatory state; females tend to have a higher ESR; ESR increases with age
Immunoglobulins (Igs)	IgA: 160–260 mg/dL IgG: 950–1,550 mg/dL IgM: 50–300 mg/dL IgD: 0–9 mg/dL IgE: 0.002–0.2 mg/dL	Assessment of immunodeficiency state and certain cancers, including multiple myeloma and macroglobulinemia; also used to assess response to immunizations
Complement system	C3: 75–150 mg/dL C4: 13–40 mg/dL	Diagnosis of systemic lupus erythematosus and other immunological disorders

the immune defect. For example, patients with defects in humoral immunity may have recurrent or chronic bacterial infections, such as meningitis or bacteremia. Repeated viral or fungal infections can indicate a defect in cell-mediated immunity. (See Chapter 45 for a review of humoral and cell-mediated immunity.)

Risk Factors for Immunocompromise

Certain factors, such as chronological age, place critically ill patients at a higher risk of immunocompromise. A patient who has a chronic disease or is already immunosuppressed may also be predisposed to further immune difficulties. Finally, certain medications and treatments can alter a patient's immunocompetence, as can his or her nutritional status and skin integrity. Nurses should be especially aware of these risk factors during their assessment of immunocompetence.

Age

The patient's chronological age influences immunocompetence. Immune response may be depressed in the very young because of the underdevelopment of the thymus gland. Older patients experience a decline in immune system function, making them more susceptible to infections. Thus, they should be closely assessed for alterations in their immunocompetence. Box 46-7 describes factors that contribute to the overall decline of immunocompetence in older people.

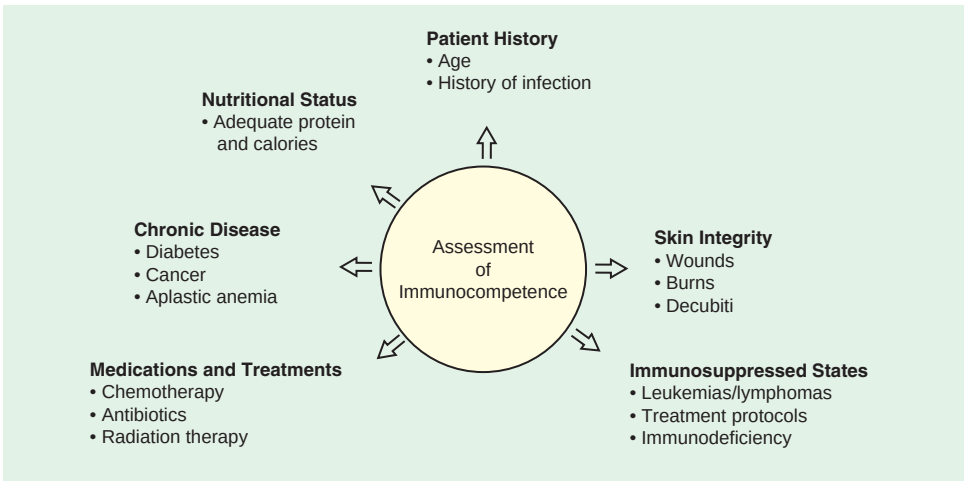


FIGURE 46-4 ▲ In addition to obtaining his or her history, assessment of the immunocompromised patient should cover six major areas.

BOX 46-6 PATIENT SAFETY

Signs of Early Septic Shock

- Fever
- Chills
- Confusion
- Irritability
- Tachycardia
- Tachypnea
- Decreased peripheral pulses
- Hypotension
- Warm, dry skin

Chronic Disease

Many chronic diseases are associated with compromised immune functioning. Diabetes, hepatic disorders, cancer, and aplastic anemia are just a few examples of diseases in which immune deficiencies occur. Because many critically ill patients have an underlying chronic disease, the existence and severity of such diseases should be considered contributing factors to immunocompromise when these patients are assessed.

Immunosuppressed States

Patients with leukemia, lymphoma, multiple myeloma, and other hematological conditions can experience impaired immunity and recurrent infections. Immunodeficiency states can be congenital or acquired. People with congenital immunodeficiencies frequently do not survive childhood. Immunodeficiency syndromes in adults may occur through a spontaneous defect in the immune system or through HIV infection (see Chapter 48).

Patients who are severely immunosuppressed have impaired responses to infectious agents and may not display the typical signs of infection. Fever and redness or pus at infection sites may be diminished because of the decreased numbers of WBCs required to promote these physical signs. Nurses should thus be extremely vigilant about monitoring for potential infection.

Medications and Treatments

Many medications affect immunocompetence. Antibiotics, such as tetracycline and chloramphenicol, impair bone marrow function. Steroids display many immunological effects, including decreased lymphocyte and antibody concentration. Patients who have received organ or bone marrow transplants (see Chapter 47) often must remain on medications (eg, cyclosporine) that severely suppress the immune system. Patients placed on treatment regimens with any immunosuppressive medications are monitored for early symptoms of infection that would indicate compromise in immune functioning.

Various treatments also impair immunocompetence. Treatment protocols for patients with cancer can lead to life-threatening complications, such as infection and sepsis. Biological therapy with interferon-α and interleukin-2 can cause leukopenia. Patients who receive multiple transfusions with RBCs can demonstrate suppressed immunity. Most chemotherapeutic agents and radiation to the pelvis, spine, ribs, sternum, skull, and metaphyses of the long bones can adversely affect the bone marrow's ability to produce WBCs. The lowest point in WBC levels, or the nadir, may not be seen until several days or weeks after the initiation of treatment. The absolute neutrophil count (ANC) is calculated in neutropenic patients to determine the degree of immunosuppression. The ANC is calculated as follows:

1. Add segmented neutrophils and band neutrophils (from the WBC differential).
2. Multiply the total WBC count by the total obtained in step 1.

Example:

Segs = 42%

Bands = 10%

Total WBC count = 4,100 cells/mm^3

42 + 10 = 52%

4,100 × 0.52 = 2,132 cells/mm^3 (ANC)

Usually, protective measures, such as those summarized in Box 46-8, are instituted for patients with an ANC of less than 1,000 cells/mm^3.[5] However, all patients in the ICU are considered at risk for immunocompromise and should have the benefit of scrupulous handwashing, rigorous monitoring, and protective interventions.

BOX 46-7 CONSIDERATIONS FOR THE OLDER PATIENT

Factors Contributing to Diminished Immunocompetence

- Decline in immune system functioning
- Decreased nutritional intake (decreased taste, poor teeth, declining appetite)
- Chronic illnesses (diabetes, chronic obstructive pulmonary disease, renal disease)
- Increased risk for malignancy
- Possible urinary incontinence
- Prostatic hypertrophy and urinary retention
- Skin breakdown and impaired wound healing
- Decreased ability to care for self
- Impaired communication
- Decreased mobility

BOX 46-8 NURSING INTERVENTIONS

For the Immunocompromised Patient*

- Provide a private room or uninfected roommate.
- Use a laminar flow or positive pressure room.
- Maintain strict handwashing with antiseptic soap.
- Use no rectal thermometers, enemas, or suppositories.
- Restrict staff and visitors with infections (or require masks).
- Provide patient with a mask when he or she goes to other departments or crowded areas.
- Permit cooked foods only.
- Allow no fresh flowers or live plants.
- Avoid sources of stagnant water (vases, water pitchers, humidifiers, denture cups).

*Precautions taken may vary according to institutional policy and the severity of immunosuppression.

Nutritional Status

The patient's nutritional status has a major impact on immune function. Inadequate intake of protein and calories can alter immune responses and resistance to infection by decreasing lymphocyte and antibody production as well as impairing wound healing. A multidisciplinary approach including a nutritionist can assist the nurse in assessing dietary intake and nutritional requirements for the critically ill immunocompromised person. Supplemental intravenous or enteral feedings may be necessary to prevent further deterioration of the body's nutritional status and ability to fight infection.

Skin Integrity

The integumentary system, including the skin and mucous membranes, provides a physical barrier to infection. Surgical or traumatic wounds, burn injuries, or pressure sores breach these physical defenses and predispose the critically ill patient to infection. Also, in a critical care setting in which intravenous and intraarterial catheters, urethral catheters, or endotracheal tubes are used, multiple portals of entry for pathogens can provide simultaneous sites for potential infection. Therefore, all wounds and portals of entry should be carefully monitored for signs and symptoms of infection.

▲ Clinical Applicability Challenges

CASE STUDY

T.R. is a 53-year-old African American male with a history of hypertension, diabetes mellitus, end-stage renal disease, and hyperlipidemia who presented to the emergency department status postdialysis with chief complaint of weakness in bilateral lower extremities. T.R. reports being "too weak to stand up" and also complains of "cloudy thinking." He also states that he has had "chills" and the "shakes" for the last 48 hours.

T.R. appears to be unbathed and in poor general health. A moist, bloody dressing is found over the catheter site. The area around the catheter insertion site is red and warm to the touch.

T.R. is febrile with a temperature of 39.8°C. Blood pressure (BP) is 85/45 mm Hg, heart rate is 168 beats/min, respiratory rate is 20 breaths/min, SpO_2 at 94% on room air. He is in no apparent distress but is lethargic. There is no jugular venous distention, lungs are clear to auscultation bilaterally, and there is no wheezing, crackles, or ronchi. A 12-lead ECG demonstrates normal sinus rhythm with a right bundle branch block and nonspecific T-wave inversion.

Results from a chemistry panel include sodium 129 mEq/L; potassium 5.0 mEq/L; chloride 96 mEq/L; bicarbonate 26 mEq/L; BUN 22 mg/dL; creatinine 2.12 mg/dL; and glucose 90 mg/dL.

On admission to the emergency department, a blood specimen is drawn for a complete blood count with differential, and the following results were reported: WBC 12.9×10^3 µL; neutrophils 91.5%, lymphocytes 1.6%; monocytes 7.4%; eosinophils 0%; and basophils 0%. The following day, after being admitted to the intermediate care unit, T.R.'s WBCs were 15.89×10^3 µL with the following differential: neutrophils 90.4%, lymphocytes 3.2%; monocytes 5.7%; eosinophils 0%; and basophils 0%. His hemoglobin and hematocrit were stable at 9.8 g/100 mL and 29%, respectively. He remained febrile with a temperature of 38.0°C and hypotensive with a BP of 90/62 mm Hg.

1. What is T.R.'s primary health problem?
2. Based on assessment of the WBC differential, what type of infection is T.R. most likely to have?
3. Identify at least three subjective findings that are consistent with T.R.'s primary health problem.
4. Identify at least four objective findings that are consistent with T.R.'s primary health problem and discuss how these findings are implicated in the primary health problem.
5. What is the significance of the abnormal hemoglobin and hematocrit values?

References

1. Kyle RA, Rajkumar SV: Criteria for diagnosis, staging, risk stratification and response assessment of multiple myeloma. Leukemia 23(1): 3–9, 2009
2. Fischbach FT, Dunning MB: A Manual of Laboratory and Diagnostic Tests, 8th ed. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2009
3. Simon D, Kunicki T, Nugent D: Platelet function defects. Haemophilia 14(6):1240–1249, 2008
4. Gupta RT, Kakarla RK, Kirshenbaum KJ, et al: D-Dimers and efficacy of clinical risk estimation algorithms: sensitivity in evaluation of acute pulmonary embolism. AJR Am J Roentgenol 193(2):425–430,2009
5. Miller J, Starks B. Deciphering clues in the CBC count. Nursing 40(7):52–55, 2010

47 Organ and Hematopoietic Stem Cell Transplantation

Sandra A. Mitchell, Jo Ann Hoffman Sikora, and Elizabeth Holderness

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Analyze the criteria used to evaluate and prepare patients for transplantation.
2. Evaluate the principles of organ and hematopoietic stem cell compatibility and immunosuppression.
3. Discuss nursing assessment and management for patients undergoing solid organ transplantation (kidney, liver, heart, pancreas, lung) or hematopoietic stem cell transplantation (HSCT).
4. Describe the early and late-phase complications of organ and HSCT.

Transplant research began in the early 1900s, but kidney transplantation did not become a realistic treatment for chronic renal failure in humans until the early 1950s. Heart and liver transplantations followed in the 1960s and since the 1980s, have steadily increased in frequency as a treatment for end-stage organ failure. Pancreas transplantation also began in the mid-1960s with good graft survival rates being achieved in the 1980s. The number of lung transplantations is small, primarily because of a lack of medically suitable donors. Table 47-1 gives survival rates for solid organ transplantations.

During the past 40 years, hematopoietic stem cell transplantation (HSCT) has evolved from an experimental treatment for patients with advanced acute leukemia into a therapeutically effective modality that is now standard therapy for selected diseases. HSCT, which is known to be curative in several malignant and nonmalignant disorders, is a transplantation using hematopoietic stem cells at various stages of differentiation and maturation. Decreased treatment-associated mortality and improved supportive care have helped make this possible.

Astute nursing care is essential to prevent treatment-related complications and death. Other factors that may affect the outcomes of HSCT include the type and stage of disease at the time of transplantation, the type of transplant (allogeneic versus autologous), the degree of human leukocyte antigen (HLA) matching in allogeneic transplants, the stem cell source, the intensity of the conditioning regimen, the ages of both the donor and the recipient, and the experience of the transplantation center. In general, the transplant-related mortality risk in allogeneic HSCT is about 20% to 30% higher than in autologous HSCT. The transplant-related mortality rate in autologous HSCT is less than 5% at most centers.

Even when a patient is cured of the original disease, he or she may experience delayed and long-term complications that can shorten or negatively affect the quality of his or her remaining life. These complications include infections, chronic graft-versus-host disease (GVHD; seen in allogeneic HSCT), thyroid dysfunction, pulmonary complications, cataracts, and the development of second malignancies. In general, autologous transplantation has fewer long-term complications, largely because no GVHD is associated with autologous transplantation. Disease-free survival 5 years after HSCT varies substantially, depending on the age of the recipient, the underlying disease, prognostic risk factors, disease status at the time of transplantation, the type of HSCT procedure, and the extent of prior treatment. Depending on these factors, disease-free survival rates vary from 10% to 75%[1–7] (Table 47-2). Advances in histocompatibility matching, immunosuppression, stem cell collection, and cryopreservation techniques, as well as the development of safer and less toxic conditioning regimens along with more effective drugs to manage posttransplantation infections and stimulate hematopoiesis, have helped increase the success of HSCT.[8]

This chapter describes the major aspects of care for patients receiving kidney, liver, heart, pancreas, and lung transplantations and HSCT. It covers principles that apply to all types of transplantation and discusses content unique to specific types of transplants.

Table 47-1 Graft and Patient 1-Year Survival Rates for Adult Solid Organ Transplants Performed in 2005–2006

Organ	Graft Survival Rate (%)	Patient Survival Rate (%)
Kidney—living donor	95	98
Kidney—deceased donor	90	95
Heart	87	87
Lung	82	84
Liver—living donor	85	90
Liver—deceased donor	82	87
Pancreas	81	98
Kidney–pancreas	92 (kidney); 86 (pancreas)	95
Intestine	72	78

Data from the 2008 Annual Report of the U.S. Organ Procurement and Transplantation Network and the Scientific Registry of Transplant Recipients: Transplant Data 1998–2007. U.S. Department of Health and Human Services, Health Resources and Services Administration, Healthcare Systems Bureau, Division of Transplantation, Rockville, MD.

▲ Indications for Transplantation

Many factors influence the indications and patient eligibility for transplantation. Currently, end-stage disease is the primary reason for most organ transplantations. HSCT is now used when bone marrow is defective or destroyed by a disease process or as a result of treating an underlying disease.[9] New information concerning patient outcomes, complications, surgical techniques, immunosuppressive drugs, and availability of organs and hematopoietic stem cells is also considered. Table 47-3 presents indications for transplantation.

Table 47-2 Disease-Free Survival 5 Years After Hematopoietic Stem Cell Transplantation

Disease and Stage	Allogeneic (% Survival)	Autologous (% Survival)
Acute myeloid leukemia		
First remission	45–70	40
First relapse, second or later remission	23–45	20–30
Refractory, multiply relapsed	10–15	<10
Acute lymphocytic leukemia		
First or second remission	30–60	40
Relapse	10	—
Chronic myelogenous leukemia		
Chronic phase	50–70	—
Accelerated or blastic phase	10–30	—
Aplastic anemia		
Untranfused	80–90	—
Transfused	50–70	—
Myelodysplastic syndrome	10–25	—
Hodgkin's lymphoma	—	50–80
Non-Hodgkin's lymphoma	40–50	30–60
Multiple myeloma	20–40	<10*

*15%–25% with tandem autologous transplantation.

Data from Tabbara IA, Zimmerman K, Morgan C, et al: Allogeneic hematopoietic stem cell transplantation: Complications and results. Arch Intern Med 162:1558–1566, 2002; Barrett JA, Chao NJA, Bishop MR: Are more patients being cured with allogeneic stem cell transplantation? American Society of Clinical Oncology, 2006 Educational Book. Alexandria, VA: American Society of Clinical Oncology, 2006; Schmit-Pokorny K: Expanding indications for stem cell transplantation. Semin Oncol Nurs 25(2):105–114, 2009; and the National Marrow Donor Program. Available at: http://www.marrow.org.

▲ Patient Evaluation and Contraindications to Transplantation

Selecting the ideal candidate for transplantation is an intricate process. To evaluate a patient's suitability for transplantation, a comprehensive multisystem analysis is performed. This includes both physiological and psychosocial factors that affect the patient's chance for a successful transplantation. During this evaluation phase, treatment of newly diagnosed conditions occurs, and clinicians make plans to ensure adequate nutrition, mobility, and muscle strength. The goal is to have the patient in the best possible physical condition for transplantation. When transplantations are performed earlier, rather than later in the disease process, there are fewer disabilities and a greater chance for survival.

Financial guidance is provided so that patients and families know what their insurance will cover and the nature and amount of their expected out-of-pocket expenses. Costs for transplantations range from $200,000 to $800,000 for the first year, including organ procurement, transplantation, and hospitalization. Medications after transplantation can cost $27,000 to $32,000 a year.[10–12] Transplantation centers may require proof of the patient's ability to cover medication expenses before accepting a patient for transplantation.

The following general criteria guide the selection of candidates for transplantation:

- Biological rather than chronological age is evaluated individually. Individuals eligible for transplantation may range from newborns to 70-year-olds. People older than age 55 may be at increased risk for complications.
- Acute or chronic infection is absent or has been treated. Localized liver infection may be an exception. Inflammatory diseases, such as systemic lupus erythematosus, do not rule out transplantation but should be quiescent at the time of the procedure.
- For the patient undergoing HSCT for a malignancy, care is taken to distinguish patients who can be saved by transplantation from those who may relapse or succumb to the rigors and toxicities of treatment.

Table 47-4 lists organ-specific criteria for transplantation. In general, evaluations common to all transplantation procedures include the following:

- ABO typing
- Tissue typing, HLA matching, mixed lymphocyte culture (MLC) matching
- Transfusion history
- Infectious disease screening (tuberculin skin test, human immunodeficiency virus [HIV], hepatitis B surface antigen, hepatitis C virus, Epstein-Barr virus, cytomegalovirus [CMV], toxoplasmosis titers, herpes simplex, varicella virus, venereal disease)
- Liver function studies

Table 47-3 Indications for Transplantation

Organ	Indications for Transplantation	Common Causes
Kidney	End-stage renal disease	Hypertension, diabetes mellitus, glomerular nephritis, urological disorders, cancer, nephrotoxins, trauma, hemolytic disorders, congenital anomalies
Liver	Adults: irreversible liver disease, malignancy, and hepatic failure resulting in synthetic liver dysfunction Children: biliary atresia, alpha-1-antitrypsin deficiency	Acute or chronic hepatitis, primary sclerosing cholangitis, primary biliary cirrhosis, hepatocellular carcinoma, Budd-Chiari syndrome, alcoholic cirrhosis
Heart	End-stage heart failure	Ischemic cardiomyopathy, idiopathic cardiomyopathy, valvular heart disease, congenital anomalies
Pancreas	Type 1 diabetes mellitus with end-stage renal disease either alone or in combination with a kidney transplant	Diabetes mellitus
Lung	Chronic obstructive pulmonary disease	Emphysema and bronchiectasis, idiopathic pulmonary fibrosis, emphysema due to alpha-1-antitrypsin deficiency, primary pulmonary hypertension
Heart–lung	Eisenmenger's syndrome	Pulmonary hypertension with irreversible right-sided heart failure not amenable to heart transplantation alone
Hematopoietic stem cell	Malignant disorders	Leukemias, myelodysplastic syndrome, Hodgkin's lymphoma, non-Hodgkin's lymphoma, multiple myeloma, and selected solid tumors (eg, renal cell tumors, germ cell tumors, neuroblastoma, pinealoblastoma)[1]
	Nonmalignant disorders	Aplastic, sickle cell, and Fanconi's anemias; selected metabolic disorders; thalassemia; and immunodeficiency syndromes[1]

- Renal function studies
- Complete blood count (CBC)
- Coagulation studies
- Gastrointestinal evaluation (depending on age and history)
- Gynecological examination
- Electrocardiogram (ECG)
- Chest radiograph
- Dental examination to rule out infection
- Social history, review of patient motivation, ability to follow postoperative regimen, and psychiatric evaluation.

Contraindications are based on conditions and behaviors that decrease the chance of survival. For solid organ transplantation, these include serious active infection or sepsis, recent cancer (unless that is the reason for transplantation), current substance abuse, HIV infection, severe cachexia, active peptic ulcer disease, psychiatric disorders that impair the ability to give informed consent or adhere to the treatment regimen, and repeated noncompliance. Table 47-4 lists these contraindications.

▲ Donor Selection

After a person is determined to be a candidate for transplantation, a donor source must be selected.

Determining Compatibility

Determination of compatibility in transplantation involves the evaluation of two major antigen systems. The primary determinant for solid organ transplantation is ABO grouping. A mismatch in compatibility may cause an immediate reaction leading to organ loss.

Organ Transplantation

The rules of compatibility that apply to the administration of blood products also apply to solid organ transplantation: type A blood has the A antigen, type B blood has the B antigen, type AB blood has both A and B antigens, and type O blood has neither antigen.

Histocompatibility testing (tissue typing) is the identification of donor and recipient antigens and the evaluation of donor antigens against recipient antibodies. This evaluation determines the compatibility between donor and recipient, which predicts the chances of graft acceptance. In the HLA antigen system, genes of the major histocompatibility complex code for the antigens that compose a person's tissue type. These genes contain information for antigens present on the surface of the nucleated cells and serve to signal the immune system in differentiating self from nonself. The major histocompatibility complex involved in the immune response includes class I antigens (A, B) and class II (DR) antigens. Class I antigens (HLAs) are present on the surface of all nucleated cells and platelets, whereas class II antigens are found on the surface of lymphocytes. Each person has six A-, B-, and DR-locus antigens that are inherited as a haplotype (ie, a single unit), receiving one HLA haplotype from each parent. Individuals have two HLA haplotypes, one for each chromosome. Offspring then share one haplotype with each parent, and on average, there is a one in four chance that they will share both haplotypes with at least one

Table 47-4 Criteria, Contraindications, and Evaluations in Transplantation

Organ	Specific Criteria	Contraindications	Specific Evaluation
Kidney	• End-stage or near end-stage renal failure (defined as a glomerular filtration rate of <10 mL/min) • Pre–end stage preferable for some patients (ie, children, patients with diabetes mellitus, and those for whom there is a living donor)	• Severe or uncorrectable coronary artery disease, peripheral vascular disease, or pulmonary disease • Severe cardiomyopathy	• Voiding cystourethrogram to evaluate for obstruction or reflux (medical history dependent) • Cardiac evaluation (age and medical history dependent)
Liver	• Malnutrition • Severe blood clotting abnormalities • Variceal bleeding • Hepatic encephalopathy • Severe, intractable ascites • Severe, intractable pruritus	• Multiple uncorrected congenital anomalies • Advanced cardiopulmonary disease • Severe pulmonary hypertension	• Abdominal computed tomography (CT) scan (to detect hepatoma) • Doppler ultrasound (to identify patency of portal vein) • Liver disease studies and autoimmune markers, such as ceruloplasmin, carcinoembryonic antigen, alpha-fetoprotein, antimitochondrial and antinuclear antibody • Endoscopic retrograde cholangiopancreatography/cholangiogram (if indicated, usually for patients with cholestasis) • Liver biopsy (if indicated) • Upper and lower endoscopy (if indicated)
Pancreas	• End-stage renal failure (combine kidney and pancreas transplant) • Absence of (or corrected) coronary artery disease	• Severe or uncorrectable coronary artery disease, peripheral vascular disease, or pulmonary disease • Previous major amputation • Blindness (not absolute contraindication) • Severe cardiomyopathy	• Thallium stress test or coronary angiogram • Cardiology consult • Gastric emptying study • Ophthalmology evaluation • Endocrine studies: glycosylated hemoglobin, serum amylase and lipase, islet cell antibody, urine, and serum peptide measurements
Heart	• Cardiac disease, New York Heart Association Class IV (or advanced III) • Condition not amenable to other forms of medical or surgical therapy • End-stage cardiac disease with less than a 25% likelihood of survival at 1 y without a transplant • Patients with potentially fatal dysrhythmia not amenable to other therapies	• Fixed pulmonary hypertension with pulmonary vascular resistance: more than 6–8 Wood units (more than 480–640 dynes/s/cm^{-5} or pulmonary arteriolar gradient >15 mm) • Recent unresolved pulmonary infarct (increased posttransplant risk for pulmonary infection) • Advanced or poorly controlled diabetes mellitus	• Right heart catheterization; full cardiac catheterization if indicated • Cardiopulmonary exercise testing (MVO_2) • Pulmonary function tests, including diffusion capacity (DLCO) • Cardiac rehabilitation consultation • Multigated acquisition (MUGA) analysis or echocardiogram
Lung	• Untreatable end-stage pulmonary disease (parenchymal or vascular) • Medical therapy ineffective • Estimated survival (without lung transplant) less than probability of survival with lung transplant	• Significant coronary artery disease • Poor nutritional status (ie, <10%–15% of ideal body weight) • Previous cardiothoracic surgery • Corticosteroid use >15 mg/d • Ventilation dependency	• Quantitative ventilation/perfusion scan • Cardiac evaluation • Full pulmonary function testing, including DLCO, arterial blood gases (ABGs), lung volume • 6-min walk test (rehabilitation assessment) • Nutritional assessment

(continued on page 1055)

Table 47-4 Criteria, Contraindications, and Evaluations in Transplantation (continued)

Organ	Specific Criteria	Contraindications	Specific Evaluation
Hematopoietic stem cells	• *Malignant disorders*: replacement of hematopoietic and immune system destroyed by high-dose chemotherapy or radiation with new immune system that can recognize malignant cells as foreign and mount immunological response against tumor • *Nonmalignant disorders:* replacement of an immune or hematopoietic system that is either defective or has failed	• Poor or no response to conventional-dose chemotherapy for malignant disorders (exception is acute leukemia that fails to respond to induction therapy [primary induction failure]; high-dose chemotherapy and allogeneic transplant is an accepted indication) • Poor performance status (using Karnofsky Performance Status scale to assess physical functioning) • Advanced cardiopulmonary or renal disease (left ventricular ejection fraction <50%; DLCO <70; creatinine clearance, 60 mL/min [exception may be multiple myeloma patients]) • Brain metastasis • Age >70 y	• Disease restaging, including CT scans, nuclear medicine scans, bone marrow aspirate and biopsy, lumbar puncture, immunoglobulin levels, cytogenetics, molecular diagnostics, and measures of minimal residual disease • DNA procurement for future engraftment studies • ABO and Rh typing • Human leukocyte antigen (HLA) typing and HLA-matched platelet transfusion support (allogeneic transplant patients only) • Chest x-ray, ECG and MUGA scan, pulmonary function tests, including DLCO, 24-h urine for creatinine clearance • Baseline CT scans of chest and sinuses, particularly if there are symptoms or a history of repeated infections • Dental evaluation, including full mouth x-rays and cleaning • Sperm/fertilized embryo banking • Autologous stem cell backup if patient is undergoing unrelated or mismatched transplantation • Consultations with radiation therapy and infectious disease

of their siblings. There is also a one in four chance that they will share neither haplotype with their siblings. Many possible alleles occur at each locus, resulting in a large number of HLA combinations. Therefore, it is rare that unrelated people have identical antigens.

The higher the number of antigens that match, the higher the likelihood of compatibility, and the lower the risk for rejection. A six-antigen match is associated with the greatest potential for successful transplantation. HLA matching is performed for both solid organ and hematopoietic stem cell transplants. It is most important in kidney and stem cell transplantation. Transplantation requires the suppression of the normal immune response with the postoperative administration of antirejection medications to prevent graft rejection. The greater the similarity of the donor and recipient tissue type, the less likely the occurrence of rejection.

In the case of living related donors, a direct white blood cell (WBC) cross-match may be performed. The sera of the donor and the recipient are tested and evaluated for cell death. For those patients not receiving living related donation, screening is routinely performed against a pool of lymphocyte samples from multiple random donors against the sera of the recipient. The percentage of samples to which the recipient reacts is referred to as the panel reactive antibody (PRA) percentage. A high PRA is predictive of a high risk for rejection, and a prospective cross-match, such as is used in living donors, would be advised. PRA should be repeated monthly because the titer may change from time to time.

Blood transfusions are avoided if possible in patients awaiting organ transplantation because of the risk for antibody production and a resultant high PRA or positive cross-match between donor and recipient. If blood transfusions are necessary, leukocyte-filtered blood should be administered.

Hematopoietic Stem Cell Transplantation

Selection of a donor for HSCT is based on the type and stage of the underlying disease, age, comorbidities, and availability of an appropriate HLA- and MLC-matched donor. MLC matching is performed to observe for interaction between the potential donor's cells and recipient cells. Low reactivity indicates greater compatibility.

There are many sources of hematopoietic stem cells. The types of HSCT may be differentiated in terms of the hematopoietic stem cell donor, the method used to collect the cells, and the intensity of the conditioning regimen (Box 47-1).

For patients who receive hematopoietic stem cells from another person (ie, allogeneic transplant), donor selection is based on the availability of HLA- and MLC-matched donors, who may or may not be related. A related donor is usually a sibling (siblings have the greatest chance of matching on both HLA and on other minor and as yet unrecognized antigens). If more than one donor is HLA-identical to the patient, donor selection is based on sex compatibility with the patient, ABO

BOX 47-1 Types of Hematopoietic Stem Cell Transplantation

Differentiated based on *donor* source for stem cells
Autologous—self
Syngeneic—identical twin
Allogeneic—nonself
- Related
- Unrelated (National Marrow Donor Program)

Cord blood
- Related
- Unrelated (cord blood bank)

Differentiated based on *method* of collecting stem cell
- Bone marrow harvest
- Peripheral blood stem cell collection by apheresis

Differentiated based on *intensity* of conditioning regimen
- Myeloablative: high doses of chemotherapy and sometimes radiation therapy given to destroy hematopoietic and immune systems of recipient. Transplantation of new stem cells allows hematopoietic and immune reconstitution. High morbidity and mortality rates restrict this treatment to younger patients and those in good medical condition.
- Reduced intensity: lower chemotherapy doses (those that do not fully destroy patient's own hematopoietic and immune systems) are given along with immunosuppression to facilitate engraftment of donor hematopoietic cells. Significant long-term risks for infection and chronic graft-versus-host disease (GVHD) persist.

compatibility with the patient, negative viral titers, overall health, younger donor age, minimal donor exposure to blood products, and donor nulliparity because all these factors are associated with an improved outcome of HSCT.

If the patient does not have a suitable family donor, a search for an unrelated donor may be undertaken. The National Marrow Donor Program (NMDP) is a federally funded registry that coordinates the donor search and matching process. The NMDP maintains the world's largest and most diverse registry of more than 6 million volunteer blood stem cell donors and more than 60,000 cord blood units donated by parents after their infant's birth. Umbilical cord blood is another potential stem cell source, particularly in pediatric allogeneic transplantation. The NMDP also works with the American Red Cross and with international registries to access 4 million additional donors and cord blood units around the world.

A difference in ABO blood groups between patient and donor does not interfere with donor selection; however, it does present unique clinical problems. The hematopoietic stem cell product may have to be depleted of red blood cells (RBCs) to prevent, during infusion, a hemolytic reaction caused by ABO antibodies still circulating in the patient's bloodstream. After engraftment and approximately 100 days after transplantation, the patient will seroconvert to the ABO type of the donor.

Living Donors

Living donors are increasingly being used in kidney, liver, pancreas, and lung transplantation. Living donors are used exclusively in HSCT. There is an increase in the use of living organ donors, but there is a dire shortage of organs for transplantation. The number of transplantation candidates is growing, and the number of cadaveric organ donors has remained relatively constant.

Once identified, a potential donor has a thorough medical evaluation to determine that the organ functions normally, there is no underlying disease, and donation would not jeopardize the donor's well-being in any obvious way. After successful completion of this evaluation, a living donor transplantation may occur.

People continue to raise ethical questions about the use of living donors. Long-term studies of living donors have shown that the risks and adverse effects of donation are rare, and, in fact, some donors report beneficial psychological effects from donating. However, some question the risk of coercion in living donors, especially when the donor is the parent of a child who will die without a transplantation. To ensure freely given and informed consent, there may be an assessment by a psychiatrist and involvement of a non–transplant-related physician, together with education and counseling of the donor.

Kidney Donor

Historically, living donors have been blood relatives because tissue matching was considered more likely. However, more recently, living kidney donors have been spouses and friends, and the results have been comparable with those obtained with living blood-related donors. Although either kidney may be used for transplantation, the left is preferred because the left renal vein is longer than the right.[13]

Liver Donor

Living donor liver transplantation involves the removal of a portion of the liver from a living adult for transplantation into a recipient. The 1-year graft survival rate in the United States is 85%.[10]

Pancreas Donor

Transplanting part of the pancreas from a living person is rarely performed. The donor must not be at risk for diabetes mellitus.

Lung Donor

Recently, the use of living related donors in lung transplantation has been successful. Either the lobe of one lung from one parent is transplanted into the child, or one lobe from each parent is used for a bilateral lobar transplantation. The major advantage of lobar transplantation is that it enables children to have the transplantation either at a time when they are in the best condition or when they become critically ill and a cadaveric donor is unavailable.

Cadaveric Donors

If a cadaveric donor is needed, the recipient is placed on the national waiting list. The National Organ Transport Act was designed to improve the organ matching and placement process. The act outlawed the sale of human organs and authorized grants to establish and operate organ procurement

organizations (OPOs). The United States is divided into approximately 60 areas; an OPO is responsible for recovering and transporting organs to transplantation hospitals in each area. The United Network for Organ Sharing (UNOS) is the private, nonprofit organization that administers organ waiting lists and allocates organs throughout the United States on behalf of the recipients.[11]

Patients are placed on the UNOS national list based on blood type and listing date. The size of hearts, lungs, and livers is important for donation. The waiting time varies by organ. Patients awaiting heart transplantation are risk stratified based on condition and classified according to status. Those awaiting heart transplantation who require inotropic medications or ventricular assist devices may have a higher priority (status 1) than those waiting at home on stable oral heart failure medications (status 2). Patients awaiting lung transplantation are ranked using a new UNOS-developed lung allocation system that uses medical information specific to each patient to estimate the severity of illness and chance of success following transplantation. The UNOS score is used to prioritize recipients who are awaiting a lung transplant. A candidate with a higher lung allocation score receives higher priority for a lung offer when a compatible lung becomes available.[11] Patients awaiting liver transplantation are also risk stratified based on the model for end-stage liver disease (MELD) score. Status rankings 1, 2A, 2B, and 3 are used based on the severity of the disease process. A patient who is projected to live less than 7 days without transplantation is status 1, whereas a patient who is living at home with chronic liver disease is status 3.[11]

Despite the continued need for transplantations, the supply of donor organs remains inadequate. Currently, according to UNOS, about 107,000 patients are awaiting organ transplantation in the United States.[11] The Scientific Registry of Transplant Recipients reported that in 2007, there were 14,339 organ donors; this number includes living and deceased donors.[10] In 2008, the reported conversion rate, which is the ratio of actual donors to identified eligible donors multiplied by 100, was 66%.[14] Lack of consent to a request for donation is the primary cause of the gap between the number of potential donors and the number of actual donors.

A major factor in the discrepancy between potential and actual donors is lack of education for potential donor families.[15] When a patient is determined to be brain dead, or support is withdrawn from a patient who is not brain dead, many hospitals alert the local OPO as a matter of policy. In some larger institutions, a nurse with advanced training in the area of organ donation and family support is trained as a donor advocate. The role of this nurse is to support the family in the grieving process while initiating the discussion of organ donation. It is important that all potential organ donors be identified. Often, potential donors are victims of trauma, cerebral aneurysm, or a variety of other circumstances. Potential donors may be precluded from donating solid organs for reasons, such as advanced age, infection, or coma, though such donors may be considered for donation of the cornea, skin, or cardiovascular tissue (such as heart valves or portions of the aorta).

Determination of Death

With current technology, death can be determined in two ways. The absence of cardiopulmonary function is the better-known method; however, the absence of brain function (brain death) also is a common method of determining death (see Chapter 36 for neurological criteria for determining death). Most patients who are organ donors are pronounced dead based on the absence of brain function. The critical care nurse should be familiar with the laws in his or her state related to "brain death" and with the institutional policies for the determination of death.

Role of the Nurse

Critical care nurses are an integral part of the organ donation team. Almost all organ donors die in critical care units; therefore, the critical care nurse is a key person in identifying potential donors. Moreover, the nurse plays an important role as advocate by making certain that all efforts are made to determine and act on the patient's wishes regarding donation. Nurses also play a vital role in supporting the family psychologically, particularly when they are trying to accept the donor's death. The role of the donor coordinator has developed to allow a nurse with specialized skills to interact with the potential donor's family during the process and to provide support for the nursing staff caring for the donor. When a decision is made for organ donation, the nurse also plays an important role in supporting the donor physiologically.

The care of the potential donor, once identified, becomes the responsibility of the OPO. A nurse who is specially trained to maintain hemodynamic stability of the donor works with the bedside nurse to care for the patient. It is essential to maintain hemodynamic stability so that the vital organs are perfused adequately. Given the massive hemodynamic shifts seen following brain death, hemodynamic management occurs in two phases. In the first phase, hemodynamic surges consequent to endogenous catecholamine mobilization produce hypotension. Short-acting agents, such as nitroprusside or esmolol, which are easily titrated and have a short duration of action, are used to control blood pressure and heart rate. Phase 2 begins after depletion of catecholamine stores, at which time the donor experiences a dramatic fall in blood pressure. Initial intervention in phase 2 involves rapid replacement of circulating blood volume with crystalloid or colloid intravenous (IV) fluids. Aggressive fluid resuscitation in this phase must account for the relative fluid volume deficit produced by a vasodilatory state from endogenous catecholamine depletion as well as the systemic inflammatory response.[16] The goal of blood pressure management in phase 2 should be to sustain a mean arterial pressure of 70 mm Hg. Recommended pharmacological therapy for vasopressor support includes dopamine or dobutamine, with target doses of less than 10 mcg/kg/min if the focus is cardiac donation. In other instances, norepinephrine may be used, with a target dose of 0.5 to 5.0 mcg/min. The administration of vasopressors and inotropes, in addition to providing cardiovascular support for the organ donor, promotes a lower incidence of kidney rejection and better long-term graft survival.[17] As the need for vasopressors and inotropes increases, the possibility for multiorgan recovery decreases.

Optimal pulmonary management, including suctioning for airway clearance and aspiration precautions, is necessary. Ventilator management should include close titration of fluids to minimize the risk for pulmonary edema. Positive end-expiratory pressure should be less than 5 cm H_2O, and

peak airway pressures should be maintained at less than 30 mm Hg if possible. Ventilation with tidal volumes of 10.0 to 12.0 mL/kg may maintain minute ventilation and attenuate risk for barotrauma.[18]

It is also essential to assess urine output hourly to detect diabetes insipidus. This is common in organ donors and is caused by failure of the posterior pituitary to produce or release antidiuretic hormones. This can lead to electrolyte imbalances. It may be necessary to order aqueous vasopressin or desmopressin acetate to reduce urine output and help maintain fluid balance.

Laboratory results, such as electrolytes, CBC, liver and renal function tests, and arterial blood gases (ABGs) values, are necessary to assess organ function and determine appropriate intervention. An ECG and echocardiogram are required for heart donation. Serial chest radiographs, bronchoscopy, sputum for Gram stain and visual inspection at the time of organ procurement are required for lung donation.

Role of the Donor Coordinator

The role of the donor coordinator developed as a result of the disparity between potential donors and the numbers of patients awaiting organ donation. The donor coordinator introduces the concept of organ donation by a nurse not associated with transplantation. This approach results in the highest rates of consent.[16] Kidneys are the most commonly transplanted organ, and heart, lung, liver, pancreas, intestine, cornea, skin, bone, and other organs or tissue may also be donated. The donor coordinator is involved in coordinating and procuring all transplantable organs. In addition, the donor coordinator usually ensures that the family has the information necessary to give informed consent and provides them with access to bereavement support. The donor coordinator also serves as a resource for the health care team and as a liaison between the transplantation program and the critical care area. Cooperation between the critical care staff and the transplantation program helps ensure that the option of donation is offered to families of all potential donors.

Preservation Time

There is a broad range of acceptable preservation times for organs. However, the goal is to transplant organs as soon as possible. Kidneys can be stored for up to 48 hours using pulsatile perfusion preservation and for 24 to 36 hours using cold storage. Livers can be stored for up to 20 hours, pancreas up to 12 hours, and hearts and lungs for 4 to 6 hours. To decrease cellular injury, organs are stored in a solution and kept in ice. The preservative solutions used are different for each organ and are based on the metabolic needs of the organ, with center-specific variability. The focus of preservation is to protect the organ from ischemic injury.[19]

▲ Assessment and Management in Organ Transplantation

The role of the transplant coordinator extends across the continuum of care from preevaluation of potential recipients through transplantation and follow-up. This person is responsible for coordinating the evaluation and teaching the recipient and family about the evaluation testing process, the listing process, and organ allocation. A major contribution is reviewing preoperative through postoperative procedures, the immunosuppressive regimen, and follow-up care. In many institutions, a nurse practitioner, who also provides medical care and follow-up for the patients, fulfills this role.

Preoperative Phase

The immediate preoperative phase, which is usually only a matter of hours, includes comprehensive laboratory studies, chest radiograph, ECG, and, for kidney transplant recipients, dialysis within 24 hours of transplantation. Laboratory studies usually include CBC, prothrombin time (PT), partial thromboplastin time (PTT), electrolytes, blood glucose, blood urea nitrogen (BUN), creatinine, liver function tests, type and cross-match, and urinalysis.

Surgical Procedure

Kidney

Typically, the kidney is placed retroperitoneally in the iliac fossa. The hypogastric or internal artery and the external iliac vein are usually used for vascular anastomosis. When it is mechanically difficult to access these vessels, as with children, it may be necessary to anastomose the renal vessels to the inferior vena cava and aorta.

Two common types of ureteral anastomoses can be performed. In the first procedure, the donor ureter is implanted into the recipient's bladder by a vertical cystotomy and a submucosal antireflux tunnel because the ureter lacks innervation and normal peristalsis. In the second type, used less frequently, the donor kidney is anastomosed at the ureteropelvic junction to the recipient ureter. An indwelling catheter is used for both types of anastomoses, and occasionally, a ureteral stent may be used. In either case, hematuria is present for several days. In the first, more common procedure, clots may be seen in the urine because of the vascular nature of the bladder. In the second, less common, procedure, the urine changes to pink within the first postoperative day because there are no sutures in the bladder.

Liver

The liver is transplanted orthotopically, that is, in its normal position after the native liver is removed. Four vascular anastomoses must be performed: the suprahepatic vena cava, the infrahepatic vena cava, the portal vein, and the hepatic artery (Fig. 47-1). Then the liver is reperfused, and the bile duct is anastomosed, usually to the recipient bile duct.[20] A T-tube is usually inserted. During liver transplantation, a rapid infusion system is used for administering blood and blood products, a cell saver is often used to limit the amount of banked blood required, and a pump for venovenous bypass is often used in adults to return blood to the heart. This is performed by inserting a catheter into the saphenous or femoral vein and another into the axillary vein (usually on the left side), which allows blood to circulate from the lower extremities back to the heart. Surgery usually takes between 8 and 16 hours.

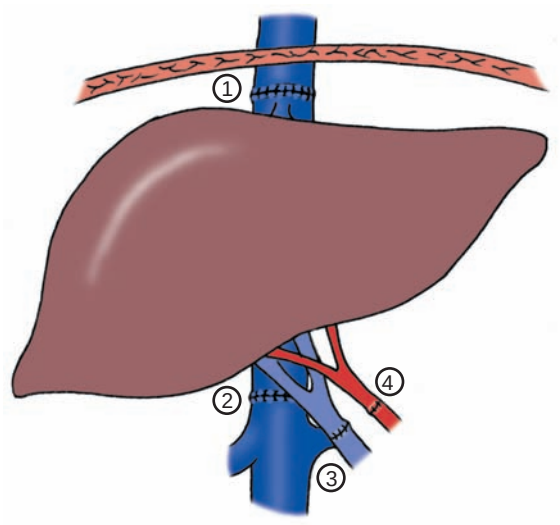


FIGURE 47-1 ▲ Diagram of vascular anastomoses in liver transplantation: (*1*) suprahepatic vena cava, (*2*) infrahepatic vena cava, (*3*) portal vein, and (*4*) hepatic artery.

Heart

Procedures for heart transplantation include orthotopic and heterotopic approaches. Orthotopic transplantation involves replacing the damaged heart with the donor heart. With heterotopic transplantation, the patient's own heart is not removed. The donor heart is positioned so that the chambers and blood vessels of both hearts can be connected, thus allowing the donor heart to assist the damaged heart.

ORTHOTOPIC TRANSPLANTATION. Orthotopic heart transplantation (OHT) is the most common heart transplantation performed. The surgeon excises the recipient's heart and implants the donor heart in its place. A median sternotomy incision is made, cardiopulmonary bypass is initiated, and the recipient's heart is removed by incising the left and right atria, pulmonary artery, and aorta. The Lower and Shumway technique has been the gold standard for OHT. The atrial septum and posterior and lateral walls of the recipient's atria are left intact, including the areas of the sinoatrial node, inferior and superior venae cavae to the right atrium, and pulmonary veins to the left atrium. The remnant atria serve as anchors for the donor heart.

The donor atria are trimmed to preserve the anterior arterial walls, sinoatrial node, and internodal conduction pathways. Then anastomoses are made between the recipient and donor left and right atria, the pulmonary arteries, and the aortas. Atrial and ventricular pacing wires are placed at the time of surgery so that temporary pacing can easily be initiated. Cardiopulmonary bypass is weaned off, and the donor heart assumes the role of providing the cardiac output (Fig. 47-2A).

An alternative to the atrial-to-atrial cuff technique is the bicaval technique (see Fig. 47-2B), which preserves atrial anatomy disrupted by the Lower and Shumway technique. The donor atria are intact, and the anastomoses are between the donor and recipient inferior and superior venae cavae rather than the atria. This avoids the loss of atrial anatomy, which has been responsible for the development of posttransplantation complications, such as mitral and tricuspid regurgitation, atrial thrombus formation, and tachydysrhythmia.[21]

HETEROTOPIC TRANSPLANTATION. Heterotopic transplantation, or piggyback procedure, is an infrequently used technique. The recipient's heart is left in place, and the donor heart is placed next to it in the right chest. The two hearts are connected in parallel by anastomoses made between the donor and recipient left and right atria, aortas, and pulmonary arteries using a synthetic tube graft. By allowing blood to flow through either or both hearts, two functional hearts work together to provide the cardiac output (Fig. 47-3).

Heterotopic transplantation may be used in patients with pulmonary hypertension in whom the donor heart alone would not have a strong enough right ventricle to pump against the increased pulmonary vascular resistance. It can also be used as a lifesaving procedure in urgent cases if the only available donor heart is too small for the size of the recipient. Limitations of heterotopic transplantation include thromboembolism from the native heart with need for anticoagulation, limited space in the chest cavity, and, in ischemic heart disease, ongoing angina and the possibility of ischemia-induced dysrhythmias in the native heart. The survival rates are less favorable for heterotopic transplantation than for orthotopic transplantation.

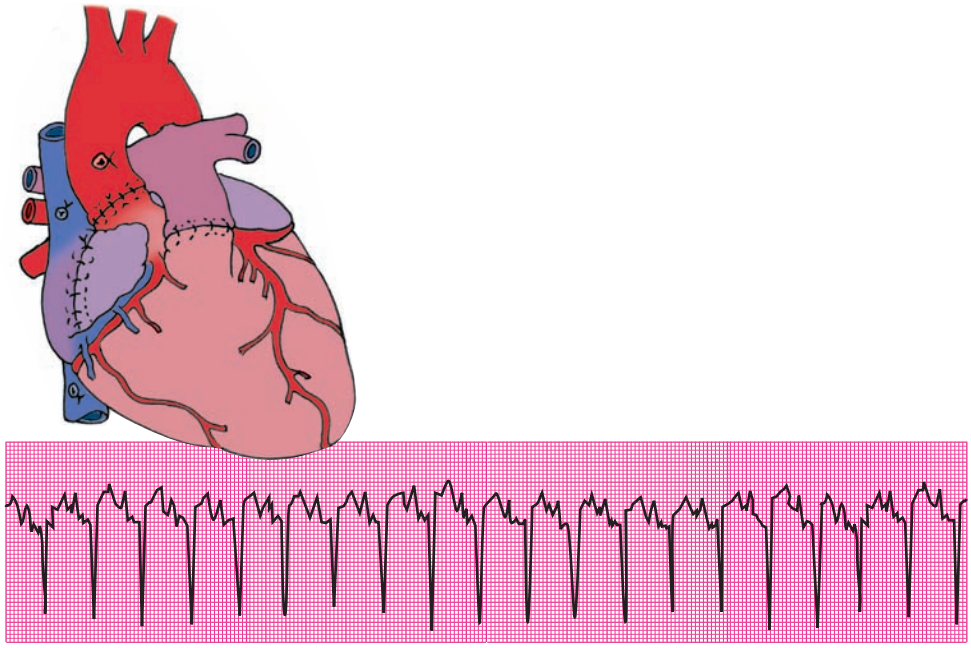
Pancreas

Transplantation of the entire pancreas is performed most commonly. Pancreas transplantation may be performed in combination with kidney transplantation for recipients with end-stage renal disease secondary to diabetes mellitus. The pancreas and kidney may be transplanted at the same time or months apart. Clinical trials are ongoing to determine the feasibility and outcomes of islet cell transplantation for patients with type 1 diabetes not controlled with insulin. With this experimental procedure, only the insulin-producing islet cells are transplanted. The recipient still requires immunosuppressive medications to prevent rejection.

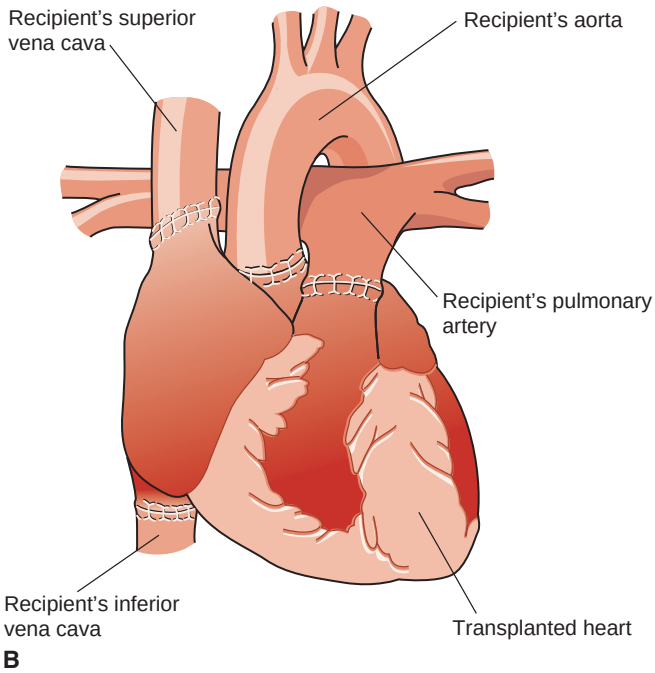
The pancreas is placed into a heterotopic position, usually the right iliac area. Techniques vary for vascular and exocrine anastomoses. The most controversial aspect of the surgical technique is the approach for draining exocrine secretions. The exocrine duct may be occluded, or exocrine secretions may drain either into the small bowel or bladder. There is no consensus about the best approach, and all have advantages and disadvantages.

Lung

Both single- and double-lung transplantations are performed. In single-lung transplantations, the left lung mainstem bronchus is longer, which makes the procedure easier technically. The preferred lung is based on perfusion abnormalities (as determined by ventilation–perfusion scan) and functional abnormalities. Anastomoses are made at the mainstem bronchus, pulmonary artery, and cuff of atrium containing pulmonary veins. Cardiopulmonary bypass is not always necessary and depends on the patient's pulmonary artery pressure, blood pressure, and gas exchange. Incision is made at the fifth intracostal space using a posterior lateral thoracotomy for single-lung transplantations, whereas a median sternotomy incision or a clamshell incision is made for double-lung transplantations. Surgeons telescope the recipient's bronchus into the donor lung or vice versa, or perform an end-to-end



A



B

FIGURE 47-2 ▲ Orthotopic method of transplantation. **A:** Lower and Shumway technique. Both the donor and the recipient sinoatrial nodes are intact (X), resulting in the electrocardiogram (ECG) tracing. Note the double P wave at the independent rates. **B:** Bicaval technique. The anastomoses are made between the inferior and superior venae cavae rather than the atria. (**B** from Smeltzer SC, Bare BG, Hinkle JL, et al: Brunner and Suddarth's Textbook of Medical-Surgical Nursing, 12th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 811.)

anastomosis with omentopexy in which an omental flap is wrapped around the tracheal anastomosis to increase blood supply to the area.

Postoperative Phase

Immediately after surgery, transplant recipients require care in a closely monitored area until their condition stabilizes. Kidney transplant recipients often go to a postanesthesia care unit and then directly to a transplant unit. Other organ recipients go to an intensive care unit (ICU) from the operating room. When a patient arrives in the postanesthesia or intensive care area, the nurse makes the following assessments:

- Blood pressure, heart rate, respirations, oxygenation and ventilator settings, temperature, central venous pressure, and cardiopulmonary hemodynamics. In renal transplant recipients, it is necessary to take blood pressure on an extremity that does not have a functioning vascular access site because even momentary interference with arterial blood flow may lead to access malfunction.
- Patient's level of consciousness and degree of pain
- Number of IV and arterial lines, noting the site, type of solution, and flow rate

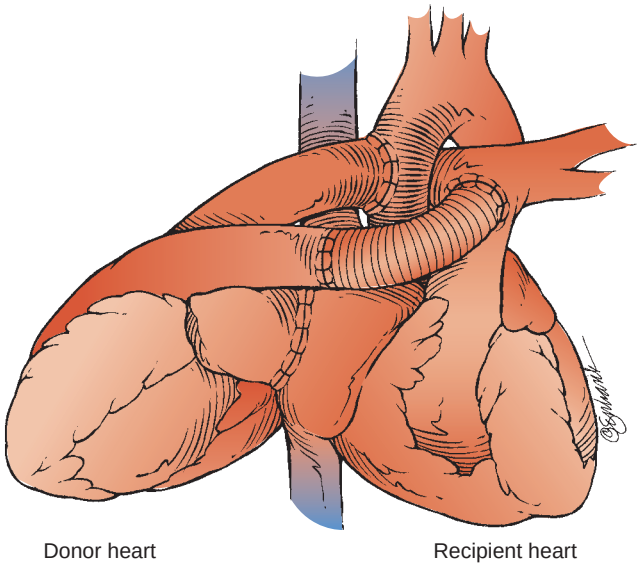


FIGURE 47-3 ▲ Heterotopic method of heart transplantation. The donor heart is anastomosed with a Dacron graft to the recipient's heart, resulting in this ECG tracing. Note the "extra" QRS at an independent rate. (From Smeltzer SC, Bare BG: Brunner and Suddarth's Textbook of Medical-Surgical Nursing, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2004, p 775.)

- Abdominal or chest dressing for drainage, noting the presence of drains and amount and type of drainage
- Presence of bladder and possible ureteral catheters and patency and urinary drainage
- Attachment of nasogastric tube to appropriate drainage system and amount and character of drainage
- Most recent hemodynamic and intraoperative laboratory results

Kidney

Care of the kidney transplant recipient emphasizes assessing renal function and administering immunosuppressive therapy. Therefore, answers to the following questions help guide care:

- Are the patient's own kidneys present in addition to the graft, and if so, how much urine do they produce daily? This information helps determine how much urine is from the transplanted kidney.
- What are the preoperative results of laboratory tests (BUN, creatinine, hematocrit)?
- How much and what kind of IV fluid has the patient received?
- What immunosuppressive drugs were given before or during surgery? What immunosuppressive therapy should be given after surgery?

Nursing responsibilities also involve the following:

- Observing the function of the transplanted kidney
- Monitoring fluid and electrolyte balance
- Helping avoid sources of infection
- Detecting early signs of complications
- Supporting the patient and family through the recovery phase

In addition, at regular intervals, the nurse evaluates the patency and the vascular access used for dialysis. This is done by placing either fingers or a stethoscope directly over the access site and feeling or listening for a characteristically loud, pulsating noise called a *bruit*. If the patient has been maintained on peritoneal dialysis and the catheter is in place, it is essential that the catheter system is sterile and capped.

RENAL GRAFT FUNCTION. The amount of urine produced by the transplanted kidney varies from a large amount (200 to 1,000 mL/h) to small amounts (<20 mL/h). The degree of renal function is related to ischemic injury in the donor kidney, usually from either hypotensive periods in a cadaveric donor or from the time the kidney is stored outside the body (preservation time). Renal function is better when kidney preservation time is less than 24 hours. Most posttransplantation dysfunction is reversible but may take up to 4 weeks to return to normal.

Assessments of renal function include periodic BUN and creatinine levels and, in some centers, a β_2-microglobulin level. The glomerular basement membrane readily filters this low–molecular-weight globulin, which the proximal renal tubules reabsorb and metabolize almost completely.

A renal scan is a radionuclide test used to determine renal perfusion, filtration, and excretion. It is usually performed in the first 24 hours to obtain baseline data and then periodically thereafter when laboratory values or clinical changes suggest an alteration in renal function.

URINARY DRAINAGE PROBLEMS. When a change in urinary output occurs, such as a large volume in 1 hour and a diminished amount in the next, mechanical factors that interfere with urinary drainage should be suspected. Clotted, kinked, or compressed tubing in the urinary drainage system may be the cause of the decreased output. When the catheter is occluded by a clot, the patient may complain of pain, feel an urgency to void, or have bloody leakage around the catheter. Milking is the preferred way to dislodge clots because irrigation, even under aseptic conditions, increases the risk for infection. However, gentle irrigation with strict aseptic technique may be necessary. Small amounts of irrigant (30 mL or less) are recommended because patients commonly have small bladders. Vigorous irrigation also could cause extravasation at the ureteral anastomosis site.

URINARY LEAKAGE. Urinary leakage on the abdominal dressing and severe abdominal discomfort or distention may indicate retroperitoneal leakage from the ureteral anastomosis site. It is important to report decreased urinary output or severe abdominal pain in the presence of good renal function and adequate pain medication because technical and surgical complications can result in loss of graft function.

HYPERKALEMIA. The most frequent electrolyte disturbance in the acute postoperative phase is hyperkalemia. If the graft functions and excretes a high volume of urine, it is usually also able to excrete the excessive serum potassium created by surgical tissue damage. If the patient is oliguric or anuric after surgery, the serum potassium may increase to

unacceptable levels. Interventions include administration of glucose and insulin to transport potassium into the cell and administration of oral polystyrene sulfonate.

Liver

Immediate postoperative care focuses on hemodynamic stability, adequate oxygenation, fluid and electrolyte balance, adequate hemostasis, and graft function. An arterial line and pulmonary artery catheter are in place. The pulmonary artery catheter readings help monitor cardiac function and fluid status because high cardiac output and low systemic vascular resistance related to the effects of end-stage liver disease continue immediately after surgery.

Vasopressors and additional fluid boluses may be required in the first 24 to 36 hours. Central venous pressure should be maintained at greater than 10 cm H_2O to balance the importance of good cardiac function against the risk for passive congestion of the liver. Hypotension is most often caused by intra-abdominal bleeding. An increase in abdominal girth or excess bloody drainage from Jackson-Pratt drains is indicative of a severe problem.

OXYGENATION. Adequate ventilation is crucial for graft perfusion and helps reduce the risk for pulmonary complications. The critical care team determines ventilator settings, and the nurse monitors the arterial (SaO_2) and mixed venous (SvO_2) oxygen saturations. Pulse oximetry may be used, but severe jaundice may interfere with saturation measurements. Postoperative pleural effusion is common owing to the presence of ascites and risk of injury to the diaphragm during surgery. A chest tube may be required for drainage.

Ventilator support is usually withdrawn when the patient is fully awake. However, if the patient is going to receive a monoclonal antibody, such as muromonab-CD3, the first dose should be given before extubation because there is a risk for pulmonary edema as a reaction to the medication.

COAGULATION. Abnormal clotting factors, bleeding at the site of anastomosis, and impaired graft function may all contribute to problems with hemostasis. Therefore, the nurse monitors PT, PTT, fibrinogen, and factor V level along with the amount, color, and consistency of bleeding from the incision and drainage tubes.

The patient may need infusions of platelets, RBCs, or cryoprecipitate. Blood products should be leukocyte reduced to avoid introduction of CMV, especially if the patient is negative for CMV. It is necessary to take care to avoid overcorrecting coagulation deficiencies, which could lead to vascular thrombosis of the extremities or the graft. As a result of the venovenous bypass system, there is also a risk for phlebitis or thrombosis in the femoral and axillary access site. This may be indicated by ipsilateral swelling in these extremities. Anticoagulation or an inferior vena cava filter may be necessary if deep venous thrombosis develops.

ELECTROLYTE BALANCE. Hyperglycemia; hyperkalemia; metabolic alkalosis; and calcium, phosphorus, and magnesium disorders may occur. Hyperglycemia is an indication that the liver is able to store glycogen and convert it to glucose. Hyperkalemia can indicate nonfunctional hepatocytes and, in turn, a nonfunctional graft. Metabolic alkalosis is related to the citrate in stored blood (metabolized to bicarbonate), hypokalemia, diuretic therapy, and the administration of large volumes of fresh frozen plasma. This usually resolves spontaneously, but hypoventilation in response to the alkalosis may slow weaning from mechanical ventilation. Calcium, phosphorus, and magnesium disturbances primarily result from the administration of fluid and blood products.

Some degree of postoperative renal dysfunction is also common due either to hepatorenal syndrome or hypotension during surgery. In addition, some immunosuppressive medications are nephrotoxic. This can affect fluid and electrolyte balance. On occasion, when dialysis is needed, continuous arteriovenous or venovenous hemofiltration is used because it interferes least with hemodynamic stability.

LIVER FUNCTION. Function of the transplanted liver can range from excellent to primary nonfunction. Although the cause of primary nonfunction is not known, it is believed to be related to preservation injury, and retransplantation is necessary. Liver function is initially assessed by bile production, coagulation factors, and later by liver function tests. Measuring bile production from the biliary drainage tube helps assess the excretory function of the liver and is a good early indicator of graft function. PT and the international normalized ratio (INR) provide a measure of the synthetic function of the liver. Aminotransferases (alanine aminotransferase and aspartate aminotransferase) provide information about the degree of hepatic injury related to preservation. In addition, improvement in the clearance of lactate, encephalopathy, and glucose metabolism are also assessments of liver function. All liver function test results are elevated initially and gradually decrease.

Heart

Postoperative care of the heart transplant recipient is similar to that for any person undergoing cardiac surgery. However, there are several major differences, including changes in cardiac rhythm and function caused by denervation of the donor heart and the potential for right ventricular failure. Only the more common orthotopic transplantation is discussed here.

REMNANT P WAVES. In the standard atrial cuff technique, the recipient sinoatrial node and portions of the recipient atria are left intact at the time of surgery. Therefore, two P waves are usually seen on the ECG. The recipient sinoatrial node initiates an impulse that depolarizes the remnant recipient atria; however, this depolarization wave usually does not cross the atrial suture line. The donor sinoatrial node initiates the impulse that causes depolarization of the entire donor heart and elicits the QRS complex. Because the two sets of atria beat independently of each other, two different P waves may appear on the ECG. Remnant P waves may be identified by their dissociation or lack of relationship to the QRS complexes. They usually occur at a slower rate than the donor P waves, and their rate may speed up or slow down because the remnant P waves are still under autonomic nervous system influence, whereas the donor P waves are denervated. The two sets of atria may also be in different rhythms. For example, the recipient atrial remnants may be in atrial fibrillation while the donor heart is in normal sinus rhythm.

EFFECTS OF DENERVATION. During removal of the donor heart, the nerve supply is severed, resulting in a lack of autonomic nervous system innervation of the transplanted

heart. Because of the loss of vagal influence, the resting sinus rate is higher than normal—usually between 90 and 110 beats/min—and heart rate variations caused by respiration do not occur.

Decreased donor sinoatrial node automaticity can also occur after transplantation as a result of injury to the node during procurement, transport, surgical procedure, or postoperative edema of the atrial suture line. Usually, these problems resolve within 1 to 2 weeks after transplantation, but temporary pacing may be needed to maintain an adequate heart rate. Atropine, which blocks vagal stimulation, is ineffective in treating bradydysrhythmias in the transplanted heart because there is no parasympathetic innervation. If the sinus rate is reduced, junctional rhythms can occur earlier than normal because of the loss of vagal tone.

Normal cardiovascular reflexes are also removed by denervation. In the normal heart, the body's increased metabolic demands cause direct compensatory stimulation of the heart by the sympathetic nervous system, which immediately increases heart rate, contractility, and cardiac output. Because direct sympathetic nervous system stimulation of the transplanted heart is absent, this response is mediated through release of circulating catecholamines from the adrenal medulla. Therefore, increases in heart rate, contractility, and cardiac output occur much more slowly than normal. With exercise, heart rate and cardiac output increase gradually over 3 to 5 minutes and remain elevated longer after exercise. Prolonged warm-up before and cooldown after exercise help compensate for these changes.

Orthostatic hypotension can occur because the normal, immediate reflex tachycardia, which compensates for venous pooling with position change, does not occur. When patients begin ambulating, they should be cautioned to change position gradually to prevent orthostatic hypotension.

Because of denervation, the cardiac effects of medications normally mediated by the autonomic nervous system are abnormal. Atropine, which increases heart rate by blocking parasympathetic influence, is ineffective. Isoproterenol, a positive chronotropic agent, has been widely used because it stimulates myocardial receptors directly for pharmacological management of symptomatic bradydysrhythmias. However, isoproterenol is not widely available. Instead, dobutamine and epinephrine, along with temporary epicardial pacing, are frequently used.

Digitalis preparations are ineffective in decreasing the heart rate or increasing the atrioventricular nodal refractory period because these effects are mediated primarily by the parasympathetic nervous system. Digitalis does increase myocardial contractility by its direct action on myocardial cells. Beta-blocking drugs or calcium channel blockers (eg, verapamil) can be used to control supraventricular tachydysrhythmias in the transplanted heart; carotid sinus pressure, the Valsalva maneuver, and digitalis are ineffective.

Finally, denervation prevents transmission of pain impulses from ischemic myocardium to the brain, so the patient does not experience angina. Severe myocardial ischemia or infarction may go unnoticed. For this reason, ECG stress testing and annual coronary angiography or coronary vascular ultrasonography are usually performed.

POTENTIAL FOR VENTRICULAR FAILURE. Posttransplantation ventricular failure, causing decreased cardiac output, occurs for the same reasons as in other cardiac surgical procedures. In addition, a prolonged ischemia time, inotropic support of the donor, or rejection can cause myocardial depression in a transplant recipient.

Right ventricular failure is the most common cause of primary graft failure after transplantation. Reasons for right-sided heart dysfunction are not entirely clear but are related to acute changes in pulmonary vascular resistance. The newly transplanted heart is required to work against elevated pulmonary pressures secondary to long-standing heart failure in the recipient.[22] Postoperative changes in pH or ABGs can cause pulmonary vascular spasm. Both pulmonary hypertension and spasm increase the pulmonary vascular resistance or resistance to ejection of blood by the right ventricle. The normal right ventricle of the donor heart may be unable to increase its output acutely to overcome a high preexisting pulmonary vascular resistance. Signs of acute right ventricular failure include elevated central venous pressure and jugular venous distention. Left ventricular cardiac output decreases because the right ventricle is unable to pump enough blood through the lungs.

Treatment of posttransplantation right-sided heart failure involves the use of drugs to decrease right heart afterload (dobutamine, milrinone, inhaled nitric oxide). Inhaled nitric oxide, a direct pulmonary vasodilator, may be administered through the ventilator circuit, thereby avoiding the systemic affects of the drugs that are administered IV. Conditions that increase right heart afterload (eg, hypoxia, acidosis, excessive blood transfusions) should be avoided.

Pancreas

The critical care of a pancreas transplant recipient is comparable to the critical care of any patient who has had major abdominal surgery. The differences relate to pancreatic function, the type of surgical procedure, and secondary effects of diabetes mellitus.

Blood glucose response usually returns within the first few postoperative hours; however, an insulin drip may be required until the blood glucose level is normal. Pancreatic function is monitored by glucose levels before and after meals, by the results of glycosylated hemoglobin, and sometimes by glucose tolerance testing. Even minor abnormalities may indicate rejection or vascular thrombosis of the graft.

When a section of duodenum is used for exocrine drainage, the prevention of infection is particularly challenging. Antibiotics are usually administered until the intraoperative culture of the duodenum is reported. When the bladder is used for exocrine drainage, bicarbonate wasting in the urine may occur. The standard method for pancreatic transplantation involves drainage of exocrine secretions into the urinary bladder with venous outflow into the systemic circulation. Despite the high success rate associated with this approach, it often leads to complications, which can include urinary tract infections, reflux pancreatitis, metabolic acidosis, and hyperinsulinemia. Depending on the patient's ability to void, the catheter may be needed for several days or possibly weeks if there is long-standing neurogenic bladder dysfunction secondary to diabetes mellitus. The surgical approach remains center and surgeon specific.

It is necessary to keep a nasogastric tube in place until bowel activity returns. This may take 3 to 5 days if there is diabetic gastroparesis. If the nasogastric tube remains in place, enteral or parenteral feedings may be necessary.

Lung

On arrival to the ICU, the lung transplant recipient is anesthetized, intubated, and mechanically ventilated. Usually, extubation occurs 24 to 36 hours after surgery when the patient's oxygenation is optimized and secretions are minimal. Extubation usually proceeds more quickly in patients with emphysema and a single-lung transplant than in patients with pulmonary hypertension, who may need a longer intubation period.

The lung transplant recipient does not have a cough reflex because of denervation. Therefore, when suctioning, the nurse avoids inserting the catheter where it can cause damage. The surgical team performs frequent bronchoscopy to ensure intact anastomoses and pulmonary toileting. A common problem in the immediate posttransplantation period is pulmonary edema due to a phenomenon known as "reperfusion" injury. As a result, it is best to maintain the patient in a relatively hypovolemic state for the first few days. Diuresis begins when the patient is hemodynamically stable.

The lungs are constantly exposed to the outside world, and therefore prevention of infection is especially important after lung transplantation. Antibiotics are given prophylactically and are determined by donor cultures, preoperative serology results, and sputum samples. Patient care measures to prevent infection include giving oral care, encouraging physical activity early in the postoperative course, and performing daily respiratory and chest physiotherapy. Continuous pulse oximetry is used to monitor oxygen saturation, and daily chest radiographs help monitor progress. Infection control measures by staff include washing hands and using aseptic suctioning technique. Staff and visitors with active infections should avoid caring for or visiting the patients.

▲ Assessment and Management in Hematopoietic Stem Cell Transplantation

HSCT involves replacing diseased, destroyed, or nonfunctioning hematopoietic cells with healthy progenitor cells, also called stem cells. Stem cells are primitive hematopoietic cells capable of self-renewal, and they are pluripotent, meaning that they are capable of maturation into an RBC, WBC, or platelet. These stem cells may be collected directly from the bone marrow spaces by a bone marrow harvest procedure, or from the peripheral blood, by apheresis. HSCT is an important advance in restoring hematopoietic function in patients whose bone marrow has been destroyed by high-dose chemotherapy and radiation therapy (see Table 47-1, p. 1052).

In both autologous and allogeneic HSCT, peripheral blood stem cells have become the preferred source of hematopoietic stem cells for grafting, although for selected allogeneic HSCT recipients, bone marrow grafting may offer specific advantages over peripheral blood stem cells. Collection of cells through apheresis is easier and less costly and may also result in a more rapid recovery of neutrophil and platelet counts. In unrelated allogeneic transplantation, the source of stem cells may be either a bone marrow harvest procedure or a peripheral blood stem cell collection. Table 47-5 presents a detailed comparison of autologous and allogeneic stem cell transplantation. Box 47-2 outlines the benefits and limitations of the various sources of stem cells for transplantation.

Table 47-5 Comparison of Autologous and Allogeneic Stem Cell Transplantation

	Autologous	Allogeneic
Indications	Hematological malignancies and solid tumors Possible role in treatment of autoimmune disorders Future role in combination with gene therapy to treat genetic disorders and HIV infection	Hematological malignancies, aplastic anemia, congenital bone marrow disorders, immune deficiency states, some inborn errors of metabolism
Source of stem cells	Stem cells collected from bone marrow or peripheral blood of the patient and then reinfused after high-dose conditioning regimen Stem cells given to rescue the patient from hematological toxicity of conditioning regimen	Marrow, peripheral blood, cord blood, family donors, unrelated donors, HLA-matched or partially matched
Preparative regimen	High-dose chemotherapy to eradicate malignant disease	High-dose chemotherapy and sometimes total-body radiation as intensive therapy for malignant disease and to provide immunosuppression to allow engraftment (makes "space" for incoming stem cells)
Posttransplantation treatment	Supportive care, transfusions, growth factors, immune manipulation	Supportive care, transfusions, growth factors, immune manipulation, prophylaxis and treatment of graft-versus-host disease (GVHD)
Risk for infectious complications	Lower risk; infections occur mainly in early posttransplantation period	Higher risk; sustained risk for infection for months or years
Major complications	Preparative regimen toxicity, disease recurrence or progression	Preparative regimen toxicity, disease recurrence or progression, GVHD, immunodeficiency
Treatment-related mortality rate	Usually <5%	5%–30% depending on many patient-, donor-, and disease-related factors

Adapted from Barrett J: In Treleaven J, Barrett AJ (eds): Hematopoietic Stem Cell Transplantation in Clinical Practice. Edinburgh, UK: Elsevier Limited, 2009.

BOX 47-2 Sources of Stem Cells: Benefits and Limitations

Peripheral Blood Stem Cell Collection/Apheresis
- Easier collection for autologous patients and potentially easier for allogeneic donors
- Shorter duration of myelosuppression
- In theory, there is thought to be less tumor contamination from peripherally derived stem cells.
- Incidence of GVHD may be same or higher

Bone Marrow Harvest
- Harvest-related pain
- General anesthesia required
- May be more cost-effective or more convenient for donors

Cord Blood
- Plentiful and relatively easy to harvest if obstetricians are trained
- Inexpensive
- Excellent source to increase pool of unrelated donors
- May be associated with less GVHD
- Currently limited to individuals who weigh less than 50 to 70 kg; with ex vivo expansion, techniques may become more widespread

Stem Cell Harvesting, Mobilization, and Collection

Stem cells are most numerous in the bone marrow spaces, and some circulate in the peripheral blood. The process of harvesting and collecting hematopoietic stem cells differs depending on whether progenitor cells will be obtained through a bone marrow harvest or collected from the peripheral bloodstream.

When stem cells are obtained from the bone marrow, the harvesting procedure is performed in the operating room with the patient anesthetized. Multiple aspirations are obtained from each posterior iliac crest using large-bore needles until a total of 2 to 3 × 10^8 nucleated cells per kilogram of recipient body weight is obtained. The total volume of aspirate is 1 to 2 L. The marrow is placed in a heparinized tissue culture medium and filtered for the removal of fat and bone particles, and the cells are taken directly to the recipient's room for infusion. The bone marrow harvest procedure usually takes 1 to 2 hours. Pressure dressings are applied to the aspirate sites, and the donor is usually admitted to the hospital for overnight observation. The harvest sites may be mildly uncomfortable for 2 to 7 days after the procedure.

Hematopoietic stem cells may also be collected from the peripheral blood. However, because stem cells are not abundant in the peripheral blood, chemotherapy or colony-stimulating factors (CSFs) (granulocyte colony-stimulating factor [G-CSF] or granulocyte–macrophage colony-stimulating factor [GM-CSF]) must be given before collection to drive progenitor cells into the peripheral circulation. This process is termed *mobilization* or *priming*. The chemotherapy that patients receive for stem cell mobilization is also useful for tumor reduction. For related and unrelated donors, colony-stimulating factors alone are used to increase the number of stem cells in the peripheral blood. Protocols vary, but G-CSF or GM-CSF is given by a subcutaneous injection daily. Stem cell collections begin after 4 or 5 days of cytokine injections.

Hematopoietic progenitor cells are collected from the peripheral blood by a method called *leukapheresis*. A commercial cell separator machine collects the progenitor cells and returns the remainder of the plasma and cellular components to the bloodstream. This is performed either through wide-bore double-lumen central catheters or large-bore antecubital IV catheters. The procedure takes approximately 3 to 4 hours, and the number of leukapheresis procedures required is determined by the number of stem cells harvested at each session. The goal is to collect 5 × 10^6 CD34-positive cells per kilogram of recipient body weight. The CD34-positive antigen is an antigen expressed on the surface of early progenitor cells. With autologous hematopoietic progenitor cells, the cells are immediately cryopreserved and stored in liquid nitrogen until the recipient is ready for reinfusion. The donor may experience a transient hypocalcemia reaction with chills, fatigue, tingling in the lips and extremities, and vertigo resulting from the citrate infusion, which is used to prevent clotting of the blood during the procedure. The symptoms can be prevented or treated by taking a calcium supplement.

Conditioning Regimen

After the progenitor cells have been obtained, the recipient begins the conditioning regimen designed to prepare him or her to receive transplanted stem cells. The goal of the conditioning regimen depends in part on whether the transplant is autologous or allogeneic and on the nature of the recipient's underlying disease. In allogeneic transplantation, the purpose of conditioning is to eradicate any malignant disease, eliminate the bone marrow to create a space for the new donor stem cells, and provide sufficient immunosuppression to allow engraftment of the transplanted stem cells. In autologous transplantation, immunosuppression is not required because the recipient of the hematopoietic stem cells is also the donor, and therefore, there is no tissue incompatibility. However, the high-dose therapy is still needed to eradicate malignant disease.

High-dose chemotherapy is based on the hypothesis that increasing the total dose or dose rate will kill more tumor cells, resulting in improved response and survival rates. Drugs with different (ie, nonoverlapping) nonhematological dose-limiting toxicities are combined in maximal doses.[23] Alkylating agents (cyclophosphamide, carboplatin, busulfan, thiotepa, cisplatin, melphalan, carmustine), etoposide, cytarabine, and sometimes total-body irradiation are used to destroy the bone marrow and eradicate disease. The regimen is administered over 2 to 8 days. The individual drugs that may be used in combination as part of the transplant conditioning regimen may have several adverse effects (Table 47-6).

The stem cell recipient is then allowed 1 to 2 rest days to clear the chemotherapeutic agents from the system before the infusion of stem cells. Bone marrow aplasia occurs within days after the conditioning regimen is completed. The acute toxicity from the regimen can last for a few weeks or until engraftment occurs.

Table 47-6 Nonhematological Adverse Effects of High-Dose Therapy Regimens

Drug or Therapy	Adverse Effects
Busulfan	Interstitial pulmonary fibrosis, hepatic dysfunction (including veno-occlusive disease of the liver), acute cholecystitis, generalized seizures, mucositis, skin adverse effects (hyperpigmentation, desquamation, acral erythema), nausea and vomiting
Carmustine (BCNU)	Hepatic, pulmonary, and central nervous system adverse effects, cardiac adverse effects (dysrhythmias and hypotension), nausea and vomiting
Cytosine arabinoside (Ara-C)	Cerebellar toxicity, encephalopathy, seizures, conjunctivitis, skin adverse effects (rash, acral erythema), nausea and vomiting, diarrhea, renal insufficiency, liver function abnormalities, pancreatitis, noncardiogenic pulmonary edema, fever, arthralgias
Cyclophosphamide	Cardiac adverse effects (cardiomyopathy, congestive heart failure, hemorrhagic cardiac necrosis, pericardial effusion, ECG abnormalities), interstitial pulmonary fibrosis, hemorrhagic cystitis, elevation in liver enzymes, nausea and vomiting, metabolic adverse effects (syndrome of inappropriate antidiuretic hormone secretion)
Carboplatinum	Nausea and vomiting, nephrotoxicity, liver function abnormalities (including veno-occlusive disease of the liver), ototoxicity
Cisplatinum	Nausea and vomiting, neurotoxicity (peripheral neuropathy, ataxia, visual disturbances), ototoxicity, renal adverse effects
Etoposide	Hypersensitivity reactions, hypotension, liver function abnormalities and chemical hepatitis, renal dysfunction, nausea and vomiting, metabolic adverse effects (metabolic acidosis), mucositis, stomatitis, painful skin rash (on the palms, soles, and periorbital area)
Ifosfamide	Hemorrhagic cystitis, nausea, vomiting
Melphalan	Acute hypersensitivity reaction, renal adverse effects, mucositis, nausea and vomiting, hepatic toxicity (including veno-occlusive disease of the liver)
Thiotepa	Hyperpigmentation, acute erythroderma, dry desquamation, liver function abnormalities (including veno-occlusive disease of the liver), mucositis, esophagitis, dysuria, hypersensitivity reactions
Total-body irradiation	Nausea and vomiting, diarrhea, fever, parotitis, xerostomia, stomatitis, erythema, pneumonitis, veno-occlusive disease of the liver

Research is under way to study whether a reduced-intensity conditioning regimen (sometimes called nonmyeloablative) followed by allogeneic HSCT provides improved transplantation outcomes for selected candidates. Reduced-intensity transplantation may provide a treatment option for patients who, because of advanced age or preexisting lung, kidney, or liver damage, cannot tolerate the toxicities of a traditional allogeneic transplant.[24] The theory behind a reduced-intensity transplantation is that the immune-mediated graft-versus-tumor effect provided by the new immune system, rather than the conditioning regimen itself, cures the disease. Specific regimens under investigation include fludarabine, single-dose total-body irradiation, and a combination of potent immunosuppressive medications. These regimens are not without risk, and patients undergoing reduced-intensity transplantation still experience many of the expected complications of myeloablative allogeneic transplantation. The problems encountered in the early posttransplantation period, such as infection, bleeding, and regimen-related toxicities, may be reduced compared with myeloablative transplantation, but the risk for GVHD and the long-term risks for infection continue to be important.

Transplantation/Hematopoietic Stem Cell Infusion

In allogeneic HSCT, the stem cells are usually infused immediately after they are collected. Autologous stem cells are cryopreserved with dimethylsulfoxide (DMSO) and must be thawed in a warm normal saline bath at the bedside immediately before reinfusion.

The actual infusion of stem cells is a relatively simple procedure, much like a blood transfusion. The cells are infused into a central venous catheter over 30 to 60 minutes, depending on the total volume of the product. Patients usually are premedicated with acetaminophen, hydrocortisone, and diphenhydramine, and they are prehydrated to maintain renal perfusion. Diuretics, mannitol, and antihypertensives may be required to prevent volume overload and manage hemodynamic changes during infusion. Vital signs are monitored, and oxygen and cardiac monitoring are readily available.

Complications of allogeneic HSCT may include pulmonary edema, hemolysis, infection, and anaphylaxis; however, these are rare. A garlicky odor or taste may occur; excretion of DMSO causes this. DMSO-associated RBC hemolysis may also occur, and patients require vigorous hydration to prevent renal toxicity. An infusion reaction that may include bradycardia (rarely heart block), hypertension, and an acute hypersensitivity reaction is another potential adverse effect of DMSO during the administration of cryopreserved autologous HSCT. During HSCT, monitoring for volume overload and for complaints suggestive of embolism, such as chest pain, dyspnea, and cough is necessary. Patients may also experience an acute hemolytic transfusion reaction if they are receiving hematopoietic stem cells from an ABO-mismatched donor.

Engraftment

After IV infusion, the hematopoietic stem cells migrate to the bone marrow spaces, where they are attracted by chemotactic factors. Engraftment occurs when the transplanted progenitor cells begin to grow and manufacture new hematopoietic cells in the bone marrow. Engraftment is generally defined as an absolute neutrophil count greater than 0.5×10^9/L for 3 consecutive days and a platelet count greater than 20×10^9/L achieved without transfusion support. The rate of engraftment depends on the source of the progenitor cells, the total progenitor cell dose, the use of colony-stimulating factors, the complications the patient experiences in the pre-engraftment period, and the choice of prophylaxis against GVHD. Time to engraftment in HSCT varies according to the origin of the hematopoietic stem cells: for bone marrow–derived stem cells, 2 to 3 weeks; for peripheral blood stem cells, 11 to 16 days; and for cord blood–derived stem cells, 26 days (average; as long as 42 days).

Patients usually receive their conditioning treatment and immediate posttransplantation care in an inpatient unit. However, improved symptom management and technological advances, including the use of hematopoietic growth factors, have allowed earlier discharge from the hospital. Box 47-3 presents the criteria for hospital discharge after HSCT. Many institutions now have outpatient care facilities available for transplant recipients that permit outpatient treatment and evaluation 7 days per week.[25]

After the transplantation but before complete hematopoietic cell engraftment, patients experience severe pancytopenia and immunosuppression, and the resulting complications may include infection and bleeding. Patients take hematopoietic growth factors (eg, G-CSF, GM-CSF) to accelerate neutrophil recovery, thereby reducing the period of pancytopenia and the highest risk for infection. While patients have pancytopenia, until their blood counts begin to recover, they typically receive broad-spectrum antimicrobial drugs directed at bacteria, viruses, and fungi, as well as blood components, such as platelets and packed RBCs.

All blood products given to HSCT recipients require filtration to remove WBCs that may transmit CMV and irradiation to prevent transfusion-associated GVHD. Studies have determined that leukodepletion by either bedside filtration during transfusion or by prestorage leukocyte depletion of blood products is an effective method of preventing transfusion-associated CMV infection in HSCT because leukocytes transmit CMV.[26] In addition, filtered blood products may also prevent febrile transfusion reactions and delay alloimmunization.[27,28] However, use of a leukocyte depletion filter does not affect the risk for transmission of viral hepatitis.

Transfusion-associated GVHD is a rare but almost uniformly fatal complication of transfusion, resulting from the infusion of immunocompetent lymphocytes capable of proliferation into an immunocompromised recipient who is unable to destroy them.[26] The infused lymphocytes recognize host tissues as foreign and mount a reaction.

To prevent transfusion-associated GVHD, all cellular blood products except for stem cell grafts and donor lymphocytes given for graft-versus-tumor effect, should be irradiated with 2500 cGy prior to transfusion.[26] The blood component should be labeled as irradiated. It is not radioactive, and no additional precautions for handling the blood product are required. Other patients can use irradiated blood products; the irradiating process does not alter the efficacy or cellular content of the product and does not harm the recipient in any way. Patients who have undergone allogeneic or autologous HSCT should receive irradiated cellular components, both before and after stem cell transplantation.[25] Most centers recommend that allogeneic stem cell recipients receive irradiated blood products for the rest of their lives. Box 47-4 presents a collaborative care guide for the patient with allogeneic HSCT.

BOX 47-3 Criteria for Hospital Discharge After Hematopoietic Stem Cell Transplantation

Discharge from hospital is usually permitted when:
- The patient has been afebrile for at least 24 hours.
- A 24-hour outpatient medical facility is available.
- Family care giver support is available.
- The patient is independent in basic activities of daily living.
- Acute posttransplantation complications have been resolved or controlled.
- The patient is achieving oral intake of at least 1,500 mL/d, or a plan to meet the patient's fluid needs through self-administration of IV fluids or daily IV fluid administration in clinic is in place.
- Nausea and vomiting are controlled.
- The patient is able to tolerate oral medications.
- Availability of appropriate transfusion support in a clinic environment and access to irradiated blood products have been determined.
- The patient's platelet count is supportable with no more frequent than daily platelet product.
- The patient's white blood cell count is greater than 1×10^9/L.
- A hematocrit of 25% is supportable with no more than daily transfusion of one unit of packed red blood cells (RBCs).

▲ Immunosuppressive Therapy

In solid organ transplantation, the transplanted organ is foreign to the recipient, whose immune system eventually will recognize this and mobilize to reject the transplanted organ. Therefore, immunosuppressive therapy is necessary to suppress the immune response so the transplanted organ will be accepted. In allogeneic HSCT, because the immune system is generated from donor cells, immunosuppressive therapy is used to prevent GVHD, a response in which donor T lymphocytes attack the recipient's cells. The challenge of immunosuppressive therapy is to provide the recipient with adequate immunosuppression without undue toxicity, unfavorable reactions, and excess susceptibility to opportunistic infections. Therapeutic regimens may be individualized based on the needs of a particular patient.

To suppress the immune response in both solid organ and hematopoietic stem cell transplant recipients, several drugs may be necessary (Table 47-7, p. 1072). A single medication usually cannot do this effectively. Therefore, immunosuppressive regimens include medications that complement each

Text continued on page 1071

BOX 47–4 COLLABORATIVE CARE GUIDE for the Allogeneic Hematopoietic Stem Cell Transplantation

OUTCOMES	INTERVENTIONS
Oxygenation/Ventilation	
The patient will demonstrate pulmonary hygiene techniques, including coughing, deep breathing, incentive spirometry, and daily exercise as tolerated. The patient will demonstrate improvement in respiratory pattern and lung sounds, with subjective reduction in complaints of dyspnea, cough, pleuritic chest pain, and weakness. The patient's risk for aspiration will be eliminated or minimized.	• Auscultate lung sounds and vital signs and pulse oximetry every 4 h and PRN. • Note skin color capillary refill and presence of central or peripheral cyanosis. • Assess the quality of respirations including use of accessory muscles, nasal flaring, and grunting. • Note cough and complaints of shortness of breath, orthopnea, or pleuritic pain. • Determine effects of medications that may affect respiratory status, including narcotics and sedatives. • Suction oropharynx or nasotrachea to remove secretions. • Encourage deep-breathing and coughing exercises, and teach patient proper use of incentive spirometry. • Encourage patient to maintain optimal level of activity, including walking, exercise bicycle, and working daily with physical therapist. • Instruct patient on methods of preventing respiratory complications related to mucositis or aspiration, such as avoiding drinking liquids after using topical local anesthetics, keeping head of bed elevated and having oral suction in a convenient location. • Implement measures to manage anemia and control bleeding, if present. • Administer supplemental oxygen as indicated. • Administer diuretics as ordered.
Circulation/Perfusion	
The patient will exhibit the absence or control of signs and symptoms of potential physiological problems: **Bleeding/hemorrhage** **Hypotension** secondary to sepsis, hemorrhage, medication side effects, or dehydration **Hypertension** secondary to medication side effects **Dehydration** secondary to decreased oral fluid intake as a result of nausea, vomiting, or mucositis, or increased fluid losses through fever, diarrhea, or insensible fluid losses through the skin	• Develop a nursing plan to manage individual cardiovascular problems. Cardiovascular problems may include hypertension, orthostatic hypotension, sepsis-induced hypotension, dysrhythmias, pericarditis, superior vena cava syndrome, thrombus formation, and myocardial infarction. • Replace fluid losses with packed RBCs or hydration. • Use appropriate measures to decrease fluid losses secondary to fever, diarrhea, vomiting, or hemorrhage. • Administer antihypertensives, diuretics, antiarrhythmics, or vasoactive drugs as ordered and monitor their effectiveness. • Provide information to patient and family regarding cardiac status, and rationale for specific nursing interventions. • Monitor platelet count and coagulation profile carefully, especially in patients experiencing hypertension. • Instruct patient about appropriate safety measures during period of altered cardiac status. Patients who are orthostatic may become dizzy when standing. Provide assistance as needed. • Although hypotension and hypertension may occur without symptoms, alert patient to these signs and symptoms, and encourage prompt reporting to the health care team. Common signs of hypertension include headache and visual disturbance. Hypotension may be associated with dizziness, visual disturbances, tachycardia, and cool or diaphoretic skin. • Emphasize the importance of taking oral antihypertensive medications when scheduled.
Fluids/Electrolytes	
The patient will exhibit the absence of or control of signs and symptoms of potential physiological problems: **Veno-occlusive disease of the liver,** as manifested by sudden weight gain, increase in bilirubin, aspartate aminotransferase, and alkaline phosphatase levels, hepatomegaly, ascites, and encephalopathy **Renal insufficiency/failure,** as manifested by a decreased urine output or rising serum creatinine	• Strictly monitor intake, output, and fluid balance every 4–8 h. • Check weight twice daily. • Measure abdominal girth daily in patients with veno-occlusive disease. • Perform pulmonary assessment, including pulse oximetry, respiratory rate, quality, and presence of adventitious breath sounds. • Perform cardiac assessment, noting presence of orthostatic hypotension, extra heart sounds, or neck vein distention. • Assess for peripheral and sacral edema. • Monitor serum electrolytes, liver function tests, and urine specific gravity once or twice daily as ordered.

(continued on page 1069)

BOX 47-4 *COLLABORATIVE CARE GUIDE for the Allogeneic Hematopoietic Stem Cell Transplantation (continued)*

OUTCOMES	INTERVENTIONS
Fluids/Electrolytes	
Dehydration secondary to decreased oral fluid intake as a result of nausea, vomiting, or mucositis, or increased fluid losses through fever, diarrhea, or insensible fluid losses through the skin. **Electrolyte disorders** secondary to steroid-induced hyperglycemia, syndrome of inappropriate antidiuretic hormone secretion, tumor lysis syndrome, or medication-induced electrolyte shifts	• Monitor for and treat steroid-induced hyperglycemia. • Adjust dose or eliminate nephrotoxic drugs in patients with renal insufficiency or renal failure. • Administer intravenous fluids as ordered to replace gastrointestinal losses and to supplement inability to consume sufficient oral fluids. • Administer electrolyte replacements as ordered. • Administer antiemetics and antidiarrheal agents as ordered. • Monitor stools for volume, consistency, color, and odor. • Monitor for fluid losses through sloughing of the skin.
Mobility/Safety	
The patient will: Maintain baseline strength and endurance and minimize focal muscle weakness, fatigue, and dyspnea Participate in measures to prevent a decrease in strength and endurance Experience minimal complications from decreased mobility Use safety measures to prevent injury	• Encourage patient to continue daily activities throughout the transplantation course. Provide rationale and explain the complications of inactivity to both patient and family. • Develop an individualized plan to address factors contributing to limited mobility, including pain, nonadherence, depression, medication side effects, nausea, generalized weakness and malaise, and altered level of consciousness. • Refer to physical and occupational therapy for assessment and treatment. • Encourage focused exercises that provide proximal muscle strengthening in patients on corticosteroids. • Encourage patient to maintain maximal independence in activities of daily living (ADLs) and self-care. • Develop a schedule that allows adequate rest by coordinating all activity and treatments (ADLs, medications, health team rounds). • Identify signs of impaired activity tolerance and set specific goals for improving tolerance/endurance in activities.
Skin Integrity	
The patient will: Maintain skin integrity Demonstrate techniques of skin care Demonstrate basic understanding of skin GVHD Exhibit control of symptoms associated with skin GVHD	• Assess skin daily for maculopapular rash, erythema, sloughing, or open lesions. • Monitor skin for the development of signs of infection such as warmth, erythema, swelling, or tenderness. • Apply skin emollients and other topical agents, including topical antimicrobial agents, as ordered. • Consider use of antipruritic or steroid topical agents to manage symptoms of itching and inflammation. • If skin breakdown occurs, consult enterostomal therapist or wound management specialist concerning nonadherent, absorptive dressings and the role of special beds/mattresses. • Monitor patient for dehydration if there are increased insensible losses of fluid through the skin. • Teach patient and family the importance of avoiding direct sun exposure and the importance of using sun block and protective clothing when outdoors because sun exposure can initiate a flare of GVHD of skin.
Nutrition	
The patient will: Have nutritional balance maintained through diet and parenteral nutrition Demonstrate understanding of dietary restrictions related to a specific gastrointestinal problem Demonstrate methods for maintaining nutritional requirements as an outpatient	• Evaluate oral mucosal integrity and note presence of oral problems. • Note subjective data, including complaints of nausea, loss of appetite, taste changes, early satiety, pain or abdominal cramping, and any precipitating factors. • Monitor calorie counts, intake and output totals, and weights to determine adequacy of nutritional intake. • Develop a multidisciplinary plan for nutritional management. Discuss approaches to use when experiencing taste changes; thick, viscous saliva and mucus; xerostomia; early satiety; nausea/vomiting; mucositis or esophagitis; and other troubling symptoms. • If patient is receiving high-dose steroids, ensure increased protein intake and calcium and vitamin D supplementation. Consider restricted concentrated carbohydrate intake if hyperglycemia is present. Consider restricted sodium intake if fluid retention is especially problematic. • Increase protein intake in patients with extensive skin or gastrointestinal GVHD.

(Continued on page 1070)

BOX 47–4 *COLLABORATIVE CARE GUIDE for the Allogeneic Hematopoietic Stem Cell Transplantation (continued)*

OUTCOMES	INTERVENTIONS
Nutrition	
	• Consider multiple vitamin without iron (to prevent iron overload), and folic acid supplementation 1 mg orally daily. • Maintain nutritional requirements through use of hyperalimentation as required. • Encourage patient to try different foods as tolerated. • Advance diet as tolerated and note response to advancement. • Encourage small, frequent meals and a bedtime snack. • Medicate for nausea or pain as needed. • Encourage family members to be supportive and patient. Pressure from family or staff can produce anxiety that will have a negative effect on eating habits.
Comfort/Pain Control	
Patient will be able to: Identify activities that increase or decrease pain Relate location and characteristics of pain as well as degree of pain relief to the health care team Participate in daily care without interference from pain or side effects of pain control measures Achieve acceptable level of pain control	• Monitor for the potential sources of pain/discomfort in the hematopoietic stem cell transplant recipient, including: Pain associated with diagnostic or therapeutic procedures Neuropathic pain secondary to immunosuppressive medications Mucositis/esophagitis Rectal pain/hemorrhoids Painful urination from hemorrhagic cystitis Abdominal pain secondary to infections, severe diarrhea/enteritis, or liver distention Cutaneous discomfort secondary to GVHD of skin • Teach patient importance of reporting pain and the effectiveness of pain relief measures to health care team. • Assess location, onset, frequency, intensity, and quality of pain. Use a 0–10 pain scale to assess patient's perception of pain and to assist in evaluating effectiveness of treatment. For patients unable to communicate, monitor changes in vital signs and observe for increased restlessness, which could indicate inadequate pain control. • Premedicate patient before potentially painful procedures. • Teach proper and safe use of patient-controlled analgesia if ordered. • For patients receiving continuous infusion, consider the need for a bolus of analgesic before disconnecting patient from continuous infusion, while disconnected, and after resuming continuous infusion. • Suggest consultation with pain management team if indicated. • Monitor narcotic dosages and patient response carefully in patients with hepatic dysfunction secondary to veno-occlusive disease or GVHD. • In the nonventilated patient, adjust analgesic dosages in the presence of markedly deceased respiratory rate or markedly altered level of consciousness.
Psychosocial—Individual and Family	
Patient and family will be able to: Identify effective and ineffective coping patterns Identify personal strengths Verbalize their needs Actively participate in problem solving Use resources and support systems to strengthen their coping skills	• Provide opportunities for patient and family to communicate with each other, as well as with psychosocial support professionals and other health care team members. • Reinforce successful coping skills (ie, clearly verbalizing needs, using activities that reduce stress, and effective patient/family/team communication). • Provide reassurance and review support systems, options, and resources available to support effective coping. • Establish trust and congruence in goals and objectives. • Provide information in a timely and specific manner. • Allow patient as much choice and control as appropriate and feasible. • Avoid the use of approaches that foster dependency (coercion, persuasion, manipulation). • Demonstrate caring, respect, and concern for patient and family. • Use positive reinforcement.

(continued on page 1071)

BOX 47–4 COLLABORATIVE CARE GUIDE for the Allogeneic Hematopoietic Stem Cell Transplantation (continued)

OUTCOMES	INTERVENTIONS
Teaching/Discharge Planning	
The patient and family will demonstrate knowledge of: The overall process of HSCT and the expected complications and self-care requirements, including preparative regimen and side effects, infusion of peripheral stem cells and side effects, engraftment, complications, discharge criteria, follow-up care, symptoms to report, protective precautions (neutropenic and thrombocytopenic precautions), dietary restrictions, and specific psychomotor skills, including central venous catheter care, medication administration, and vital sign monitoring The structure of the inpatient unit, unit routines, and the programs and resources available to make their inpatient stay more comfortable and facilitate their self-care The importance of the active involvement of patient and family in the daily care and decision making throughout the HSCT process On discharge, patient and family will demonstrate knowledge of: Signs and symptoms that should be reported immediately to the health care team, including fever, chills, skin rash, bleeding, nausea, vomiting, diarrhea or abdominal pain, shortness of breath, cough, dyspnea, and an inability to take oral medication or consume sufficient fluids Names of medication, rationale for each medication, the required schedule for administration, and potential side effects Contact telephone numbers for day and after-hours care Psychomotor skills necessary for administration of oral and IV medications, self-care of central venous catheter, and any other self-care skills such as administration of total parenteral nutrition and home IV hydration	• Orient patient to unit, room environment, ancillary services, and general routines. The patient and family may be initially overwhelmed by the amount of new information and may require reinforcement of information. • Allow patient and family to share concerns and request additional information about the transplantation process. • Encourage questions throughout the transplantation process, and provide clarification on all aspects of treatment. • Explain the need for active patient and family involvement in daily care and decision making throughout the HSCT process. • Teach the importance of daily hygiene, diligent oral care, daily exercise, nutrition, and other routines. • Encourage patient to maintain open communication with health care team. • Integrate discharge teaching while fostering patient family participation in daily care. • Provide patient with written instructions on outpatient self-care guidelines, medication schedule, and other self-care activities. • Refer to home health agency for continued education and support of patient at home, as indicated. • Provide teaching/instruction using methods adapted to patient's learning style. Discuss with patient how he or she learns best: by doing, by listening, or by watching. • Document teaching provided, areas requiring continued reinforcement and follow-up, and learning outcomes achieved in the patient's record.

other and increase the effectiveness of the immunosuppression. The foundation of most immunosuppressant regimens for solid organ transplantation is triple, or three-drug, therapy. The combination of drugs used for organ transplantation and HSCT may differ.

Triple therapy is a combination of low-dose prednisone, azathioprine or mycophenolate mofetil (MMF), and cyclosporine A or tacrolimus. By combining these three agents, the dose of each drug is lower so that patients experience fewer adverse effects than they would from one drug alone. For example, the risk for aseptic necrosis, diabetes mellitus, cataracts, and gastrointestinal complications attributed to chronic steroid therapy is greatly reduced with the combination therapy. Because the dosage of azathioprine is low, the potential for hepatotoxicity and leukopenia is decreased. Problems associated with higher doses of cyclosporine A, including lymphoma, hirsutism, hepatotoxicity, gingival hyperplasia, seizures, and gastrointestinal disturbances, occur at a lower frequency.

Quadruple, or sequential, therapy is a combination of the same three drugs that are used in triple therapy (prednisone, azathioprine or MMF, and either cyclosporine A or tacrolimus) plus antithymocyte antibody preparations or monoclonal antibody, monomurab-CD3. The calcineurin inhibitors, cyclosporine A or tacrolimus, are withheld until renal function is present. All four drugs are given for several days, after which the polyclonal or monoclonal antibody preparation is discontinued. A triple-drug regimen is then continued for maintenance therapy.

Because of the nephrotoxicity of calcineurin inhibitors and the risks for drug accumulation and resultant toxicity in the absence of renal function, not using the calcineurin inhibitors in the early posttransplantation period has advantages. Quadruple therapy permits both broad and specific immunosuppression while limiting toxicity until renal function has improved. However, it does have a disadvantage: the potential inability to use the polyclonal or monoclonal antibody preparation for treatment of rejection episodes or as "rescue" therapy.

Text continued on page 1078

Table 47-7 Selected Immunosuppressive Drugs Used in Solid Organ and Hematopoietic Stem Cell Transplantation

Drug	Mechanism of Action	Dosing/Administration	Adverse Reactions	Comments
Cyclosporine A (Sandimmune, Neoral)	Prevents IL-2 gene expression and thus impairs IL-2 synthesis and activation of T lymphocytes	Solid organ transplant dosing: Initial dosing of 4 mg/kg/d (IV); maintenance dosing of 5–15 mg/kg/d (PO). HSCT dosing: usually 1.5 mg/kg IV every 12 h, 0.75 mg/kg every 6 h or 3 mg/kg/d as a continuous infusion Dosage is adjusted to achieve therapeutic levels. IV to PO conversion: approximately 1:3 Dosage dependent on achieving and sustaining therapeutic blood levels based on laboratory evaluation Therapeutic monitoring not required once drug dosage is being tapered	Metabolic: hyperkalemia and hyperglycemia, hypomagnesemia, hyperlipidemia, hyperuricemia, diabetes mellitus Neurotoxicity: headache, tremor, insomnia, paresthesia, dizziness, seizures GI: diarrhea, nausea, constipation, anorexia, vomiting, abdominal pain, ascites, elevated liver function tests Renal: elevated creatinine, nephrotoxicity Cardiovascular: hypertension, chest pain Hematological: anemia Cutaneous: acneiform rash, striae Other: peripheral edema, infection, impaired wound healing, osteoporosis, gingival hyperplasia, flushing, sweating, hirsutism	• Bioavailability differs between oral solution and capsule formulation. Once regimen is established, patients should be instructed not to change formulation or brand. • Take with food. • Instruct patient on importance of strict adherence to administration schedule and to notify health care team immediately if unable to take due to GI side effects. • Monitor serum creatinine, blood urea nitrogen (BUN), potassium, magnesium, glucose, and triglyceride levels. • Avoid potassium-sparing diuretics. • Replete electrolytes as indicated. • Coadministration with grapefruit juice may increase cyclosporine levels and should be avoided. • Drug–drug interactions can lead to subtherapeutic or toxic cyclosporine levels. Drugs that inhibit or induce cytochrome P-450 are most responsible (see Table 47-14, p. 1088). • Cyclosporine trough levels to be drawn before administration of morning dose. Therefore, doses are usually timed for 10:00 AM and 10:00 PM to allow trough blood draw at morning clinic visit. Instruct patient to bring dose to clinic and to administer once trough level drawn. • Should not be used simultaneously with tacrolimus. • Tacrolimus should be discontinued 24 h before starting cyclosporine. In the presence of increased tacrolimus levels, initiation of cyclosporine should usually be further delayed. • Doses should be adjusted for renal dysfunction. • Monitor levels carefully in patients with renal or hepatic dysfunction.
Tacrolimus (Prograf)	Impaired synthesis of IL-2 and prevents T-lymphocyte proliferation; interferes with the gene transcription for a variety of cytokines including IFN-γ, TNF-α	Solid organ transplant dosing: 0.10 mg/kg/d (IV); 0.05–0.2 mg/kg/d (PO) HSCT dosing: usually 1–2 mg PO every 12 h; 0.05–0.1 mg/kg/d as a continuous IV infusion, with dosage adjusted to achieve therapeutic levels	Metabolic: hyperkalemia and hypokalemia, hyperglycemia, hypomagnesemia, hyperlipidemia, hypophosphatemia, diabetes mellitus Neurotoxicity: headache, tremor, insomnia, paresthesia, dizziness, seizures GI: diarrhea, nausea, constipation, anorexia, vomiting, abdominal pain, ascites, elevated liver function tests	• Take on empty stomach. • Instruct patient on importance of strict adherence to administration schedule and to notify the health care team immediately if unable to take due to GI side effects. • Monitor serum creatinine, BUN, potassium, magnesium, phosphorus, glucose, and triglyceride levels. • Avoid potassium-sparing diuretics. • Replete electrolytes as indicated • Coadministration with grapefruit juice may increase tacrolimus levels and should be avoided.

		IV to PO conversion: approximately 1:4 Dosage dependent on achieving and sustaining therapeutic blood levels based on laboratory evaluation Therapeutic monitoring not required once drug is being tapered	Renal: elevated creatinine, nephrotoxicity Cardiovascular: hypertension, chest pain Hematological: anemia, leukocytosis, thrombocytopenia Cutaneous: pruritus, acneiform rash Pulmonary: pleural effusion, atelectasis, dyspnea Other: peripheral edema, infection, impaired wound healing, osteoporosis	• Drug–drug interactions can lead to subtherapeutic or toxic tacrolimus levels. Drugs that inhibit or induce cytochrome P-450 are most responsible (see Table 47-14, p. 1088). • Tacrolimus trough levels to be drawn before administration of morning dose. Therefore doses are usually timed for 10:00 AM and 10:00 PM to allow trough blood draw at morning clinic visit. Instruct patient to bring dose to clinic and to administer once trough level drawn. • Should not be used simultaneously with cyclosporine. • Cyclosporine should be discontinued 24 h before starting tacrolimus. In the presence of elevated cyclosporine levels, initiation of tacrolimus is delayed. • Doses should be adjusted for renal dysfunction. • Monitor levels carefully in patients with renal or hepatic dysfunction.
Methylprednisolone (IV) Prednisone (PO)	Decreases cytotoxic T-cell proliferation, inhibits production of IL-1 and IFN-g, prevents production of IL-2, inhibits neutrophils function by stabilizing leukocyte lysosomal membrane and inhibiting chemotaxis	Dosage varies according to institutional protocols Dosage: 0.5–2 mg/kg/d every 12 h, with tapering schedule determined based on starting dose and patient response During solid organ transplant rejection: Methylprednisolone may be given in IV bolus up to 1 g/dose	Metabolic: fluid and electrolyte imbalance, diabetes mellitus, hyperlipidemia Neurotoxicity: tremors, seizures, headache, difficulty concentrating, insomnia GI: GI irritation Cardiovascular: hypertension, dysrhythmias Cutaneous: bruising, fragile skin Neurotoxicity: tremors, seizures, headache Other: hunger, peripheral edema, infection, impaired wound healing, hirsutism, osteoporosis, weight gain, steroid myopathy, cataracts/ glaucoma, cushingoid changes, psychiatric disturbances (steroid psychosis, mood changes, confusion)	• Usually used in combination with cyclosporine or tacrolimus. • Consult physical therapy for proximal muscle strengthening exercise program. • Instruct patient in strategies to prevent or treat hyperglycemia, and in diabetic self- management. • Administer oral corticosteroids with food/milk to minimize GI upset. • Administer H_2 blockers or proton pump inhibitor, as ordered. • May increase tacrolimus or cyclosporine levels. • Report complaints of visual changes and consult ophthalmology. • For patients on long-term steroids or otherwise at risk for or experiencing osteopenia (eg, patients with acute lymphocytic leukemia, postmenopausal), ensure regular dual-energy x-ray absorptiometry scans, calcium and vitamin D supplementation, and specific treatment for osteopenia with antiresorptive agents such as pamidronate, alendronate. • Tapering calendar specifying the dosage to be taken each day can help facilitate adherence in patients who are on tapering doses of steroids, or an alternate-day steroid regimen.
Mycophenolate mofetil (MMF) (CellCept)	Antimetabolite that selectively inhibits the proliferation of T and B lymphocytes by interfering with purine nucleotide synthesis	Dosage: 1–1.5 g IV or PO every 12 h depending on institutional guidelines	Metabolic: hyperkalemia and hypokalemia, hyperlipidemia, hypophosphatemia, hyperglycemia Neurotoxicity: headache, insomnia, tremors, seizures GI: diarrhea, nausea, constipation, anorexia, vomiting, abdominal pain, hepatotoxicity Renal: elevated creatinine, nephrotoxicity Cardiovascular: hypertension, hypotension, dysrhythmias Hematological: anemia, leukocytosis, thrombocytopenia Cutaneous: acneiform rash Pulmonary: cough, dyspnea Other: fever, edema, pain, infection, muscle weakness, anxiety, depression	• MMF should be taken on an empty stomach. • Monitor complete blood count (CBC) at regular intervals and adjust dosage for pancytopenia, as ordered. • Monitor liver function tests (bilirubin and serum aminotransferases) at regular intervals, adjust dosage for liver function abnormalities, as ordered. • Monitor serum levels of the MMF metabolic to guide treatment in patients with renal dysfunction. • In the setting of renal impairment, or when coadministered with probenecid, acyclovir, or ganciclovir, the drug concentrations of MMF and of these drugs may increase. • There may be decreased MMF absorption when coadministered with magnesium oxide, aluminum- or magnesium-containing antacids, or cholestyramine.

(Continued on page 1074)

Table 47-7 **Selected Immunosuppressive Drugs Used in Solid Organ and Hematopoietic Stem Cell Transplantation** (continued)

Drug	Mechanism of Action	Dosing/Administration	Adverse Reactions	Comments
Methotrexate	Antimetabolite that inhibits dihydrofolate reductase, thereby hindering DNA synthesis and cell reproduction, and thus inhibiting lymphocyte proliferation	Institutional protocols vary Usual dose: 5–15 mg/m^2 given IV on days 1, 3, 6, and 11 after transplantation	Myelosuppression, mucositis, photosensitivity, interstitial pneumonitis, hepatotoxicity, nephrotoxicity	• Dose and schedule for methotrexate prophylaxis for GVHD vary by institution. Common regimen is 5–15 mg/m^2 on days 1, 3, 6 and 11 posttransplantation. • Doses may be adjusted or held for severe mucositis and renal or hepatic insufficiency. Doses may need to be adjusted for hypoalbuminemia. • Wait until at least 24 h after stem cell infusion to give day +1 dose.
Rituximab (Rituxan)	Rituximab binds specifically to the B-lymphocyte cell surface antigen CD20, inducing B-cell death. CD20 is a protein located on normal pre-B and mature B lymphocytes	375 mg/m^2 by IV infusion given weekly for 4 wk Administer slowly. The first infusion of rituximab should be initiated at a rate of 50 mg/h. If hypersensitivity or infusion-related events do not occur, the infusion rate should be increased by 50 mg/h increments every 30 min to a maximum of 400 mg/h. Subsequent infusions can be initiated at 100 mg/h, with increases of 100 mg/h every 30 min to a maximum of 400 mg/h. Protect from sunlight. Do not filter	Mild to moderate infusional toxicities consisting of fever and chills/rigors in the majority of patients during the first rituximab infusion. Other frequent infusion reaction symptoms include nausea, pruritus, angioedema, asthenia, hypotension, headache, bronchospasm, throat irritation, rhinitis, urticaria, rash, vomiting, myalgia, dizziness, and hypertension. These reactions generally occur within 30–120 min of beginning the first infusion and resolve with slowing or interruption of the rituximab infusion and with supportive care (diphenhydramine, acetaminophen, IV saline solution, and vasopressors). The incidence of infusion reactions is highest during the first infusion (77%) and decreases with each subsequent infusion. Other adverse effects include lymphopenia and decreased serum immunoglobulins, which can contribute to risk for serious infections, including hepatitis B reactivation with fulminant hepatitis, progressive multifocal leukoencephalopathy, and other opportunistic infections. Adverse effects also include cardiac dysrhythmias, renal toxicity, bowel obstruction and perforation; neutropenia and anemia; and cough, rhinitis, bronchospasm, dyspnea, interstitial pneumonitis, and bronchiolitis obliterans.	• Premedication consisting of acetaminophen and diphenhydramine should be considered before each infusion. • Medications and equipment necessary for the management of hypersensitivity/anaphylaxis should be readily available (eg, epinephrine, antihistamines, corticosteroids, oxygen). • Assess baseline vital signs. Check CBC with differential and renal function. • Because of the potential for transient hypotension during infusion antihypertensive medication should not be taken 12 h prior to infusion. • The infusion should be interrupted if infusion-related reactions occur. Mild side effects usually resolve with temporary discontinuation of the infusion or administration of meperidine, diphenhydramine, or other supportive therapy such as bronchodilators or IV saline. • Patients experiencing chills should be offered blankets and comfort measures until the reaction subsides. Administration of acetaminophen as often as every 3 h as needed is recommended. • Most patients who experience non–life-threatening reactions are able to complete the full course of therapy. Once the infusion-related side effect has completely resolved and the patient is comfortable (usually about 30 min), rituximab may be restarted at half the previous infusion rate. After restarting the infusion, recurrence of the side effect usually does not occur. • Patients and family members should be advised concerning infusion-related side effects and informed that driving or operating equipment cannot be resumed until the effects of sedation are gone. • Women of childbearing age should receive instructions to ensure that they are using effective contraceptive methods during therapy and for 12 mo after therapy is completed. Infant nursing should be discontinued until circulating levels are no longer detectable, which is usually 12 mo after therapy is completed. • The safety of immunization with live viral vaccines following rituximab therapy has not been studied, and vaccination with live virus vaccines is not recommended.

Daclizumab (Zenapax)	Monoclonal antibody against the IL-2 receptor expressed on activated T cells. Binds to the IL-2 receptor in a nonactivating fashion, competing with IL-2 and thereby inhibiting IL-2 driven proliferation of the activated T lymphocyte IL-2–induced proliferation of activated (antigen-stimulated) T lymphocytes is a critical step in proliferation and ultimately tissue destruction	Institutional protocols vary Usual dose: 1 mg/kg by IV administration	Constipation, nausea, vomiting, diarrhea, abdominal pain, abdominal distention, edema, tremor, headache, dizziness, nephrotoxicity, chest pain, tachycardia, fever, pain, fatigue, hypertension, hypotension, dyspnea, pulmonary edema, coughing, musculoskeletal pain, back pain	• Anaphylactoid reactions after the administration of daclizumab have not been observed but can occur after the administration of proteins. Medications for the treatment of severe hypersensitivity reactions should be available for immediate use. • The calculated volume of daclizumab should be mixed with 50 mL of sterile 0.9% sodium chloride solution and administered through a peripheral or central vein over a 15-min period. Once the infusion is prepared, it should be administered IV within 4 h. If it must be held longer, it should be refrigerated between 2°C–8°C (36°F–46°F) for up to 24 h. After 24 h, the prepared solution should be discarded. • No incompatibility between daclizumab and PVC or polyethylene bags or infusion sets has been observed. • No dosage adjustment is necessary for patients with severe renal impairment.
Infliximab (Remicade)	Monoclonal antibody against TNF-α Binds to soluble and membrane-bound TNF-α, producing reduction in serum IL-1 and reduced levels of nitric oxide synthase	Institutional protocols vary Usual dose: 10 mg/kg by IV administration Administer over at least 2 h Must be given with a low protein-binding filter of 1.2 mcm or less	Headache, nausea, abdominal pain, fatigue, fever, and coughing Infusion reactions, including fever, chills, chest pain, hypotension, headache, and urticaria can occur during the infusion and for up to 2 h after the infusion is complete. There is no increase in the incidence of reactions after the initial infusion. Delayed, serum sickness–like reactions including myalgias, arthralgias, fever, rash, sore throat, dysphagia, and hand and facial edema can be seen 3–12 d after infusion. Patients may develop human antichimeric antibody.	• Monitor patient for development of infusional toxicities. • Consider premedication with acetaminophen and diphenhydramine (Benadryl). • Initiate therapy at 10 mL/h × 15 min, increase to 20 mL/h × 15 min, and then increase to 40 mL/h × 15 min, then 80 mL/h × 15 min, then 150 mL/h × 30 min, and then 250 mL/h × 30 min to complete infusion in 2 h. • Stop or slow infusion and give diphenhydramine (Benadryl), acetaminophen, or Solu-Cortef to treat mild to moderate infusion reaction. Resume infusion at 10 mL/h once reaction is controlled or abates. • Medications for treating hypersensitivity reactions (eg, acetaminophen, antihistamines, corticosteroids, or epinephrine) and supplemental oxygen should be available for immediate use in the event of a reaction. • Incompatible with PVC equipment or devices. Use glass infusion bottles and polyethylene-lined administration sets.
Muromonab-CD3 (Orthoclone OKT3)	Febrile reactions; fever, chills, tremor Respiratory: dyspnea, chest pain, wheezing, pulmonary edema GI: nausea, vomiting, diarrhea Anemia, thrombocytopenia	2.5–5 mg/d IV bolus over 30–60 s, for 10–14 d	Reactions are greatest with first dose and occur within 30–60 min. To minimize first dose reaction, pretreat with methylprednisolone, acetaminophen, and diphenhydramine hydrochloride. Monitor vital signs every 15 min for 2 h, then every 30 min first two doses. Have emergency equipment and cooling blanket available. Repeat administrations may cause serious reactions if antibodies develop.	Muromonab-CD3 (Orthoclone OKT3)

(Continued on page 1076)

Table 47-7 Selected Immunosuppressive Drugs Used in Solid Organ and Hematopoietic Stem Cell Transplantation *(continued)*

Drug	Mechanism of Action	Dosing/Administration	Adverse Reactions	Comments
Alemtuzumab (Campath)	Monoclonal antibody directed against the cell surface antigen CD52, which is expressed on B and T lymphocytes	Institutional protocols vary Usual dose: 20 mg/d IV given over several hours for 5 d, beginning before transplantation	Infusional toxicities may be severe, and include fever and rigors in more than 80% of patients. Other adverse effects include neutropenia, anemia, thrombocytopenia, nausea, vomiting, rash, fatigue, and hypotension.	• Premedicate patient with acetaminophen and diphenhydramine • Medications for treating hypersensitivity reactions (eg, acetaminophen, antihistamines, corticosteroids, or epinephrine) and supplemental oxygen should be available for immediate use in the event of a reaction. • Consider treatment with meperidine to control infusional rigors. • Administer fluid bolus as ordered to treat hypotension. • Produces profound and rapid lymphopenia; therefore, patients require broad antifungal, antibacterial, antiviral, and antiprotozoal prophylaxis for at least 4 mo after treatment.
Antithymocyte globulin (ATG, Atgam, equine) (Thymoglobulin, rabbit)	Polyclonal immunoglobulin composed of horse or rabbit antibodies capable of destroying human leukocytes	Institutional protocols vary Usual dose: 10–40 mg/kg/d for equine ATG, and 2.5 mg/kg/d for rabbit ATG	Seizures, laryngospasm, anaphylaxis, pulmonary edema, leukopenia, and thrombocytopenia. ATG is a foreign xenogeneic protein and an antibody, which may cause serum sickness, including myalgias, arthralgias, fever, rash, sore throat, dysphagia and hand and facial edema.	• Skin test for hypersensitivity to animal serum may be performed before initial dose Monitor patient closely both during infusion and after infusion for signs of serum sickness and anaphylaxis. Consider premedication with corticosteroids, acetaminophen, and H_1 and H_2 blockers. Medications for treating hypersensitivity reactions (eg, acetaminophen, antihistamines, corticosteroids, or epinephrine) and supplemental oxygen should be available for immediate use in the event of a reaction. • Because transient and at times severe thrombocytopenia may occur after ATG administration, in patients with platelet counts <100,000, monitor platelet count 1 h after ATG administration, and transfuse platelets as indicated.
Cyclophosphamide (Cytoxan)	Leukopenia, thrombocytopenia Increased susceptibility to infections Metabolite of cyclophosphamide is a direct irritant to bladder mucosa and may cause hemorrhagic cystitis.	1–2 mg/kg	Given in place of azathioprine in patients with hepatotoxicity. Mesna may be administered to neutralize the toxic metabolite of cyclophosphamide (acrolein) Administer on awakening to avoid accumulation of metabolites in bladder while sleeping. Observe for hematuria. Urine output of at least 100 mL/h should be maintained for at least 72 h after Cytoxan is administered to promote adequate dilution of renally excreted metabolites that produce hemorrhagic cystitis.	Cyclophosphamide (Cytoxan)

Azathioprine (Imuran) (IV or PO)	Bone marrow suppression: leukopenia, thrombocytopenia, anemia, pancytopenia Rash Alopecia Liver damage, jaundice Increased susceptibility to infection	Regulated to keep WBC 5,000–10,000; drug usually stopped when WBC 3,000 or less Initial 2–5 mg/kg of body weight Maintenance: 2–3 mg/kg of body weight During rejection: maximum of 3 mg/kg of body weight dose not usually increased with rejection	Imuran dose is lowered when given concurrently with allopurinol as allopurinol delays metabolism of azathioprine	Azathioprine (Imuran) (IV or PO)
Sirolimus (Rapamune)	Structurally similar to tacrolimus and cyclosporine; however, it has a distinct immunosuppressant activity Inhibits response of B and T lymphocytes to cytokine stimulation by IL-2 and inhibits antibody production by B cells	Long half-life permits once-daily dosing Monitor trough blood levels	Hyperlipidemia, thrombocytopenia, leukopenia, headache, nausea, anorexia, dizziness	• May suppress hematopoietic recovery if used in patients who have recently undergone high-dose therapy. • Oral bioavailability is variable and is improved with high-fat meals. • Like tacrolimus and cyclosporine, sirolimus is metabolized through the cytochrome P-450 3A system. • Always administer doses in the evening to minimize impact of drowsiness on lifestyle and safety. • Teach patient to use caution when taking thalidomide with other drugs that can cause drowsiness or neuropathy. • Teach patient to rise slowly from a supine position to avoid lightheadedness. • Teach patient to report immediately signs or symptoms suggestive of peripheral neuropathy, including numbness or tingling in the hands or feet or the development of skin rash or skin lesion. These may require immediate cessation of the drug until the patient can be evaluated for the neuropathy or skin rash. • Teach patient to use protective measures (eg, sunscreens and protective clothing) against exposure to ultraviolet light or sunlight. • Control or manage constipation with a stool softener or mild laxative. • May impair wound healing
Methoxsalen (Oxsoralen)	When photoactivated by ultraviolet light, drug inhibits mitosis by binding covalently to pyrimidine bases in DNA	400 mcg/kg PO 1.5–2 h before exposure to ultraviolet light		• Patients who have received cytotoxic chemotherapy or radiation and who are taking methoxsoralen are at increased risk for skin cancers, and long-term use may increase the risk of skin cancer. • Toxicity increases with concurrent use of phenothiazines, thiazides, and sulfanilamides. • Severe burns may occur from sunlight or ultraviolet A exposure if dose or treatment frequency is exceeded. • Pretreatment eye examinations are indicated to evaluate for the presence of cataracts. Repeat eye examinations should be performed every 6 mo while patients are undergoing psoralen and ultraviolet A therapy.

These dosage ranges are general guidelines. Significant variations in dosages occur based on institutional practices, other drugs being used in combination, type of transplantation, and patient response to the drugs.

The cyclosporine preparations Neoral and Sandimmune are not bioequivalent and cannot be used interchangeably. Gengraf and Neoral are bioequivalent.

HTN, hypertension; WBC, white blood cell; ANC, absolute neutrophil count; GI, gastrointestinal; IFN, interferon; IL, interleukin; IV, intravenous; PO, oral; PVC, polyvinyl chloride; TNF, tumor necrosis factor.

▲ Complications of Transplantation

Complications after solid organ transplantation and HSCT are usually due to graft function, problems with immunosuppression, or the adverse effects of the transplantation preconditioning regimen. Other common complications after HSCT are GVHD and infection.

Organ Transplantation

Recipients of kidney, liver, heart, pancreas, and lung transplants are at risk for a number of complications, including acute and chronic organ rejection, infection, and bleeding.

Organ Rejection

The early recognition and management of organ rejection and its associated problems are key priorities for the critical care nurse. The transplanted organ represents a continuous source of HLA alloantigens capable of inducing a rejection response. This immunological response involves the recognition of HLA antigens of donor endothelial tissues cells by recipient lymphocytes or antibodies. The allograft continuously activates the immune response, resulting in lifelong overproduction of cytokines, constant cytotoxic activity, and sustained alteration in the graft vasculature. Transplantation of a vascular organ induces sensitization through direct stimulation of circulating host immune cells as they encounter donor antigens on allograft cell surfaces. Rejection leads to subsequent destruction of the antigen-bearing graft. The pathophysiological mechanisms of graft rejection are depicted in Figure 47-4.

Both donor and host factors contribute to the immune response of rejection. The major donor factor is the expression of antigens on the donor tissue and the presence of antigen-presenting cells within the transplanted graft. The major host factor is prior sensitization against ABO and HLA antigens expressed in the graft.[29]

Because the transplanted organ is not immunologically identical to the recipient, it acts as an antigen or foreign substance and triggers the immune system to reject it. Rejection can vary in degree from mild to severe and may be irreversible. Rejection may occur at any time, but the risk is highest in the first 3 months after transplantation. It is important to maintain therapeutic levels of immunosuppression and to provide patient and family education about the importance of taking all medications as instructed and the rationale for routine laboratory monitoring of the levels of immunosuppressive drugs. The earlier and more severe the rejection episode, the worse the prognosis for graft survival. Biopsy of the transplanted organ is usually needed to diagnose rejection definitively. Four types of rejection are defined: hyperacute, accelerated, acute, and chronic, although all types do not occur in all transplanted organs.

Hyperacute Rejection

Hyperacute rejection occurs in the operating room immediately after transplantation. It is a humoral immune response in which the recipient has preformed antibodies that immediately react against antigens of the donor organ. Vascular damage occurs, resulting in severe thrombosis and graft necrosis. In kidney and heart transplantation, hyperacute rejection always results in graft failure and the need for retransplantation. Fortunately, hyperacute rejection is uncommon and can usually be prevented by pretransplantation cross-matching.

Accelerated Rejection

Accelerated rejection is defined only in kidney transplantation, and it occurs within 1 week after transplantation. Clinically, the patient may have anuria, increased BUN and creatinine levels, and pain at the graft site. Accelerated rejection is due either to preformed antibodies against the donor antigens in the recipient's blood or to lymphocytes in the recipient that are already sensitized to some of the donor antigens. Like hyperacute rejection, accelerated rejection is seen infrequently because of improved tissue typing and cross-matching. It is treated aggressively with immunosuppressants and usually results in loss of the transplanted kidney.

Acute Rejection

Acute rejection occurs within the first 3 months after transplantation. This is the most common type of rejection, and most patients experience at least one episode. Acute rejection occurs when antigens on the donor organ trigger lymphocytes to mature into helper T cells. The helper T cells increase the production of cytotoxic killer T cells, which bind to the transplanted organ and damage it by secreting lysosomal enzymes and lymphokines. Acute rejection is also the type of rejection that responds best to immunosuppressive therapy.

Chronic Rejection

The pathophysiology of chronic rejection is not completely understood. Most likely it is a combination of a cell-mediated response and a response to circulating antibodies. Second in frequency to acute rejection, chronic rejection usually occurs from 3 months to years after transplantation and is accompanied by deteriorating organ function.

KIDNEY. Acute rejection occurs after the first postoperative week. It is the most frequently seen form of rejection and the type that responds best to therapy. Changes in laboratory values are the earliest and most reliable indicators that graft function is deteriorating. Clinical manifestations of rejection are more subtle and may not be seen. The patient may experience any, all, or none of the following laboratory findings during an acute rejection episode:

- Increased serum creatinine, BUN, serum β_2-microglobulin levels
- Decreased creatinine clearance
- Decreased urine creatinine level
- Possibly decreased urine sodium level
- Decreased blood flow identified by renal scan

Clinical manifestations of rejection include:

- Decreased urine output
- Weight gain

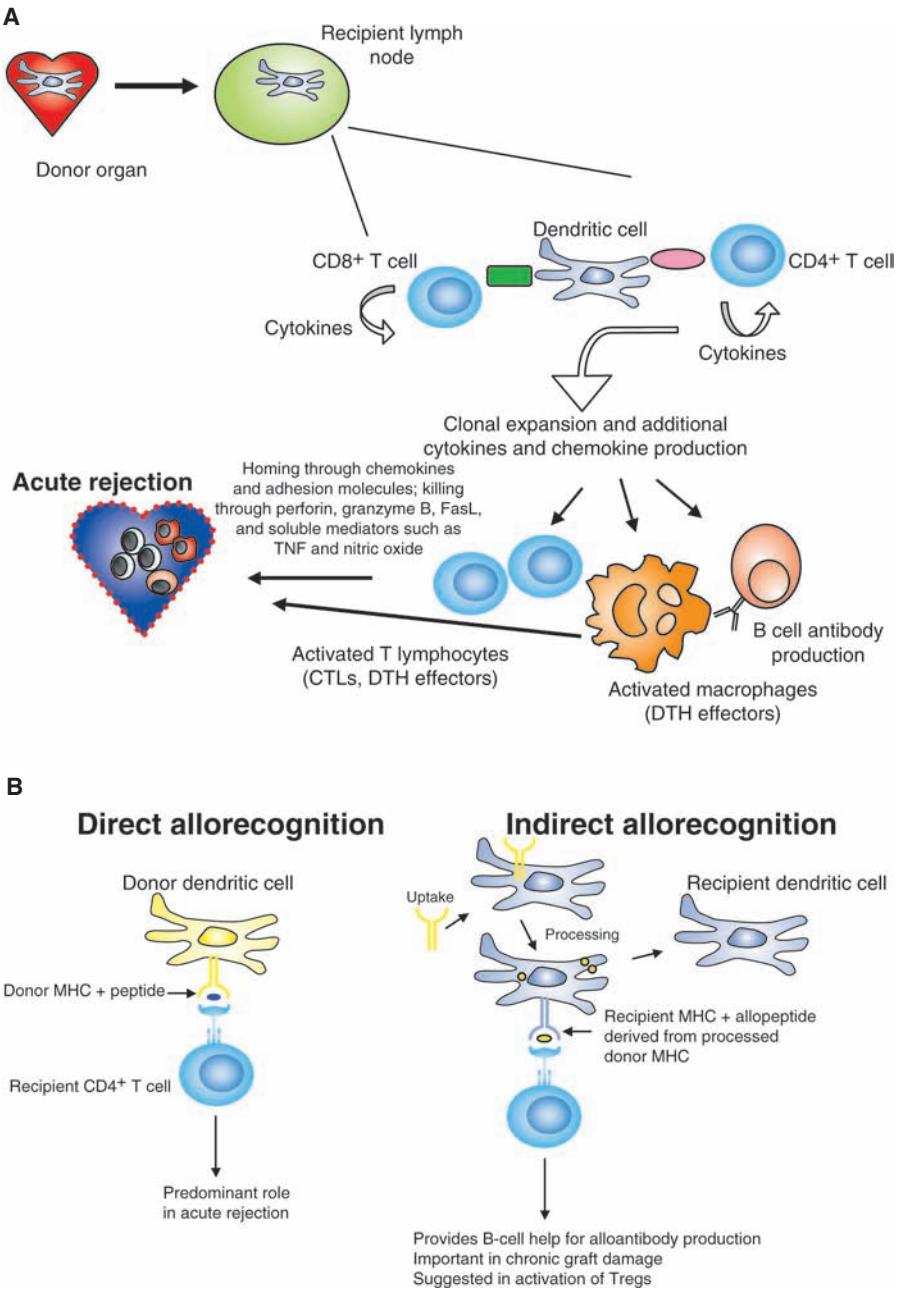


FIGURE 47-4 ▲ Mechanisms of graft rejection. **A:** Within 24 to 48 hours after engraftment, dendritic cells that normally reside within the donor organ migrate to regional recipient lymphoid tissue. In the lymph node, they stimulate alloreactive $CD4^+$ and $CD8^+$ T cells. Activated T cells, particularly $CD4^+$ cells, produce cytokines (eg, interleukin-2, interleukin-4, interferon-γ), and both populations respond by proliferating and differentiating. Activated T lymphocytes can cause graft destruction by direct lysis (cytotoxic T lymphocytes, or CTLs) or by local production of cytokines, a delayed-type hypersensitivity (DTH) reaction. Cytokines also promote macrophage and eosinophil activation and recruitment, and these cells can also secrete soluble inflammatory mediators that kill their targets. Last, activated T cells provide help for alloantibody production by B cells. **B:** There are two types of allorecognition. Direct allorecognition occurs when T cells recognize intact foreign major histocompatibility complex (MHC) molecules (as depicted in part *A*). This is thought to be the dominant initiator of acute graft rejection. T cells may also recognize peptide fragments derived from processing of donor antigens presented on self-MHC molecules. This is termed *indirect allorecognition*, and it is believed to be important in chronic graft dysfunction, in part perhaps because of its role in providing T-cell help for alloantibody production by B cells. Indirect allorecognition is also implicated in activating regulatory T cells, which may act to limit graft damage and promote tolerance. (From Lechler RI, Sykes M, Thomson AW, et al: Organ transplantation: How much of the promise has been realized? Nat Med 11(6):605–613, 2005.)

- Edema
- Temperature at least 100°F (37.8°C)
- Tenderness over the graft site, with possible swelling of the kidney
- General malaise
- Increased blood pressure

Chronic rejection is the result of repeated episodes of acute rejection in which the vessels become infarcted owing to the vasculitis, and the renal tissue becomes scarred. This gradually leads to deteriorating kidney function. The symptoms are similar to those of acute rejection except for fever and graft enlargement. Laboratory findings are similar to those of acute rejection but also include signs of chronic renal failure, such as a declining hematocrit and calcium–phosphorus imbalance. The rate of deterioration can vary from months to years. A transplant nephrectomy is not usually required unless the kidney becomes necrotic and life threatening.

LIVER. Acute rejection in liver transplantation is suspected when liver function tests, specifically PT/INR (most sensitive), aminotransferases, alkaline phosphatase, and total bilirubin, are increased. Clinical signs, such as decreased bile production and perigraft tenderness, may or may not occur.[20] Chronic rejection is believed to be due to multiple acute rejection episodes or a positive cross-match. A definitive diagnosis is made when a biopsy shows portal and bile duct inflammation and inflammatory cells, such as T lymphocytes.[20]

HEART. Although acute rejection is often asymptomatic, subtle signs and symptoms may include decreased cardiac output, atrial flutter or fibrillation, elevated WBC count, and low-grade fever. Endomyocardial biopsy is performed weekly for the first month and then less frequently to diagnose rejection. Acute rejection is a major cause of death in the first year after transplantation. Chronic rejection is the leading cause of death after the first year of cardiac transplantation. The prevalence within 5 years of transplantation is at least 60%. This cell-mediated rejection causes progressive myocardial fibrosis, leading to heart dysfunction. The lesions in allographic vasculopathy are concentric, not focal, unlike in typical atherosclerosis, and allographic vasculopathy can often be missed on standard cardiac catheterization. Angina cannot be used as a warning sign for coronary artery disease because the heart is denervated. Instead, decreased exercise tolerance during stress testing or intravascular ultrasonography is used for diagnosis.[30]

PANCREAS. Rejection is a major cause of graft loss in pancreas transplantation. This may be attributed to the difficulty in diagnosing rejection. Elevated blood glucose levels are a late sign and may occur too late to initiate successful treatment. When the bladder is used for exocrine drainage, urinary amylase levels reflect rejection before hyperglycemia becomes obvious. In combined kidney–pancreas transplantation, an elevated serum creatinine level may indicate rejection, although rejection can occur in one organ and not the other. Some experts state that chronic pancreas rejection may not occur in combined kidney–pancreas transplantation. The best pancreas transplant survival rate occurs when the procedure is performed simultaneously with kidney transplantation[10] (see Table 47-1, p. 1052). Needle biopsy during cystoscopy is used for definitive diagnosis.

LUNG. The signs and symptoms of lung transplant rejection are difficult to distinguish from pulmonary infection. Decreased lung function (ie, forced expiratory volume), dyspnea, cough, decreased breath sounds, fever, and tachypnea may occur in both rejection and infection. Immediately after surgery, rejection may also be confused with volume overload, reperfusion injury, or ischemic injury secondary to preservation. Chest radiographs showing interstitial and perihilar edema may be signs of rejection and signal the need for biopsy. Even so, biopsies must be carefully interpreted to rule out infectious complications, such as infection with CMV or *Pneumocystis carinii*, which can have histological findings similar to those of acute rejection.

Chronic rejection is known as obliterative bronchiolitis and occurs in approximately 15% to 25% of lung transplant recipients. Acute rejection and infection are believed to play a role in obliterative bronchiolitis. Acute rejection remains common, with 36% of lung transplant patients experiencing at least one episode of acute rejection in the first year after transplant.[30]

Infection

Infection is the most common posttransplantation complication. Alterations in the integrity of mucosal barriers and severe neutropenia from the pretransplantation conditioning regimen produce an environment conducive to serious bacterial and fungal infection.

The causative agents are often from the patient's own flora, particularly from the gastrointestinal tract and integumentary system. Pathogens may be bacteria, fungi, viruses, and even protozoa. The latter three groups of organisms are referred to as opportunistic pathogens. Normally harmless and found in humans and in the environment, they pose serious threats to patients with compromised immune systems. They take advantage of the decreased host defenses—hence the term "opportunistic." Examples of opportunistic infections include herpes simplex and herpes zoster viruses, CMV, *Candida albicans*, *P. carinii*, *Aspergillus* species, and *Cryptococcus* species.

All transplant recipients are at risk for bacterial infections from intravascular lines and urinary drainage catheters, but organ transplant recipients can also acquire postoperative wound and lung infections. Usually broad-spectrum antibiotics are given prophylactically for 48 hours after organ transplantation or until invasive lines and drains are removed. HSCT recipients receive antibiotics prophylactically for months after transplantation. Table 47-8 lists infections caused by bacteria and other organisms that commonly occur after allogeneic HSCT.

Recipients of organ transplants are at high risk for infection during the first 3 months after transplantation because they receive high dosages of immunosuppressants. Infections in the post–stem cell transplantation period usually follow a predictable pattern based on the recovery of the immune system. Therefore, recipients of HSCT are at high risk for infection during the first month, which is the pre-engraftment phase, because of neutropenia. HSCT recipients may receive colony-stimulating factors to reduce their risk for infection by accelerating WBC recovery. They remain at high risk for infection if they are receiving immunosuppressive medications to prevent or treat GVHD.

Table 47-8 Infections After Allogeneic Hematopoietic Stem Cell Transplantation

Period of Neutropenia (Days 0–30)	Period of Acute GVHD (Days 30–100)	Period of Chronic GVHD (Days 100+)
Gram-negative bacteria	Gram-negative bacteria	Encapsulated bacteria
Gram-positive bacteria	Gram-positive bacteria	Varicella-zoster virus
Herpes simplex	Cytomegalovirus (CMV)	CMV
Candida species	Polyomavirus (BK virus)	*P. carinii*
Aspergillus species	Adenovirus	*Aspergillus* species
	Varicella-zoster virus	
	Candida species	
	Aspergillus species	
	Pneumocystis carinii	
	Toxoplasma gondii	

Based on information from: Wingard JR, Hsu J, Hiemenz JW: Hematopoietic stem cell transplantation: An overview of infection risks and epidemiology. Infect Dis Clin North Am 24(2), 257–272, 2010.

During the first month, the predominant fungal infections in recipients of HSCT are *Aspergillus* species and *Candida* species, for which amphotericin B or fluconazole may be used prophylactically. The most frequent viral infection is herpes simplex, and 80% of the patients who were seropositive before transplantation experience a reactivation of herpes simplex unless they receive acyclovir prophylactically.[31]

After the first month, the most common infection in all transplant recipients is CMV. The consequences of CMV infection include enteritis, retinitis, pneumonitis, and marrow suppression. To prevent CMV infection, patients who are CMV seronegative should receive only CMV-negative blood products. Many centers require that all blood transfusions be filtered. Current recommendations are to treat solid organ transplant recipients, who are CMV negative and who receive a CMV-positive organ, prophylatically with oral valacyclovir (Valtrex) for 3 to 6 months after transplantation.[32] Ganciclovir may be appropriate for patients who are CMV seropositive and who are receiving increased immunosuppression for an episode of acute organ rejection. Close monitoring of HSCT recipients for CMV reactivation, as demonstrated by a rising level of CMV antigen by polymerase chain reaction in the blood, is imperative, with early preemption. Prevention is important in heart transplant recipients because there is a connection between CMV and coronary artery disease.[33] CMV may affect many organ systems; therefore, signs and symptoms of hepatitis, retinitis, enteritis, pneumonitis, fever, chills, and malaise may occur.[34]

A small number of HSCT recipients contract severe and potentially fatal infections 3 months or more after transplantation, during the late recovery phase, because of cellular and humoral immune deficiencies. The most frequent causes of these infections are *Pneumococcus* species, *Staphylococcus aureus*, *Candida* species, and varicella-zoster virus.

If infection develops in immunosuppressed patients, the usual signs and symptoms may be absent. In these patients, even a small increase in temperature (99°F [37.2°C]) may be significant. Daily monitoring of the WBC count is necessary. After organ transplantation, the leukocyte count is usually slightly elevated because of surgery and steroid treatment. However, infection may be present if the elevation persists, a rapid elevation occurs after a decline, or there is an increase in the percentage of immature WBCs (bands) noted on the differential.

It is essential to prevent infection in transplant recipients, who are immunosuppressed and may be neutropenic. Important nursing responsibilities include maintaining protective environments, practicing consistent and thorough provider handwashing and good oral and skin hygiene, monitoring vital signs frequently, and performing head-to-toe assessments. In some centers, additional protective measures include protective isolation systems, air filtration, gut and skin decontamination, and low-microbial diets. The benefit of these interventions has been debated, and their application is institution or protocol specific.[35,36]

In combined kidney–pancreas transplantation, immunosuppressive drugs may be discontinued in the presence of a severe infection to mobilize the patient's immune system. Consequently, the graft may be lost to save the patient. In heart, lung, and liver transplantation, immunosuppression may be decreased but must be continued.

Bleeding

Bleeding, oozing from the surface of the transplanted organ, or the presence of hematoma or lymphocele may occur after surgery. The heart transplant recipient is at risk for bleeding because the pericardial sac has stretched to accommodate an enlarged heart. When a smaller, healthy heart is implanted, the larger pericardial sac becomes a reservoir that can conceal postoperative bleeding. This may result in cardiac tamponade. Long-term coagulation therapy and liver congestion from pretransplantation heart failure also increase the risk for bleeding.

After liver transplantation, bleeding may occur as a result of coagulopathy because of liver dysfunction or from small vessels that continue to bleed after surgery. When the bladder drainage technique is used for exocrine drainage in pancreas transplantation, patients may have postoperative hematuria if the transplanted duodenal segment becomes ulcerated or if cystitis develops. Electrocautery using cystoscopy may be required for severe bleeding.

Gastrointestinal Complications Related to Steroid Therapy

Chronic steroid therapy increases the risk for peptic ulceration and erosive gastritis because it increases the secretion of hydrochloric acid and pepsinogen. Massive gastrointestinal bleeding may occur not only from steroid therapy but also from stress and decreased tissue viability caused by long-term protein restriction. For these reasons, patients usually are given histamine-2 (H_2) receptor antagonists (eg, nizatidine or ranitidine) or proton pump inhibitors (eg, omeprazole). The degree of renal function, together with concurrent medications, dictates which class of agent is selected for gastric cytoprotection.

Other serious gastrointestinal complications include acute pancreatitis, diverticulitis, *Candida* infection, esophagitis, obstruction from bowel adhesions, and ulcerative colitis. Infection becomes an added risk if the patient has an intestinal perforation. Ischemic bowel disease has been observed in the early posttransplantation period as a result of dehydration or ischemia resulting from low cardiac output.

More than one complication may occur simultaneously. In addition, signs and symptoms of gastrointestinal bleeding or perforation may be obscured by the anti-inflammatory effects of steroids. Therefore, complaints and changes in the patient's progress require thorough and prompt assessment.

The gastrointestinal tract of the patients who have had HSCT may also suffer the effects of the total-body irradiation and chemotherapy used in the preparatory regimen. Symptoms may include mucositis, nausea, vomiting, diarrhea, cramping, dyspepsia, anorexia, taste changes, and xerostomia.

Hematopoietic Stem Cell Transplantation

Graft Failure

Graft failure is usually defined precisely by institutional protocols. However, all definitions include a complete absence of engraftment or a seemingly initial hematopoiesis after transplantation, with later decreasing blood cell counts and an absence of hematopoiesis.[37] The clinical features of graft failure include neutropenia, anemia, and thrombocytopenia occurring beyond the initial period expected as a result of high-dose chemotherapy or chemoradiation therapy.

The overall incidence of graft failure is less than 5%, and it occurs most often in patients with aplastic anemia or those receiving unrelated donor transplants. The etiology is multifactorial. An important component in the evaluation is differentiating graft failure from disease recurrence or drug-induced myelosuppression. Table 47-9 describes graft failure in terms of its risk factors, possible causes, and therapeutic and preventive measures.

The HSCT recipient may be more susceptible to the myelosuppressive effects of various drugs after the transplantation. Drugs with a potential to cause myelosuppression should be used very cautiously, if at all, to minimize the risk for

Table 47-9 Graft Failure After Hematopoietic Stem Cell Transplantation: Risk Factors, Causes, and Preventive Measures

	Risk Factors	Causes	Preventive/Supportive Measures
Autologous stem cell transplants	• Patients with acute myelocytic leukemia, patients extensively pretreated • Low cell dose • Purged marrow • Marrow-suppressive drugs • Viral infection	• Defective marrow microenvironment with stromal cell damage • Collection of damaged stem cells due to extensive previous treatment • Drug-induced myelosuppression • Viral effects on stroma of bone marrow	• Harvest autologous stem cells early, before multiple cycles of potentially stem cell–toxic regimens have been given • Maximize number of infused cells (minimum number of autologous peripheral blood stem cells that results in consistent engraftment is at least 1×10^6 $CD34^+$ cells/kg of body weight, below which engraftment may be incomplete or there may be failure of engraftment) • Avoid myelosuppressive drugs after transplantation • Adjust doses of medications for renal dysfunction • Treat/prevent viral infections • Keep unmanipulated cells as backup in case or purged or manipulated grafts • Ensure that there is no folate or vitamin B_{12} deficiency • Administer G-CSF and rEPO as needed
Allogeneic stem cell transplants	• Diseases associated with a defective marrow microenvironment, including aplastic anemia and myelofibrosis • Stem cell source from HLA-mismatched, unrelated, or cord blood donor • Pretransplant transfusions, especially from a related donor • T-cell depletion, low cell dose, purging • Patients whose clinical condition precludes a sufficiently intensive conditioning regimen • Inadequate posttransplantation immunosuppression • Marrow-suppressive drugs • Viral infection, including infection with CMV	• Defective marrow microenvironment with stromal cell damage • Histocompatibility barriers • Allosensitization by transfusions • Damaged or inadequate number of stem cells infused • Persistence of host hematopoiesis • Persistence of immunocompetent host lymphocytes • GVHD-associated damage to bone marrow microenvironment • Drug-induced myelosuppression • Viral effects on stroma of bone marrow	• Avoid pretransplantation transfusions, especially from relatives • Select histocompatible donors • Ensure that the conditioning regimen is adequately immunosuppressive • Provide sufficient stem cell dose (minimum number of allogeneic peripheral blood stem cells that results in consistent engraftment is at least 2×10^6 $CD34^+$ cells/kg of body weight, below which engraftment may be incomplete or there may be failure of engraftment) • Use posttransplantation immunosuppression with cyclosporine, tacrolimus, or methotrexate • Avoid all myelosuppressive drugs after transplantation • Adjust doses of medications for renal dysfunction • Treat/prevent viral infections • Ensure that there is no folate or vitamin B_{12} deficiency • Administer G-CSF, rEPO as needed • Consider cryopreserving autologous peripheral blood stem cells preallograft for possible use in the event of graft failure and overwhelming clinical problems such as hemorrhage or life-threatening infection

rEPO, recombinant erythropoietin; G-CSF, granulocyte colony-stimulating factor.

Based on information from references Rees C, Beale P, Judson I: Theoretical aspects of dose intensity and dose scheduling. In Barrett J, Treleaven J (eds): The Clinical Practice of Stem Cell Transplantation. Oxford, UK: Isis Medical Media, 1998, pp 17–29; Potter M: Graft failure. In Treleaven J, Barrett AJ (eds): Hematopoietic Stem Cell Transplantation in Clinical Practice. Edinburgh, UK: Elsevier Limited, 2009, pp 381–385; Lowe T, Bhatia S, Somlo G: Second malignancies after allogeneic hematopoietic cell transplantation. Biol Blood Marrow Transplant 13(10):1121–1134, 2007.

drug-induced graft failure. In patients with delayed engraftment, it is prudent to review all medications and consider eliminating those that are not absolutely essential.

Veno-Occlusive Disease of the Liver

Veno-occlusive disease of the liver (also called sinusoidal obstructive syndrome) is a potentially fatal liver disease that occurs in 15% to 20% of recipients of HSCT. Veno-occlusive disease is a complication of the conditioning regimen and usually develops within 2 weeks of transplantation. The risk for veno-occlusive disease is increased in patients who have received total-body irradiation. Veno-occlusive disease may be severe; some studies report an incidence of 25% and a mortality rate close to 50%.[38–40]

Veno-occlusive disease occurs when fibrous material accumulates, resulting in obstruction of small venules in the liver. Subsequently, portal hypertension, acute liver congestion, and destruction of liver cells develop. Liver disease ranges from mild to severe, and severe liver failure may occur. In addition, veno-occlusive disease affects the kidneys; there is a decrease in renal blood flow, causing further water and sodium retention. Mild veno-occlusive disease persists until liver tissue heals and resumes normal function, usually 10 to 14 days after onset. Severe disease may result in multisystem failure.

Clinical manifestations of veno-occlusive disease usually begin during the first 3 weeks after transplantation and are characterized by hyperbilirubinemia, rapid weight gain, ascites, right upper quadrant pain, hepatomegaly, splenomegaly, and jaundice.[40] Treatment is supportive and focuses on maintaining intravascular volume and renal perfusion while minimizing fluid accumulation.[41] This may require central venous pressure monitoring and mechanical ventilation, as well as pulmonary artery pressure monitoring if excess fluids accumulate in the lungs. Sodium restriction is warranted, and spironolactone administration is necessary to decrease extravascular accumulation. Other supportive strategies may include renal-dose dopamine infusion, avoidance of diuretics that deplete intravascular volume, and chest physiotherapy to avoid pulmonary atelectasis.[40]

Strategies for preventing veno-occlusive disease of the liver are currently undergoing investigation. These include anticoagulation with heparin; fibrinolytics, such as tissue plasminogen activator or antithrombin III concentrates, defibrotide, prostaglandin E; and ursodeoxycholic acid (Actigall).[38–40,42,43]

Pulmonary Complications

Pulmonary complications develop in 30% to 60% of patients after HSCT.[44] Pulmonary complications may result from (1) infection, pulmonary edema, aspiration pneumonia, acute respiratory distress syndrome, and septic shock and (2) lung damage from total-body irradiation or pulmonary toxic chemotherapy agents.[41,44–48] Table 47-10 lists the pulmonary complications of HSCT.

Table 47-10 Pulmonary Complications of Hematopoietic Stem Cell Transplantation

Time Line	Complication
Acute (before day 30)	Pulmonary edema (secondary to fluid overload, cardiac dysfunction, or allergic reaction to medications/therapy) Oropharyngeal mucositis Aspiration pneumonia Pulmonary hemorrhage/diffuse alveolar hemorrhage Bacterial or fungal pneumonia Atelectasis Pleural effusion Recall radiation pneumonitis Allergic bronchospasm Transfusion-associated lung injury ARDS and septic shock
Early (before day 100)	Idiopathic interstitial pneumonitis Pulmonary embolism Viral pneumonia (CMV, herpes simplex virus, varicella-zoster virus, respiratory syncytial virus, adenovirus, parainfluenza, influenza) Protozoal pneumonia (*Pneumocystis carinii* pneumonia) Fungal pneumonia Bacterial pneumonia Transfusion-associated lung injury ARDS and septic shock
Late (after day 100)	Idiopathic interstitial pneumonitis Bacterial, fungal, or viral pneumonia Bronchiolitis obliterans/GVHD of lung

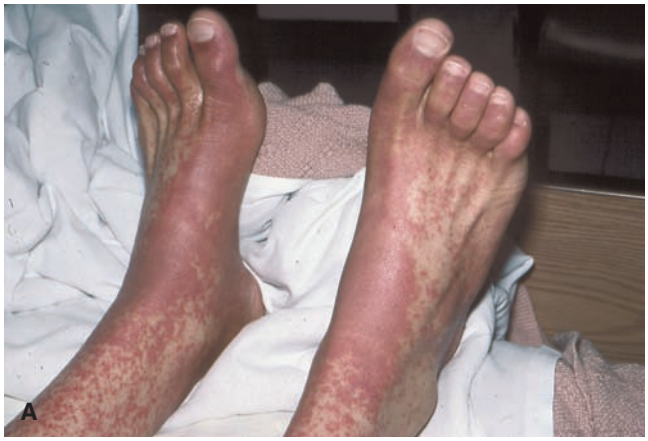
ARDS, acute respiratory distress syndrome.

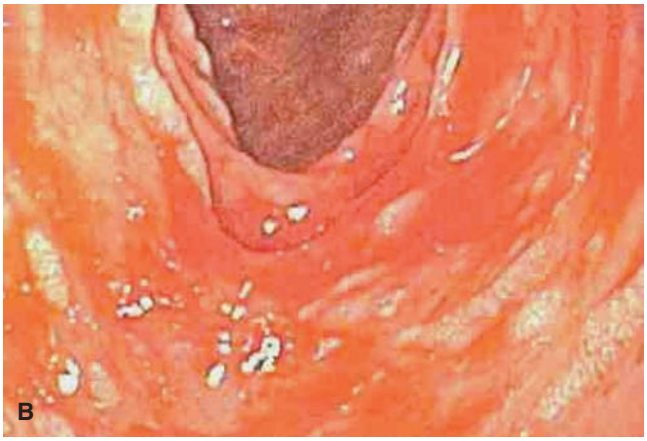
Graft-Versus-Host Disease

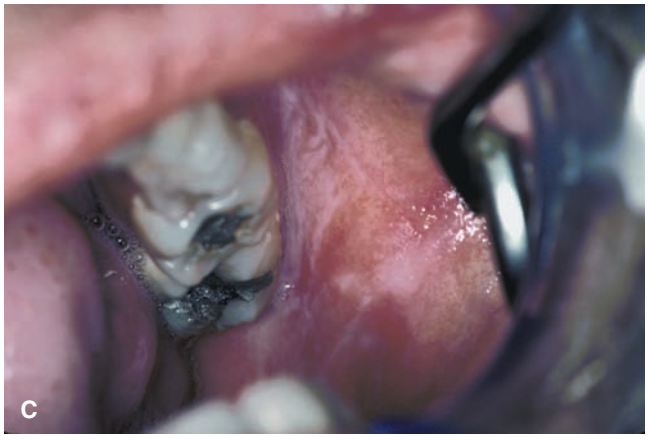
GVHD, which is unique to allogeneic HSCT, results when the infused donor stem cells (graft) recognize the recipient (host) as foreign tissue. The graft then mounts an immunological response attacking the host tissues, resulting in a T-cell–mediated reaction in the skin (rash), gastrointestinal tract (enteritis), and liver (elevated liver function test results). Figure 47-5 shows examples of the skin and gastrointestinal tract manifestations that may occur in acute GVHD.

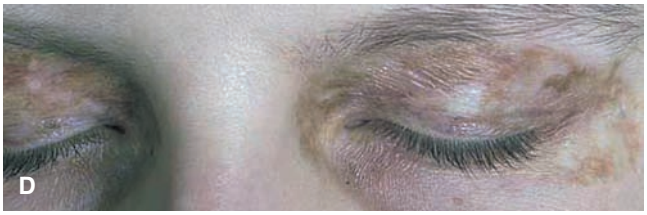
The incidence of GVHD is 30% to 60% in cases involving histocompatible, sibling-matched allografts, with more GVHD occurring when there is greater HLA mismatch between the donor and recipient.[49,50] The mortality rate due directly or indirectly to GVHD may reach 50%.[50] Risk factors other than histoincompatibility include sex mismatching; donor parity; older age; posttransplantation infectious complications, especially viral infections; the use of donor lymphocyte infusions after transplantation; and the type of GVHD prophylaxis used.[50]

GVHD is a serious complication, but it also has a beneficial effect in controlling the patient's malignancy in that immunocompetent donor cells are able to recognize the patient's malignant cells as foreign and eliminate them. This effect was originally identified in leukemia patients and was termed *graft-versus-leukemia effect*. Leukemia relapse was seen less often in patients with GVHD than in those without GVHD. The absence of GVHD in autologous transplant recipients is suspected to play a role in the higher disease relapse rates these patients experience. Recently, researchers are applying the graft-versus-malignancy effect to prevent disease recurrence after stem cell transplantation by the infusion of donor









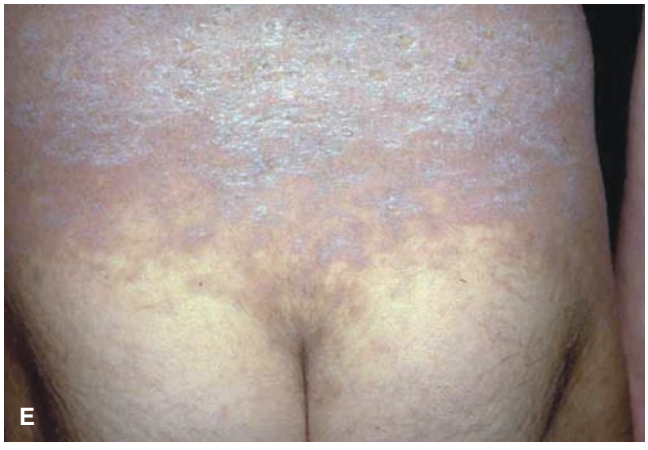


FIGURE 47-5 ▲ Acute and chronic graft-versus-host disease (GVHD). **A:** GVHD of the skin is characterized by fine, discrete or confluent, erythematous macules and papules. Lesions may be pruritic or slightly tender with palpation. Earliest skin findings are usually seen on the face, palms and soles, and upper trunk. **B:** GVHD of the gastrointestinal tract. Images obtained during endoscopy demonstrate tissue edema, extensive erythema, and mucosal ulcerations. **C:** Oral lichen planus changes in a patient with chronic GVHD more than 130 days after allogeneic peripheral blood stem cell transplantation. Note the confluent, smooth, white papules that create a lacy pattern on the buccal mucosa. **D:** Chronic GVHD of the skin with irregularly shaped, deeply hyperpigmented macular lesions. Note the atrophy of the dermal and subcutaneous tissues with paper-thin skin giving an easily wrinkled or shiny appearance. The term *poikiloderma* is used to describe the classic features of patchy hypopigmentation and hyperpigmentation, dermal atrophy, and telangiectasias (small-diameter linear blood vessels seen on the skins surface.) **E:** Lichenoid chronic GVHD of the skin of the lumbar region with flat-topped, violaceous papules; the surface is shiny and has a lacy white pattern. The eruption is confluent in some areas, and hypertrophic plaques have developed. Postinflammatory hyperpigmentation may develop. (**B**, photo courtesy of Bruce Greenwald, MD, University of Maryland Medical System, Baltimore, MD; **C**, photo courtesy of Jane Fall-Dickson, RN, PhD, AOCN, National Institutes of Health, Bethesda, MD; **D** and **E**, photos courtesy of T. L. Diepgen and G. Yihune, Dermatology Online Atlas [http://www.dermis.net/doia].)

lymphocytes. This approach is called donor lymphocyte infusion. Research is also under way to devise strategies to induce GVHD in autologous recipients of HSCT.[51]

Acute Graft-Versus-Host Disease

Acute GVHD may occur as early as 7 to 21 days after transplantation but peaks 30 to 40 days after transplantation. Acute GVHD targets the skin, liver, and gastrointestinal system. Skin reactions, which often occur first, include an itchy maculopapular, erythematous rash on the palms, soles, ears, face, and trunk. This may resolve or progress to generalized erythroderma and desquamation. Gastrointestinal symptoms include nausea, vomiting, anorexia, abdominal cramping, and large-volume diarrhea that is green and watery. Stool may be guaiac-positive as a result of intestinal mucosa sloughing. An enlarged liver, right upper quadrant pain, jaundice, and elevated bilirubin and alkaline phosphatase levels may occur. The severity and extent of acute GVHD are evaluated using a grading system (Table 47-11).

Table 47-11 Staging and Grading System for Acute Graft-Versus-Host Disease

Clinical Staging of Individual Organ Manifestations		
Organ	**Stage***	**Description**
Skin†	+1	Maculopapular eruption over <25% of body area
	+2	Maculopapular eruption over 25%–50% of body area
	+3	Generalized erythroderma
	+4	Generalized erythroderma with bullous formation and often with desquamation
Liver	+1	Bilirubin 2.0–3.0 mg/dL
	+2	Bilirubin 3.1–6.0 mg/dL
	+3	Bilirubin 6.1–15 mg/L
	+4	Bilirubin >15 mg/dL
Gut	+1	Diarrhea <500 mL/d
	+2	Diarrhea 500–999 mL/d, or persistent nausea with histological evidence of GVHD in the stomach or duodenum
	+3	Diarrhea 1,500 or more mL/d
	+4	Severe abdominal pain, with or without ileus

Overall Grade			
Grade	**Skin‡**	**Liver**	**Gut**
I	+1 to +2	0	0
II	+1 to +3	+1 and/or	+1
III	+2 to +3	+2 to +3 and/or	+2 to +3
IV	+2 to +4	+2 to +4 and/or	+2 to +4

*Criteria for staging minimal degree of organ involvement required to confer that stage.

†Use rule of nines or burn chart to determine extent of rash.

‡If no skin disease is present, the overall grade is the highest single organ stage.

Chronic Graft-Versus-Host Disease

Chronic GVHD usually occurs in patients who have had acute GVHD, although it can occur in the absence of acute GVHD. Chronic GVHD typically occurs between 100 and 400 days after transplantation. Among patients who survived 150 days after allogeneic stem cell transplantation, researchers observed chronic GVHD in 33% to 49% of HLA-identical related transplants and in 64% of matched unrelated donor transplants.[52]

Risk factors for chronic GVHD include previous acute GVHD, older recipient age, and sex mismatching (female donor and male recipient).[53] The incidence of chronic GVHD may also be higher in recipients of peripheral blood stem cells than in recipients of bone marrow–derived stem cells.[52] Another significant risk factor for the development of chronic GVHD is a continuing need for corticosteroids for control of GVHD by day 100 after transplantation.[54]

Clinical manifestations of chronic GVHD, which may be limited or extensive, are present in the skin, liver, eyes, oral cavity, lungs, gastrointestinal system, neuromuscular system, and a variety of other body systems. Although the onset of chronic GVHD typically occurs much later than acute GVHD, generally 100 to 400 days after transplantation, there is a growing recognition that acute and chronic GVHD are best differentiated by their features, not their onset. Features of acute GVHD, including erythematous skin rash, liver function test abnormalities, nausea, vomiting, diarrhea, and abdominal pain, may occur after donor lymphocyte infusion. Similarly, pigmentary skin changes, sclerotic skin features, bronchiolitis obliterans, keratoconjunctivitis sicca, and oral dryness, all considered to be characteristics of chronic GVHD, may be observed in patients before day 100 after transplantation. A new paradigm for identifying acute and chronic GVHD and for diagnosing and staging chronic GVHD is evolving[55]; classification is based on the signs and symptoms of acute or chronic GVHD rather than on the number of days after transplantation. In addition, contemporary classification systems now identify an overlap syndrome in which diagnostic or distinctive features of chronic GVHD and acute GVHD appear together. Table 47-12 summarizes the clinical features, screening and evaluation, and interventions recommended for patients with chronic GVHD.[52,56–67]

Treatment and Prophylaxis of Graft-Versus-Host Disease

The first and most important way to limit GVHD is to find an HLA-matched donor. Despite such optimal matching of donor and recipient, further strategies to limit GVHD must be taken. The two major approaches to the prophylaxis of GVHD after HSCT are T-cell depletion of the graft and pharmacological therapy to prevent and treat GVHD.

T cells play a major role in the recognition of self from nonself proteins, and decreasing the number of T cells in the

Table 47-12 Chronic Graft-Versus-Host Disease: Clinical Manifestations, Screening/ Evaluation, and Interventions

Organ/System	Clinical Manifestations	Screening Studies/Evaluation	Interventions
Dermal	Dyspigmentation, xerosis (dryness), erythema, hyperkeratosis, pruritus, sclerosis, lichenification, onychodystrophy (nail ridging/ nail loss), alopecia	Clinical examination Skin biopsy—3-mm punch biopsy from forearm and posterior iliac crest areas	• Systemic Immunosuppressive therapy • PUVA; extracorporeal photopheresis • Topical tacrolimus ointment (Protopic) • Topical treatment with steroid creams, moisturizers/ emollient, antibacterial ointments to prevent suprainfection; aggressive lubrication of the skin • Because the sweat glands are affected, avoid overheating because heat prostration and heat stroke can occur • Avoid sunlight exposure, use sunblock lotion and wear a large hat that shades the face when outdoors
Oral	Lichen planus, xerostomia, ulceration	Oral biopsy from inner lower lip or buccal mucosa	• Steroid mouth rinses, oral PUVA, pilocarpine for xerostomia, fluoride gels/rinses to decrease caries • Careful attention to oral hygiene; regular dental evaluations
Ocular	Keratitis, sicca syndrome	Schirmer's test, ophthalmic evaluation	• Regular ophthalmological follow-up • Preservative-free tears • Temporary or permanent lacrimal duct occlusion • Fluid-ventilated, gas-permeable scleral lens prosthesis • Consider trial of cyclosporine ophthalmic emulsion (Restasis)
Hepatic	Jaundice, abdominal pain	Liver function tests (alanine and aspartate aminotransferases, alkaline phosphatase, bilirubin)	• Consider bile acid displacement therapy with ursodeoxycholic acid (Actigall) 300 mg PO three times a day
Pulmonary	Obstructive/restrictive pulmonary disease, shortness of breath, cough, dyspnea, wheezing, fatigue, hypoxia, pleural effusion	Pulmonary function studies, peak flow, ABG, chest CT	• Prevent and treat pulmonary infections, including *Pneumocystis carinii* and *Streptococcus pneumoniae* • Aggressively investigate changes in pulmonary function because these may represent chronic GVHD of lung/bronchiolitis obliterans • Encourage smoking cessation
Gastrointestinal	Nausea, odynophagia, dysphagia, anorexia, early satiety, malabsorption, diarrhea, weight loss	Stool cultures, esophagogastroduodenoscopy, colonoscopy, nutritional assessment, fecal fat excretion studies, serum amylase, D-xylose absorption test, CT of the abdomen	• Referral to gastroenterologist; consultation with nutritionist and nutrition support • Consider empirical trial of pancreatic enzyme supplementation • Aggressive management of gastrointestinal symptoms such as nausea and vomiting • Consider the use of cholestyramine (Questran) in the management of diarrhea • Consider a trial of oral beclomethasone, budesonide, or both
Nutritional	Protein and calorie deficiency, malabsorption, dehydration, weight loss, muscle wasting	Weight, fat store measurement, prealbumin	• Nutritional monitoring, supplementation, symptom specific interventions • Trial of megestrol (Megace) or other approaches to appetite stimulation (eg, mirtazapine [Remeron] or similar antidepressants; dronabinol [Marinol])
Genitourinary	Vaginal sicca, vaginal atrophy, stenosis, dyspareunia, vulvodynia	Pelvic examination; tissue biopsy	• Consider trial of mucosal application of corticosteroid ointment, cyclosporine ointment, or tacrolimus ointment • Vaginal dilators • Vaginal lubricants • Sexual counseling
Immunological	Hypogammaglobulinemia, autoimmune syndromes, development of autoantibodies	Quantitative immunoglobulin levels, CD4/CD8 lymphocyte subsets	• IV immunoglobulin supplementation as indicated, and prophylactic antimicrobials (rotating antibiotics for recurrent sinopulmonary infections, PCP prophylaxis, topical antifungals) • Screening for CMV and other opportunistic infections with frequent surveillance cultures and antigen detection • Consider vaccination against influenza and pneumococcus
Musculoskeletal	Myositis fascitis, contractures, debility, muscle cramps/ aches, carpal spasm	MRI measured range of motion, walk time, grip strength, creatine kinase, aldolase; performance status, formal quality-of-life evaluation (eg, FACT-BMT), formal evaluation of rehabilitation needs (CARES)	• Physical therapy for stretching and endurance • Correct electrolyte imbalances • Consider clonazepam, magnesium supplementation treatment for muscle cramping or myalgias

MRI, magnetic resonance imaging; CT, computed tomography; PCP, *Pneumocystis carinii* pneumonia; PUVA, psoralen and ultraviolet A.

graft before transplantation may decrease the incidence and severity of GVHD. Methods of T-cell depletion involve physical, immunological, and pharmacological techniques. The desired outcome is a reduction or elimination of T cells capable of initiating life-threatening GVHD. However, T cells also play a role in engraftment, and T-cell depletion carries greater risks for infection, graft failure, and disease relapse.

A variety of immunosuppressive agents, alone or in combination, have been used prophylactically for acute GVHD.[50] Immunosuppressive medications minimize the ability of the newly developing donor immune system to recognize the host or patient as foreign and limit the immune response. Immunosuppressive drugs may need to be taken for months or years after an allogeneic HSCT. Immunosuppression may involve a single drug (often tacrolimus or cyclosporine A) or a combination of drugs (methotrexate, tacrolimus, cyclosporine A, steroids, MMF, antithymocyte globulin [ATG]), sometimes in combination with T-cell depletion.[50,68] Table 47-7 on pages 1072–1077 presents the immunosuppressive agents commonly used in both HSCT and solid organ transplantation settings and the associated nursing implications.[67,69–77]

For patients at higher risk for GVHD, especially those undergoing matched unrelated HSCT, more intensive strategies for GVHD prevention are necessary. Many drug–drug interactions are associated with cyclosporine A and tacrolimus. Table 47-13 lists drugs that may interact with cyclosporine A and tacrolimus.[69] It is important to instruct patients to take their immunosuppressive medication exactly as directed and to contact their physician before starting any new medication.

Prospective, randomized trials have demonstrated that combination therapy is superior to single-drug therapy in preventing acute GVHD. However, to date, research has not shown that any one prophylactic regimen is superior in preventing acute GVHD or improving overall outcome.[50,68] The most widely used pharmacological regimen for the prophylaxis of acute GVHD is a combination of methotrexate and either cyclosporine A or tacrolimus.[78] Other drugs included in some GVHD prophylaxis regimens are corticosteroids, ATG, daclizumab, and MMF.[68,78–80] Table 47-14 presents several sample regimens for GVHD prophylaxis.

If grade II to IV acute GVHD develops, treatment is usually required.[56] Corticosteroids are the main component of therapy, along with continuing treatment with the immunosuppressive agent used for prophylaxis (tacrolimus or cyclosporine A). Corticosteroids are the main component of therapy, along with continuation of the immunosuppressive agents used for initial prophylaxis.[68,78–80] High doses of methylprednisolone (1 to 20 mg/kg/d) may be used. However, these high-dose regimens are associated with fatal infections and cannot be administered for more than a few days, and the dose of methylprednisolone is rapidly tapered to 2 mg/kg/d in divided doses. Once maximal improvement is achieved, the steroid dosage is tapered over 8 to 20 weeks, based on patient response.

For patients with GVHD in whom initial therapy has failed, a variety of salvage or secondary regimens, including MMF, infliximab, and daclizumab, are available. Once chronic GVHD develops, the usual therapy involves steroids, cyclosporine A, tacrolimus, and a variety of other immunosuppressive agents.[52,63,65,66,72,81] Gut rest, pain control, and antimicrobial prophylaxis coupled with hyperalimentation, if needed, are important aspects of the supportive care of patients with acute GVHD.[50] The outcome of treatment of acute GVHD is predicted by the overall grade of acute GVHD; higher overall grades are associated with poorer outcomes.[52,53] Response to treatment is another key deter-

Table 47-13 Drugs That May Alter Levels of Cyclosporine and Tacrolimus

Effects	Known Interactions	Suspected Interactions
Increase serum levels	Erythromycin Clarithromycin Itraconazole Fluconazole Ketoconazole Corticosteroids	H_2 antagonists Cephalosporins Thiazide diuretics Furosemide Acyclovir Warfarin Calcium channel blockers (ie, diltiazem, verapamil, nicardipine) Oral contraceptives Doxycycline Metoclopramide Coadministration with grapefruit juice
Decrease serum levels	Phenytoin or phenobarbital Rifampin or isoniazid Sulfadiazine + trimethoprim (IV)	Sulfinpyrazone Carbamazepine Anticonvulsants
Cause additive nephrotoxicity	Amphotericin B Aminoglycosides Melphalan Trimethoprim-sulfamethoxazole	Nonsteroidal anti-inflammatory drugs
Alter immunosuppressive effects		Propranolol Verapamil Etoposide

Based on information from Evans SO: The transplant pharmacopeia. In Treleaven J, Barrett AJ (eds): Hematopoietic Stem Cell Transplantation in Clinical Practice. Edinburgh, UK: Elsevier Limited, 2009, pp 331–342.

Table 47-14 Examples of Commonly Used Drug Regimens for Prophylaxis of Acute GVHD

Regimen	Dosing Schedule
Cyclosporine/steroids	Cyclosporine 3 mg/kg/d IV infusion from day –2, taper 10% weekly starting day +180* Methylprednisolone 0.25 mg/kg twice a day, days +7 to +14; 0.5 mg/kg twice a day, days +15 to +28; 0.4 mg/kg twice a day, days +29 to +42; 0.3 mg/kg twice a day, days +43 to +58; 0.25 mg/kg twice a day, days +59 to +119; and 0.1 mg/kg daily, days +120 to 180
Cyclosporine/ methotrexate/ steroids	Cyclosporine 5 mg/kg/d IV infusion from day –2, taper 20% every 2 wk starting day +84* Methotrexate 15 mg/m^2 on day +1, 10 mg/m^2 on days +3 and +6 Methylprednisolone 0.25 mg/kg twice a day, days +7 to +14; 0.5 mg/kg twice a day, days +15 to +28; 0.4 mg/kg twice a day, days +29 to +42; 0.3 mg/kg twice a day, days +43 to +58; 0.25 mg/kg twice a day, days +59 to +119; and 0.1 mg/kg daily, days +120 to 180
Tacrolimus/ minimethotrexate	Tacrolimus 0.03 mg/kg/d infusion from day –2, taper 20% every 2 wk starting day +180* Methotrexate 5 mg/m^2 on days +1, +3, +6, and +11
ATG/ cyclosporine/ methotrexate	ATG 20 mg/kg IV days –3, –2, and –1 Cyclosporine 5 mg/kg/d IV infusion from day –1, taper 10% weekly starting day +180* Methotrexate 10 mg/m^2 on days +1, +3, +6, and +11

*Either tacrolimus or cyclosporine has been used with this methotrexate or steroid dose schedule.

minant of outcome, and mortality is greatest in patients who do not achieve a complete response to the initial treatment strategy for acute GVHD.[54]

▲ Long-Term Considerations

Organ transplantation can lead to long-term survival. Increasing numbers of recipients lead healthier and longer lives. However, complications may occur long after transplantation.

Long-term care focuses on monitoring the patient's progress and adherence to the health care regimen. In solid organ transplant recipients, a major cause of graft loss in the long term is failure of patients to adhere to the medication regimen. Patients must also be monitored for the development of late complications, including infections, hypertension and cardiovascular disease, chronic rejection, and recurrence of the original disease, such as hepatitis in liver transplantation and recurrent glomerulonephritis in kidney transplantation. There is also increased incidence of posttransplantation lymphoproliferative disease in solid organ transplant recipients who are receiving long-term immunosuppression.[82,83]

Weight gain can be a significant complication after transplantation as a result of steroid use or because of general improved well-being related to the organ transplantation. Osteoporosis secondary to high steroid use is also a long-term issue for organ transplant recipients, more often for heart, liver, and stem cell transplant recipients than for kidney transplant recipients.

The refinement and success of HSCT has resulted in a large population of patients who have achieved control of their underlying disease. However, these patients must often deal with long-term sequelae and late effects of HSCT. In addition to chronic GVHD and infectious risks, they may experience a wide range of complications (Box 47-5).[67,84–102] Most transplantation centers have unique requirements for continued follow-up care that depends on protocols. The nature of the patient's complications determines the frequency of clinic visits. Patients and clinicians can use the guidelines provided at http://www.cibmtr. org/PUBLICATIONS/guidelines.html to direct long-term follow-up care after HSCT. Table 47-15 presents guidelines for screening and management of late effects in HSCT recipients.[67,84,86,87,89–92,94–102]

BOX 47-5 Early and Late Complications of Autologous and Allogeneic Hematopoietic Stem Cell Transplantation

Early (Occurring Before Day 1100)

Regimen-related toxicity
- Hemorrhagic cystitis
- Veno-occlusive disease of the liver
- Pulmonary complications
- Renal complications
- Neurological complications

Nutritional complications
Idiopathic pneumonitis
Graft failure
Infection
- Viral
- Bacterial
- Fungal

Graft-versus-host disease
Relapse

Late (Occurring After Day +100)

Regimen-related toxicity
- Cataracts
- Neurological conditions (peripheral and autonomic neuropathies)
- Gonadal dysfunction
- Endocrine dysfunction

Immunodeficiency
Infection
Musculoskeletal
- Osteoporosis
- Avascular necrosis

Chronic GVHD
Relapse of malignancy
Secondary malignancy

Table 47-15 Evaluation and Screening of Late Effects of Hematopoietic Stem Cell Transplantation

System/Dimension	Possible Late Effects	Evaluation and Screening
Disease status	Relapse/recurrence	Determined based on site of original disease, but may include CT scans, bone marrow aspirate and biopsy, lumbar puncture, cytogenetics, and engraftment studies Evaluation for minimal residual disease (if available)
Engraftment	Graft failure/marrow dysfunction with cytopenia	CBC with differential Bone marrow aspirate and biopsy Engraftment studies: to detect differences between DNA of donor and recipient and thus establish engraftment: variable nucleotide tandem repeats or restriction fragment length polymorphisms; cytogenetic studies may also be used to establish engraftment if the donor and recipient are of opposite sexes
Immunological function/recovery	Disorders of B- and T-lymphocyte quantity and function Hypogammaglobulinemia	CD4/CD8 lymphocyte subsets Quantitative immunoglobulin levels Vaccination titers
Cardiopulmonary effects	Interstitial pneumonitis Bronchiolitis obliterans Hypertension, cardiomyopathy, pericardial damage, peripheral vascular disease, coronary artery disease	Chest x-ray Pulmonary function tests with diffusing capacity of lungs for carbon monoxide Electrocardiogram (ECG) Echocardiogram History and physical examination
Neurological effects	Peripheral and autonomic neuropathies Cognitive changes, shortened attention span, difficulty with concentration Leukoencephalopathy Ototoxicity	Health history Neurological examination Neuropsychological testing Rehabilitation medicine Audiological testing
Gastrointestinal effects	Liver dysfunction Malabsorption syndromes	Liver function tests Hepatitis B serologies, hepatitis C polymerase chain reaction qualitative
Genitourinary effects	Renal dysfunction Radiation nephritis Hematuria, proteinuria Cancer of the bladder	BUN, creatinine Urinalysis with microscopy 24-h urine for creatinine clearance, total protein, if indicated
Endocrine		
Thyroid function	Hypothyroidism	TSH, T_3, T_4, free T_4
Gonadal function	Decreased production of gonadal hormones	LH, FSH, estradiol (women) Pelvic examination LH, FSH, testosterone (men)
Hypothalamic–pituitary	Abnormal pituitary gland function	Prolactin levels, FSH, LH, TSH
Ophthalmic	Cataracts	Ophthalmological examination to include slit-lamp examination and Schirmer's test
Dental/oral cavity	Sicca syndrome Caries Periodontal disease Xerostomia Oral malignancy	Regular dental evaluations Careful attention to oral hygiene Fluoride gels/rinses
Musculoskeletal	Osteoporosis Avascular necrosis Myopathy	Dual-energy x-ray absorptiometry scan MRI if pain in a joint, limited range of motion, or a limp MRI, neurological examination, electromyogram
Second malignancy	Nonmelanoma skin cancer	Complete physical examination with biopsy of suspect lesions; skin photographs may also help to monitor status
	Breast cancer	Mammogram, self-examination
	Thyroid cancer	History and physical examination, ultrasound, ^{131}I scan
	Acute leukemia	CBC with differential
	Myelodysplastic syndrome	Bone marrow aspirate and biopsy (if CBC abnormal)
	PTLD	CT scans if PTLD suspected
	Cancer of the uterine cervix	Gynecological examination with Papanicolaou smear
	Cancer of the bladder	Urinalysis with micro to detect microhematuria, urine cytology, follow-up cystoscopy

(continued on page 1090)

Table 47-15 Evaluation and Screening of Late Effects of Hematopoietic Stem Cell Transplantation (continued)

System/Dimension	Possible Late Effects	Evaluation and Screening
Integumentary	Increased incidence of benign and malignant nevi	Complete physical examination Skin biopsy of suspect lesions
Psychological/rehabilitation, quality of life	Changes in body image, roles, family relationships, lifestyle, occupation, discrimination, overcoming stigma, living with compromises, coping with symptoms	Assessment of individual adjustment, achievement of normal developmental tasks, marital stress, sexual function, body image, rehabilitation needs, symptom distress through systematic and structured evaluation

CBC, complete blood count; CT, computed tomography; FSH, follicle-stimulating hormone; LH, luteinizing hormone; MRI, magnetic resonance imaging; PTLD, posttransplantation lymphoproliferative disease; T_3, triiodothyronine; T_4, thyroxine; TSH, thyroid-stimulating hormone.

▲ Clinical Applicability Challenges

CASE STUDY

Mr. S. is a 25-year-old man who began experiencing shortness of breath 1 year ago. A chest radiograph revealed an enlarged cardiac silhouette. An echocardiograph demonstrated an ejection fraction of 15%, with dilated left and right ventricles, and no valvular abnormalities. The patient was diagnosed with idiopathic cardiomyopathy. The patient has no other medical problems. The patient was seen by a cardiologist and began medical management of his heart failure. The patient continued to have fatigue, shortness of breath at rest, and lower extremity swelling despite medical therapy (New York Heart Association Class IV failure). The patient was evaluated for heart transplantation and a left ventricular assist device (LVAD). The patient was deemed a candidate for heart transplantation, but because of the long waiting list and the continued failure of medical therapy, the decision was made to place an LVAD as a bridge to transplantation. The patient recovered well after placement of LVAD and was discharged home and was listed 1B on the heart transplant list.

Mr. S. was readmitted to the hospital after his LVAD placement for acute gastrointestinal bleeding secondary to gastritis. Since the patient was having complications from the anticoagulation that is needed for the LVAD, his transplantation status was upgraded to 1A. Fortunately, a suitable heart became available within a month.

Mr. S. underwent orthotopic heart transplantation. A standard biatrial approach was used. When he was separated from cardiopulmonary bypass, he was receiving dobutamine 6.0 mcg/kg/min, epinephrine 0.05 mcg/kg/min, vasopressin 0.04 units/min, and isoproterenol 5 mcg/min. After bypass, nitric oxide was introduced through the ventilator for an elevated pulmonary pressure of 48/26 mm Hg. He remained intubated from the operative procedure and was transferred to the cardiac surgery ICU. Mr. S. was extubated on postoperative day 1.

Mr. S. had no bleeding complications in the ICU. Weaning from the vasopressin and epinephrine occurred within the first 24 hours. He remained on dobutamine for right ventricular heart support, and he continued on isoproterenol for chronotropic support. After stable pulmonary pressures and adequate oxygenation were achieved, weaning from the nitric oxide occurred over 24 hours. Stable pulmonary pressures were maintained. Immediately after the operation, he was started on Solu-Medrol, 125 mg IV for three doses, as well as mycophenolate mofetil (MMF) (CellCept) for immunosuppression. After the IV therapy was completed, oral doses of steroids were given. Tacrolimus (Prograf) was held for the first 24 hours to ascertain that renal function was normalized. Tacrolimus was then initiated at low doses, with close attention to urinary output and renal indices. Blood urea nitrogen and creatinine values remained within normal limits, and the tacrolimus was increased with a goal level of 12 to 15 ng/mL. The transplant team had difficulty getting tacrolimus levels increased despite quickly increasing his doses. Because of cytomegalovirus (CMV) mismatch, he was started on prophylactic valganciclovir (Valcyte) 900 mg twice daily. CMV by polymerase chain reaction was checked weekly to evaluate for early CMV reactivation. Mr. S. was slowly weaned completely from dobutamine over the week after transplantation. He also had very high glucose levels and remained on an insulin drip and was also started on insulin glargine (Lantus).

One week postoperatively, the patient underwent endomyocardial biopsy and right heart catheterization. At this time, measurements of cardiac function were right atrial (RA) pressure, 8 mm Hg; right ventricular (RV) pressure, 31/14 mm Hg; pulmonary artery (PA) pressure, 21/11 mm Hg; pulmonary artery occlusion pressure (PAOP), 8 mm Hg; and cardiac index, 3.2 L/min/m^2.

(continued on page 1091)

CASE STUDY *(Continued)*

On the date of biopsy, the tacrolimus level was 8 ng/mL. The patient's immunosuppression medications included prednisone 30 mg twice daily, MMF (CellCept) 1,000 mg twice daily, and tacrolimus 6 mg twice daily. The endomyocardial biopsy was a grade 1A rejection. The transplant team increased the MMF (CellCept) to 1,500 mg twice daily, and the tacrolimus dose was aggressively titrated to maintain a level in the range of 12 to 15 ng/mL.

One week later, the patient underwent repeat right heart catheterization and endomyocardial biopsy. On the date of biopsy, the tacrolimus level was 12.6 ng/mL. The biopsy revealed a grade 0 rejection. The patient was discharged to home on a prednisone taper, MMF 1,500 mg twice daily, and tacrolimus 9 mg three times daily.

1. What are the unique priorities for care in a postoperative heart transplant recipient?.
2. How is CMV transmitted during transplantation procedures, and why is CMV an important consideration relative to transplantation outcomes? Also, what are the current treatment recommendations?
3. Why did the patient need an insulin drip postoperatively when he had no history of diabetes mellitus? What patient education would be important for this patient with regard to his newly diagnosed diabetes?
4. Why is it a priority to maintain tacrolimus levels of 12 to 15 ng/mL in the postoperative heart transplant recipient?

References

1. Leung AY, Kwong YL: Haematopoietic stem cell transplantation: Current concepts and novel therapeutic strategies. Br Med Bull 93: 85–103, 2009
2. Brunstein CG, Baker KS, Wagner JE: Umbilical cord blood transplantation for myeloid malignancies. Curr Opin Hematol 14:162–169, 2007
3. Chantry AD, Snowden JA, Craddock C, et al: Long-term outcomes of myeloablation and autologous transplantation of relapsed acute myeloid leukemia in second remission: A British Society of Blood and Marrow Transplantation registry study. Biol Blood Marrow Transplant 12:1310–1317, 2006
4. Koreth J, Cutler CS, Djulbegovic B, et al: High-dose therapy with single autologous transplantation versus chemotherapy for newly diagnosed multiple myeloma: A systematic review and meta-analysis of randomized controlled trials. Biol Blood Marrow Transplant 13:183–196, 2007
5. Nademanee A, Forman SJ: Role of hematopoietic stem cell transplantation for advanced-stage diffuse large cell B-cell lymphoma-B. Semin Hematol 43:240–250, 2006
6. Tabbara IA, Zimmerman K, Morgan C, et al: Allogeneic hematopoietic stem cell transplantation: Complications and results. Arch Intern Med 162:1558–1566, 2002
7. Yakoub-Agha I, Mesnil F, Kuentz M, et al: Allogeneic marrow stem-cell transplantation from human leukocyte antigen-identical siblings versus human leukocyte antigen-allelic-matched unrelated donors (10/10) in patients with standard-risk hematologic malignancy: A prospective study from the French Society of Bone Marrow Transplantation and Cell Therapy. J Clin Oncol 24:5695–5702, 2006
8. Barrett JA, Chao NJA, Bishop MR: Are more patients being cured with allogeneic stem cell transplantation? American Society of Clinical Oncology, 2006 Educational Book. Alexandria, VA: American Society of Clinical Oncology, 2006
9. Schmit-Pokorny K: Expanding indications for stem cell transplantation. Semin Oncol Nurs 25(2):105–114, 2009
10. Scientific Registry of Transplant Recipients. 2008. Retrieved May, 2010, from http://www.ustransplant.org
11. United Network for Organ Sharing. Retrieved May, 2010, from http://www.unos.org
12. Medicare. Retrieved May, 2010, from http://www.medi care.org
13. Danovitch GM (ed): Handbook of Kidney Transplantation. Philadelphia, PA: Lippincott Williams & Wilkins, 2009
14. Klein AS, Messersmith EE, Ratner LE, et al: Organ donation and utilization in the United States, 1999–2008. Am J Transplant 10:973–986, 2010
15. Volk, ML, Warren, GJ, Anspach, RR, et al: Attitudes of the American public toward organ donation after uncontrolled (sudden) cardiac death. Am J Transplant 10:675–680, 2010
16. DuBose J, Salim A: Aggressive organ donor management protocol. J Intensive Care Med 23:367–375, 2008
17. Schnuelle P, Gottmann U, Hoeger S, et al: Effects of donor pretreatment with dopamine on graft function after kidney transplantation: A randomized controlled trial. JAMA 302:1067–1075, 2009
18. Mascia L, Mastromauro I, Viberti S, et al: Management to optimize organ procurement in brain dead donors. Minerva Anestesiol 75:125–133, 2009
19. Sharpiro R, Halloran PF: Organ Preservation-Can we do Better? Am J Transplant 8(3):479–480, 2008
20. Rudow DL, Goldstein MJ: Critical care management of the liver transplant recipient. Crit Care Nurs Q 31(3):232–243, 2008
21. Jacob S, Sellke F: Is bicaval orthotopic heart transplantation superior to the biatrial technique? Interact Cardiovasc Thorac Surg 9(2): 333–342, 2009
22. Poston RS, Griffith BP: Heart transplantation. J Intensive Care Med 19(1):3–12, 2004
23. Rees C, Beale P, Judson I: Theoretical aspects of dose intensity and dose scheduling. In Barrett J, Treleaven J (eds): The Clinical Practice of Stem Cell Transplantation. Oxford, UK: Isis Medical Media, 1998, pp 17–29
24. Alousi A, de Lima M: Reduced-intensity conditioning allogeneic hematopoietic stem cell transplantation. Clin Adv Hematol Oncol 5(7): 560–570, 2007
25. Schmit-Pokorny K, Franco T, Frappier B, et al: The cooperative care model: An innovative approach to deliver blood and marrow stem cell transplant care. Clin J Oncol Nurs 7(5):509–514, 556, 2003
26. Fox MC: Transfusions. In Burt RK, Deeg HJ, Lothian S, et al (eds): Bone Marrow Transplantation. Austin, TX: Landes Bioscience, 1998, pp 54–68
27. Davey DB, Crawford J: Hematologic support of the cancer patient. In Berger AM, Shuster JL, Von Roenn JH (eds): Principles and Practice of Supportive Oncology, 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2006, pp 727–740
28. Dodds A: ABO incompatibility and blood product support. In Atkinson K, Champlin R, Ritz J, et al (eds): Clinical Bone Marrow and Blood Cell Transplantation, 3rd ed. Cambridge, UK: Cambridge University Press, 2004, pp 1077–1087

29. Keown PA, McMaster WR, McManus BM: Tools to identify organ rejection and immune quiescence for biological understanding and personalized medical care. Biomark Med 4(1):115–121, 2010
30. Christie JD, et Al: The Registry of the International Society for Heart and Lung Transplantation: Twenty-sixth Official Adult Lung and Heart-Lung Transplantation Report—2009. J Heart Lung Transplant 28: 1031–1049, 2009
31. Burt RK, Walsh T: Infection prophylaxis in bone marrow transplant recipients: Myths, legends and microbes. In Burt RK, Deeg HJ, Lothian S, et al (eds): Bone Marrow Transplantation. Austin, TX: Landes Bioscience, 1998, pp 438–451
32. Kotton CN, et al: International consensus guidelines on the management of cytomegalovirus in solid organ transplantation. Transplantation 89:779–795, 2010
33. Schmauss D, Weiss M: Cardiac allograft vasculopathy. Circulation 117:2131–2141, 2008
34. Stratta RJ, Pietrangeli C, Baillie GM: Defining the risks for cytomegalovirus infection and disease after solid organ transplantation. Pharmacotherapy 30(2):144–157, 2010
35. Fishman JA: Introduction: Infection in solid organ transplant recipients. Am J Transplant 9 (Suppl 4):S3–S6, 2009
36. Zitella LJ, Friese CR, Hauser J, et al: Putting evidence into practice: Prevention of infection. Clin J Oncol Nurs 10(6):739–750, 2006
37. Mielcarek M, Awaya N, Torok-Storb B: Mechanisms of failure of sustained engraftment. In Atkinson K, Champlin R, Ritz J, et al (eds): Clinical Bone Marrow and Blood Cell Transplantation, 3rd ed. Cambridge, UK: Cambridge University Press, 2004, pp 151–159
38. Ho VT, Linden E, Revta C, et al: Hepatic veno-occlusive disease after hematopoietic stem cell transplantation: Review and update on the use of defibrotide. Semin Thromb Hemost 33(4):373–388, 2007
39. McDonald GB: Hepatobiliary complications of hematopoietic cell transplantation, 40 years on. Hepatology 51(4):1450–1460, 2010
40. Strasser SI, McDonald GB: Gastrointestinal and hepatic complications. In Appelbaum FR, Forman SJ, Negrin RS, et al (eds): Thomas' Hematopoietic Cell Transplantation, 4th ed. Hoboken, NJ: Blackwell Publishing, 2009, pp 1434–1455
41. Saria MG, Gosselin-Acomb TK: Hematopoietic stem cell transplantation: Implications for critical care nurses. Clin J Oncol Nurs 11: 53–63, 2007
42. Imran H, Tleyjeh IM, Zirakzadeh A, et al: Use of prophylactic anticoagulation and the risk of hepatic veno-occlusive disease in patients undergoing hematopoietic stem cell transplantation: A systematic review and meta-analysis. Bone Marrow Transplant 37(7):677–686, 2006
43. Tay J, Tinmouth A, Fergusson D, et al: Systematic review of controlled clinical trials on the use of ursodeoxycholic acid for the prevention of hepatic veno-occlusive disease in hematopoietic stem cell. Biol Blood Marrow Transplant 13(2):206–217, 2007
44. Yoshihara S, Yanik G, Cooke KR, et al: Bronchiolitis obliterans syndrome (BOS), bronchiolitis obliterans organizing pneumonia (BOOP), and other late-onset noninfectious pulmonary complications following allogeneic hematopoietic stem cell transplantation. Biol Blood Marrow Transplant 13(7):749–759, 2007
45. Watkins TR, Chien JW, Crawford SW: Graft versus host-associated pulmonary disease and other idiopathic pulmonary complications after hematopoietic stem cell transplant. Semin Respir Crit Care Med 26(5):482–489, 2005
46. Afessa B, Peters SG: Major complications following hematopoietic stem cell transplantation. Semin Respir Crit Care Med 27(3): 297–309, 2006
47. Majhail NS, Parks K, Defor TE, et al: Diffuse alveolar hemorrhage and infection-associated alveolar hemorrhage following hematopoietic stem cell transplantation: Related and high-risk clinical syndromes. Biol Blood Marrow Transplant 12(10):1038–1046, 2006
48. Soubani AO, Uberti JP: Bronchiolitis obliterans following haematopoietic stem cell transplantation. Eur Respir J 29(5):1007–1019, 2007
49. Choi SW, Levine JE, Ferrara JL: Pathogenesis and management of graft-versus-host disease. Immunol Allergy Clin North Am 30(1): 75–101, 2010
50. Jacobsohn DA, Vogelsang GB: Acute graft versus host disease. Orphanet J Rare Dis 2:35–39, 2007
51. Bolanos-Meade J, Garrett-Mayer E, Luznik L, et al: Induction of autologous graft-versus-host disease: Results of a randomized prospective clinical trial in patients with poor risk lymphoma. Biol Blood Marrow Transplant 13(10):1185–1191, 2007
52. Baird K, Pavletic SZ: Chronic graft versus host disease. Curr Opin Hematol 13:426–435, 2006
53. Holler E: Risk assessment in haematopoietic stem cell transplantation: GvHD prevention and treatment. Best Pract Res Clin Haematol 20:281–294, 2007
54. Lee SJ: New approaches for preventing and treating chronic graft-versus-host disease. Blood 105(11):4200–4206, 2006
55. Filipovich AH, Weisdorf D, Pavletic S, et al: National Institutes of Health consensus development project on criteria for clinical trials in chronic graft-versus-host disease. I. Diagnosis and staging working group report. Biol Blood Marrow Transplant 11:945–956, 2005
56. Demarosi F, Lodi G, Carrassi A, et al: Oral malignancies following HSCT: Graft versus host disease and other risk factors. Oral Oncol 41(9):865–877, 2005
57. Akpek G, Valladares JL, Lee L, et al: Pancreatic insufficiency in patients with chronic graft versus host disease. Bone Marrow Transplant 27:163–166, 2001
58. Aristei C, Allessandro M, Santucci A, et al: Cataracts in patients receiving stem cell transplantation after conditioning with total body irradiation. Bone Marrow Transplant 29:503–507, 2002
59. Baker KS, DeFor TE, Burns LJ, et al: New malignancies after blood or marrow stem-cell transplantation in children and adults: Incidence and risk factors. J Clin Oncol 21:1352–1358, 2003
60. Grigg AP, Angus PW, Hoyt R, et al: The incidence, pathogenesis, and natural history of steatorrhea after bone marrow transplantation. Bone Marrow Transplant 31:701–703, 2003
61. Lash AA: Sjögren's syndrome: Pathogenesis, diagnosis and treatment. Nurse Pract 26(8):50–58, 2001
62. Lee SJ, Vogelsang G, Flowers ME: Chronic graft versus host disease. Biol Blood Marrow Transplant 9(4):215–233, 2003
63. Vogelsang G: How I treat chronic graft-versus-host disease. Blood 97:1196–1201, 2001
64. Wagner JL, Flowers MED, Longton G, et al: Use of screening studies to predict survival among patients who do not have chronic graft-versus-host disease at day 100 after bone marrow transplantation. Biol Blood Marrow Transplant 7:239–240, 2001
65. Couriel D, Carpenter PA, Cutler C, et al: Ancillary therapy and supportive care of chronic graft-versus-host disease: National Institutes of Health consensus development project on criteria for clinical trials in chronic graft-versus-host disease. V: Ancillary Therapy and Supportive Care Working Group Report. Biol Blood Marrow Transplant 12:375–396, 2006
66. Pusic I, Vogelsang G, Pavletic S: Chronic Graft-vs-host disease. In Wingard JR, Gastineau DA, Leather HL, et al (eds): Hematopoietic Stem Cell Transplantation. Bethesda, MD: AABB, 2009, pp 345–364
67. Mitchell SA: Graft versus host disease. In Ezzone SA (ed): Peripheral Blood Stem Cell Transplant: Guidelines for Oncology Nursing Practice. Pittsburg, CA: Oncology Nursing Society Press, 2004
68. Deeg HJ: How I treat refractory acute graft versus host disease. Blood 109:4119–4126, 2007
69. Leather HL: Drug interactions in the hematopoietic stem cell transplant (HSCT) recipient: What every transplanter needs to know. Bone Marrow Transplant 33:137–152, 2004
70. Srinivas TR, Meier-Kriesche HU, Kaplan B: Pharmacokinetic principles of immunosuppressive drugs. Am J Transplant 5:207–217, 2005
71. Cutler C: Acute graft-vs-host disease. In Wingard JR, Gastineau DA, Leather HL, et al (eds): Hematopoietic Stem Cell Transplantation: A Handbook for Clinicians. Bethesda, MD: AABB, 2009, pp 331–343
72. Wolff D, Steiner B, Hildebrandt G, et al: Pharmaceutical and cellular strategies in prophylaxis and treatment of graft-versus-host disease. Curr Pharm Des 15(17):1974–1997, 2009
73. McPartland KJ, Pomposelli JJ: Update on immunosuppressive drugs used in solid-organ transplantation and their nutrition implications. Nutr Clin Pract 22(5):467–473, 2007

74. Evans SO: The transplant pharmacopeia. In Treleaven J, Barrett AJ (eds): Hematopoietic Stem Cell Transplantation in Clinical Practice. Edinburgh, UK: Elsevier Limited, 2009, pp 331–342
75. Henry L, Loader G: Nutrition support. In Treleaven J, Barrett AJ (eds): Hematopoietic Stem Cell Transplantation in Clinical Practice. Edinburgh, UK: Elsevier Limited, 2009, pp 344–354
76. Beauchesne PR, Chung NS, Wasan KM: Cyclosporine A: A review of current oral and intravenous delivery systems. Drug Dev Industr Pharm 33:211–220, 2007
77. Potter M: Graft failure. In Treleaven J, Barrett AJ (eds): Hematopoietic Stem Cell Transplantation in Clinical Practice. Edinburgh, UK: Elsevier Limited, 2009, pp 381–385
78. Chao NJ, Sullivan KM: Pharmacologic prevention of acute graft-versus-host disease. In Appelbaum FR, Forman SJ, Negrin RS, et al (eds): Thomas' Hematopoietic Cell Transplantation, 4th ed. Hoboken, NJ: Blackwell Publishing, 2009, pp 1257–1274
79. Kim SS: Treatment options in steroid-refractory acute graft-versus-host disease following hematopoietic stem cell transplantation. Ann Pharmacother 41(9):1436–1444, 2007
80. Bolanos-Meade J, Vogelsang GB: Acute graft-versus-host disease. Clin Adv Hematol Oncol 2(10):672–682, 2004
81. Seeley K, DeMeyer E: Nursing care of patients receiving Campath. Clin J Oncol Nurs 6(3):138–143, 2002
82. Frey NV, Tsai DE: The management of posttransplant lymphoproliferative disorder. Med Oncol 24(2):125–136, 2007
83. Lowe T, Bhatia S, Somlo G: Second malignancies after allogeneic hematopoietic cell transplantation. Biol Blood Marrow Transplant 13(10):1121–1134, 2007
84. Carpenter PA: Late effects of chronic graft-versus-host disease. Best Pract Res Clin Haematol 21(2):309–331, 2008
85. Aziz NM: Cancer survivorship research: State of knowledge, challenges and opportunities. Acta Oncol (Stockholm, Sweden) 46:417–432, 2007
86. Baker KS, Gurney JG, Ness KK, et al: Late effects in survivors of chronic myeloid leukemia treated with hematopoietic cell transplantation: Results from the Bone Marrow Transplant Survivor Study. Blood 104:1898–1906, 2004
87. Bhatia S, Robison LL, Francisco L, et al: Late mortality in survivors of autologous hematopoietic-cell transplantation: Report from the Bone Marrow Transplant Survivor Study. Blood 105:4215–4222, 2005
88. Quinney B: Psychologic and supportive care issues in the transplant setting. In Treleaven J, Barrett AJ (eds): Hematopoietic Stem Cell Transplantation in Clinical Practice. Edinburgh, UK: Elsevier Limited, 2009, pp 369–377
89. Cohen JM, Cooper N, Chakrabarti S, et al: EBV-related disease following haematopoietic stem cell transplantation with reduced intensity conditioning. Leuk Lymphoma 48:256–269, 2007
90. Doyle C, Kushi LH, Byers T, et al: Nutrition and physical activity during and after cancer treatment: An American Cancer Society guide for informed choices. CA Cancer J Clinicians 56:323–353, 2006
91. Gillis TA, Donovan ES: Rehabilitation following bone marrow transplantation. Cancer 92(4 Suppl):998–1007, 2001
92. Guise TA: Bone loss and fracture risk associated with cancer therapy. Oncologist 11:1121–1131, 2006
93. Poppelreuter M, Weis J, Mumm A, et al: Rehabilitation of therapy-related cognitive deficits in patients after hematopoietic stem cell transplantation. Bone Marrow Transplant 41(1):79–90, 2008
94. Kinch A, Oberg G, Arvidson J, et al: Post-transplant lymphoproliferative disease and other Epstein-Barr virus diseases in allogeneic haematopoietic stem cell transplantation after introduction of monitoring of viral load by polymerase chain reaction. Scand J Infect Dis 39: 235–244, 2007
95. Lee SJ, Schover LR, Partridge AH, et al: American Society of Clinical Oncology recommendations on fertility preservation in cancer patients. J Clin Oncol 24:2917–2931, 2006
96. Lenssen P, Akers S: Nutrition support of the hematopoietic cell transplant recipient. In Appelbaum FR, Forman SJ, Negrin RS, et al (eds): Thomas' Hematopoietic Cell Transplantation, 4th ed. Hoboken, NJ: Blackwell Publishing, 2009, pp 1551–1569
97. Carson K, Mehta J, Singhal S: Reimmunization after stem cell transplantation. In Treleaven J, Barrett AJ (eds): Hematopoietic Stem Cell Transplantation in Clinical Practice. Edinburgh, UK: Elsevier Limited, 2009, pp 363–368
98. Roziakova L, Mladosievicova B: Endocrine late effects after hematopoietic stem cell transplantation. Oncol Res 18(11–12):607–615, 2010
99. Rizzo JD, Wingard JR, Tichelli A, et al: Recommended screening and preventive practices for long-term survivors after hematopoietic cell transplantation: Joint recommendations of the European Group for Blood and Marrow Transplantation, the Center for International Blood and Marrow Transplant Research, and the American Society of Blood and Marrow Transplantation. Biol Blood Marrow Transplant 12:138–151, 2006
100. Flowers MED: Chronic graft-versus-host disease. In Treleaven J, Barrett AJ (eds): Hematopoietic Stem Cell Transplantation in Clinical Practice. Edinburgh, UK: Elsevier Limited, 2009, pp 410–407
101. Socié G, Bahtia S, Tichelli A: Late effects. In Treleaven J, Barrett AJ (eds): Hematopoietic Stem Cell Transplantation in Clinical Practice. Edinburgh, UK: Elsevier Limited, 2009, pp 467–491
102. Baker KS, Ness KK, Steinberger J, et al: Diabetes, hypertension, and cardiovascular events in survivors of hematopoietic cell transplantation: A report from the bone marrow transplantation survivor study. Blood 109:1765–1772, 2007

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

48 Common Immunological Disorders

Michael V. Relf, Brenda K. Shelton, and Kimmith M. Jones

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Utilize epidemiological evidence to describe the current human immunodeficiency virus (HIV) and acquired immunodeficiency syndrome (AIDS) epidemics in the United States.
2. Describe the immunopathogenesis and natural history of HIV infection and AIDS.
3. Explain standard precautions and transmission-based precautions and their implementation in the intensive care unit.
4. Discuss the use of diagnostic testing and antiretroviral therapy in the management of HIV infection and AIDS.
5. Describe the pathophysiological processes of the oncological emergencies.
6. Discuss appropriate assessment data for each oncological emergency derived from patient history and physical examination, clinical manifestations, and diagnostic studies.
7. Explain the anticipated medical management and rationale for the treatment of selected oncological emergencies.
8. Describe relevant aspects of nursing management for each of the oncological emergencies.

Normally, an intact immune system provides protection from disease—both infectious and noninfectious diseases such as cancer. When one or more components of this system are weakened or adversely altered, a person becomes susceptible to a variety of diseases including opportunistic infections and some cancers. Primary immunodeficiency syndromes, typically single-gene disorders, mostly occur in males due to the fact that many are X-linked inherited syndromes.[1,2] The overall incidence of symptomatic primary immunodeficiency is estimated to be 1 in 10,000. Examples of primary immunodeficiencies include agammaglobulinemia (a failure of B-lymphocyte precursors to mature into B lymphocytes and ultimately plasma cells), X-linked agammaglobulinemia or Bruton's disease, Wiscott-Aldrich syndrome (involving both T and B lymphocytes and platelets), and severe combined immunodeficiency in which there is an absence of both T-lymphocyte and B-lymphocyte function. Spotlight on Genetics 48-1 focuses on the hematological condition, polycythemia vera, and Spotlight on Genetics 48-2 discusses ankylosing spondylitis, a form of chronic inflammatory arthritis.

An immunodeficiency is known as secondary if it is acquired later in life. Secondary immunodeficiencies have a variety of etiologies including malignancies (chronic lymphocytic leukemia, myeloma), drugs (cytotoxic agents, immunosuppressants used in organ transplantation, and steroids), viruses, nutritional deficits, metabolic disorders, and severe protein loss.[2]

This chapter is divided into two parts that focus on two areas of secondary immunodeficiency: (1) HIV infection and AIDS and (2) emergent situations precipitated by commonly occurring neoplastic disorders. The reader is encouraged to review Chapter 45, especially the material relating to the immune mechanisms (humoral and cell-mediated immunity, complement system, and phagocytosis/chemotaxis/opsonization). This review will help the reader appreciate the pathophysiological changes occurring in the conditions discussed in this chapter.

HUMAN IMMUNODEFICIENCY VIRUS INFECTION

Impaired cellular immunity is the underlying pathophysiological consequence of AIDS, which is caused by the HIV. In 1981, the first case reports of the illness known today as AIDS were reported to the Centers for Disease Control and Prevention (CDC).[3] Since early in the HIV/AIDS epidemics, case surveillance definitions have been utilized by public health professionals and health care providers to diagnose,

SPOTLIGHT ON GENETICS 48-1

HEMATOLOGICAL SYSTEM–POLYCYTHEMIA VERA

- Polycythemia vera is a condition characterized by an increased number of red blood cells within the bloodstream and approximately 1 in 200,000 individuals are diagnosed each year.
- Mutations in the *JAK2* and *TET2* genes are associated with polycythemia vera. The function of the *TET2* gene is unknown. The *JAK2* gene provides instructions for making a protein that promotes the growth and division (proliferation) of cells.
- Polycythemia vera begins with one or more mutations in the DNA of a single hematopoietic stem cell, although it remains unclear exactly what initiates the disorder. A mutation in the *JAK2* gene seems to be particularly important for the development of polycythemia vera, as nearly all affected individuals have a mutation in this gene. *JAK2* gene mutations result in the production of a *JAK2* protein that is constantly turned on (constitutively activated), which improves the cell's ability to survive and increases production of blood cells
- Genetic and lab testing is available to diagnose Polycythemia Vera

Genetic Home Reference—http://ghr.nlm.nih.gov—Accessed July 14, 2011

Tefferi A, Pardanani A, Lim K-H, et al: TET2 mutations and their clinical correlates in polycythemia vera, essential thrombocythemia and myelofibrosis. Leukemia 23(5):905–911, 2009

SPOTLIGHT ON GENETICS 48-2

IMMUNE SYSTEM—ANKYLOSING SPONDYLITIS

- Ankylosing spondylitis is a form of chronic inflammatory arthritis that primarily affects the spine. It is part of a family of diseases called spondyloarthropathies, which affect between 3.5 and 13 per 1,000 people.
- A mutation in the *HLA-B* gene increases the risk of developing ankylosing spondylitis. Variations in several additional genes, including *ERAP1*, *IL1A*, and *IL23R*, have also been associated with ankylosing spondylitis.
- The *HLA-B* gene is part of a family of genes called the human leukocyte antigen (HLA) complex, which provide instructions for making a protein that plays an important role by helping the immune system distinguish the body's own proteins from proteins made by foreign invaders. Although *ERAP1*, *IL1A*, and *IL23R* influence immune system performance, it is unclear how variations affect risk of developing ankylosing spondylitis.
- Genetic testing such as targeted mutation analysis is available to diagnose ankylosing spondylitis.

Genetic Home Reference—http://ghr.nlm.nih.gov—Accessed July 14, 2011

Chen C-H, Lin K-C, Yu DTY, et al: Serum matrix metalloproteinases and tissue inhibitors of metalloproteinases in ankylosing spondylitis: MMP-3 is a reproducibly sensitive and specific biomarker of disease activity. Rheumatology 45(4):414–420, 2006

stage, and describe the natural history of HIV infection. In 2008, the HIV classification system and surveillance case definitions were revised and combined into a single case definition for HIV categorized by increasing severity—stage I, stage II, stage III (AIDS), or as stage unknown (Table 48-1).[4]

Since the 1980s, HIV infection has transformed from a life-limiting illness to a chronic illness manageable with strict adherence to antiretroviral therapy (ART) and other therapeutic interventions including the prophylaxis and treatment of opportunistic infections. Despite significant treatment advances, it remains an incurable disease that is highly stigmatized in the United States and around the world.[5] Without treatment, a person infected with HIV will progress to AIDS in about 10 years. However, with early diagnosis and treatment, survival for patients receiving ART and other therapies can be extended, on average, to 39 years.[6] Contemporary evidence across multisite studies demonstrates that the higher the $CD4^+$ T-cell lymphocyte count and the lower the viral load, there are decreased morbidity and mortality rates as well as a lower risk for progression to AIDS.

Caring for persons with HIV or AIDS requires collaborative, interdisciplinary care throughout the course of the illness.[5] Early in the AIDS epidemic, many believed it to be futile and a waste of resources to admit persons living with AIDS patients to an intensive care unit (ICU).[7] Today, however, as persons living with HIV and AIDS are living longer, they are being admitted to the ICU for management of AIDS-related opportunistic infections, complications associated with ART, and for medical problems unrelated to HIV infection.[8] When persons living with HIV or AIDS are admitted to the ICU, there are several challenging issues including legal statutes concerning HIV testing and disclosure, the administration of ART, risk for significant drug interactions between ART and medications commonly used in the ICU, and controversies surrounding the use of ART in the ICU.[9]

Critical care nurses need to be familiar with ART (1) to recognize life-threatening toxicities associated with this class of drugs, (2) to avoid drug interactions between ARTs and other classes of drugs that are common and potentially life-threatening, and (3) to avoid promoting ART drug resistance—a situation that could have profound negative consequences for the person living with HIV or AIDS after discharge from the ICU.[10] Physiological problems potentially experienced by persons living with HIV or AIDS requiring ICU care include immune reconstitution syndrome; acute respiratory distress syndrome from a variety of pathogens; atherogenic metabolic complications associated with ART; end-stage liver disease secondary to viral hepatitis; toxic effects, complications, and drug interactions associated with hepatitis C coinfection treatment (pegylated interferon and ribavirin); lactic acidosis associated with nucleoside reverse transcriptase inhibitors (NRTIs); and end-stage renal disease secondary to HIV-associated nephropathy; hepatitis B or C; diabetes; and hypertension.[9]

For some persons, a hospitalization or critical illness may result in them being diagnosed with HIV or AIDS. As many as 40% of patients with HIV are unaware of their status when they are admitted to the ICU.[9] To increase the rate

Table 48-1 Surveillance Case Definition for Human Immunodeficiency Virus (HIV) Infection Among Adults and Adolescents (Aged ≥13 years)—United States, 2008

Stage	Laboratory Evidence*	Clinical Evidence
Stage 1	Laboratory confirmation of HIV infection *and* $CD4^+$ T-lymphocyte count of ≥500 cells/mcL *or* $CD4^+$ T-lymphocyte percentage of ≥29	None required (but no AIDS-defining condition)
Stage 2	Laboratory confirmation of HIV infection *and* $CD4^+$ T-lymphocyte count of 200–499 cells/mcL *or* $CD4^+$ T-lymphocyte percentage of 14–28	None required (but no AIDS-defining condition)
Stage 3 (AIDS)	Laboratory confirmation of HIV infection *and* $CD4^+$ T-lymphocyte count of <200 cells/mcL *or* $CD4^+$ T-lymphocyte percentage <14†	*or* documentation of and AIDS-defining condition (with laboratory confirmation of HIV infection)†
Stage unknown‡	Laboratory confirmation of HIV infection *and* no information on $CD4^+$ T-lymphocyte count or percentage	*and* no information on presence of AIDS-defining conditions

*The $CD4^+$ T-lymphocyte percentage is the percentage of total lymphocytes. If the $CD4^+$ T-lymphocyte count and percentage do not correspond to the same HIV infection stage, select the more severe stage.

†Documentation of an AIDS-defining condition (Appendix A) supersedes a $CD4^+$ T-lymphocyte count of ≥200 cells/mcL and a $CD4^+$ T-lymphocyte percentage of total lymphocytes of ≥14. Definitive diagnostic methods for these conditions are available in Appendix C of the 1993 revised HIV classification system and the expanded AIDS case definition (CDC. 1993 Revised classification system for HIV infection and expanded surveillance case definition for AIDS among adolescents and adults. MMWR Recomm Rep 41[No. RR-17], 1992) and from the National Notifiable Diseases Surveillance System. Available at: http://www.cdc.gob/epo/dphsl/casedet/case_definitions.htm.

‡Although cases with no information on $CD4^+$ T-lymphocyte count or percentage or on the presence of AIDS-defining conditions can be classified as stage unknown, every effort should be made to report $CD4^+$ T-lymphocyte counts or percentages and the presence of AIDS-defining conditions at the time of diagnosis. Additional $CD4^+$ T-lymphocyte counts or percentages and any identified AIDS-defining conditions can be reported as recommended. (Council of State and Territorial Epidemiologists. Laboratory reporting of clinical test results indicative of HIV infection: new standards for a new era of surveillance and prevention [Position Statement 04-ID-07]; 2004. Available at http://www.cste.org/ps/2004pdf/04-ID-07-final.pdf)

From Morb Mort Wkly Rep 57(RR10):1–8, 2008.

of HIV testing in health care settings, foster earlier detection of HIV infection, identify and counsel persons with unrecognized HIV infection while linking them to clinical and prevention services, and further reduce the rate of perinatal or vertical (mother-to-child) transmission, the CDC issued *Revised Recommendations for HIV testing of adults, adolescents, and pregnant women in health-care* settings in 2006 advocating the routine testing of all people who visit any health care setting.[11] On World AIDS Day 2010 (December 1, 2010), the Emerging and Infectious Diseases Expert Panel of the American Academy of Nursing endorsed the role of nursing in supporting routine HIV testing in all health care settings and increasing efficiencies in administrative operations for widespread testing.[12] Knowledge of an individual's HIV serostatus impacts the differential diagnosis and may influence the diagnostic and treatment options including patient outcome. Therefore, it is essential for critical care nurses to understand how HIV infection, as well as its sequelae and treatment, serves as a comorbid condition of the critically ill person or the primary admission diagnosis to the ICU.

▲ Epidemiology

In July 2010, CDC estimated that more than 1 million people were living with HIV in the United States.[13] One in five of those people was living with HIV but unaware of the infection. As a consequence of increased survival, the prevalence of HIV and AIDS is increasing in the United States while the number of new infections remains too high, with an estimated 56,000 Americans becoming newly infected with HIV annually. The majority of AIDS cases continue to occur in gay, bisexual, and other men who have sex with men (MSM). Half of new HIV infections annually and nearly half of the people living with HIV/AIDS are among MSM. However, heterosexuals and injection-drug users also continue to be diagnosed with HIV and AIDS. Like MSM, African Americans/Blacks are disproportionately affected by HIV and AIDS. Although African Americans/Blacks account for approximately 12% of the U.S. population, they account for nearly half of all new infections each year and half of the people living with HIV in the United States. The states and dependent areas with the highest rates (per 100,000) of adults and adolescents living with an AIDS diagnosis at the end of 2007 are, in descending order, the District of Columbia (1781.1), New York (456.9), Maryland (353.8), U.S. Virgin Islands (353.5), Puerto Rico (337.2), Florida (316), New Jersey (263.2), Delaware (261.6), Connecticut (253.7), and Georgia (250.8).[14]

▲ Immunopathogenesis of HIV

The immunocompromise associated with AIDS is caused by the viral agent HIV.[15,16] HIV is a single-stranded RNA virus that is part of the *Retroviridae* family in the genus *Lentivirus*. Two species of HIV are known to exist and include HIV-1 and HIV-2. HIV-1 and HIV-2 are human

primate lentiviruses while their genetic cousins, simian immunodeficiency virus 1 and 2 (SIV-1 and SIV-2), are nonhuman primate lentiviruses. Recent evidence documents extensive diversity of HIV-1 in the human primate population by 1960 and that the common ancestor to HIV-1 was spreading among humans 60 to 80 years before AIDS was first recognized in the 1980s.[17,18]

HIV is transmitted from person to person by blood and bodily fluids (semen, vaginal secretions, breast milk).[16] A variety of cells are susceptible to HIV infection including cells of the hematopoietic system, central nervous system (CNS), skin, gastrointestinal tract, myocardium as well as the dendritic cells, renal tubular cells, hepatocytes, Kupffer cells, pulmonary fibroblasts, cervix, prostate, testes, and dental pulp fibroblasts.[16] Influencing that transmission of HIV includes the biologic properties of the virus, its concentration in the bodily fluids, and the nature of the host's susceptibility at the cellular and immunological levels. Sexually transmitted infections (STIs) increase both the amount of infectious virus and the number of infected cells in the genitals and enhance HIV transmission. The lack of circumcision in males has been associated with an increased risk of infection related to the large number of dendritic cells in the foreskin and also with increased rates of transmission among uncircumcised men to their sexual partners.[16]

Viral Replication

HIV is composed of nine genes responsible for invading host cells and replicating. HIV is composed of a small outer envelope, an inner core of genetic material (RNA), and three enzymes necessary for reproduction: reverse transcriptase, integrase, and protease (controlled by the pol—or polymerase—gene). Like all RNA viruses, HIV cannot reproduce on its own. It must attach to and invade other cells to reproduce. Figure 48-1 illustrates the process of viral replication.

After HIV enters the bloodstream, infection occurs with transmission of HIV across a mucosal barrier attaching itself to dendritic cells or Langerhans cells.[15] Propagation of HIV occurs initially in partially activated $CD4^+$ T cells followed by massive propagation in activated $CD4^+$ T cells of the gut-associated lymphoid tissue. Then, dissemination of HIV to other secondary lymphoid tissues ensues with establishment of stable HIV viral reservoirs following.

Having an affinity for $CD4^+$ receptors, HIV attaches to the $CD4^+$ receptors on the T lymphocytes, fusing with the host cell, shedding its outer envelope and then integrating its viral core into the DNA of the host's cell. The viral RNA is incorporated into the DNA of the T-lymphocyte host with help from the enzyme integrase. After integration, viral RNA is transcribed into the host's DNA by way of the enzyme reverse transcriptase. After incorporation into the cell's DNA structure, the enzyme protease breaks the components down into functional pieces, which are then assembled into structurally intact, new infectious units called virions. This process tricks the host's cells into making components for more virions.

Eventually, the new virions undergo a process of coating and are then expelled from the host cell by budding.

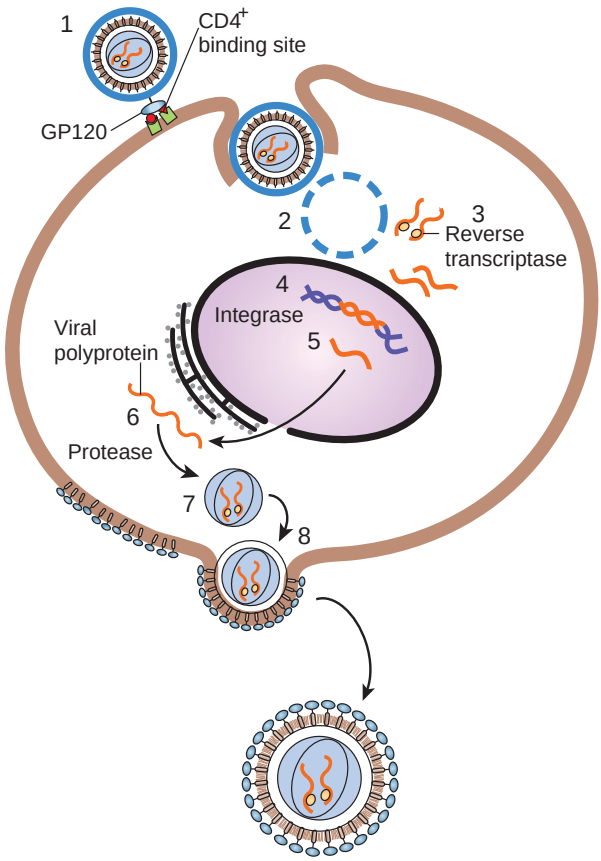


FIGURE 48-1 ▲ Process of human immunodeficiency virus (HIV) replication. (*1*) Attachment of HIV to a $CD4^+$ receptor. (*2*) Internalization and uncoating of the virus with viral RNA and reverse transcriptase. (*3*) Reverse transcription, which produces a mirror image of the viral RNA and double-stranded DNA molecule. (*4*) Integration of viral DNA into host DNA using the integrase enzyme. (*5*) Transcription of the inserted viral DNA to produce viral messenger RNA. (*6*) Translation of viral messenger RNA to create viral polyprotein. (*7*) Cleavage of viral polyprotein into individual viral proteins that make up the new virus. (*8*) Assembly and release of the new virus from the host cell. (Adapted from Porth C: Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009.)

During budding, the parent cell releases a daughter cell with its cytoplasmic material, which begins existence as a separate cell. These daughter cells disseminate through the bloodstream and infect other cells. Approximately 30% of the viral burden in a person who is HIV positive is regenerated daily. This budding process—which weakens the cell wall of the original $CD4^+$ T cells leading to cellular instability in combination with the direct cytopathic effects of HIV on $CD4^+$ cells and progenitor cells, the effect of HIV on cell membrane permeability, induction of apoptosis (programmed cell death) associated with immune activation, destruction of the bone marrow and lymphoid tissues as well as cytokine dysregulation, anti-$CD4^+$ cell cytotoxic activity, and anti-$CD4^+$ autoantibodies—results in a decline in the number of circulating $CD4^+$ T-cell lymphocytes. Ultimately, these complex mechanisms destroy cellular immune functioning resulting in immunosuppresion.[16]

Immune Defects

Patients with HIV infection exhibit impaired activation of both cellular and humoral immunity.[15,16] HIV primarily infects the helper CD4+ T-cell lymphocytes of the immune system. As discussed in Chapter 45, these T cells play a major role in the overall immune response. Infection of the helper T cells with HIV results in profound lymphopenia with decreased functional abilities, including decreased response to antigens and loss of stimulus for T- and B-cell activation. In addition, the cytotoxic activity of the killer cells (CD8+ T cells) is impaired. Functional abilities of macrophages also are affected leading to decreased phagocytosis and diminished chemotaxis. In humoral immunity, there is diminished antibody response to antigens, along with dysregulation of antibody production. The total effect of these immune defects ultimately results in immunosuppression increasing susceptibility to opportunistic infections and neoplasms. Figure 48-2 presents a summary of the immune defects associated with AIDS.

HIV Transmission and Natural History

HIV is a fragile virus and cannot survive long outside the body. Survival time depends on the size of the liquid droplet in which it exists—the larger the droplet, the longer HIV can remain alive. As the droplet dries, HIV dies. HIV has been isolated from all types of body fluids and tissues. However, not all body fluids have been implicated in the transmission of HIV. The four fluids from which large amounts of virus have been isolated and have been implicated in transmission include blood, semen, vaginal fluid, and breast milk.

The infectiousness of a fluid depends on the amount of virus present (viral load) in the fluid and the ability of that fluid to reach the target cell. For HIV to cause infection, it must leave the body of the infected host, successfully enter the new host's bloodstream, and attach itself to a CD4+ receptor site. The likelihood that this series of events will occur is low, especially because a certain amount of virus is required to cause an infection. The notion that HIV does not transmit easily is based on evidence suggesting that the probability of transmission ranges from 0.0001 to 0.0040 per sexual contact.[15] Small tears in the anus or vagina provide a portal of entry for virus present in blood, semen, and vaginal fluid. The virus in breast milk can enter through cuts or irritation in the gastrointestinal tract of the infant.

There are three known modes of HIV transmission:[16]

- Unprotected vaginal or anal sexual contact with an infected person with unprotected receptive anal intercourse being the riskiest sexual behavior
- Inoculation with infected blood or blood products, which includes accidental needlestick injuries and risk for transmission with needle sharing by injection-drug users
- Vertical transmission from mother-to-child during delivery or through breast-feeding

After infection with HIV, a person does not immediately test positive for HIV. Seroconversion is the development of antibodies as a consequence of antigenic stimulation from HIV associated with exposure, which can be detected in the blood. In other words, seroconversion is the change from an HIV-negative result to an HIV-positive result. During the seroconversion process, the body recognizes HIV as an invader and develops antibodies, which are then detectable by enzyme-linked immunosorbent assay (ELISA). In most people, seroconversion occurs 2 to 8 weeks after exposure to HIV (average is 25 days).[19] During this time, a person can unknowingly transmit the virus and may falsely test negative by an ELISA screening test. In 97% of persons, seroconversion occurs within 3 months, but in extremely rare cases, it can take up to 6 months for antibodies to develop.[19]

Once infected with HIV, manifestations of acute or primary HIV infection, also known as acute antiretroviral syndrome, occurs 2 to 4 weeks later. The manifestations of acute HIV infection can present like other infectious viral disease including mononucleosis and influenza. The most frequent manifestations associated with acute HIV infection include fever (96%), adenopathy (74%), pharyngitis (70%), rash (70%), myalgias (54%), and, less frequently, diarrhea (32%), headache (32%), nausea/vomiting (27%), hepatosplenomegaly (14%), weight loss (13%), thrush (12%), and neurological symptoms (12%).[20] During this acute HIV infection phase, there is usually a high viral load. During this phase of HIV infection, it is not uncommon for an individual to be unaware of his/her HIV serostatus and to test negative or indeterminate for HIV. Several studies suggest that during

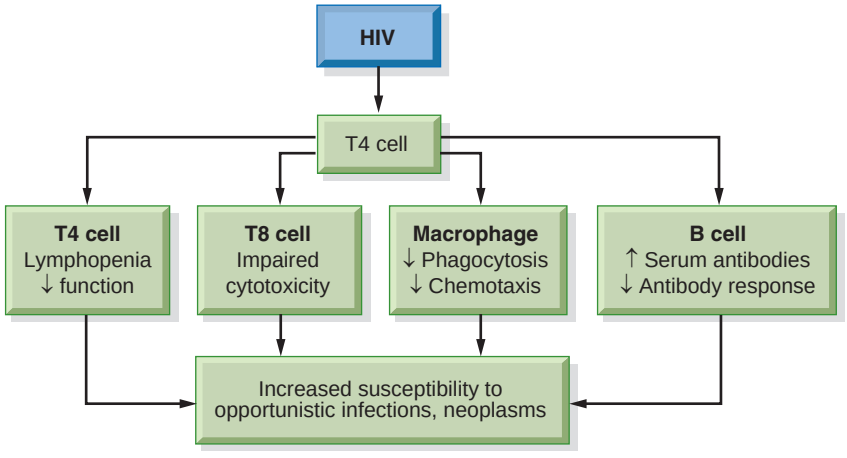


FIGURE 48-2 ▲ Summary of immune defects in AIDS.

BOX 48-1 Summary of Isolation Precautions

Standard Precautions: "Based on the principle that all blood, bodily fluids, secretions, excretions except sweat, non-intact skin and mucous membranes may contain transmissible infectious agents"

Common Organisms

Hepatitis A virus
Hepatitis B/D virus
Hepatitis C virus
Human immunodeficiency virus (HIV)
Pneumocystis jiroveci

Key Elements

Hand Hygiene

- Before direct care contact
- Wash hands after touching blood, body fluids, secretions, excretions, and contaminated items, regardless of whether gloves are worn.
- Wash hands immediately after gloves are removed, between patient contacts, and whenever indicated to prevent transfer of microorganisms to other patients or environments. Use plain soap for routine handwashing and an antimicrobial or waterless antiseptic agent for specific circumstances.
- Wear clean, nonsterile gloves when touching blood, body fluids, excretions or secretions, contaminated items, mucous membranes, and nonintact skin. Change gloves between tasks on the same patient as necessary, and remove gloves promptly after use.
- Restrict use of artificial nails and nail polish.

Use of Personal Protective Equipment

- Wear mask, eye protection, or face shield during procedures and care activities that are likely to generate splashes or sprays of blood or body fluids. Use gown to protect skin and prevent soiling of clothing.

Health Care Delivery System

- Ensure that used patient care equipment that is soiled with blood or identified body fluids, secretions, and excretions is handled carefully to prevent transfer of microorganisms or cleaned and appropriately reprocessed if used for another patient.
- Use adequate environmental controls to ensure that routine care, cleaning, and disinfection procedures are followed.
- Handle, transport, and process linen soiled with blood and body fluids, excretions, and secretions in a manner that prevents skin and mucous membrane exposures, contamination of clothing, and transfer of microorganisms.

Safe Injection Practices

- Use previously identified techniques and equipment to prevent injuries when using needles, sharps, and scalpels and place these items in appropriate puncture-resistant containers after use.

Airborne Precautions: Used in addition to standard precautions for "patients known or suspected with organisms transmitted by airborne droplets that remain suspended in the air and can be widely dispersed by air current"

Common Organisms

Mycobacterium tuberculosis
Varicella
Measles
SARS

Key Elements

- Place the patient in private room with an isolation sign on the door that has monitored negative air pressure in relation to surrounding areas, 6–12 air changes per hour, and appropriate discharge of air outside or monitored filtration if air is recirculated.
- Keep the door closed to the patient room at all times and the patient in the room whenever possible.
- Use respiratory protection when entering room of patient with known or suspected TB. If patient has known or suspected rubeola (measles) or varicella (chickenpox), respiratory protection should be worn unless person entering room is immune to these diseases.
- Transport the patient out of the room only when necessary, and place a surgical mask on the patient if possible.
- Consult Centers for Disease Control and Prevention Guidelines for additional prevention strategies for TB.

Droplet Precautions: Used in addition to standard precautions for "patients known or suspected to be infected with microorganisms transmitted by droplets generated during coughing, sneezing, talking, or during cough producing procedures"

Common Organisms

Meningitis
Pertussis
Influenza (A, B, C, Avian, H1N1)
Mumps
Rubella
Mycoplasma

Key Elements

- Place the patient in private room with an isolation sign on the door, if available, or cohort patients if necessary. Door closed at all times.
- Spatial separation of at least 3 ft from other patients/visitors. Health care providers should wear a mask when working within 3 ft of the patient.
- Transport the patient out of the room only when necessary, and place a surgical mask on the patient if possible.

(*continued on page 1100*)

BOX 48-1 Summary of Isolation Precautions (continued)

Contact Isolation: Used in addition to standard precautions for "patients known or suspected to be infected or colonized with epidemiologically important microorganisms that can be transmitted by direct contact with the patient when performing care activities or indirect contact (touching) with environmental surfaces or patient care items"

Common Organisms	Key Elements
Clostridium difficile	• Place the patient in private room with an isolation sign on the door, if available, or cohort patients if necessary. Door closed at all times.
Respiratory syncytial virus	
Pediculosis	• Change gloves after having contact with infective material. Remove gloves before leaving the patient environment, and wash hands with an antimicrobial or waterless antiseptic agent.
Scabies	
Multidrug resistant staphylococcus aureus (MRSA)	• Wear a gown if contact with infectious agent is likely or patient has diarrhea, ileostomy, colostomy, or wound drainage not contained by a dressing.
Vancomycin resistant enterococcus (VRE)	• Limit movement of the patient out of the room.
Gram-negative bacteria	• When possible, dedicate the use of noncritical patient care equipment to a single patient to avoid sharing equipment.

Adapted from Siegel JD, Rhinehart E, Jackson M, et al; the Healthcare Infection Control Practices Advisory Committee: 2007 Guideline for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings, June 2007. Available at: http://www.cdc.gov/ncidod/dhqp/pdf/isolation2007.pdf

this acute HIV infection phase, as many as 40% of lifetime transmissions occur. As previously stated, over 20% of all HIV-infected persons in the United States are not aware of their current serostatus and therefore have the potential to transmit the virus to another person. Although ELISA plays a valuable role in screening, it is not a confirmatory test for the diagnosis of HIV. Later in this chapter, other diagnostic tests used in HIV testing will be discussed.

The risk for HIV transmission to health care workers is low if standard precautions are followed[21] (Box 48-1). Occupational exposure can occur through a percutaneous injury (needlestick), contact with mucous membrane, or contact with nonintact skin (chapped, abraded, or affected by dermatitis).[22] The estimated average risk for HIV transmission after a percutaneous injury is 0.3%. The estimated average risk for HIV transmission after a mucous membrane exposure is 0.09%.[22] To provide a mechanism for comparison, if a health care worker were to receive a percutaneous needlestick from a host who was antigen positive for hepatitis B virus (HBV), hepatitis C virus (HCV), and HIV, the health care worker would have a 37% to 62% risk for acquiring HBV if previously not vaccinated and a 1.8% risk for acquiring HCV in comparison to only a 0.3% risk for acquiring HIV. Factors that can influence HIV transmission in a health care setting include contact with a device that is visually contaminated with blood, exposure to a large quantity of blood, participating in a procedure in which a needle is placed in an artery or vein, and deep injuries.[22]

For many years, occupational and nonoccupational postexposure prophylaxis (PEP) has been offered to health care workers and persons with exposure to HIV. PEP should be started as soon as possible after exposure to HIV, preferably within hours but no later than within 72 hours.[22] After 72 hours, PEP is not recommended. The decision about when to initiate PEP is based on the type of exposure, risk assessment of the incident, and risk assessment for HIV. Combination therapy using two or three medications is recommended in PEP; the number of medications depends on the type of exposure and the risk status of the source. Whether an occupational or nonoccupational exposure, PEP is prescribed for 28 days and requires the person to maintain strict adherence, which may be difficult if toxicities or side effects develop. Laboratory tests for toxicity should occur at the time of exposure and 2 weeks later and consist of a complete blood count (CBC) and renal and hepatic function tests. At the time of the exposure, persons exposed should have serological testing followed by repeat testing at 6 weeks, 12 weeks, and 6 months after exposure.[22] The National HIV/AIDS Clinicians' Consultation Center is a 24-hours-a-day, 7-days-a-week resource to all health care professionals and includes a PEP hotline to help manage exposure not only to HIV but also to HBV and HCV (see http://www.nccc.ucsf.edu/home for more information).

Since there has not been a decline in the number of new infections in the United States, scientists, clinicians, public health specialists, and advocacy groups have debated the value of preexposure chemoprophylaxis as a mechanism to prevent HIV infection. In a large, multisite clinical trial reported in December 2010, scientists identified that use of oral ART provided protection against the acquisition of HIV infection among MSM study participants.[23] Although not current standard of care, findings from this study may change prevention guidelines for high-risk populations in the future.

Regardless of the etiology of HIV infection, once infected with HIV, individuals will progress from the acute primary HIV infection phase to an asymptomatic phase. During this period, HIV viral replication continues resulting in a decline of immune functioning. As the number of $CD4^+$ T cells declines, a number of infectious and noninfectious diseases begin to present. Table 48-2 describes the complications common at various $CD4^+$ T-cell counts. If not treated, the cellular immune system will continue to decline and the person living with HIV will continue to be at risk for or experience an increased number of infectious and noninfectious diseases. Once the $CD4^+$ T-cell count reaches 200 cells/mm^3, the infected person is considered immunosuppressed and at very high risk for a number of opportunistic infections and other problems (Box 48-2 provides a list of AIDS-defining conditions).

Table 48-2 Correlation Between CD4⁺ T-Cell Count and HIV Complications

CD4⁺ T-cell count* (as cells/mm³)	Infectious Complications	Noninfectious† Complications
>500	Acute retroviral syndrome Candidal vaginitis	Persistent generalized lymphadenopathy Guillain-Barré syndrome Myopathy Aseptic meningitis
200–500	Pneumococcal and other bacterial pneumonia Pulmonary tuberculosis (TB) Herpes zoster Oropharyngeal candidiasis (thrush) Cryptosporidiosis, self-limited Kaposi's sarcoma Oral hairy leukoplakia	Cervical intraepithelial neoplasia Cervical cancer B-cell lymphoma Anemia Mononeuronal multiplex Idiopathic thrombocytopenic purpura Hodgkin's lymphoma Lymphocytic interstitial pneumonitis
<200	*Pneumocystis jiroveci* pneumonia Disseminated histoplasmosis and coccidioidomycosis Miliary/extrapulmonary TB Progressive multifocal leukoencephalopathy	Wasting Peripheral neuropathy HIV-associated dementia Cardiomyopathy Vacuolar myelopathy Progressive polyradiculopathy Non-Hodgkin's lymphoma
<100	Disseminated herpes simplex Toxoplasmosis Cryptococcosis Cryptosporidiosis, chronic Microsporidiosis Candidal esophagitis	
<50	Disseminated cytomegalovirus Disseminated *Mycobacterium avium* complex	Primary central nervous system (CNS) lymphoma

*Most complications occur with increasing frequency at lower CD4⁺ T-cell counts.

†Some conditions categorized as noninfectious are often microbially mediated, such as lymphoma (Epstein-Barr virus), and cervical carcinoma (human papillomavirus).

From Bartlett JG, Gallant JE, Pham P: The Management of HIV Infection. Durham, NC: Knowledge Source Solutions, LLC., 2009, p 3.

▲ Assessment

History and Physical Examination

The spectrum of clinical findings associated with acute HIV infection, HIV and AIDS ranges from flulike symptoms, to a period of no symptoms, to a variety of infections and symptoms associated with decreasing immunocompetence, to unquestionable AIDS. Patients with HIV infection may become seriously ill, requiring frequent hospitalizations and care in the ICU. In the past, critical care nurses frequently encountered patients with AIDS experiencing life-threatening opportunistic infections. Today, as patients with HIV and AIDS are living longer, critical care nurses are more frequently caring for this population as they experience other critical illnesses associated with aging including cardiovascular disease, renal problems, trauma, or consequences of ART in addition to the complications of AIDS.

Pneumocystis pneumonia (PCP), which is caused by *Pneumocystis jiroveci* (formerly known as *Pneumocystis carinii*), is the most common opportunistic infection requiring admission to the ICU.[24] The organism is considered a fungus based on its genetic makeup. Even though the name has changed, the abbreviation PCP is still used to describe the pathogen. The most common presenting symptoms include fever, exertional dyspnea, nonproductive cough, and a normal chest radiograph progressing to severe hypoxemia and respiratory failure.

The major indication for critical care of patients with PCP is impending or actual respiratory failure. Symptoms of respiratory compromise often are more severe than diagnostic studies, such as chest radiographs and blood gas values, indicate. Therefore, early aggressive therapy for PCP using intravenous (IV) trimethoprim and sulfamethoxazole (Bactrim, Septra) and corticosteroids is the treatment of choice. Corticosteroids are given to reduce the inflammation caused by the death of *P. jiroveci* in the lungs. Even with urgent, aggressive treatment, many patients require mechanical ventilation for progressive alveolar hypoventilation. Adverse reactions to trimethoprim and sulfamethoxazole, including nausea and vomiting, maculopapular rash, bone marrow suppression, anorexia, headache, crystalluria, and fever, reportedly occur in more than 50% of patients.

Patients with AIDS may also experience complex neurological conditions including cryptococcal meningitis, toxoplasmosis, histoplasmosis, Creutzfeldt-Jakob disease (CJD) leading to progressive multifocal leukoencephalopathy, and CNS lymphomas. Like PCP, a priority is to reduce immunosuppression by initiating ART so that these opportunistic

BOX 48-2 Case Definition of AIDS for Surveillance Purposes: Indicator Conditions

- Candidiasis of bronchi, trachea, or lungs
- Candidiasis, esophageal
- Cervical cancer, invasive
- Coccidioidomycosis, disseminated or extrapulmonary
- Cryptococcosis, extrapulmonary
- Cryptosporidiosis, chronic intestinal (>1 month duration)
- Cytomegalovirus (CMV) disease (other than liver, spleen, or nodes)
- CMV retinitis (with loss of vision)
- Encephalopathy, HIV-related
- Herpes simplex: chronic ulcer(s) (>1 month duration); or bronchitis, pneumonitis, or esophagitis
- Histoplasmosis, disseminated or extrapulmonary
- Isosporiasis, chronic intestinal (>1 month duration)
- Kaposi's sarcoma
- Lymphoma, Burkitt's (or equivalent)
- Lymphoma, immunoblastic (or equivalent)
- Lymphoma, of brain, primary
- *Mycobacterium avium* complex or *Mycobacterium kansasii*, disseminated or extrapulmonary
- *Mycobacterium tuberculosis*, any site (pulmonary or extrapulmonary)
- *Mycobacterium*, other species or unidentified species, disseminated or extrapulmonary
- *Pneumocystis jiroveci* pneumonia
- Pneumonia, recurrent bacterial
- Progressive multifocal leukoencephalopathy (PML)
- *Salmonella* septicemia, recurrent
- Toxoplasmosis of brain
- Wasting syndrome due to HIV
- $CD4^+$ count 200 cells/mL or less

Source: Centers for Disease Control and Prevention. Revised surveillance case definitions for HIV infection among adults, adolescents, and children <18 months and for HIV infection and AIDS among children aged 18 months to less than 13 years—United States, 2008. Morb Mortal Wkly Rep 57(RR10):1–8, 2008.

infections can be avoided. For the critical care nurse, treatment is usually associated with managing the neurological compromise that includes increased intracranial pressure, seizures, and hemiparesis.

Although the early research related to HIV and AIDS was originally conducted in men, the body of knowledge examining HIV and AIDS in women is significant—partially as a result of the natural history study of HIV in women also known as the Women's Interagency HIV Study.[25] Many recent studies, which have included women, suggest that men and women do not differ in terms of the general characteristics of HIV disease, except for the HIV-infected woman's increased risk for cervical dysplasia.[6] The clinical course of infection, including time from HIV infection to AIDS, risk factors for HIV seroconversion, number and type of opportunistic illnesses, protection against infections, and the effectiveness of potent antiretroviral agents, all appear similar in both men and women.

No organ system escapes involvement in HIV infection. Single infections may develop in critically ill patients with AIDS, but patients often have multiple infections simultaneously that require a variety of treatment strategies. The decrease in immune system functioning causes the multisystem manifestations to develop, resulting in an increase in opportunistic infections. Figure 48-3 presents manifestations of HIV infection and AIDS. Box 48-3 lists examples of nursing diagnoses for patients with HIV infection.

Laboratory and Diagnostic Studies

Tests Used to Detect HIV

Several serological tests are used to determine whether a person has been exposed to HIV. The most widely used test for screening is the ELISA, which determines the presence of antibodies for HIV. The results of this rapid and inexpensive test are available in usually less than 1 hour. ELISA screening tests can be performed on either blood/plasma/serum or oral fluid.[20] Results are reported as either reactive (positive) or nonreactive (negative) and have a sensitivity (true positive) rate of 99.5% and specificity (true negative) rate of 99.994%.[20] Unfortunately, the presence of other antibodies may lead to a false-positive result, which means that the test result was HIV positive, but the person is actually HIV negative. With ELISA, a positive/reactive ELISA is always repeated, and if the second ELISA is positive, confirmatory testing with Western blot is required.

Due to the "window period" associated with seroconversion, during the acute HIV infection phase, low production of antibodies at the time the test is performed or recency of infection may lead to a false-negative result. A false-negative result means that the test result was HIV negative but that the person is actually HIV positive; therefore, persons should not be considered HIV negative until there are repeated negative results in a 6-month period. During this "window period," if a person engages in high-risk behaviors associated with HIV, another 6-month window is required to determine the individual's HIV status.

Rapid HIV testing is based on ELISA technology. Instead of sending the specimen to the laboratory and then obtaining results days later, as was standard procedure in the past, rapid HIV testing with ELISA yields results within 20 minutes. Frequently, this type of testing is done in emergency departments (EDs) and primary health care environments including clinics. Current rapid HIV tests can be performed with either serum or saliva samples. Any positive ELISA, regardless of specimen type, still requires confirmatory testing with Western blot. In June 2010, the FDA approved a fourth-generation HIV test. This contemporary HIV test incorporates p24 antigen testing with standard antibody testing and enables earlier and more accurate HIV detection—particularly during the window period.

All positive ELISAs are confirmed by a Western blot. The Western blot analysis is the most widely used confirmatory test and is highly sensitive and specific. The Western blot identifies the presence of antibodies to HIV-1 and/or HIV-2 proteins (depending on the specific assay). The Western blot screens for the following proteins: core (p17, p24, p55), polymerase (p31, p51, p66), and envelope (gp41, gp120, gp160).[20] In analyzing test results from a Western blot, the test is considered to be negative if no bands are present. To be positive, the Western blot results must include gp120/160 and either gp41 or p24. An indeterminate Western blot is the presence of any band not meeting positive criteria.

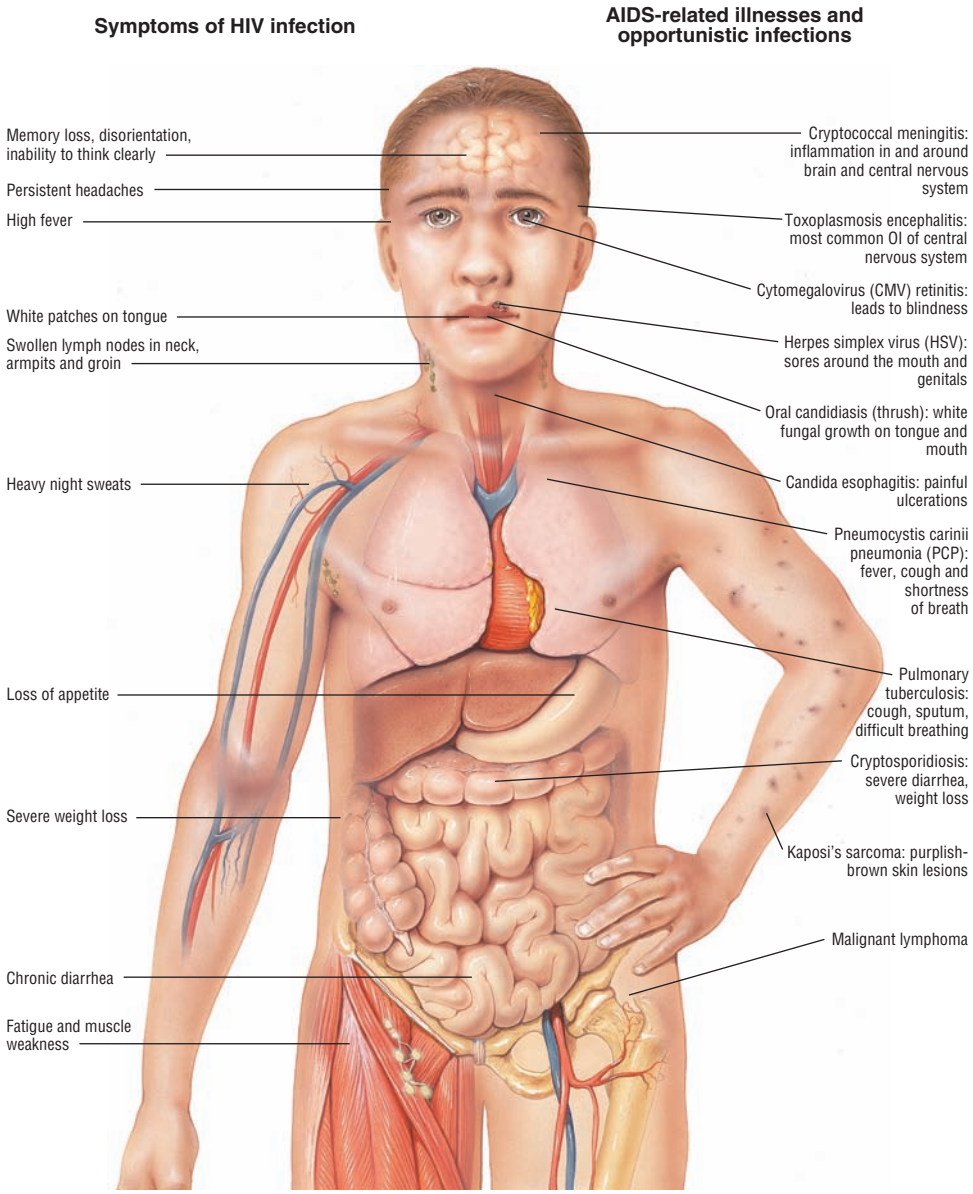


FIGURE 48-3 ▲ Manifestations of HIV infection and AIDS. (From Anatomical Chart Company: Atlas of Pathophysiology, 3rd ed. Springhouse, PA: Springhouse, 2010, p 267.)

Several alternative FDA-approved HIV detection methods are available, but usually are not routinely used in screening adults. The polymerase chain reaction (PCR) test is frequently used in newborns and children to screen for HIV infection. Because it tests for genetic material of HIV rather than antibodies to the virus, it can identify HIV infection at a much earlier stage. Therefore, in adults, this test is frequently done when there has been a high-risk occupational or nonoccupational exposure.

As a consequence of the 2006 CDC recommendations, screening for HIV should be routinely performed in all health care settings for persons between the ages of 13 and 64. Further, all patients initiating treatment for tuberculosis (TB) or seeking treatment for STIs should be routinely screened. All persons likely to be at high risk for HIV should be tested annually. According the CDC, "persons likely to be at high risk include injection-drug users and their sex partners, persons who exchange sex for money or drugs, sex partners of HIV-infected persons, and MSM or heterosexual persons who themselves or whose sex partners have had more than one sex partner since their most recent HIV test."[20](p 7)

Tests Used to Evaluate Progression of HIV Infection

HIV nucleic acid testing, also called viral load testing, in combination with the $CD4^+$ T-cell lymphocyte count is currently the best method available to determine progression on the HIV disease continuum. The viral load measures the amount of viral particles in a cubic mm (mm^3) of blood. The higher the viral load, the more HIV present to cause immune destruction and the more rapid the progression to AIDS.[20] Three methods can be used to determine the viral load: HIV RNA PCR, branched-chain DNA (bDNA), and nucleic acid sequence–based amplification. PCR is the most common method. The test results are used to determine the best time to begin ART and when a change in therapy may be indicated.

BOX 48-3 EXAMPLES OF NURSING DIAGNOSES

For the Patient With HIV or AIDS

- Risk for Infection related to immunodeficiency
- Risk for Impaired Gas Exchange related to
  - Alveolar–capillary membrane changes with *Pneumocystis jiroveci* infection
  - Infectious processes (TB, pneumococcal pneumonia)
  - Acute respiratory distress syndrome
  - Pneumothorax
- Risk for Deficient Fluid Volume related to diarrhea, dysphagia, sepsis
- Anxiety related to critical illness, fear of death
- Social Isolation related to family/community/health care provider knowledge deficit about risk for transmission and HIV-related stigma
- Deficient Knowledge related to antiretroviral therapy, prophylactic medications
- Risk for Decreased Cardiac Tissue Perfusion related to HIV-associated cardiomyopathy, nephropathy
- Risk for Activity Intolerance related to anemia, HIV-associated fatigue
- Risk for Bleeding related to thrombocytopenia, coagulopathy, intracranial hemorrhage

The $CD4^+$ T-cell count and percentage is another important evaluation tool used to stage HIV disease and to make decisions concerning the initiation of ART and prophylactic treatment for opportunistic organisms.[26] The normal $CD4^+$ T-cell count is around 1,000 cells/mm^3 in adults, and the count declines over time in the person with HIV not on treatment (Fig. 48-4). There is an inverse relationship between the viral load and $CD4^+$ T-cell count (Fig. 48-5). As HIV/AIDS progresses, the number of $CD4^+$ T cells declines, and the amount of HIV in the blood increases.

Other tests used to evaluate HIV infection include CBC, rapid plasma reagin (to screen for syphilis), chest radiograph, serum chemistries, Papanicolaou (Pap) tests to screen for cervical cancer in women and to screen for anal carcinoma in both men and women, purified protein derivative skin test (to screen for TB), hepatitis serology (to screen for HBV and HCV), toxoplasmosis serology, and cytomegalovirus (CMV) antibody serology.

▲ Management

Management of patients with HIV disease involves a complex, multisystem assessment, including diagnostic tests that establish a baseline and determine the appropriateness of therapy. Prognosis is based on the type and number of opportunistic infections that occur and the degree of immunocompromise. Patients with multiple opportunistic infections tend to be more seriously immunosuppressed and have a poorer prognosis.

Control of Opportunistic Infection

The primary goal of management in critically ill HIV-infected patients is the prevention or resolution of infections whether opportunistic, community-acquired, and/or health care associated. Opportunistic infections are the leading cause of death in patients with HIV infection; therefore, prevention is the cornerstone of treatment. Treatment of opportunistic infections is aimed at support of the involved system or systems. Treatment guidelines have been developed for prophylaxis against several organisms associated with HIV and AIDS. In April 2009, the *Guidelines for Prevention and Treatment of Opportunistic Infections in HIV-Infected Adults and Adolescents* were published.[26] The current organisms for which prophylaxis is strongly recommended include *P. jiroveci*, *Mycobacterium tuberculosis*, and *Toxoplasma gondii*. Organisms that should be considered for prophylaxis include CMV, *Mycobacterium avium* complex, and varicella-zoster.

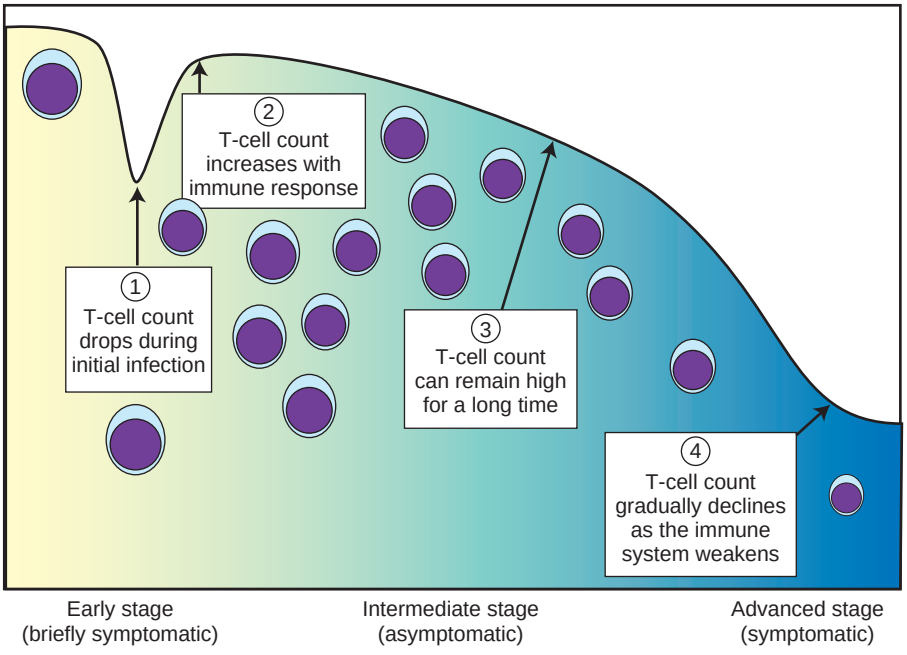


FIGURE 48-4 ▲ Use of T-cell count (CD4) to stage HIV infection. (*1*) The T-cell count drops during initial infection because the virus is destroying the T cells. (*2*) Once the immune system starts to fight back, the T-cell count increases. T-cell counts can go up and down at different times during HIV disease, but they do not return to where they were before infection. (*3*) T-cell counts can remain fairly high for a long time—sometimes for years—but steadily lose ground. (Redrawn from Glaxo Wellcome: HIV: Understanding the Disease. Research Triangle Park, NC: Author, 1995.)

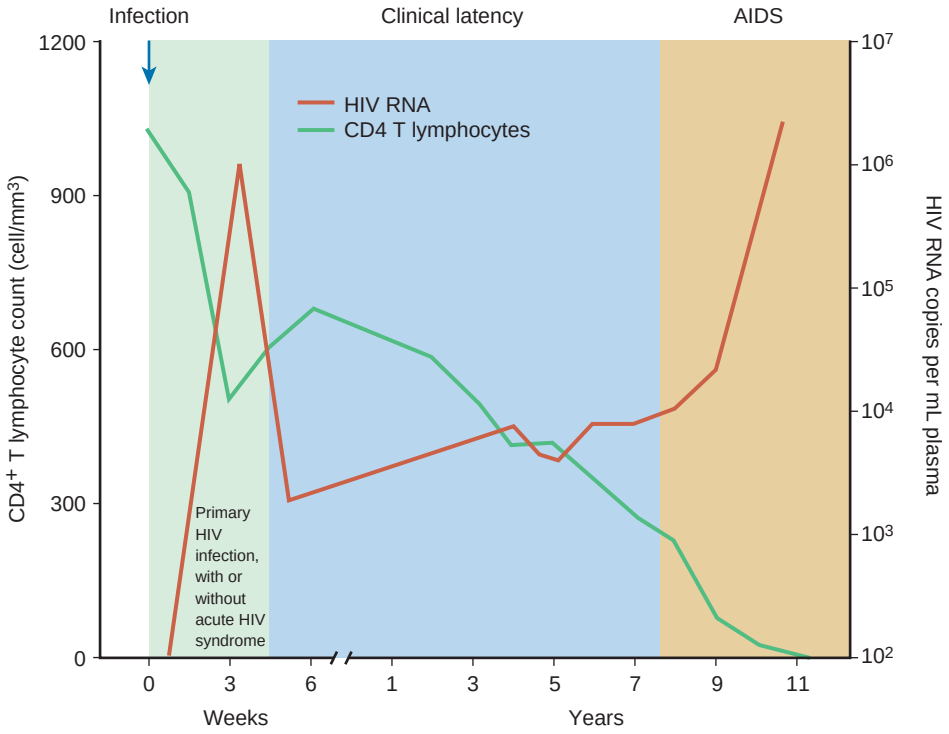


FIGURE 48-5 ▲ Typical course of HIV infection. (From Understanding Disease and Management. Available at: http://www.Roche-hiv.com; modified from Fauci AS, Pantaleo G, Stanley S, et al: Immunopathogenic mechanisms of HIV infection. Ann Intern med 124(7):654–663, 1996.)

Further, vaccination guidelines should be implemented to prevent vaccine preventable diseases including hepatitis A, hepatitis B, seasonal influenza, and *S. pneumoniae*.

Maintenance of safe infection control measures to prevent unnecessary contamination and complications in the ICU are essential when working with patients with HIV infection and AIDS. For current treatment guidelines associated with prophylaxis against common opportunistic infections associated with HIV and AIDS, critical care nurses are encouraged to collaborate with the interdisciplinary team and review data published on the HIV/AIDS pages of the CDC Web site (please refer to http://www.cdc.gov/hiv/default.htm for more information).

The use of ART has had a significant impact on the treatment of opportunistic infections. The number of opportunistic infections has significantly decreased because of advances in ART.[25,26] Prophylactic or suppressive treatment for PCP, toxoplasmosis, CMV infection, M. *avium* complex infection, leishmaniasis, cryptococcosis, and candidal thrush may be discontinued if the patient's $CD4^+$ T-cell count increases sufficiently and is sustained, usually above 200 cells/mm^3.[26] Administration of ART also can rapidly reduce viral load, thereby decreasing the incidence of complications.

Antiretroviral Therapy

Persons living with HIV or AIDS who receive ART and maintain strict adherence can survive for many years (average is 39 years) after being infected with the virus.[6] The classes of antiretrovirals used to treat HIV include the nucleoside/nucleotide reverse transcriptase inhibitors (NRTIs), nonnucleoside reverse transcriptase inhibitors (NNRTIs), protease inhibitors (PIs), fusion inhibitors (FIs), integrase strand transfer inhibitors (INSTIs), and entry inhibitors/CCR5 antagonists.[27] These drug groups act by blocking the fusion of HIV with $CD4^+$ receptors (FIs) or work at different points along the replication cycle (INSTIs, NRTIs, NNRTIs) or inhibit the development of new virions (PIs). Many FDA-approved medications in these classes are available for use (Table 48-3).[15]

The decision to initiate or change a person's therapy is determined by the presence or absence of symptoms along with the degree of immunosuppression that the patient is experiencing and the viral burden. The guidelines are based on the virological, immunological, and clinical status of the patient.[27] In the critical care environment, there is considerable debate about the appropriateness of initiating ART as well as using ART because of the potential for significant adverse effects (Table 48-4) and drug interactions (Table 48-5). Therefore, it is important for critical care nurses to collaborate with the interdisciplinary team, especially the infectious diseases specialist, to obtain parameters for initiating and withholding ART medications when caring for critically ill persons living with HIV or AIDS. In regard to ART, either therapy needs to be fully administered or entirely withheld. If not, there is significant potential for viral resistance to develop.

For some persons with advanced AIDS, initiation of ART can lead to immune reconstitution inflammatory syndrome (IRIS). In this syndrome, an increase of symptoms consistent with an acute inflammatory or infectious process are present but not explained by a new infectious diagnosis. Clinically, a paradoxical worsening or new onset of infectious manifestations usually occurs as a result of improved immune functions and renewed inflammatory responses, an increase in the production of memory and naïve T cells, enhanced lymphoproliferation, increased interleukin-2 responses, and reduced production of some cytokines. The most

Table 48-3 Food and Drug Administration–Approved Antiretroviral Drugs

Generic Name	Brand Name
Entry Inhibitors (EIs) or CCR5 Antagonists	
Maraviroc, MVC	Selzentry
Fusion Inhibitors (FIs)	
Enfuvirtide, T-20	Fuzeon
Integrase Strand Transfer Inhibitors (INSTIs)	
Raltegravir, MK-0518	Isentress
Nonnucleoside Reverse Transcriptase Inhibitors (NNRTIs)	
Delavirdine, DLV	Rescriptor
Efavirenz, EFV	Sustiva
Etravirine, ETR, ETV	Intelence
Nevirapine, NVP	Viramune
Nucleoside/Nucleotide Reverse Transcriptase Inhibitors (NRTIs)	
Abacavir, ABC	Ziagen
Abacavir, lamivudine	Epzicom
Abacavir, zidovudine, and lamivudine	Trizivir
Didanosine, ddI, dideoxyinosine	Videx, Videx EC
Emtricitabine, FTC	Emtriva, Coviracil
Emtricitabine, tenofovir DF	Truvada
Lamivudine, 3TC	Epivir
Lamivudine, zidovudine	Combivir
Stavudine, d4T	Zerit
Tenofovir DF, TDF	Viread
Zidovudine, AZT, ZDV	Retrovir
Protease Inhibitors (PIs)	
Amprenavir, APV	Agenerase
Atazanavir, ATV	Reyataz
Darunavir, TMC	Prezista
Fosamprenavir, FPV	Lexiva
Indinavir, IDV	Crixivan
Lopinavir, ritonavir, LPV/r	Kaletra
Nelfinavir, NFV	Viracept
Ritonavir, RTV	Norvir
Saquinavir, SQV	Fortovase, Invirase
Tipranavir, TPV	Aptivus

Retrieved December 1, 2010, from http://aidsinfo.nih.gov/.
AIDsinfo, A Service of the U.S. Department of Health and Human Services.

frequent infectious processes associated with IRIS are TB, *Mycobacterium avium* complex, cryptococcosis, CMV, herpes zoster, HBV/HCV, and CJD. During IRIS, supportive therapy is indicated and aggressive treatment of the infectious diseases is indicated until the patient is clinically stabilized.

Potent Combination Antiretroviral Therapy

Potent combination ART, also known as highly active ART, became the standard of practice in 1996. According to guidelines published by CDC in October 2011,[27] ART should be initiated in all patients with a history of an AIDS-defining illness (see Box 48-2, p. 1102) or with a $CD4^+$ T-cell count below 350 cells/mm^3. For patients with a $CD4^+$ T-cell count between 350 and 500 copies/mm^3, ART is recommended. In patients with HIV-associated nephropathy and HBV coinfection, regardless of the $CD4^+$ T-cell count, ART should be initiated. For patients with $CD4^+$ T-cell counts about 500 cells/mm^3, the panel was equally divided with 50% favoring starting ART and 50% recommending ART as optional at this stage.

Contemporary evidence indicates that monotherapy with single NRTI, dual-NRTI, and triple-NRTI regimens are not recommended.[27] The three preferred ART regimens for treatment-naïve patients (those who have not previously taken ART) recommended by CDC in October 2011 include:

- Nonnucleoside reverse transcriptase inhibitor + 2 nucleoside reverse transcriptase inhibitors
- Protease inhibitor (preferably boosted with ritonavir) + 2 nucleoside reverse transcriptase inhibitors

Table 48-4 Adverse Events Associated With Antiretroviral Therapy Requiring Intensive Care Unit

Adverse Event	Associated ART
Bleeding events	TPV/r: Reports of intracranial hemorrhage PIs: increased bleeding in hemophiliac patients
Bone marrow suppression	Zidovudine (ZDV)
Cardiovascular effects	Myocardial infarction (MI) and cerebrovascular accident: associated with PI but not NNRTIs use in cohort study MI only: association between recent ABC and didanosine (ddI) use found in observational cohort; association not seen in randomized studies of ABC
CNS effects (may include one or more of the following: drowsiness, somnolence, insomnia, abnormal dreams, dizziness, impaired concentration and attention span, depression, hallucination, exacerbation of psychiatric disorders, psychosis, suicidal ideation)	Efavirenz (EFV)
Hypersensitivity with hepatic failure	Nevirapine (NVP)
Hepatotoxicity	All NNRTIs; all PIs; most NRTIs; maraviroc
Hyperlipidemia	All PIs (except unboosted atazanavir [ATV]); stavudine (d4T); EFV > NVP
Hypersensitivity	ABC
Insulin resistance/diabetes mellitus	Thymidine analogs (ZDV, d4T); some PIs linked to insulin resistance and diabetes mellitus (but unclear if a class effect)
Lactic acidosis/hepatic steatosis ± pancreatitis (severe mitochondrial toxicities)	NRTIs, especially d4T, ddI, ZDV
Nephrolithiasis/ urolithiasis/crystalluria	Indinavir (IDV), ATV, fosamprenavir (FPV)
Nephrotoxicity	IDV, tenofovir (TDF)
Neuromuscular weakness syndrome (ascending)	Stavudine (d4T)
Pancreatitis	ddI alone; ddI + d4T, hydroxyurea (HU), ribavirin (RBV), or TDF; rare reports with Lopinavir/r (LPV/r)
Stevens-Johnson syndrome (SJS)/toxic epidermal necrosis (TEN)	NVP more than DLV, EFV, ETR; also reported with APV, FPV, ABC, DRV, ZDV, ddI, IDV, LPV/r, ATV

Source: Panel on Antiretroviral Guidelines for Adults and Adolescents. Guidelines for the use of antiretroviral agents in HIV-1-infected adults and adolescents. Department of Health and Human Services. 1–167, 2011. Available at http://www.aidsinfo.nih.gov/ContentFiles/AdultandAdolescentGL.pdf. Accessed January 30, 2012.

- Integrase strand transfer inhibitor + 2 nucleoside reverse transcriptase inhibitors

In regard to the treatment-experienced person living with HIV, or those who have previously taken ART, the current understanding of viral dynamics during treatment suggests that "most first-line antiretroviral regimens should be able to suppress virus indefinitely, assuming that the optimal regimen is selected and assuming that the patient can adhere to that regimen indefinitely."[27] (p 65)

Regardless of the combination selected, ART has numerous treatment goals.[27] First, it is essential to maximally and durably suppress the HIV viral load to undetectable levels for as long as possible. Second, with viral suppression, it is then possible to restore and preserve immunological function. Third, with the suppression of the viral load, it is also possible to reduce HIV disease transmission by positive persons. Fourth, with viral suppression and immune reconstitution, it is possible to reduce HIV-associated morbidity and mortality ultimately improving survival. Finally, and equally important, ART can help to improve the quality of life of the person living with HIV or AIDS.

The decision to initiate ART or modify the regimen is complex and based on many factors. It is these issues, as well the significant risk for drug interactions and low trough levels associated with poor absorption, that ARTs are carefully considered in the critical care environment. According to the CDC,[27] (p 38) decisions about ART regimen selection should also be individualized in collaboration with the patient partner and should critically examine a number of factors, including:

- Comorbid conditions (eg, cardiovascular disease, chemical dependency, liver disease, psychiatric disease, renal diseases, or TB)
- Potential adverse drug effects
- Potential drug interactions with other medications
- Pregnancy or pregnancy potential
- Results of genotypic drug resistance testing
- Gender and pretreatment CD4 T-cell count if considering nevirapine
- HLA-B*5701 testing if considering abacavir

Table 48-5 Drugs That Should Not Be Used With Protease Inhibitors, Non-Nucleoside Reverse Transcriptase Inhibitors, or CCR5 Antagonist

Drug Categories										
Antiretrovirals[1,2]	**Cardiac Agents**	**Lipid-Lowering Agents**	**Antimyco-bacterials**	**Gastrointestinal Drugs**	**Neuroleptics**	**Psychotropics**	**Ergot Derivatives (Vasoconstrictors)**	**Herbs**	**Antiretroviral Agents**	**Others**
ATV ± RTV	None	Lovastatin Simvastatin	Rifampin Rifapentine[3]	Cisapride[5]	Pimozide	Midazolam[6] Triazolam	Dihydroergotamine Ergonovine Ergotamine Methylergonovine	St. John's wort	ETR NVP	Alfuzosin Irinotecan Salmeterol Sildenafil for PAH
DRV/r	None	Lovastatin Simvastatin	Rifampin Rifapentine[3]	Cisapride[5]	Pimozide	Midazolam[6] Triazolam	Dihydroergotamine Ergonovine Ergotamine Methylergonovine	St. John's wort	None	Alfuzosin Salmeterol Sildenafil for PAH
FPV ± RTV	Flecainide Propafenone	Lovastatin Simvastatin	Rifampin Rifapentine[3]	Cisapride[5]	Pimozide	Midazolam[6] Triazolam	Dihydroergotamine Ergonovine Ergotamine Methylergonovine	St. John's wort	ETR	Alfuzosin Salmeterol Sildenafil for PAH
LPV/r	None	Lovastatin Simvastatin	Rifampin[4] Rifapentine[3]	Cisapride[5]	Pimozide	Midazolam[6] Triazolam	Dihydroergotamine Ergonovine Ergotamine Methylergonovine	St. John's wort	None	Alfuzosin Salmeterol Sildenafil for PAH
SQV/r	Amiodarone Dofetilide Flecainide Lidocaine Propafenone Quinidine	Lovastatin Simvastatin	Rifampin[4] Rifapentine	Cisapride[5]	Pimozide	Midazolam[6] Triazolam Trazodone	Dihydroergotamine Ergonovine Ergotamine Methylergonovine	St. John's wort Garlic supple-ments	None	Alfuzosin Salmeterol Sildenafil for PAH
TPV/r	Amiodarone Flecainide Propafenone Quinidine	Lovastatin Simvastatin	Rifampin Rifapentine[3]	Cisapride[5]	Pimozide	Midazolam[5] Triazolam	Dihydroergotamine Ergonovine Ergotamine Methylergonovine	St. John's wort	ETR	Alfuzosin Salmeterol Sildenafil for PAH
EFV	None	None	Rifapentine[3]	Cisapride[5]	Pimozide	Midazolam[5] Triazolam	Dihydroergotamine Ergonovine Ergotamine Methylergonovine	St. John's wort	Other NNRTIs	None
ETR	None	None	Rifampin Rifapentine[3]	None	None	None	None	St John's wort	Unboosted PIs, ATV/r, FPV/r, or TPV/r; other NNRTIs	Carbamazepine Phenobarbital Phenytoin Clopidogrel
NVP	None	None	Rifapentine[3]	None	None	None	None	St John's wort	ATV ± RTV other NNRTIs	Ketoconazole
RPV	None	None	Rifabutin Rifampin Rifapentine[3]	Proton pump inhibitors	None	None	None	St John's wort	Other NNRTIs	Carbamazepine Oxcarbazepine Phenobarbital Phenytoin
MVC	None	None	Rifapentine[3]	None	None	None	None	St John's wort	None	None

Source: Panel on Antiretroviral Guidelines for Adults and Adolescents. Guidelines for the use of antiretroviral agents in HIV-1-infected adults and adolescents, p K-17. Department of Health and Human Services. Retrieved March 27, 2012, from http://www.aidsinfo.nih.gov/ContentFiles/AdultandAdolescentGL.pdf

- Coreceptor tropism assay if considering maraviroc
- Patient adherence potential
- Convenience (eg, pill burden, dosing frequency, and food and fluid considerations)

The $CD4^+$ T-cell count is one of the most important markers in determining whether to start treatment.[27] The most recent treatment guidelines recommend initiating ART in all patients with a $CD4^+$ T-cell count less than 350 cells/mm^3 and to those patients who have a history of an AIDS-defining illness (see Box 48-2, p. 1102). ART therapy should also be initiated, regardless of $CD4^+$ T-cell count, in patients who are pregnant, have HIV-associated nephropathy, or are infected with HBV warranting treatment. For patients with a $CD4^+$ T-cell count between 350 and 500 cells/mm^3, ART is recommended.

HIV viral load levels are used to evaluate the effectiveness of ART. Once therapy has begun, a decrease in HIV viral load should occur within 2 to 8 weeks. According to the CDC, "the minimal change in viral load considered to be statistically significant (two standard deviations) is a threefold, or a 0.5 $\log_{10}$ copies/mL change."[27, p. C-6] One key goal of therapy is suppression of viral load to below the limits of detection (below 40 to 75 copies/mL by most commercially available assays). For most individuals who are adherent to their antiretroviral regimens and who do not harbor resistance mutations to the prescribed drugs, viral suppression is generally achieved in 12–24 weeks.[27] Viral load assessment should be repeated every 3 to 4 months to monitor effectiveness.

There are times when the treatment does not achieve the desired effect. This is termed antiretroviral treatment failure. There are three types of treatment failure: virological failure, immunological failure, and clinical progression. Virological failure is defined as a viral load that fails to decrease to nondetectable levels. Immunological failure is defined as a lack of the immune system to increase the $CD4^+$ T-cell count by 25 to 50 cells/mm^3 over the patient's baseline within the first year of treatment or a drop in the $CD4^+$ T-cell count to below the patient's baseline. Clinical progression is defined as the occurrence or recurrence of an opportunistic illness after 3 months of receiving treatment with antiretroviral agents.[27] When failure occurs, virological failure is most commonly the first to develop. Virological failure is typically followed by immunological failure and then clinical progression. The time interval between each is extremely varied and may be several years.

Drug Resistance Testing

Genotypic and phenotypic resistance assays are frequently used in the provision of HIV care to assess viral strains and inform treatment decisions.[27] Genotypic assays assess mutations in relevant sections of the viral genome while phenotypic assays determine the ability of HIV to grow in the presence of different drug concentrations. Genotypic resistance testing is the most commonly used resistance assay in clinical practice. Genotypic assay results can be obtained within a few days after obtaining a blood sample, are more affordable, and have an increased sensitivity for detecting mixtures of wild-type virus and resistant virus.[27] A genotype assay should not be obtained if the viral load is less than 1,000 copies/mm.[27] Genotypic resistance testing helps to reduce the chance that antiretroviral treatment failure will occur since ART is based on identified mutations and individual drug/class resistance profiles.

ONCOLOGICAL COMPLICATIONS AND EMERGENCIES

Oncological emergencies are potentially life-threatening complications that occur as a result of malignancy or its treatment.[1,2] As many as 20% of people diagnosed with cancer have at least one oncological emergency during the course of their disease.[2,3] The incidence of these emergencies increases as patients with cancer live longer and develop complications that relate to progressive or advanced disease.[4,5] The nurse must recognize disease-related and patient-specific risk factors for the development of critical illness and plan appropriate assessment and intervention strategies for the most common oncological emergencies. These emergencies are classified by pathophysiological mechanisms: hematological, anatomical–structural, and metabolic. Hematological complications involving bone marrow dysfunction, such as engraftment syndrome and leukostasis, commonly occur in neoplastic disorders. Disorders related to tumor or treatment-related bone marrow suppression include anemia, bleeding, and infections. Anatomical–structural disorders, such as cardiac tamponade, carotid artery rupture, hepatic veno-occlusive disease, obstruction of the superior vena cava, pleural effusion, spinal cord compression (SCC), and tracheobronchial obstruction, are the result of tumor invasion or treatment-related destruction of normal anatomical structures. Metabolic disruptions from cancer or its treatment, such as hypercalcemia, syndrome of inappropriate antidiuretic hormone (SIADH) secretion, and tumor lysis syndrome, may involve hormone stimulation, procoagulant activity, and electrolyte imbalances.[6]

▲ General Principles in the Critical Care of Patients With Cancer

Patients with cancer present unique concerns for the critical care nurse. A knowledge of preexisting illness, nature of the malignancy, treatment-related considerations, and prognostic implications must be incorporated into patient care.[1,5,7] In addition, it is necessary to appreciate the psychosocial factors related to caring for patients with a chronic disease.[1] Box 48-4 provides guidelines for evaluating an oncological emergency.[1,8,9] Ideally, before any acute event, the oncologist or primary care physician has discussed end-of-life care and the oncological crises that should be treated and those that should not be treated with the patient and family members.[5,10] However, if this is not the case, the critical care nurse is an important liaison between the primary care physician and the intensive care physician. Each time a patient with cancer presents with

BOX 48-4 Guidelines for Evaluating an Oncological Emergency

Symptoms and Signs
1. Are the symptoms and signs related to the tumor or to complications of treatment?
2. How quickly are the symptoms of the oncological emergency progressing?

Natural History of the Primary Tumor
1. Is there a previous diagnosis of malignancy?
2. What is the disease-free interval between the diagnosis of the primary tumor and onset of the emergency?
3. Has the emergency developed in the setting of terminal disease?

Efficacy of Available Treatment
1. Has there been no prior therapy or extensive pretreatment?
2. Should treatment be directed at the underlying malignancy or the urgent complications?
3. Will the patient's general medical condition influence the ability to administer effective treatment?

Treatment and Goals
1. What is the potential for cure?
2. Is prompt palliation required to prevent further debilitation?
3. What is the risk versus benefit ratio of treatment?
4. Should treatment be withheld if there is a minimal chance of response to available antitumor therapies?

Used with permission from Murphy GP, Lawrence W, Lenhard RE: Clinical Oncology. Atlanta, GA: American Cancer Society, 1995, p 597.

critical illness, malignancy-associated prognostic variables and information regarding treatment of the presenting condition should be used to advise the patient of the best course of action.[1,8,9] Clearly there are times when the risk/benefit ratio of a lifesaving measure does not warrant its use, yet there are also many other situations in which a lifesaving intervention in a hopelessly ill patient may significantly enhance the quality of remaining life.[1,8–11] For example, a patient with advanced cancer may present with a potentially life-threatening pericardial effusion that can be effectively treated with insertion of a pericardial catheter. This patient may require a limited amount of intensive care after catheter insertion and fluid drainage, but the symptom relief may be advantageous in enhancing the quality of the last few months of the patient's life. Aggressive management of most oncological emergencies is indicated if a histological diagnosis of cancer has not been established, if the patient has a good prognosis or can achieve prolonged palliation with treatment, or if there is the possibility of restoring functional status.[1,5,7,8,11,12] The guidelines in Box 48-4 are presented as a list of clinical questions that should be considered when deciding whether to provide critical care interventions for the patient with cancer.[8,13]

To provide high-quality, individualized care to patients with oncological emergencies, the critical care nurse should know a few facts regarding critical illness and the patient with cancer. Box 48-5 presents important conclusions drawn from multiple studies of critical illness in patients with cancer.[1,5,7–9,12,14–16] Box 48-6 lists nursing diagnoses for patients with oncological emergencies.

BOX 48-5 Critical Care of Cancer Patients: Conclusions From the Literature

Incidence of Critical Illness
- Affects about 20% of patients
- More common in patients with hematological malignancy
- Most common critical illnesses include respiratory distress, refractory hypotension, and oncological emergencies.

Prognostics of Critical Illness
- Survival is better in the newly diagnosed patient who has not yet received antineoplastic therapy.
- Survival from specific interventions:

Cardiac arrest:	<2% alive to discharge
Mechanical ventilation:	12%–45% alive to discharge (worst prognosis in patients receiving blood and marrow transplant; best prognosis in patients with a newly diagnosed solid tumor)
Dialysis:	21%–40% alive to discharge (prognosis has been improving with use of continuous renal replacement therapies)

- Most important predictor of survival: status of the underlying malignant disease
- Other poor prognostic variables: age extremes, concomitant health problems, severity of cancer, aggressiveness/potency of treatment, and reversibility of the specific crisis.

▲ Hematological Complications

Bone Marrow Suppression

Cancer and its treatment often cause suppression of hematopoietic cell production or differentiation. Causes of bone marrow suppression are commonly associated with cancerous invasion of the bone, chemotherapy and some radiation treatments, or hematopoietic stem cell transplantation. The clinical consequences are symptoms related to decreased red blood cell production (anemia), decreased platelet production (thrombocytopenia), and decreased white blood cell (WBC) production (leukopenia). Table 48-6 is a summary of the key clinical features of these three types of bone marrow suppression.[1,17–19] These disorders are

BOX 48-6 EXAMPLES OF NURSING DIAGNOSES

For Patients With Oncological Emergencies
- Risk for Infection related to impaired or deficient leukocytes
- Risk for Injury related to thrombocytopenia and bleeding
- Risk for Ineffective Peripheral Tissue Perfusion due to reduced erythrocytes, or vascular disruption by tumor
- Impaired Gas Exchange due to cancer involvement of the lungs or alterations in fluid status
- Risk for Imbalanced Fluid Volume due to disease or treatment
- Fatigue due to malignant illness or its treatment
- Impaired Physical Mobility due to disease complications
- Anxiety related to fear of disease or treatment
- Deficient Knowledge related to disease or anticancer therapies
- Ineffective Coping related to severity of illness or prognosis
- Ineffective Role Performance due to chronic serious illness

not uniquely oncological, but they are common in patients with cancer and influence the patient's response to other critical illnesses.

Other causes of bone marrow suppression must be considered if cancer-related etiological factors are not present. When serum tests are unclear in elucidating the etiology of bone marrow suppression, a bone marrow aspirate or biopsy may be performed to confirm whether the pathophysiological process arises in the cell production phase. This test may require sedation and a local anesthetic before a large coring needle is used to remove the liquid red bone marrow from either the hip or sternum. Bone marrow biopsy determines whether the bone marrow defect is present during the cellular production phase, and it may be a basis for clinical management.[1,19] Management of bone marrow suppression as a cluster involves determining whether the cause is time-limited and ascertaining the amount of supportive therapy required. Treatment may include administration of bone marrow growth factors specific to the deficient cellular component,[20] infusion of blood components,[17] or prophylactic clotting enhancement, and antimicrobial therapy to prevent life-threatening bleeding or infectious complications.[17,21]

Engraftment Syndrome

Engraftment syndrome, also known as cytokine release syndrome, cytokine storm, hemophagocytic syndrome, and macrophage activation syndrome, is a recently identified disorder that occurs infrequently in association with the return of bone marrow growth after treatment of hematological malignancies and hematopoietic stem cell transplantation.[22–24] Patients most at risk for engraftment syndrome are women, those with acute leukemia (especially lymphocytic subtype), those who have just had allogeneic hematopoietic stem cell transplantation (especially with human leukocyte antigen (HLA)-mismatched donors), and those who have had transplantation for autoimmune disorders or solid tumors.[22–28]

Table 48-6 Key Clinical Features of Bone Marrow Suppression

Feature	Anemia	Thrombocytopenia	Leukopenia
Definition	General criteria • Hemoglobin <12 mg% • RBC count < 3.0 × 10^6/mm^3 • Hematocrit < 32% Specific to types of anemia • Aplastic anemia • Nutritional anemia • Hemolytic anemia	Classified according to severity of thrombocytopenia and risk of bleeding: • Mild: <100,000/mm^3 • Mild: <1,000/mm^3 • Moderate: <50,000/mm^3 • Severe: <20,000/mm^3	Classified according to severity of leukopenia and risk for infection Decreased granulocytes (granulocytopenia) classified by severity of ANC • Moderate: <500/mm^3 • Severe: <100/mm^3 Decreased lymphocytes (lymphocytopenia) classified by severity of absolute lymphocyte count • Mild: <250 cells/mm^3 • Moderate: <100 cells/mm^3 • Severe: <50 cells/mm^3
Pathophysiology			
Etiology/contributing factors	General • Bone marrow suppression (eg, chemotherapy, radiation to axial skeleton) • Nutritional deficits—iron, protein, B vitamins • Medications (estrogens, allopurinol [Zyloprim]) Aplastic anemia • Congenital disorders (eg, Fanconi's syndrome, maternal ingestion of thiazides) • Viral infection • Medications Nutritional anemia • Iron deficiency • B-vitamin deficiency Hemolytic anemia • Immune hemolysis (viral illness, autoimmune disease) • Sickle cell anemia • PNH	• Bone marrow suppression (eg, chemotherapy, radiation to axial skeleton) • Medications (nonsteroidal anti-inflammatory drugs) • Large-bore intravenous lines (eg, IABP) • High metabolic rate (eg, fevers)	• Bone marrow suppression (eg, chemotherapy, radiation to axial skeleton) • Nutritional deficits • Medications

(continued on page 1112)

Table 48-6 Key Clinical Features of Bone Marrow Suppression (continued)

Feature	Anemia	Thrombocytopenia	Leukopenia
Clinical manifestations	• Due to decreased oxygen carrying and tissue delivery: fatigue, oliguria, chest pain, decreased bowel sounds, and constipation • Due to decreased body insulation and vascular volume: hypothermia, hypotension, orthostasis • Due to compensation for inadequate oxygen delivery to the tissue: tachycardia, tachypnea, cool extremities	• Due to decreased platelet plugging for normal vascular wear and tear: gum oozing, petechiae, occult blood in urine and stool • Related to inadequate platelet response to injury: ecchymoses, hematomas, bleeding around procedure sites, frank hematuria, or gastrointestinal bleeding	Granulocytopenia • Due to decreased phagocytic properties and recognition of invading microbes: fever, pain at site of potential infection, bacterial and fungal infecting organisms (after 7–10 d of granulocytopenia) • Related to diminished inflammatory response: lack of localized erythema, swelling, or exudates Lymphocytopenia • Due to decreased cellular immune responses and recognition of foreign tissue or proteins: tissue anergy to pathogens, (opportunistic and viral infections more common)
Diagnostic tests	General • RBC count • Hematocrit and hemoglobin • RBC morphology Aplastic anemia • Bone marrow aspirate and biopsy Nutritional anemia • Ferritin level • Transferrin level • Total iron-binding capacity • Folate level • Vitamin B_{12} level Hemolytic anemia • Total and direct bilirubin • Erythrocyte sedimentation rate • RBC morphology • Hemoglobin electrophoresis (sickle cell, PNH) • Indium-tagged RBC survival studies	• Platelet count • Bleeding time tests platelet quality to identify whether symptoms may be partly related to platelet function rather than number	• White blood cell count is initial screening tool, but analysis of actual cell count may be helpful • ANC demonstrates the true number of granulocytes available for phagocytic activity • Absolute lymphocyte count demonstrates the true number of lymphocytes available for recognition of foreign tissue and proteins
Common nursing problems	• Fatigue • Activity intolerance • Hypoxemia • Digestion disorders	• Bleeding • Altered body image	• Infection • Risk for hemodynamic instability
Medical management	• Erythropoietin injections • RBC transfusions • Energy conservation	• Interleukin-11 (Oprelvekin) injections • Platelet transfusions • Bleeding precautions • Thrombopoietin (Nplate®)	• Granulocyte colony-stimulating factor (G-CSF) or granulocyte–macrophage colony-stimulating factor (GM-CSF) injections • Broad-spectrum antimicrobial therapy

ANC, absolute neutrophil count; PNH, paroxysmal nocturnal hemoglobinuria; IABP, intra-aortic balloon pump; RBC, red blood cell.

Patients who have had early engraftment after high-dose marrow-ablative treatment are also at risk for engraftment syndrome.[27]

Pathophysiology

Regrowth of bone marrow cells, particularly myelocytes, results in release of inflammatory cytokines that produce vasodilation and capillary leaking similar to sepsis.[27] Lymphocytes and myelocytic precursors engraft the bone marrow earliest, and patients often still appear leukopenic at the onset of symptoms. Patients with engraftment syndrome often present with signs and symptoms similar to infection at a time when their blood counts are still low, and they are equally at risk for engraftment syndrome and severe infection. Therefore, it is extremely difficult to distinguish the two disorders.

Assessment

HISTORY. Engraftment syndrome often begins with fever, total-body erythema or rash, and symptoms of respiratory distress,[27,29] and these symptoms may be the only manifestations. However, many patients exhibit additional signs or symptoms of cytokine effects, such as oliguria or hematuria with elevated creatinine, abdominal discomfort with elevated aminotransferases, and gastrointestinal bleeding.[27–30] The Spitzer criteria for diagnosis includes these symptoms and the presence of the inflammatory marker of increased C-reactive protein.[27] The onset of symptoms is rapid, usually occurring over 24 to 48 hours, and symptoms dissipate after the neutrophils engraft and the WBC count reaches about 2,500 to 3,000/mm.[4,26,27,29] Box 48-7 outlines key clinical manifestations that distinguish sepsis and engraftment syndrome.

DIAGNOSTIC STUDIES. There is no clearly definitive diagnostic test that can differentiate engraftment syndrome from sepsis, which it closely resembles. Even elevations of C-reactive protein are more reflective of inflammation than a true diagnostic test of differentiation.[27] The cornerstones of diagnosis are the constellation of clinical symptoms, subsequent increase in WBC count in patients with previous leukopenia, and absence of a positive microbial culture. New markers for bacterial of fungal pathogens such as beta-D-glucan and procalcitonin are inconclusive at this time.

Management

Engraftment syndrome is managed supportively and conservatively. Patients are presumed septic and treated with broad-spectrum antimicrobial agents. Acetaminophen and diphenhydramine are administered as needed for erythema and pruritus. Hepatic dysfunction requires cautious monitoring and adjustment of fluids and medication doses as appropriate. IV fluids are used to prevent vasodilatory hypotension, but, occasionally, vasoconstricting agents such as phenylephrine or norepinephrine are necessary.[23] Rapid-acting IV corticosteroids have been used effectively when clinical symptoms are strongly suggestive of this disorder.[29] Mechanical ventilation and renal replacement therapy are initiated as indicated, with the understanding and presumption that the syndrome is usually very short-lived.

Complications

The long-term outcome for most patients with engraftment syndrome is excellent, and there are no significant clinical sequelae.[23,26,27,29] Rarely do patients die or have long-term ischemic organ damage. In situations in which negative sequelae have occurred, it has been difficult to determine whether engraftment syndrome or sepsis was the primary pathophysiological process. For example, patients with a rapid onset and progression of respiratory distress syndrome may die of refractory hypoxemia, yet whether this has been caused by undiagnosed and untreated infection or engraftment syndrome cannot be determined.

Leukostasis

Leukostasis is a disorder of excess circulating immature WBCs, resulting in hyperviscosity and microvascular occlusions.[31–34] Cancers such as acute leukemias are the primary cause of leukostasis, although the similar proliferative lymphomas, such as Burkitt's and lymphoblastic lymphoma, have also been reported as triggering malignancies.[31,32] The incidence of symptomatic leukostasis in acute leukemia is estimated to be 5% to 30%,[2,34,35] and its association with high circulating immature WBCs confers a poor prognosis.[31,35,36] Initial mortality in patients with this syndrome is estimated to be approximately 40%.[33,37]

Pathophysiology

Excess numbers of circulating WBCs, such as commonly occur in patients with acute leukemia, can cause a hyperviscosity syndrome that may lead to microcirculatory occlusion with ischemia and vessel rupture. Several types of leukemia can cause elevated WBC counts, with greatest risk with

BOX 48-7 Distinguishing Between Sepsis and Engraftment Syndrome

Sepsis	Engraftment Syndrome
• Fever, variable clinical features	• Fever, sudden onset, often high and continuous
• Variable symptom onset	• Sudden symptom onset over 24–48 h near engraftment period
• Variable skin manifestations	• Erythema with or without pruritic total-body rash
• Dyspnea, often with distinct infiltrates on chest radiography	• Dyspnea; bilateral diffuse alveolar infiltrates on chest radiography
• Thrombocytopenia; occasional mucous membrane bleeding	• Gastrointestinal bleeding
• Hypotension-related oliguria and elevated creatinine	• Unprecipitated oliguria, elevated creatinine, hematuria
• Hypotension-related hepatomegaly, elevated aminotransferases	• Unprecipitated hepatomegaly, elevated aminotransferases

acute monocytic leukemias of the M3v, M4, M5 subtypes, or acute lymphoblastic leukemia with 11q23 chromosome abnormality.[2,38] The immature myelocytes (blasts) found in acute nonlymphocytic leukemia have the greatest propensity for "stickiness" due to adhesion molecules and their interaction with endothelial vessel linings and are most likely to cause leukostasis.[2,33,35] Risk for leukostasis is considered greatest when the WBC count is greater than 100,000/mm^3, although significant clinical symptoms may be present even when counts are in the 50,000/mm^3 range, especially if the WBC count is increasing rapidly or the cells are immature.[2,31,33] Vascular occlusion of the lungs and brain is most common, although coronary artery occlusion, renal failure, and splenic or bowel infarctions have been reported.[32,33,39] Hypoxic vasodilation is thought to worsen the clinical effects of vascular occlusions in the brain.[39,40]

Assessment

HISTORY. Patients with leukostasis usually first present with respiratory or neurological symptoms.[31,34,35] Onset of symptoms is acute (several hours to 1 day). Severe respiratory distress with hypoxemia and inflammatory alveolar infiltrates are the hallmarks of pulmonary involvement. It is difficult to determine the severity of hypoxemia because the immature WBC blasts consume the oxygen in the arterial blood gas (ABG) specimen, making the arterial blood oxygen level appear even lower than suspected based on clinical evidence. Oxygen saturation may be low (eg, 82% to 90%), but ABG oxygen levels may be only 30 mm Hg.[2,34,41] It is believed that immediate icing and rapid transit may reduce but not eliminate this testing problem. Neurological leukostasis presents as mental status changes with clear focal deficits; vascular occlusions cause thrombotic or embolic strokes.[35,39,40] Other clinical findings also associated with a poorer outcome in these patients include older age, monocytic cell line, bilirubin greater than 1.5 mg/dL, serum creatinine greater than 1.2 mg/dL, lactic dehydrogenase more than 2,000 IU, and thrombocytopenia less than 50,000/mm^3.[35]

DIAGNOSTIC STUDIES. Leukostasis may be suspected in high-risk groups, but the diagnosis is primarily made on the basis of clinical manifestations. In many instances, the existence of pathophysiological complications such as infarction or stroke may validate the presumed diagnosis. Patients have specific diagnostic tests performed to assess their presenting symptoms. Chest radiography is often sufficient to diagnose pulmonary leukostasis. A head computed tomography (CT) scan may reveal neurological leukostasis. Ultrasonography and magnetic resonance imaging (MRI) may also be performed. In pulmonary leukostasis, ABG results are used with a clear understanding of their diagnostic limitations.[2,31,38]

Management

In leukostasis, the preferred management is to identify high-risk or early symptomatic patients and perform rapid cytoreduction, such as leukapheresis or high-dose chemotherapy, before the cells cause organ damage.[2,31,32,36,37] Some clinicians have noted that although leukapheresis does not have an impact on mortality, it does notably affect the number of WBCs and their influence on end-organ damage.[32,37,42] Therapeutic leukapheresis removes 20,000 to 40,000 WBCs per treatment, and once- or twice-daily treatments are often required until the WBC count is less than 30,000 to 40,000/mm.[31,33,37,38,42] If leukapheresis cannot be performed immediately, large amounts of IV fluids should be administered to dilute the blood and enhance renal excretion of metabolic toxins.[2] If leukapheresis is still not possible within a 12- to 24-hour period, and the patient's symptoms continue to worsen, exchange transfusions may be necessary.[33,42]

Many patients also receive immediate concomitant chemotherapy to prevent rapid cell regrowth or spontaneous tumor lysis syndrome with renal failure.[31,34] If possible, it is preferred to complete the necessary leukapheresis cycles before starting chemotherapy; however, many patients are too sick for such treatment. When this occurs, the critical care nurse must administer antimicrobial agents or chemotherapy between leukapheresis treatments. Despite some early controversies, chemotherapy may be administered concomitantly with continuous renal replacement therapy (CRRT) as the clearance rate with this dialysis therapy is less than normal kidney clearance so there are no worries that the chemotherapy is being removed by the dialysis treatment.[43] This is not the case with hemodialysis, which does remove many chemotherapy agents. Low-dose cranial radiation (100 to 300 Gy) was once believed to stabilize cell membranes and destroy malignant cells, but this practice is now not recommended.[2,43] Patients with leukostasis receive supportive drug therapy with agents such as antimicrobials, diuretics, bronchodilators, phosphate-binding agents, rasburicase, and allopurinol, which are aimed at stabilizing their symptoms.

It is important to recognize interventions that worsen the hyperviscosity of leukostasis and avoid these actions. Patients with acute leukemia are often anemic, but blood products should be administered with extreme caution and in combination with crystalloid fluids to avoid increased blood viscosity.[2,43] Diuretics may be given to enhance renal excretion of uric acid associated with tumor cells lysis, but also only in combination with crystalloid fluids to maintain normal vascular osmolarity. Supportive interventions to reduce intracranial hemorrhage may include elevating the head of the bed and administering corticosteroids. Definitive treatment requires administration of chemotherapy directed against the leukemia. Box 48-8 lists nursing interventions aimed at reducing the risk for leukostasis-related complications.

BOX 48-8 Nursing Interventions for Leukostasis

- Recognize patients at risk for leukostasis—acute myelocytic leukemia (with circulating blasts), white blood cell count more than 100,000/mm^3, renal dysfunction, dehydration.
- Administer large volumes of intravenous fluids to dilute cells and aid excretion of lysis components.
- Perform cytoreduction with leukapheresis or rapid-acting chemotherapy (eg, cyclophosphamide) as soon as possible.
- Treat organ system–specific leukostatic symptoms (eg, elevation of head of bed, bronchodilators).
- Administer drugs to reduce effects of tumor lysis: phosphate-binding drugs, allopurinol, rasburicase.
- Administer blood components cautiously early in the disease when hyperviscosity is problematic.
- Plan assessment interventions aimed at monitoring for ischemia or infarction of the body organs.

Complications

Even in the face of appropriate, definitive treatment, patients with leukostasis may experience stroke, respiratory failure, bowel infarction, renal failure, or myocardial infarction. In many, some degree of reversible organ ischemia develops, requiring supportive treatment.[31,33]

Typhlitis/Necrotizing Enterocolitis

Pathophysiology

Patients with severe or prolonged neutropenia are at risk for acute gastrointestinal symptoms related to the presence and activity of microbes within the gastrointestinal tract.[21] Resident Gram-negative organisms can produce serious clinical disease in patients without granulocytes. Microbial infiltration of less perfused areas of the bowel, such as the cecum and appendix, produces an acute inflammation of the bowel wall, edema, and paralytic ileus.[21] This has been named neutropenic enterocolitis, which is interchangeable with the term typhlitis.[44] Gases produced by these bacteria can also lead to air in the bowel wall, ischemia, and possible infarction, which is then termed necrotizing enterocolitis or pneumatosis intestinalis. While reported more frequently in children, high-dose marrow suppressing therapy in any individual can lead to typhlitis.[45,46] Although older studies describe incidence rates of 20% to 40% of patients treated for leukemia or undergoing hematopoietic stem cell transplantation, recent reports suggest the incidence may vary from less than 1% to approximately 6% of such cases.[21,45,47] Despite a serious clinical presentation of acute abdominal infection, more than half of patients had negative blood cultures.[45]

Assessment

HISTORY. Patients with prolonged and/or severe neutropenia are at risk for developing typhlitis, although other specific risks include presence of an appendix and prior bowel surgery.[44] Medications that have been linked to increased incidence of typhlitis include carboplatin, cyclophosphamide, cytosine arabinoside, daunomycin, docetaxel, doxorubicin, idarubicin, methotrexate, paclitaxel, pegylated asparaginase, topotecan, and vincristine.[45] Unusual incidents of its occurrence with alemtuzumab,[48] interferon alfa,[49] and pemetrexed[50] have also been reported. Since the consistent administration of oral antimicrobials to sterilize the gut, nonadherence with these medications has also been associated with development of this disorder. Patients present with diffuse abdominal pain that is semilocalized in the right upper or middle quadrant of the abdomen.[51–53] Guarding, abdominal distention, diarrhea, and reduced bowel sounds are common, and rebound tenderness or gastrointestinal bleeding signals a more severe disease that has progressed to bowel ischemia and infarction.[21] Most patients demonstrate symptoms of acute abdominal sepsis such as fever, fluid shifting with oliguria, weight gain, and hypotension, although in antibiotic-controlled disease, these septic symptoms may be intermittent.[21,52,53]

DIAGNOSTIC STUDIES. Initial screening tests include an abdominal flat plate x-ray to detect possible air under the diaphragm, and a CT scan with contrast, while also evaluating lactic acid and amylase levels for signs of bowel ischemia.[21,44,54] An enlarged and edematous cecum is considered diagnostic, although other tests are better at predicting the severity of bowel wall injury or ischemia.[42,52,54]

Management

Since recognition of this complication and its pathophysiological origin, many clinicians have undertaken two primary preventive measures. First, high-risk patients are administered oral antimicrobial antibiotics (usually in liquid form) that destroy intestinal bacteria, producing sterile conditions within the bowel.[44] Secondly, patients with anticipated severe neutropenia are administered prophylactic hematopoietic growth factors, unless acute myeloid leukemia where there is concern the growth factors may enhance tumor growth.[21] These patients may perhaps then have reduced neutropenia. In patients who develop less severe typhlitis, potent broad-spectrum antimicrobials aimed to destroy a variety of both Gram-positive and Gram-negative bacteria, gut rest, and observation may be warranted and adequate until the patient's own WBCs repopulate their bone marrow and can destroy an existing infection.[44,45,53] Throughout the continuum of this complication, lactic acid levels are followed and IV fluids are administered to maintain gut perfusion.[49,55] Vasopressor agents are avoided if at all possible. In more serious cases, granulocyte transfusions have been administered in an attempt to boost the patient's immune activity.[21] In severe, sepsis-related disease or pneumatosis, emergent surgical resection may be necessary.[49,51,55]

Complications

This continuum of bowel wall abnormalities may lead to acute bowel perforation, bowel infarction or gastrointestinal bleeding.[51,55] These serious consequence often herald poor outcomes as the patients are also too high of risk to consider surgical resection of the bowel, even though this intervention may be the only potentially effective measure to arrest the sepsis.

▲ Anatomical–Structural Complications

Cardiac Tamponade

Cardiac tamponade is the result of accumulation of excess pericardial fluid or the presence of a tumor that compresses the heart. At autopsy, as many as 20% of people with cancer are found to have cardiac or pericardial metastases.[56]

Pathophysiology

The pericardium is a double-walled sac that surrounds the heart and great vessels. A visceral layer lines the surface of the heart, and the parietal layer (or outer layer) moves freely. The pericardium supports the heart in a stable position and provides a frictionless sac for cardiac contractions. The pericardial cavity lies between the two layers and contains 10 to 50 mL of serous fluid.

Neoplastic cardiac tamponade results from the formation and accumulation of excessive amounts of fluid in the pericardial sac. This emergent condition may also be caused by encasement of the heart by tumor or postradiation

pericarditis. The severity of the tamponade is in direct proportion to the rate of fluid formation and the volume of fluid accumulated. Slow accumulation may stretch the pericardium so that cardiac contractility is not adversely affected for months.[56,57] Normal diastolic filling is impaired by elevated pericardial pressures, and stroke volume is reduced. As stroke volume continues to fall, hypotension, compensatory tachycardia, and equalization and elevation of the mean left atrial, pulmonary arterial and venous, right atrial, and vena caval pressures occur. In an attempt to maintain arterial pressure, increase blood volume, and improve venous return, tachycardia and peripheral vasoconstriction develop. If the tamponade goes undiagnosed or untreated, circulatory collapse ensues.

Cancers of the esophagus or lung grow by direct extension into the pericardium, whereas distant primary cancers (eg, renal cell) metastasize to the pericardium through the bloodstream. Large chest tumors may also cause pericardial effusion due to lymphatic obstruction of pericardial fluid recirculation. The primary tumors most commonly associated with pericardial effusion are tumors of the breast, lung, or esophagus; lymphoma; gastrointestinal carcinomas; melanoma; sarcoma; and leukemia.[58–60] Radiation pericarditis may be a causative factor, especially if the patient's heart was in the treatment field and if the total dose of radiation to this field exceeded 4,000 rad (40 Gy).[61] Biotherapeutic agents such as interleukin-2 (Aldesleukin) and interferon-alpha cause increased capillary permeability and clinically significant pericardial effusions.[62] Pericardial effusion has also been reported in patient receiving arsenic trioxide,[57,63,64] cyclophosphamide,[64] and cytosine arabinoside.[57]

Assessment

HISTORY. Signs and symptoms reflect the rapidity with which the fluid accumulates in the pericardial sac and, in the patient with cancer, are mainly those of right-sided heart failure due to slow accumulation.[20] Signs of tamponade include rapid, weak pulse; distant heart sounds; distended neck veins during inspiration (Kussmaul's sign); pulsus paradoxus (inspiratory decrease in arterial blood pressure of >10 mm Hg from baseline); ankle or sacral edema; edema; ascites; hepatosplenomegaly; hepatojugular reflex; lethargy; and altered level of consciousness.[56,65–67] The patient may complain of dyspnea, cough, and retrosternal pain that is relieved by leaning forward. On occasion, a patient with a large effusion experiences epigastric pain, hiccups, hoarseness, nausea, and vomiting.[56]

DIAGNOSTIC STUDIES. A variety of studies are used to determine the presence and severity of cardiac tamponade. A chest film is used to determine the presence of cardiac enlargement, mediastinal widening, or hilar adenopathy.[68] The electrocardiogram (ECG) may show nonspecific abnormalities, including low QRS complex voltage in limb leads, sinus tachycardia, precordial ST-segment elevations, and T-wave changes.[56] Echocardiography is the most sensitive and most specific noninvasive test for the presence of tamponade and is used routinely in most settings.[65,69] Two distinct echoes may be identified, one from effusion and the other from the posterior heart border. Spaces between these echoes indicate the size of the effusion or the thickness of the pericardium. Catheterization of the right side of the heart reveals pericardial tamponade or constriction but is performed infrequently because echocardiograms are routinely available.[70] Clarification of specific pathological features and tumor involvement of the pericardium itself cannot be determined by echocardiogram and may require CT or MRI imaging.[65,71,72] Pericardiocentesis gives a positive cytological result in the patient with metastatic cancer.[73,74]

Management

First, volume expansion is necessary because it increases venous pressure so that it is greater than the pericardial pressure, allowing increased venous return and improved cardiac output.[56] Oxygen administration is required, although assisted or mechanical ventilation may increase thoracic pressures, further impeding venous return and worsening the tamponade.

The definitive treatment for pericardial effusion is fluid drainage. Acute or life-threatening symptoms are indications for emergent pericardial drainage by needle or catheter pericardiocentesis (Box 48-9). Without definitive treatment to alleviate the fluid in the pericardium, cardiac arrest occurs. Tamponade is likely to recur in 24 to 48 hours if treatment to prevent pericardial fluid reaccumulation is not initiated quickly.

Factors that clinicians should consider when selecting a therapeutic option include the sensitivity of the primary tumor to specific treatment modalities, previous treatment, and the patient's life expectancy.[58] If effective drugs are available (eg, as in lymphoma and small cell lung cancer), systemic chemotherapy may be initiated after the patient is clinically stable.[62] This treatment may also be effective in patients with leukemia, lymphoma, and breast cancer who have pericardial effusion. In radiosensitive tumors such as lymphoma and breast cancer, radiation therapy may be the treatment of choice.[68] Research has shown that radiation therapy may control more than 50% of malignant pericardial effusions.[75] Insertion of a pericardial catheter guided by fluoroscopy or echocardiography to permit rapid fluid drainage is often the preferred immediate treatment.[71,75] The catheter may remain in place while anticancer therapy begins. Pericardial sclerosis through the pericardial catheter, which is rarely used, can control tamponade by causing adherence of the two pericardial layers and inhibiting fluid accumulation.[58,75,76] Intrapericardial chemotherapy with agents such as cisplatin[74] and bleomycin[60] has also been infrequently reported. Intraoperative fluid drainage or shunting has been used with moderate success in extensive malignant disease.[77,78] In patients with a longer life expectancy and adequate performance status, an inferior pericardiotomy may be performed thoracoscopically. In this procedure, a pleural–pericardial window is created, which provides immediate relief of cardiac compression and tissue specimens for histo-

BOX 48-9 PATIENT SAFETY

Signs and Symptoms of Neoplastic Cardiac Tamponade

- Cyanosis, dyspnea with hypoxemia, impaired consciousness, or shock
- Pulsus paradoxus exceeding 50% of the pulse pressure
- Decrease of more than 20 mm Hg in pulse pressure
- Central venous pressure exceeding 13 mm Hg

logical diagnosis. Fewer than 5% of patients have recurrence of symptoms after this procedure. Pericardectomy is necessary if radiation-induced pericardial disease is not responsive to conservative medical management. This procedure should not take place if an extensive pericardial tumor is present because surgical morbidity and mortality rates are high.[79]

Carotid Artery Rupture

Causes of a carotid artery rupture (or "blowout") are tumor erosion and rupture of the carotid artery. Such rupture results in the loss of large amounts of blood that, without rapid intervention, becomes life-threatening hemorrhage. Patients at risk for this oncological emergency are primarily those with cancer of the head and neck, especially after surgery or radiation, or with a wound infection.[80–82] Affected patients occasionally have thyroid cancer, lymphoma, or melanoma.[80,81] Patients with a palpable pulse on top of a tumor, or in close proximity to it, have a greater risk for carotid vessel erosion.[80,81]

Pathophysiology

Rupture of a carotid artery is likely to occur when that vessel is weakened by invasion of tumor or by surgical manipulation. Other causes of vessel weakness include simultaneous infection or skin flap necrosis.

Assessment

The rupture of the artery may occur suddenly with forceful expulsion of large volumes of blood from the damaged vessel; however, the first sign of erosion or rupture usually is a small trickle of blood from the neck area or unexplained oral bleeding.[80] If the skin over the artery is intact, the patient may have darkened or ecchymotic skin changes, swelling, difficulty swallowing or breathing, retrosternal or high epigastric chest pain, and mental status changes. A unilateral headache or visual disturbance may also signal carotid artery bleeding. Box 48-10 lists the cardinal signs and symptoms of carotid artery rupture.

Management

Patients identified at high risk for carotid artery rupture may have vascular stents placed during surgery as a preventive measure. In addition, IV access should be in place, and blood should be typed and available for immediate transfusion.[81,83] Gauze, irrigation saline solution, and vascular clamps should be readily available at all times. The first emergency intervention in cases of suspected carotid artery rupture is constant digital pressure with saline-soaked cotton dressing wrapped around the two middle fingers and applied directly to the area over the artery.[84] The nurse must not lessen pressure to see whether the bleeding has stopped or attempt to apply a hemostat. Either of these steps increases the likelihood of further blood loss. Maintenance of the airway is essential. Only after the patient is in the operative suite and the operative area has been prepared should the pressure be released. The surgical treatment of choice is ligation of the damaged artery. Embolization or stent placement may be alternatives.[84,85] Chapter 22 contains a detailed discussion of carotid artery surgery with assessment and nursing care.

BOX 48-10 PATIENT SAFETY

Cardinal Signs and Symptoms of Carotid Artery Rupture

- Oozing blood from neck wound
- Unexplained oral bleeding
- Ecchymoses over neck region
- Sudden neck edema
- Retrosternal or epigastric chest pain
- Sense of impending doom, anxiety, or restlessness

Complications

The overall mortality rate of carotid artery rupture is 40% to 60%.[80] About 60% of patients who survive this complication have long-term neurological deficits, the most common of which is hemiparesis. The risk for hemiparesis is reduced by the prevention of shock and replacement of fluid for adequate perfusion of the brain through the opposite internal carotid artery.[80]

Hepatic Veno-Occlusive Disease (Sinusoidal Obstruction Syndrome)

Hepatic veno-occlusive disease, also known as sinusoidal obstruction syndrome, is occlusion of the venous vessels of the liver. The disease is a complication of high-dose radiation therapy and chemotherapy.[86,87] Its incidence is as low as 5% to 10% in patients receiving some chemotherapy[88] and monoclonal antibody regimens but as high as 30% to 40% in some receiving hematopoietic stem cell transplants.[88,89] Although sinusoidal syndrome is most likely to develop in patients receiving high-dose alkylating agents (eg, cyclophosphamide, busulfan) or abdominal radiation, it also occurs in patients receiving the leukemic monoclonal antibody gemtuzumab (Mylotarg)[86,90] and oxaliplatin (Aloxatin).[91] Other risk factors in patients with cancer are extensive pretreatment, older age, and previous history of hepatitis.[90]

Pathophysiology

Through uncertain mechanisms, etiological agents cause fibrotic changes in the endothelial layer that lines the walls of the veins and sinusoids in the liver, resulting in narrowed and stiff-walled venules that have a tendency for thrombosis. Venous flow through the liver is reduced, and there is congestion and eventual pressure-related hepatic damage.[87,88]

Assessment

HISTORY. The earliest manifestations of hepatic veno-occlusive disease are fluid retention, elevated serum bilirubin, and nonspecific abdominal pain.[88,89] The onset of these symptoms occurs an average of 8 to 20 days after therapy; the time varies with the causative agent.[88] The clinical course begins primarily as one of portal hypertension with ascites, painful hepatomegaly, and right-sided heart failure; it progresses over 1 to 3 weeks to include hepatic destruction with coagulopathies, thrombocytopenia, hyperammonemia, metabolic alkalosis, increased vagal tone, and hepatorenal failure.[88,89] Box 48-11 lists early and late clinical findings in hepatic veno-occlusive disease. Most patients with hepatic veno-occlusive disease have mild, reversible disease, and only 10% to 20% have severe, life-threatening manifestations.[88]

BOX 48-11 PATIENT SAFETY

Signs and Symptoms of Hepatic Veno-Occlusive Disease

Early Findings

- Weight gain
- Fluid retention, edema
- Painful hepatomegaly
- Increased total and direct bilirubin
- Increased aspartate aminotransferase and alkaline phosphatase

Late Findings

- Coagulopathies, thrombocytopenia
- Hyperammonemia
- Metabolic alkalosis
- Hepatorenal syndrome
- Right-sided heart failure
- Elevated aminotransferases
- Increased vagal tone

DIAGNOSTIC STUDIES. The first and most specific diagnostic test is the elevation of total and indirect bilirubin.[88] Aspartate transaminase (previously known as serum glutamic oxaloacetic transaminase) and alkaline phosphatase also increase early, and when progressive liver failure develops, hepatic aminotransferases also increase. Abdominal ultrasonography confirms hepatic enlargement and is used to rule out causal conditions, such as cholestasis and hepatic abscess.[88] The new addition of ultrasonographic Doppler technology can provide more conclusive evidence of hepatic veno-occlusive disease with validation of venous wall turgidity and estimated portal pressures.[89]

In late or severe disease, the platelet count decreases, and coagulation studies such as prothrombin time or partial thromboplastin time are prolonged. A definitive diagnosis may be made only on the basis of liver biopsy. When it is necessary to differentiate hepatic veno-occlusive disease from other clinically similar processes, such as graft-versus-host disease, liver biopsy shows venule fibrosis.[92]

Management

Because the pathological mechanisms of hepatic veno-occlusive disease are uncertain, therapy is still presumptive and not clearly effective. Once hepatic veno-occlusive disease is suspected, supportive therapies, such as balancing fluid administration and diuresis, are implemented.[93] Transjugular intrahepatic portosystemic shunt procedures have been used in an attempt to enhance portal blood outflow.[93] Patients may require platelet and fresh frozen plasma transfusions, vasopressors, and ammonia-lowering therapies, such as lactulose.[94] Researchers have noted modest reports of successful symptom resolution with high-dose methylprednisolone, glutamine with high-dose vitamin E, ursodiol, activated factor VII, defibrotide, and tissue plasminogen activator.[93,95] Defibrotide, an oligonucleotide derived from porcine intestinal mucosa that demonstrates simultaneous antithrombotic properties with microvascular protective features, producing minimal hemorrhagic risk, is the most promising and well-studied agent.[96,97] Renal replacement therapy is often required; continuous venovenous hemofiltration is often the preferred method of therapy because these patients exhibit increased vagal tone and a tendency for vasodilatory hypotension. Some experts advocate early implementation to preserve renal function and reduce the need for respiratory support.[87] Before the advent of CRRT, mechanical ventilation to control fluid imbalance–induced respiratory distress was often necessary.

No methods to prevent hepatic veno-occlusive disease have yet proved successful. Results of studies of low-dose heparin (subcutaneously or intravenously), prostaglandin E_1, and defibrotide as potential preventive agents have so far been inconclusive.[88,98,99]

Complications

Patients with mild to moderate hepatic veno-occlusive disease experience complete reversal of the pathological process. It is uncertain whether supportive therapies have any influence on this outcome. Well-controlled studies clearly show that mortality is high in patients with total bilirubin levels greater than 15 to 18 mg/dL, renal dysfunction, high fibrinogen D-dimers, or a hepatic pressure gradient of greater than 20 mm Hg.[90,93]

Superior Vena Cava Syndrome

Superior vena cava syndrome (SVCS)—obstruction of the superior vena cava—results in venous blockage that produces pleural effusion and facial, chest, arm, and neck edema.[100,101] Severe obstruction results in impaired cardiac filling, and the venous congestion has also been associated with tracheal obstruction and cerebral edema.

Pathophysiology

The superior vena cava is a thin-walled, low-pressure blood vessel in the mediastinal cavity that collects blood from the venous vessels that drain the head and neck and the upper thoracic cavity. The mediastinum is a rigid anatomical structure that contains the trachea, the vertebral column, the sternum and ribs, and the lymph nodes.

Most cases of SVCS result from mediastinal malignancies or involved lymph nodes that cause extrinsic compression or invade the vessel.[100–102] More than 75% of cases are secondary to small cell or squamous cell lung cancers, and 10% to 15% are secondary to mediastinal lymphomas.[100–104] Obstruction of the vessel lumen by a thrombus may also occur; it is most commonly caused by a central venous catheter or a hypercoagulability syndrome due to cancer.[100–102,105] Box 48-12 summarizes the risk factors for SVCS.

BOX 48-12 Risk Factors for Superior Vena Cava Syndrome

- Chest, neck, or epigastric tumors (eg, lung cancer, breast cancer, lymphoma, head and neck cancer, thyroid cancer, gastric cancer, esophageal cancer, pancreatic cancer, metastatic renal cell cancer, metastatic colorectal cancer, melanoma)
- Devices in the superior vena cava (eg, large-bore, multilumen central lines, especially if placed in the subclavian site)
- Hypercoagulability syndromes (disseminated intravascular coagulation, hypercoagulability of malignancy [eg, mucin-producing adenocarcinomas, brain tumors, Trousseau's syndrome])

Assessment

HISTORY. Signs and symptoms of SVCS depend on the rapidity of compression of the superior vena cava. If it is compressed gradually and collateral circulation develops, indications of SVCS may be more subtle.[102,104] Initial symptoms are most prominent in the early morning and include periorbital and conjunctival edema, facial swelling, and Stokes' sign (tightness of the shirt collar).[101] These signs may disappear after the patient has been upright for a few hours. The patient may also complain of visual disturbances and headache. Altered consciousness and focal neurological signs may result from brain edema and impaired cardiac filling. Late signs and symptoms include distention of the veins of the thorax and upper extremities[104] dysphagia, dyspnea, cough, hoarseness, and tachypnea. All patients, including children, most commonly visit health care providers because of dyspnea.[101,103] Pleural effusions are present in approximately 60% of cases, compounding respiratory symptoms and providing a complex dimension for treatment planning.[106] Most pleural effusions are transudative and related to obstruction of pleural and lymphatic outflow.

DIAGNOSTIC STUDIES. Until recently, diagnostic evaluation of SVCS required multiple tests to validate the location, size, and vena cava involvement of tumors or thrombus. Conventional chest CT with IV contrast, venography, angiography, and radionuclide scans were necessary. Currently, the spiral CT scan with contrast, which provides accurate information about tumor location and involvement of the vena cava, may be the only diagnostic test performed.[107] However, biopsy or cytological tests may be required to establish a diagnosis in many patients because this syndrome is the presenting symptom at the time of diagnosis of cancer.[108]

Management

The primary treatment of choice for SVCS caused by a tumor is radiation therapy. Dosage depends on the size of the tumor and its radiosensitivity. Radiation therapy is initially given in high daily fractions (total dose of 30 to 50 rad) for 14 to 21 days, and symptom relief occurs in 7 to 14 days.[101] Radiation therapy is palliative for SVCS in 70% of patients with lung cancer and for more than 95% of patients with lymphoma.[103] Radiation of the mediastinal, hilar, and supraclavicular lymph nodes and any adjacent parenchymal lesions is appropriate in patients with locally advanced non–small cell lung cancer.

Patients who receive radiation therapy experience increased cough within 3 days of the start of therapy. During the initial 7 to 10 days, secretions are increased because of inflammation, but a dry irritation then develops, resulting in a dry, hacking cough with few secretions but possible bleeding.[100] Chemotherapy may be the treatment of choice for SVCS in patients with disseminated disease, such as small cell anaplastic carcinoma or lymphoma. The agents used most often include high-dose regimens containing cyclophosphamide, cisplatin or carboplatin, bleomycin, etoposide, and doxorubicin.[100,103] The most common adverse effects of these agents include bone marrow suppression, cardiac toxicity, and renal dysfunction.

Treatment of SVCS caused by a thrombus around a central venous catheter may include antifibrinolytics or anticoagulants and possibly surgical removal of the catheter.[109] In any case, chest and neck central venous catheter placements should be avoided until effective treatment has been delivered.

In some circumstances, the placement of stents or vascular grafts in the superior vena cava provides immediate symptomatic relief while patients receive definitive therapy.[108,110–112] It is unclear whether long-term anticoagulation is required. Caution must be taken and intensive observations are made to enhance early detection of bleeding as the tumor shrinks.[113] Supportive care is essential. Maintenance of a patent airway is of the highest priority.[100] Because many patients have severe dyspnea, they are unable to lie flat for their radiation therapy, and short-term airway intubation may be necessary. Clinicians may prescribe oxygen therapy, diuretics, steroids, and heparin, and their administration requires careful observation of patient response. If necessary, administration of corticosteroids for 3 to 7 days to decrease the edema associated with the disease and treatment is warranted.[108] The nurse teaches the patient not to bend over and to avoid Valsalva maneuvers. When the patient is in bed, the head should be at least in a semi-Fowler's position. Elevation of the arms on pillows helps alleviate swelling; however, elevation of the legs is not helpful because this increases fluid volume in the torso.

Complications

Several complications may occur in patients with SVCS. Right-sided heart failure is the most common.[100] Such heart failure is usually self-limiting and is treated symptomatically with fluid restrictions, diuretics, and digoxin. Vessel rupture in SVCS when a tumor invades the vena cava is a great risk because the tumor shrinks with treatment. The incidence of vessel rupture is highest in patients with esophageal and lung cancer; peak incidence is 3 to 4 weeks after initiation of therapy.[100] Warning signs of vessel rupture are acute and sudden dyspnea, hypoxia, cough, and vascular collapse. Radiation pneumonitis, an inflammatory response in the radiation field that correlates with breath sound and radiographic changes reflective of alveolar capillary permeability, may occur 2 to 8 weeks after start of therapy in patients who receive chest radiation for SVCS.[100] Treatment of radiation pneumonitis involves corticosteroids and supportive therapy. SVCS recurs in 10% to 30% of patients.[101]

Pleural Effusion

Pathophysiology

There is normally 30 to 150 mL of fluid between the visceral and parietal pleura that helps maintain a negative pleural pressure to facilitate lung expansion with minimum work of breathing. A pleural effusion is excess accumulation of fluid in the pleural space with subsequent impaired lung expansion and hypoxemia. When lymphatic obstruction (particularly of the thoracic duct), venous congestion, pleural inflammation, or excess capillary permeability occurs, the amount of fluid increases or does not drain properly.[114]

Although many nonmalignant conditions (eg, congestive heart failure, hypothyroidism) may cause pleural effusion, malignant conditions involving lymphatic obstruction or infiltration with malignant cells may also have the same result. Pleural effusions that result from volume overload, capillary permeability, or lymphatic obstruction produce a transudate characterized by the presence of albumin and the

absence of cell fragments or enzymes in the pleural fluid.[115,116] Malignant cell infiltration or pleuritic infection causes pleural inflammation and exudates characterized by the release of red blood cells, WBCs, and lactate dehydrogenase into the pleural fluid.[116] As many as 50% of patients with cancer experience pleural effusions during the course of their disease, particularly in cancers of the lung or breast.[114,117,118] The presence of pleural effusion is associated with a shorter life expectancy, averaging 3 to 12 months after diagnosis.[117,119,120] Predictors for better outcome include specific types of cancer and patients with higher performance status at diagnosis of effusion.[120]

Accumulation of pleural fluid leads to increased (more positive) pleural pressure. Higher pleural pressures increase the work of breathing, and collapsed alveoli cause decreased gas exchange and hypoxemia.[114]

Assessment

HISTORY. The clinical findings in pleural effusion are related to the two major physiological mechanisms: increased work of breathing and alveolar collapse. Excess pleural pressures decrease lung compliance ("stiff lungs"). Patients feel short of breath and must use their accessory muscles to breathe, and chest excursion on the affected side is reduced. When patients are in an upright position, the force of gravity pulls down the fluid, and breath sounds are diminished to the level of fluid. The pleural fluid takes up space in the chest, impeding lung expansion with consequent alveolar collapse. Symptoms that relate to this pathological process are diminished breath sounds, unequal chest excursion, tracheal shift away from the effusion, and signs of hypoxemia (eg, dyspnea, anxiety, confusion, oliguria, decreased bowel sounds).[114,118]

DIAGNOSTIC STUDIES. The first diagnostic test performed to confirm the presence of pleural effusion is an upright chest radiograph or CT.[121,122] The fluid accumulates in the lower lung, causing a blunted diaphragmatic dome and decreased radiolucence in the lower lung. Fluid accumulation often produces a meniscus of decreased radiolucence and a thickened lateral pleural lining, indicating fluid tracking up the side.[121] When alternative diagnoses such as hemothorax, infection, or tumor infiltrates are possible, the CT scan may be more accurate. After a pleural effusion is confirmed, a cytological evaluation, which involves extraction of a sample of fluid and sending it for fluid chemistry and cytology, is necessary. Pleural fluid is categorized as transudative or exudative, which provides clues to the cause of the effusion. Cytological studies require at least 50 mL fluid to confirm the presence or absence of malignant cells; the results of which influence treatment decisions.[123,124] Multiple specimens may be necessary to confirm malignancy; it is estimated that cytology may be positive only 30% of the time when malignant cells are present.[122,123] When fluid cytology is inconclusive, pleural biopsy may be helpful in diagnosis of malignant infiltration.[125]

Management

The treatment of pleural effusion depends on the etiological mechanism, rapidity of symptom onset, and degree of respiratory compromise.[114,126] Because many patients with malignant pleural effusion have limited survival, the selection of treatment that enhances quality of life with minimal time required for recovery is optimal. When pleural effusions are small or have a nonmalignant cause, observation without definitive treatment may be indicated. Aggressive antineoplastic therapy may be indicated when a large tumor causes lymphatic obstruction, heart failure, or pneumonitis that in turn causes pleural effusion.

When malignant cells are present in the pleural fluid, management may be determined by overall treatment goals. Repeated therapeutic thoracenteses are often the preferred initial choice; this assumes that the ultimate cause of the pleural effusion is being treated or that the patient's life expectancy does not warrant more interventional measures.[114,118,122,126] When the patient's life expectancy is longer, and pleural effusions do not resolve with anticancer therapy and intermittent thoracenteses, treatment includes long-term chest catheter drainage or pleurodesis. Long-term pleural drainage via a soft tunneled catheter (eg, Pleurex® catheter) allows patients to have a means of draining excess fluid while remaining at home.[127–131] When drainage slows and patients become a good candidate for pleurodesis, they are admitted to the hospital for this procedure. Alternatively, patients may be admitted for placement of a traditional chest catheter and once drainage slows, pleurodesis may be performed. Pleurodesis, also called pleural sclerosing, involves intrapleural administration of a chemical (eg, doxycycline, bleomycin) or a mechanical agent (eg, talc slurry) to alter the pH of the pleural fluid and cause inflammatory adherence of the visceral and parietal pleura to each other.[123] Sclerosed pleura do not have the normal lubricating pleural fluid, and restrictive lung disease is the long-term consequence. Pleurodesis is successful only about 67% of the time, necessitating the availability of additional treatment options. Box 48-13 presents nursing interventions related to managing pleurodesis.

Pleurectomy is a thoracic surgical procedure that removes the entire pleura. Pleurectomy is effective but can be difficult to perform when long-term inflammation and pleurodesis attempts cause a friable pleura that

BOX 48–13 NURSING INTERVENTIONS

For Pleurodesis

- Be certain pleural drainage from chest tube in previous 24 hours is less than 150 mL.
- Obtain sclerosing agent (bleomycin, doxycycline, or talc slurry) and postsclerosing flush solution (preservative-free sterile water or normal saline solution or lidocaine [Xylocaine] 1% or 2%).
- Plan to inject sclerosing agent into the chest drainage tube.
- Set up an extra chest drainage system with tubing to connect if tubing leaks after injecting sclerosing agent.
- Clamp tubing for 4 to 6 hours after instillation of sclerosing agent.
- Have patient be as mobile as possible, but scheduled body rotation is not necessary to ensure distribution of the agent throughout the pleura.
- Unclamp chest tube and observe drainage (effective 67% to 70% of time). If effective, drainage is minimal to absent.
- Assist with removal of chest tube and monitor patient for air tracking and pneumothorax, or reaccumulation of fluid.

is not easily separated. Chronic, long-term pleuroperitoneal shunts or implanted access devices have been used, but development of fibrin sheaths on the catheters often causes occlusion.[118,126] In cases where catheter drainage is unsuccessful, intracavitary chemotherapy with cisplatin, interferon, or pemetrexed have been used with moderate success[122,123,126,132]

Complications

Untreated pleural effusions that continue to accumulate lead to clinically significant alveolar collapse and respiratory failure, which may be caused by loss of gas-exchanging airways or mediastinal shifting with major airway obstruction. Progressive hypoxemia leads to profound respiratory acidosis and ischemic organ failure. Evacuation of an extensive and long-standing pleural effusion may result in reexpansion pulmonary edema or hypotension from fluid shifts.[133]

Spinal Cord Compression

Spinal cord compression (SCC) occurs when tumor cells or collapsed vertebrae in the epidural space exert pressure on the spinal cord, which may result in permanent dysfunction (including paralysis) if not diagnosed and treated promptly. Epidural tumors are found in more than 5% of patients with metastatic disease at autopsy.[134] The most common cause of SCC is compression fracture and vertebral collapse.[135] Factors associated with effective local control and long-term survival after SCC include favorable histological diagnosis, no visceral metastases, and a long-course radiation therapy schedule.[134,135]

Pathophysiology

Two major pathophysiological mechanisms are likely to result in SCC: (1) tumors arising within the epidural space through vertebral or lymphatic spread and (2) bony metastasis causing vertebral collapse with spinal cord and nerve root compression.[134,135] Permanent neurological damage from proximal tumors may also occur if spinal circulation is compromised such as in prolonged ischemia or hemorrhage.[136] Other disorders producing signs and symptoms of cord compression are paraneoplastic syndromes, radiation myelopathy, herpes zoster, pain from a pelvic or long bone metastasis, or cytotoxic drug effects.[136] Table 48-7 presents the tumors most likely to cause cord compression and the location of compression.

Assessment

HISTORY. When a primary tumor presses on the spinal cord, signs and symptoms usually develop slowly. Problems develop more rapidly with metastatic disease. Most patients with SCC complain of progressive central or radicular back pain that often is aggravated by weight bearing, lying down, coughing, sneezing, or performing the Valsalva maneuver. Sitting relieves the pain.[137,138]

The earliest neurological symptoms are sensory changes, such as numbness, paresthesia, and coldness.[138,139] Compression occurs most often in the thoracic section of the spinal cord, causing neurogenic bladder with urinary retention and incontinence. Patients may also lose the urge to defecate and are unable to bear down. Men on occasion lose the ability to have or maintain an erection. Metastases to the cauda equina frequently produce impaired urethral, vaginal, and rectal sensations; bladder dysfunction; decreased

Table 48-7 Spinal Cord Compression: Etiology and Clinical Presentation

Location of Lesion	Common Malignant Etiologies	Physical Symptoms	Autonomic Symptoms
Cervical spine	• Head and neck cancers • Melanoma	• Radicular pain in the neck, occipital region, and shoulders (pain is often provoked by neck movement) • Quadriplegia • Upper extremity weakness (may be spastic or atrophic) • Sensory loss in area of weakness • Weakness or paralysis of the diaphragm may occur with lesion at or above C4 (may be unilateral or bilateral)	• Hypotension • Bradycardia • Loss of temperature autoregulation • Autonomic hyperreflexia • Gastric hypersecretion and paralytic ileus • Reflex bowel, bladder, and penile erection • Hoffman's sign (flicking of the middle finger induces flexion of the ipsilateral thumb or index finger)
Thoracic spine	• Breast cancer • Gastric cancer • Lung cancer • Lymphoma • Pancreatic cancer	• Pain (may be local, radicular, or both) • Paraplegia • Sensory loss below the level of the lesion • Reflex abnormalities distal to the lesion	• Venous stasis and associated complications • Reflex bowel, bladder, and penile erection
Lumbar spine	• Ovarian cancer • Renal cell cancer • Prostate cancer	• Bowel and bladder dysfunction • Extensor plantar response	• Venous stasis and associated complications • Reflex bowel, bladder, and penile erection
Cauda equina	• Bladder cancer • Prostate cancer	• Pain (may be local, referred, or radicular) • Sphincter disturbances • Loss of buttock and leg sensation • Lower extremity weakness/paralysis	• Areflexic bowel, bladder, and penile erection

BOX 48-14 CONSIDERATIONS FOR THE OLDER PATIENT

Spinal Cord Compression

Signs and symptoms of SCC often begin as subtle and nonspecific back pain and sensory changes. The older patient may have concomitant diabetes mellitus or osteoarthritis that produces overlapping symptoms, delaying diagnosis of the oncological complication. In addition, older persons often have bowel and bladder changes causing constipation or urinary incontinence, mimicking the more serious autonomic changes that occur in later SCC. People at high risk for SCC, such as those with known bone metastases, should be taught the importance of reporting and having evaluated all back pain and sensory changes, especially in the lower extremities. In SCC, palpable vertebral tenderness is more often present than with other nononcological disorders.

sensation in the lumbosacral dermatomes; and saddle anesthesia.[139] Box 48-14 describes spinal cord considerations in the older adult.

It is possible to determine the level of cord compression by the patient's report of pain during straight leg raising, neck flexion, or vertebral percussion. The upper limit of the sensory level is usually one or two vertebral bodies below the site of compression.[139] Lessened rectal tone and perineal sensation are observed with autonomic dysfunction. Deep tendon reflexes can be brisk with cord compression and diminished with nerve root compression.

Once patients experience pain, motor weakness and ataxia often follow.[139] They may complain that the arms or legs feel heavy. Some patients lose the ability to sense light touch, pain, and temperature. Over time, weakness may progress to spasm, paralysis, and muscle atrophy; sensations of deep pressure and position may disappear.

DIAGNOSTIC STUDIES. A screening spinal radiograph will detect up to 80% of vertebral fractures and high risk for SCC and may be used for initial, rapid evaluation in high-risk patients, particularly when an MRI is not readily available.[140] MRI is the diagnostic test of choice due to high sensitivity for neurological tissues. MRI examinations can clearly demonstrate all epidural deposits as well as complete or partial block of the spinal cord.[141,142] A myelogram or CT scan may reveal spinal tumors, but these studies are less sensitive for diagnosing the presence and extent of cord compression. Lumbar puncture, which is used to obtain cerebrospinal fluid, reveals malignant cells in the presence of epidural disease.[141]

Management

Factors considered in the selection of the best therapeutic option are the level of cord compression, the rate of neurological deterioration, and previous use of radiation therapy.[134] Corticosteroids decrease peritumoral edema and neurological dysfunction. Dexamethasone, 10 mg as an initial dose, is administered to patients with neurological symptoms before emergency diagnostic procedures are performed and is continued during radiation therapy (4 to 20 mg every 6 hours) and then tapered.[134,135,140,143] It is not clear whether such steroid therapy affects final patient outcome.

Radiation therapy is appropriate when the tumor is determined to be radiosensitive and should be initiated as soon as the diagnosis of cord compression has been confirmed.[137] Radiation portals include the entire area of blockage and two vertebral bodies above and below this area. More than 50% of patients with rapid neurological deterioration improve with radiation therapy; however, patients with autonomic dysfunction or paraplegia have a poor prognosis with any therapy.[140,143] Laminectomy, with or without placement of stabilization rods in the nearby vertebral bodies, may result in immediate decompression of the spinal cord and nerve roots.[144,145] The posterior approach is preferred but is often difficult because most metastases arise in the vertebral bodies anterior to the spinal cord.[141,145] The anterior approach is warranted for people with tumors that are believed to be resectable, making the clinical risks worth the aggressive surgical intervention.[144] Postoperative radiation therapy is used to shrink residual tumor, relieve pain, and improve the patient's functional status. Surgery is usually contraindicated if there is a collapsed vertebral body or if there are several areas of cord compression. If there is no previous histological diagnosis of cancer, or if infection or epidural hematoma must be ruled out, then laminectomy can be used for both diagnosis and treatment. If high cervical cord compression precludes surgery, a neurologist should stabilize the patient's neck in halo traction to prevent respiratory paralysis.[145] If the patient continues to deteriorate neurologically despite high doses of steroids and radiation therapy, then emergency decompression may be necessary.[144] In some people, stabilization of the spine with vertebroplasty with or without kyphoplasty represents a less invasive and equally effective short-term resolution for acute cord compression and its associated pain.[145–147] Injection of physiological cement (kyphoplasty) to reexpand collapsed vertebrae has been used to successfully prevent progression to SCC.[147] If the tumor is chemosensitive, chemotherapy concurrently with or soon after completion of radiation therapy or surgery may be appropriate. Chemotherapy may also be effective in patients with multiple myeloma who have had previous radiation therapy.[145] Systemic chemotherapy or hormonal therapy may be useful in certain types of tumors, such as lymphoma or prostatic cancer.

Pain management includes the administration of appropriate analgesics, bed rest, and patient support during position changes and transfer. Range-of-motion exercises are useful in patients with motor and sensory deficits. Bowel retraining and intermittent urinary catheterization may be necessary. Frequent skin care is essential. Surgical wounds are particularly susceptible to skin breakdown (with possible wound dehiscence) because of limited mobility and the effects of concomitant corticosteroid therapy.[138]

Tracheobronchial Obstruction

Pathophysiology

Obstruction of the trachea or major branches of the bronchi with tumor results in respiratory distress and hypoxemia. The severity of symptoms depends on the rapidity of obstruction and degree of closure.[148,149] Tumors most likely to cause airway obstruction are lung cancer and lymphoma, although other metastatic tumors (eg, head and neck cancer, melanoma, renal or breast cancer) and nonmalignant disorders (eg, amyloidosis, bronchomalacia) may also cause airway obstruction.[149] Rare instances of leukemia and lymphomatous infiltrates causing airway obstruction have also been reported.[150]

Assessment

HISTORY. Patients with tracheobronchial obstruction present with varying degrees of dyspnea depending on the amount and location of the obstruction and the rapidity of onset. Some patients with slowly developing tumors have compensated respiratory acidosis and minimal symptoms even with nearly complete obstruction. Other patients, especially those with lymphoma or small cell lung carcinoma, have rapidly growing tumors and severe symptoms even when the airway is less than 75% obstructed. Stridor is present in tracheal obstruction, and wheezing with unequal chest excursion is seen with bronchial obstruction.[149] Some patients presenting with severe respiratory distress actually have only partial airway obstruction, but the resultant narrowed airway leads to concomitant atelectasis or trapped secretions with pneumonia that may be mistaken as more severe tumor obstruction.[151]

DIAGNOSTIC STUDIES. Bronchoscopy makes it easy to detect tracheal or bronchial obstruction and grade its severity. However, bronchoscopy does not always reveal whether the airways are compressed extrinsically or invaded with tumor.[148] Bronchoscopy is used with spiral CT scans to provide a comprehensive description of the obstructive process that is used to guide therapy.[152]

Management

Clinically significant obstruction of the major airways always necessitates immediate treatment, although the therapeutic plan varies according to tumor-specific factors and therapeutic goals. Emergent treatment of airway occlusion–induced hypoxemia or hypercapnia may require nasal inhalation or heliox-based nebulizer treatments. A combination of oxygen and helium that is lighter than pure oxygen, heliox enhances movement of the air beyond the area of obstruction and provides palliative relief until more aggressive operative measures are possible.[153] If air movement is adequate, bronchodilators and corticosteroids are administered to enhance ventilation, and if simultaneous pneumonia is suspected, antimicrobial therapy is instituted.[149]

Effective treatment for endobronchial tumors includes laser, cautery, photodynamic therapy, and endobronchial brachytherapy.[154–157] These therapies for tumors invading the major airways are highly successful for prolonging life as well as improving its quality. Most procedures entail use of a rigid bronchoscope under anesthesia, and patients usually experience a rapid recovery with little more than a sore throat and annoying cough for a few days afterward.[156,157] Endobronchial brachytherapy involves endotracheal intubation with precisely directed radiation therapy through an endobronchial catheter.[158] In laser therapy, electrocautery, photodynamic therapy, and endobronchial brachytherapy, close observation for airway bleeding is necessary, and clinicians may prescribe cough suppressants or low-dose corticosteroids to reduce the incidence of bleeding.[155,156] Airway opening with tracheal or bronchial stents may provide temporary symptomatic relief while definitive anticancer treatment is implemented for palliative relief of symptoms.[154,159–162] For insertion of an airway stent, a rigid bronchoscope and light anesthesia are necessary, and multiple bronchoscopic procedures to assess or adjust placement are required. The most common problem with stents, especially if placed before shrinking the tumor, is displacement because the airway naturally opens with the reduction of tumor. Displaced stents usually cause severe and sudden respiratory distress and require immediate interventional adjustment. In rare circumstances, or when stenting is not possible, patient positioning to shift the chest tumor off the major airway (eg, prone positioning) may provide temporary symptomatic relief while cancer therapy is used to shrink the tumor.[159,160]

Complications

Two severe complications that may occur are total airway occlusion and hemorrhage caused by tumor erosion into the nearby pulmonary vessels. Treatment of total obstruction is the same as that of partial obstruction when an improvement in symptoms can be reasonably expected as a result of therapy. Emergent extracorporeal membrane oxygenation (ECMO) has also been used to bridge until the tumor shrinks. Treatment of hemorrhage, when recognized before massive bleeding occurs, may involve embolization. If severe hemorrhage occurs, it is necessary to insert a dual-lumen endotracheal tube or single-sided intubation and occlude the bleeding lung while ventilating the good lung until surgical repair can be performed. Airway obstruction may also lead to erosion through the airway and accompanying pneumothorax. In these circumstances, supportive therapy, such as chest tube insertion, may be used but is rarely helpful.

▲ Metabolic Complications

Hypercalcemia

Hypercalcemia exists when the corrected serum calcium level is above 11 mg/dL (normal range, 8.5 to 10.5 mg/dL). This is the most common metabolic oncological emergency that develops when the bones release more calcium into the extracellular fluid than can be filtered by the kidneys and excreted in the urine.[163–166]

Pathophysiology

Ninety-nine percent of the calcium in the body is in an insoluble form in the bones. The remaining 1% is freely exchangeable calcium. The calcium of importance is the ionized calcium, which must be maintained within a precise range. Serum calcium levels are regulated by parathyroid hormone and calcitonin.[163,167] The release of parathyroid hormone from the parathyroid glands stimulates an increase in serum calcium levels, whereas the release of calcitonin produces a decrease in serum calcium levels.[164]

Destruction of the bone by metastatic invasion is believed to be the most common cause of malignant hypercalcemia; however, 20% of patients with solid tumors usually associated with hypercalcemia do not show evidence of bony involvement.[163,168] In some cancers, tumor cells secrete certain humoral substances, such as parathyroid hormone–like substances or osteolytic prostaglandins. In patients with multiple myeloma, the abnormal plasma cells produce osteoclast-activating factor (OAF); however, hypercalcemia does not develop in these patients unless they have inadequate renal function.[169] Patients with T-cell lymphoma have severe hypercalcemia related to the ectopic production of OAF, colony-stimulating factor, interferon-γ, and an active vitamin D metabolite.[170] Additional causes of hypercalcemia in the presence of malignancy include immobilization,

BOX 48-15 Causes of Hypercalcemia in Malignancy

- Bone demineralization due to bone metastases (most common in breast, colorectal, lung, and renal cell cancer)
- Tumor production of a parathormone-like substance (thyroid cancer, multiple myeloma, leukemia, lymphoma, gastric cancer, pancreatic cancer, lung cancer)
- Renal insufficiency
- Immobilization
- Dehydration

renal insufficiency, thiazide diuretics, high dietary calcium or vitamin D intake, and low phosphate levels.[164,167,171] Box 48-15 lists causes of hypercalcemia in malignancy.

Hypercalcemia develops in as many as 40% to 50% of women with metastatic breast cancer. There is a risk for bone metastasis, and estrogen and antiestrogens stimulate breast cancer cells to produce osteolytic prostaglandins and to increase bone resorption.[167] In cases of nonbone sources of hypercalcemia, it is often viewed as a hallmark of the presence of tumor. The incidence and severity may dramatically decrease when the patient's tumor is quiescent and become severe or difficult to manage when the tumor is active.[169]

Assessment

HISTORY. The severity of signs and symptoms of hypercalcemia often correlates with the serum calcium level. Common presenting symptoms include nausea, constipation, polyuria, and mental status changes. Most patients present with somnolence, combativeness, or confusion.[172]

DIAGNOSTIC STUDIES. Elevated serum calcium and elevated ionized calcium are the hallmark diagnostic findings in hypercalcemia. The serum calcium measurement is often reported as an absolute number without considering that only the calcium bound to albumin is counted. The serum calcium may be corrected for a low albumin by subtracting the patient's albumin from low normal, multiplying this number by a correction factor of 0.8, and adding this number to the reported calcium.[173,174] Serum ionized calcium levels are accurate, but because the normal value is 1.0 mEq/L (± 0.02), it is a less sensitive indicator of clinically significant hypocalcemia.[174]

In addition to increased calcium levels, there are also elevations in alkaline phosphatase and immunoreactive parathyroid hormone.[173,174] Serum phosphate and serum potassium are decreased. Symptomatic patients usually have ECGs that show a bradycardia and prolonged PR, QRS, and QT intervals.[172,174]

Management

Medical management of hypercalcemia involves the use of IV fluids and drug therapy to enhance renal excretion of calcium and to decrease bone resorption. Acute hypercalcemia is initially treated with IV normal saline (0.9% NaCl) solution to dilute calcium levels and increase urinary calcium excretion.[165,175] When hypercalcemia is life-threatening, aggressive hydration (250 to 300 mL/h) and IV loop diuretics such as furosemide are necessary.[175] Hemodialysis with calcium-free dialysate has been used successfully for emergency management of life-threatening hypercalcemia.[176]

In most patients, treatment with hydration, diuretics, appropriate antitumor therapy, and mobilization is effective. Patients who do not respond to these therapies require hypocalcemic therapy indefinitely. Bisphosphonates are most frequently used. Currently, the most potent bisphosphonate available is zoledronic acid. It is administered as an 8-mg 15-minute IV infusion daily for 3 days unless serum calcium levels decrease before that time.[175] Until recently, the mainstay of bisphosphonate therapy was pamidronate, and some clinicians may still prefer it. It is licensed to be given as a 90-mg, 24-hour infusion with continued hydration, possibly diuretics, and careful monitoring of calcium levels. However, because of clinical safety and efficacy studies, many clinicians give this dose over 90 to 120 minutes. An FDA-licensed monoclonal antibody targeting rank ligand for prevention of bone breakdown, denosumab, is also available, but its use in hypercalcemia has been limited.[177] In cases unresponsive to bisphosphonates, calcitonin, corticosteroids, or strontium-98 may be useful.[81,164,165,175,178] If possible, patients should ambulate to prevent osteolysis. It is necessary to eliminate constipation, which is usually caused by an increased level of calcium in the blood. Reduced oral intake of calcium or increased salt intake may be of some help. Patients should not take medications, such as thiazide diuretics and vitamins A and D, because they elevate the calcium level.[179] Close monitoring of fluid status is essential. Patients may receive up to 10 L of IV fluids daily, and the nurse should carefully measure intake and output. In addition, careful observation for overhydration is important. Potassium supplements may be necessary. Hypercalcemia is a common oncological emergency that can be prevented or diminished in a large number of patients with the appropriate prophylactic bisphosphonates, education, and precautions.[81,165,172,179] Box 48-16 presents a teaching guide for patients with hypercalcemia.

BOX 48-16 TEACHING GUIDE Malignancy-Associated Hypercalcemia

Patients at high risk for malignancy-associated hypercalcemia include those with:

- Bone metastases (most common in breast, lung, and colon cancer)
- Lung cancer
- Gastrointestinal cancers (gastric, pancreatic, colon)
- Hematological cancers (leukemia, lymphoma, multiple myeloma)
- Renal (kidney) cancer
- Thyroid cancer

Patients with the risk factors below need to be instructed that additional factors increase their risk for developing hypercalcemia. These factors include:

- Lack of physical activity
- Low fluid status
- Poor kidney function

Critical care nurses can teach patients methods for preventing hypercalcemia as follows:

- Drink at least six to eight glasses of water every day.
- Eat salty foods.
- Remain physically active.
- Limit dairy products and vitamin D–enriched foods, such as milk, cheese, and yogurt.

Complications

Permanent renal tubular abnormalities may develop in patients with prolonged hypercalcemia.[171] Sudden death from cardiac dysrhythmias may result from an acute increase in serum calcium. Long-term bisphosphonate use has been associated with severe osteonecrosis of the jaw. Specific risk factors for this complication are not yet clear.[180]

Syndrome of Inappropriate Antidiuretic Hormone (SIADH) Secretion

SIADH secretion is a clinical disorder characterized by excess stimulation of pituitary excretion of antidiuretic hormone (ADH). Under normal circumstances, the posterior pituitary gland releases ADH in response to changes in plasma osmolality (concentration of solutes) and circulating blood volume. ADH release causes decreased urine production and volume and increased water resorption. SIADH has several specific causes related to cancer and its treatment.[181] The clinical consequences of SIADH and its management strategies are discussed in Chapter 44.

Pathophysiology

When thoracic or mediastinal tumors press on major cardiac vessels, the obstruction may impede cardiac output. The posterior pituitary gland perceives this to be a fall in circulatory volume and compensates by inappropriately secreting ADH, which in turn suppresses urinary output. The resulting volume expansion improves cardiac output but leaves the patient with a relative sodium deficiency (dilutional hyponatremia).

In addition to the pressure of thoracic or mediastinal tumors on cardiac vessels, cancers and treatment-related factors can also precipitate SIADH. Small cell lung cancers or mixed cellularity lung cancers, pancreatic, renal, gastric, head and neck, thyroid cancer, neuroendocrine, and melanoma release an ADH-like substance.[181–185] Certain chemotherapeutic agents, such as cyclophosphamide, ifosphamide, imatinob, vincristine, vinorelbine and alemtuzumab, as well as morphine, may stimulate ADH release or potentiate its effects on the kidneys.[181,182,186–189] One study views ongoing evidence of SIADH after antineoplastic treatment as a poor prognostic sign, often a subtle indicator of persistent tumor.[190] Enhancing this confusing clinical picture may be that brain injury, pulmonary infection and HIV disease also cause SIADH.[191]

Management

Treatment of the underlying malignancy is of primary importance in cancer-related SIADH.[187] Clinical evidence of excess ADH is present until the primary tumor stops compressing the major cardiac vessels or producing ADH-like substances. Antineoplastic therapy may include chemotherapy, radiation therapy, or corticosteroids. Fluid intake limited to 500 to 1,000 mL/d should result in a corrected fluid balance in 7 to 10 days.[182,184,192] Demeclocycline, an antibiotic that inhibits ADH secretion, may be effective; patients with chronic SIADH may receive demeclocycline, 900 to 1,200 mg/d.[192] Adverse effects include diarrhea, nausea, dysphagia, and photosensitivity. New specific vasopressin-receptor antagonists, called vaptans, enhance water diuresis without sodium loss and show promise in the treatment of this disorder.[192]

Diuretics are not necessary except in severe circumstances because they may produce additional electrolyte imbalances. However, the patient who is comatose or convulsing should receive 3% IV hypertonic saline solution and a potent loop diuretic, such as furosemide.[182,184] Fluid imbalances and hyponatremia may be severe enough to warrant initiation of mechanical ventilation; the need for this aggressive respiratory support is the most predictive of a mortality rate of 22% to 40%.[190]

Tumor Lysis Syndrome

Tumor lysis syndrome is a metabolic imbalance caused by rapid cancer cell death occurring with clinical significance in 2% to 4% of high-risk patients.[193] Most patients experience this complication 1 to 5 days after initiation of therapy in patients with chemosensitive or radiosensitive tumors.[194,195] However, there are documented instances of tumor lysis syndrome in rapidly proliferating disease such as acute leukemia or high-grade lymphoma even before treatment initiation.[195]

Patients at greatest risk for tumor lysis syndrome are those with bulky tumors having a high growth rate (eg, acute leukemia or Burkitt's lymphoma) and those with highly radiosensitive or chemosensitive tumors, such as small cell lung cancer and most malignant lymphomas.[194–197] Patients with preexisting renal dysfunction may be at greatest risk owing to their difficulty in clearing the metabolic waste products fast enough to prevent clinical complications.[195,198] Other patients at high risk are those with Merkel's tumor, testicular cancer, hepatoblastoma, and medulloblastoma.[195]

Pathophysiology

Rapid cell death causes the release of intracellular contents (potassium, phosphorus, and nucleic acids) into the circulating serum. The normal filtration mechanisms in the kidneys should immediately detect the levels of metabolic waste products and attempt to excrete them. If production is more rapid than excretion or renal insufficiency is present, accumulation of electrolytes and uric acid occurs in the serum. The most common abnormalities include hyperkalemia, hyperphosphatemia, and hyperuricemia.[195] High phosphorus causes the kidneys to excrete calcium, causing hypocalcemia. Hyperuricemia causes deposition of uric acid crystals in the urinary tract and may lead to renal failure.[43,195,197,198]

Assessment

HISTORY. Signs and symptoms of tumor lysis syndrome are related to the specific electrolyte imbalances involved and renal dysfunction. Hyperkalemia, hyperphosphatemia, hypocalcemia, hyperuricemia, and acidosis may occur. Box 48-17 lists the typical clinical signs and symptoms associated with the metabolic abnormalities of tumor lysis syndrome.[195]

BOX 48-17 PATIENT SAFETY

Signs and Symptoms of Tumor Lysis Syndrome

Hyperkalemia
- Peaked T waves on electrocardiogram (ECG)
- Dysrhythmias (tachycardia, ventricular ectopy/torsade de pointes [especially when potassium levels are >6.8 mEq/L])
- Muscle flaccidity, weakness
- Hyperactive bowel sounds, abdominal cramping, and diarrhea

Hyperphosphatemia
- Muscle weakness
- Bone marrow suppression (thrombocytopenia, leukopenia)
- Bone demineralization with tendency for pathological fractures
- Renal dysfunction

Hypocalcemia
- Muscle tetany
- Seizures
- Short PR and QT intervals on ECG
- Dysrhythmias (tachycardia, ventricular ectopy/torsade de pointes)
- Hyperactive bowel sounds, abdominal cramping, and diarrhea

Hyperuricemia
- Uric acid crystals in urine
- Hematuria
- Oliguria, anuria
- Flank pain
- Renal failure

Acidosis
- Tachypnea
- Hypotension

DIAGNOSTIC STUDIES. The electrolyte panel analysis is used to identify key abnormalities in patients at risk for tumor lysis syndrome. Elevated serum potassium, phosphate, uric acid, blood urea nitrogen, and creatinine, with low calcium, are reported. Acidosis may be present in patients with severely compromised renal function. The urinary uric acid/creatinine ratio is greater than 1. Renal ultrasonography is used to exclude ureteral obstruction.[199]

Management

Treatment involves recognition of high-risk patients and promoting prevention through aggressive hydration, as well as administration of phosphate-binding agents and allopurinol for at least 48 hours before beginning chemotherapy. It is necessary to avoid agents that block tubular reabsorption of uric acid (eg, aspirin, radiographic contrast, probenecid, thiazide diuretics). The goal is to keep the serum uric acid level within normal limits. Electrolyte disturbances are specifically treated as needed.[193,200]

IV fluids are given to ensure a urine volume of more than 3 L/d. In the past, IV sodium bicarbonate (4 g initially, then 1 to 2 g every 4 hours) has been administered to alkalinize the urine and reduce uric acid crystallization in the kidney tubules. Clinicians are now less likely to initiate alkalinization if the phosphate is high because calcium phosphate precipitation is equally likely to cause renal failure.[196,197,200] To measure urine output more accurately, insertion of a Foley catheter into the bladder is usually necessary. If oliguria or anuria develops, ureteral obstruction must be excluded. Phosphate-binding agents such as aluminum hydroxide are given every 2 to 4 hours in an effort to keep phosphate levels below 4 mg/dL.[195] Concomitant diuresis or medications such as Kayexalate that enhance gastrointestinal excretion of potassium may effectively manage elevated serum potassium levels not prevented with hydration. Allopurinol, a xanthine oxidase inhibitor that blocks uric acid production, is administered in doses ranging from 300 to 900 mg/d. Because it is now available in an IV form given as 200 to 400 mg/m^2/d, rapid normalization of uric acid levels is an achievable objective.[195] Its greatest limitation is that it cannot assist in breakdown or clearance of already existing uric acid.[201] Rasburicase (Elitek), acts like the natural enzyme urate oxidase to oxidize uric acid to allantoin for excretion.[201,202] Rasburicase is used cautiously in patients at risk for glycoprotein deficiency (GPD) because of increased risk for hemolytic anemia. Severe hypersensitivity reactions have also been reported and warrant careful observation during infusion. The uric acid serum blood levels drawn on patients receiving rasburicase must be placed in an iced blood tube and transported to the laboratory on ice to ensure accurate levels.[200–203] If diuresis does not occur within a few hours after the initiation of treatment, renal replacement therapy is needed. An initial hemodialysis treatment usually reduces the patient's uric acid levels by 50%, but most patients then receive several additional days of CRRT until electrolyte abnormalities and hyperuricemia resolve.[43,193] A low-calcium dialysate is used to prevent calcium phosphate precipitation. If peritoneal dialysis is used, albumin is added to the dialysate to increase uric acid protein binding and removal.

The focus of nursing care is on careful monitoring of fluid therapy, intake and output, and electrolyte balance. The use of prophylactic allopurinol, aggressive hydration, and early intervention with CRRT has reduced the incidence and severity of tumor lysis syndrome.[195]

▲ Clinical Applicability Challenges

CASE STUDY

M.S. is a 62-year-old woman, with a 2-month history of intermittent dyspnea, occurring with increasing frequency and less provocation. She is currently presenting to the ED with acute exacerbation of symptoms and new onset of epigastric pain. Her medical history reveals past mild hypertension controlled by diet and exercise. She also has a history of light smoking of less than two packs of cigarettes per week for 15 years.

On presentation, the patient has visible respiratory distress. She is diaphoretic, has circumoral cyanosis, is using her accessory muscles to breathe, has better chest excursion on the left, and is sitting upright on the Gurney. Physical examination reveals:

Neurological assessment: M.S. is anxious but oriented to person, place, and time and has equal limb movement and sensation with some stiffness and aching of the right arm. She also reports epigastric pain rating 4 on a 1 to 10 scale (with 10 the greatest pain) radiating to the back.

Respiratory assessment: Labored respirations at 34/min, greatly diminished breath sounds throughout the right lung field, bilateral wheezes in upper lobes, egophony auscultated on the right side, and tracheal shift to the left. A small, approximately 2-cm mass is palpated in the right supraclavicular area.

Cardiovascular assessment: Muffled heart sounds, with point of maximal impulse shifted to the sixth intercostal space, lateral one third of the clavicular line. There is global edema of the upper extremities and neck and right-sided jugular vein distention. Pulse is 132 beats/min and irregular, and the blood pressure is 160/96 mm Hg in the right arm and 142/88 mm Hg in the left.

Gastrointestinal/genitourinary assessment: Abdomen is soft and nontender other than the epigastric distress. Patient reports moderate nausea and anorexia that has become more pronounced within the past week. Bowel sounds are diminished but active in all four quadrants. No urinary distress is reported and last urine output was 4 hours ago.

Chest x-ray shows a right mediastinal lung mass measuring approximately 4 × 4 cm with moderate pleural effusion, venous congestion, and enlarged cardiac silhouette.

Arterial blood gas values on 100% nonrebreather mask: pH 7.31, pCO_2 54, pO_2 56, and HCO_3 28.

M.S. is transferred to the ICU after peripheral IV insertion, verification of normal 12-lead electrocardiogram, initiation of the cardiac monitor showing atrial fibrillation at a rate between 120 and 134 beats/min, initiation of ipratropium bromide (Atrovent) bronchodilator therapy, and indwelling catheter after administration of furosemide (Lasix) 40 mg. She was also febrile at presentation and specimens for culture were obtained followed by administration of acetaminophen (Tylenol) and piperacillin–tazobactam (Zosyn).

Upon arrival in the ICU, the nurse confirms these findings and verifies that the only other laboratory abnormalities are white blood cell count 18,500, Hgb 11.0, Na 132, Cl 95, K 3.4, and Mg 0.9.

A chest computed tomography (CT) with contrast is ordered but delayed because the patient is unable to lie flat for the procedure. Electrolyte replacement of potassium and magnesium is initiated. The ICU nurse suggests insertion of a central line for blood drawing, and potential multiple medications, but physicians are reluctant at this time suspecting probable superior vena cava syndrome (SVCS).

Discussion of best management of the atrial fibrillation is debated. It is decided that the rhythm disturbance is likely acute in onset and anticoagulation is considered risky until more diagnostic tests are performed. After diuresis, electrolyte replacement, and antibiotics have been in place for 12 to 24 hours, they will reevaluate the patient unless her condition dramatically worsens. If her condition improves, the CT scan for assessment of SVCS, pleural effusions, possible pulmonary embolism, and tracheobronchial obstruction will be performed. If her condition does not improve, it is likely that endotracheal intubation with mechanical ventilation and sedation will be used for stabilization. An echocardiogram is ordered to assess the clinical impact of the pericardial effusion, and a bronchoscopy is planned for the following day to assess the degree of airway obstruction and accessibility for tissue biopsy diagnosis.

1. In a patient with a known large mediastinal tumor, several oncological emergencies may occur. Describe the emergent conditions M.S. is experiencing in the case study as well as the physiological basis of these emergencies.

2. Patients and families often are surprised by the sudden occurrence of an oncological emergency. Develop a teaching plan for M.S. and her family.

3. Explore the psychosocial challenges of treating a patient with newly diagnosed cancer presenting initially in the ICU.

4. M.S. requires additional evaluation for cytopathological diagnosis and even potential treatment. How can the nurse best assist in achieving the CT scan and potential emergent radiation or chemotherapy?

References

Human Immunodeficiency Virus Infection

1. Morimoto Y, Routes JM: Immunodeficiency overview. Prim Care 35(1):159–173, 2008
2. National Library of Medicine/National Institutes of Health: Immunodeficiency disorders. Retrieved December 1, 2010 from http://www.nlm.nih.gov/medlineplus/ency/article/000818.htm
3. Balt CA, Phillips J; Craig (Co-chairs), Members: Agnoli M, Donovan M, Ghigliotty B, Hunter H, Jefferies C, Miramontes H, Santillan-Rabe M, Raper JL, Relf M, Rolfson N, Santhanam H, & Staats J. HIV/AIDS Nursing: Scope and Standard of Practice. Silver Spring, MD: American Nurses Association & Association of Nurses in AIDS Care, 2007
4. Centers for Disease Control and Prevention. Revised surveillance case definitions for HIV infection among adults, adolescents, and children <18 months and for HIV infection and AIDS among children aged 18 months to <13 years—United States, 2008. MMWR Morb Mortal Wkly Rep 57(RR10):1–8, 2008
5. The White House Office of National AIDS Policy. National HIV/AIDS Strategy for the United States. July 2010
6. Centers for Disease Control and Prevention, National Center for HVI/AIDS, Viral Hepatitis, STD and TB Prevention, Division of HIV/AIDS Prevention: HIV testing in the US. CDC Vital signs. December 2010
7. Sprung CL, Steinberg A: Acquired immunodeficiency syndrome and critical care. Crit Care Med 18(11):1300–1302, 1990
8. Davaro RE, Thirumalai A: Life-threatening complications of HIV infection. J Intensive Care Med 22(2):73–81, 2007
9. Huang L, Quartin A, Jones D, et al: Intensive care of patients with HIV infection. N Engl J Med 355:173–181, 2006
10. Corona A, Raimondi F: Caring for HIV-infected patients in the ICU in the highly active antiretroviral therapy era. Curr HIV Res 7(6):569–579, 2009
11. Centers for Disease Control and Prevention: Revised recommendations for HIV testing of adults, adolescents, and pregnant women in healthcare settings. MMWR Recomm Rep 55(RR14):1–17, 2006
12. American Academy of Nursing: American Academy of Nursing supports recommendations that all public health and health care settings develop a system of routine testing for HIV infection. Accessed December 1, 2010. Available at: http://www.aannet.org/i4a/headlines/headlinedetails.cfm?id=289
13. Centers for Disease Control and Prevention: HIV in the United States. Retrieved December 1, 2010, from http://www.cdc.gov/hiv/resources/factsheets/PDF/us.pdf
14. Centers for Disease Control and Prevention: Rates of adults and adolescents living with an AIDS diagnosis, year end 2007—United States and dependent areas. Retrieved December 1, 2010, from http://www.cdc.gov/hiv/topics/surveillance/resources/slides/general/slides/general_29.pdf
15. Moir S, Chun TW, Fauci AS: Pathogenic mechanisms of HIV disease. Annu Rev Pathol 6:223–248, 2011
16. Levy JA: HIV pathogenesis; 25 years of progress and persistent challenges. AIDS 23:147–160, 2009
17. Sharp PM, Hahn BM: Prehistory of HIV-1. Nature 455:605–606, 2008
18. Worobey M, Gemmel M, Teuwen DE, et al: Direct evidence of extensive diversity in HIV-1 in Kinshasa in 1960. Nature 455:661–664, 2008
19. Centers for Disease Control and Prevention: Basic information about HIV and AIDS. Retrieved December 1, 2010, from http://www.cdc.gov/hiv/topics/basic/print/index.htm
20. Bartlett JG, Gallant JE, Pham P: The Management of HIV Infection. Durham, NC: Knowledge Source Solutions, LLC, 2009
21. Siegel JD, Rhinehart E, Jackson M, et al; the Healthcare Infection Control Practices Advisory Committee: 2007 Guideline for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings, June 2007. Retrieved December 1, 2010, from http://www.cdc.gov/ncidod/dhqp/pdf/isolation2007.pdf
22. Centers for Disease Control and Prevention: Updated U.S. Public Health Service guidelines for the management of occupational exposures to HIV and recommendations for Postexposure Prophylaxis. MMWR Recomm Rep 54(No. RR-9), 2005
23. Grant RM, Lama J, Anderson PL, et al: Preexposure chemoprophylaxis for HIV prevention in men who have sex with men. N Engl J Med 363(27):2587–2599, 2010
24. Carr RL, Dodge R: Care of the AIDS patient with Pneumocystis pneumonia. Dimens Crit Care Nurs 28(6):264–269, 2009
25. Women's Interagency HIV Study (WIHS): Retrieved December 1, 2010, from https://statepiaps.jhsph.edu/wihs/index.htm
26. CDC, NIH, the HIV Medicine Association of the Infectious Diseases Society of America: Guidelines for the prevention and treatment of opportunistic infections in HIV-infected adults and adolescents. MMWR Recomm Rep 58(RR04):1–198, 2009
27. Panel on Antiretroviral Guidelines for Adults and Adolescents: Guidelines for the use of antiretroviral agents in HIV-1-infected adults and adolescents. Department of Health and Human Services. Retrieved March 27, 2012. Available at http://www.aidsinfo.nih.gov/ContentFiles/AdultandAdolescentGL.pdf. Accessed May 15, 2012

Oncological Complications and Emergencies

1. Shelton BK: Admission criteria and prognostication in patients with cancer admitted to the intensive care unit. Crit Care Clin 26:1–20, 2010
2. Behl D, Hendrickson AW, Moynihan TJ: Oncologic emergencies. Crit Care Clin 26(1):181–206, 2010
3. Azoulay E, Afessa B: The intensive care support of patients with malignancy: Do everything that can be done. Intensive Care Med 32(1):3–5, 2006
4. Lim Z, Pagliuca A, Simpson S, et al: Outcomes of patients with haematological malignancies admitted to the intensive care unit: A comparative review of allogeneic haematopoietic stem cell transplantation data. Br J Haematol 136(3):448–450, 2007
5. Raoof ND, Groeger JS: You never know-one of your patients with cancer might surprise you. Crit Care Med 35(3):965–966, 2007
6. Chernecky CC, Murphy-Ende K (eds): Acute Care Oncology Nursing, 2nd ed. St Louis, MO: Saunders Elsevier, 2008, pp 26–34
7. LeCuyer L, Chevret S, Thiery G, et al: The ICU trial: A new admission policy for cancer patients requiring mechanical ventilation. Crit Care Med 35:808–814, 2007
8. Freedman N, Hansen-Fletcher J: Intensive care for oncology patients: Short term prognosis. UpToDate online 18.1, last literature review January 2010; last updated January 25, 2010. Accessed May 25, 2010
9. Markou N, Demopoulou E, Myrianthefs P: The critically ill patient with cancer—Indications for intensive care unit admission and outcomes. J BUON 13(4):469–478, 2008
10. Gaeta S Price KJ: End-of-life issues in critically ill cancer patients. Crit Care Clin 26(1):219–228, 2010
11. Marik PE: Management of patients with metastatic malignancy in the intensive care unit. Am J Hosp Palliat Care 23(6):479–482, 2006
12. Thakker SG, Fu AZ, Sweetenham JW, et al: Survival and predictors of outcome in patients with acute leukemia admitted to the intensive care unit. Cancer 112(110):2233–2240, 2008
13. Shelton BK: Critical care of cancer patients. Crit Care Connect SCCM 4(4):1, 5, 2005
14. Van Gestel JPJ, Bollen CW, van der Tweel I, et al: Intensive care unit mortality trends in children after hematopoietic stem cell transplantation: A meta-regression analysis. Crit Care Med 36(10):2898–2904, 2008
15. Walter KL, Siegler M, Hall JB: How decisions are made to admit patients to medical intensive care units (MICUs): A survey of MICU directors at academic medical centers across the United States. Crit Care Med 36(2):414–420, 2008
16. Soares M, Depuydt PO, Salluh IF: Mechanical ventilation in cancer patients: Clinical characteristics and outcomes. Crit Care Clin 26(1):41–58, 2010
17. Carlson KS, DeSancho MT: Hematological issues in critically ill patients with cancer. Crit Care Clin 26(1):107–132, 2010
18. Drews RE: Hematologic consequences of malignancy: Anemia. UpToDate version 18.1 online, last Updated October 5, 2009. Accessed May 6, 2010
19. Shelton BK, Rome SI, Lewis SL: Nursing assessment: Hematologic system. In Lewis SL, Heitkemper MM, Dirkson SR, et al (eds): Medical-Surgical Nursing: Assessment and Management of Clinical Problems, 7th ed. Philadelphia, PA: Elsevier, 2007, pp 665–683

20. Shelton BK: General toxicity: Myelosuppression and secondary malignancies. In Gobel B, Triest S, Vogel W (eds): Advanced Oncology Certification Review and Resource Manual. Pittsburgh, PA: Oncology Nursing Society Press, 2010
21. Thirmula R, Ramaswamy M, Chawla S: Diagnosis and management of infectious complications in critically ill patients with cancer. Crit Care Clin 26(1):59–92, 2010
22. Sreedharan A, Bowyer S, Wallace CA, et al: Macrophage activation syndrome and other systemic inflammatory conditions after BMT. Bone Marrow Transplant 37(7):629–634, 2006
23. Takagi S, Masuoka K, Uchida N, et al: High incidence of haemophagocytic syndrome following umbilical cord blood transplantation for adults. Br J Haematol 147:543–553, 2009
24. Lee YH: Pre-engraftment syndrome in hematopoietic stem cell transplant. J Korean Med Sci 23(1):98–103, 2008
25. Rimkus C: Acute complications of stem cell transplant. Semin Oncol Nurs 25(2):129–138, 2009
26. Keung YK, Beaty MW, Peltenati M, et al: Possible role of engraftment syndrome and autologous graft-versus-host disease in myelodysplastic syndrome after autologous stem cell transplantation: Retrospective analysis and review of the literature. Clin Lymphoma Myeloma Leuk 10(2):129–133, 2010
27. Carreras E, Fernandez-Ariles F, Silva L, et al: Engraftment syndrome after auto-SCT: Analysis of diagnostic criteria and risk factors in a large series from a single center. Bone Marrow Transplant 45(9):1417–1422, 2010
28. Gorak E, Geller N, Srinivasan R, et al: Engraftment syndrome after nonmyeloablative allogeneic hematopoietic stem cell transplantation: Incidence and effects on survival. Biol Blood Marrow Transplant 11:542–550, 2005
29. Dai E, Couriel D, Kim SK: Bilateral marginal keratitis associated with engraftment syndrome after hematopoietic stem cell transplantation. Cornea 26(6):756–758, 2007
30. Alkhatib AA, Boynton KK, Badheeb AM: Colitis secondary to engraftment syndrome in a patient with autologous peripheral blood stem cell transplant. Dig Dis Sci 55(5):1500–1501, 2010. DOI: 10.1007?S10620-0841-1
31. Schiffer CA: Hyperleukocytosis and leukostasis. UpToDate online version 18.1, last literature review January 2010; last updated December 2009
32. Adams BD, Baker R, Lopez JA, et al: Myeloproliferative disorders and the hyperviscosity syndrome. Emerg Med Clin North Am 27:459–476, 2009
33. Blum W, Porcu P: Therapeutic apheresis in hyperleukocytosis and hyperviscosity syndrome. Semin Thromb Hemost 33(4):350–354, 2007
34. Chang M, Chen T, Tang J, et al: Leukapheresis and cranial irradiation in patients with hyperleukocytic acute myeloid leukemia: No impact on early mortality and intracranial hemorrhage. Am J Hematol 82:976–980, 2007
35. Piccirillo N, Laurent L, Chiusolo P, et al: Reliability of leukostasis grading score to identify patients with high risk hyperleukocytosis. Am J Hematol 84(6):381–382, 2009
36. Oliveira LC, Romano LG, Prado-Junior BP, et al: Outcome of acute myeloid leukemia patients with hyperleukocytosis in Brazil. Med Oncol 27(4): 1254–1259, 2010
37. Bug G, Anargyrou K, Tonn T, et al: Impact of leukapheresis on early death rate in adult acute myeloid leukemia presenting with hyperleukocytosis. Transfusion 47(10):1843–1850, 2007
38. Marbello L, Ricci F, Nosari AM, et al: Outcome of hyperleukocytic adult acute myeloid leukaemia: A single-center retrospective study and review of literature. Leuk Res 32(8):1221–1227, 2008
39. Ruggierio A, Attina G, Piastra M, et al: Severe hyperleukocytosis and multifocal intracranial haemorrhage: Not always a fatal outcome. Int J Hematol 90(1):87–90, 2009
40. Chou SHY, Singhal AB: Multiple punctate cerebral hemorrhages in acute leukemia with blast crisis. Neurology 68(12):953, 2007
41. Lele AV, Mirski MA, Stevens RD: Spurious hypoxemia. Crit Care Med 33(8):1854–1856, 2005
42. Balint B, Ostojic G, Pavlovic M, et al: Cytapheresis in the treatment of cell-affected blood disorders and abnormalities. Transfus Apher Sci 35:25–31, 2006
43. Choi KA, Lee JE, Kim YG, et al: Efficacy of continuous venovenous hemofiltration with chemotherapy in patients with Burkitt lymphoma and leukemia at high risk for tumor lysis syndrome. Ann Hematol 88(7):639–645, 2009
44. Sultan K, Vasudeva R: Neutropenic enterocolitis. EMedicine. Updated last July 9, 2009. Accessed May 23, 2010
45. Osenga KL. Typhlitis in pediatrics. In Chernecky CC, Murphy-Ende K (eds): Acute Care Oncology Nursing, 2nd ed. St Louis, MO: Saunders Elsevier, 2009, pp 560–568
46. Ullery BW, Pieracci FM, Rodney JR, et al: Neutropenic enterocolitis. Surg Infect 10(3):307–314, 2009
47. Mullassery D, Bader A, Battersby SJ, et al. Diagnosis, incidence, and outcomes of suspected typhlitis in oncology patients—Experience in a tertiary pediatric surgical center in the United Kingdom. J Pediatr Surg 44(2):381–385, 2009
48. Marie I, Robaday S, Kerleau M, et al: Typhlitis as a complication of alemtuzumab therapy. Haematologica 92(5):e62–e63, 2007
49. Kasturi KS, Mummadi RR, Sood GK: Neutropenic enterocolitis. An unusual complication of HCV combination therapy with PEG-IFN and ribavirin. Eur J Inter Med 19(5):372–373, 2008
50. Tiseo M, Gelsomino F, Bartolotti M, et al: Typhlitis during second-line chemotherapy with pemetrexed in non-small cell lung cancer (NSCLC): A case report. Lung Cancer 65(2):251–253, 2009
51. Aksoy DY, Tanriover MD, Uzun O, et al: Diarrhea in neutropenic patients: A prospective cohort study with emphasis on neutropenic enterocolitis. Ann Oncol 18(1):183–189, 2007
52. Davila ML: Neutropenic enterocolitis: Current issues in diagnosis and management. Curr Infect Dis Rep 9(2):116–120, 2007
53. Song LW, Marcon NE: Typhlitis (neutropenic enterocolitis). UpToDate Version 19.2, last updated November 23, 2009. Accessed January 25, 2012
54. Spencer SP, Power N, Reznek RH: Multidetector computed tomography of the acute abdomen in the immunocompromised host: A pictorial review. Curr Probl Diagn Radiol 38(4):145–155, 2009
55. Badgwell BD, Cormier JN, Wray CJ, et al: Challenges in surgical management of abdominal pain in the neutropenic cancer patient. Ann Surg 248(1):104–109, 2008
56. Shelton BK: Pericardial effusion and tamponade. In Chernecky CC, Murphy-Ende K (eds): Acute and Critical Care of Cancer Patients, 2nd ed. St Louis, MO: Saunders Elsevier, 2009, pp 45–58
57. Sampat K, Rossi A, Garcia-Gutierez V, et al: Characteristics of pericardial effusions in patients with leukemia. Cancer 116(10):2366–2371, 2010
58. Borlaug BA, DeCamp MM, Gangadharan SP: Neoplastic pericardial disease. UpToDate online version 18.1, last updated June 19, 2009. Accessed May 23, 2010
59. Sweetenham JW: Highly aggressive lymphomas in adults. Hematol Oncol Clin North Am 22:965–978, 2008
60. Kunitoh H, Tamura T, Shibata T, et al: A randomized trial of intrapericardial bleomycin for malignant pericardial effusion with lung cancer (JCOG9811). Br J Cancer 100(3):464–469, 2009
61. Quraishi AR, Khan AA, Kazmi KA, et al: Clinical and echocardiographic characteristics of patients with significant pericardial effusion requiring pericardiocentesis. J Pak Med Assoc 55(2):66–70, 2005
62. Imazio M, Demichelis B, Parrini I, et al: Relation of acute pericardial disease to malignancy. Am J Cardiol 95(11):1393–1394, 2005
63. Ueda K, Nagai, S, Miyashita S, et al: Arsenic-induced pericardial and pleural effusion without acute promyelocytic leukemia differentiation syndrome. Leuk Res 34:e25–e26, 2010
64. McArdle JR: Critical care outcomes in the hematologic transplant recipient. Clin Chest Med 30:155–167, 2009
65. Corey GR: Diagnosis and treatment of pericardial effusions. UpToDate online version 19.3, January 2012; last updated April 25, 2011. Accessed January 26, 2012
66. Billikanty S, Bashir R: Images in cardiovascular medicine: Echocardiographic demonstration of electrical alternans. Circulation 113(24): e866–e868, 2006

67. Roy C, Minor M, Brookhart M, et al: Does this patient with pericardial effusion have cardiac tamponade? N Engl J Med 297(16):1810–1818, 2007
68. Choe KS, Salama JK: Advances in radiotherapy for tumors involving the mediastinum. Thorac Surg Clin 19:133–141, 2009
69. Savides TJ. EUS for mediastinal disease. Gastrointest Endosc 69(2):S97–S99, 2009
70. Mulvagh SL, Rakowski H, Vannan MA, et al: American Society of Echocardiography Consensus Statement on the clinical applications of ultrasonic contrast agents in echocardiography. J Am Soc Echocardiogr 21(11): 1179–1201, 2008
71. Hoey ETD, Mankad K: Computed tomography-guided pericardiocentesis: utility in the management of malignant pericardial effusion. Am J Emerg Med 28:388e1–388.e3, 2010
72. Misselt AJ, Harris SR, Glockner J, et al: MR imaging of the pericardium. Magn Reson Imaging Clin N Am 16:185–199, 2008
73. Cooper CA. Centesis studies in critical care. Crit Care Nurs Clin North Am 22:95–108, 2010
74. Oida T, Mimatsu K, Kano H, et al: Pericardiocentesis with cisplatin for malignant pericardial effusion and tamponade. World J Gastroenterol 16(6):740–744, 2010
75. Becit N, Unlu Y, Ceviz M, et al: Subxiphoid pericardiostomy in the management of pericardial effusions: Case series analysis of 368 patients. Heart 91:785–790, 2005
76. Lestuzzi C, Lafaras C, Bearz A, et al: Malignant pericardial effusion: Sclerotherapy or local chemotherapy. Br J Cancer 101:734–735, 2009
77. Masullo D, Benedetto PD, Pinto G: Intraoperative strategy in patients with extended involvement of mediastinal structures. Thorac Surg Clin 19:113–120, 2009
78. Motas C, Motas N, Rus O, et al: Left paraxiphoidian approach for drainage of pericardial effusions. Interact Cardiovasc Thorac Surg 10:4–5, 2010
79. Cullinane CA, Paz IB, Smith D, et al: Prognostic factors in the surgical management of pericardial effusion in the patient with concurrent malignancy. Chest 125(4):1328–1334, 2004
80. Frawley T, Begley C: Causes and prevention of carotid artery rupture. Br J Nurs 15(22):1198–1202, 2006
81. Sargent C: Carotid Artery Rupture. In CC Chernecky, K Murphy-Ende (eds): Acute Care Oncology Nursing, 2nd ed. St Louis, MO: Saunders Elsevier, 2009, pp 59–66
82. Brockstein BE, Vokes EE: Overview of the complications of head and neck cancer and its therapy. UpToDate online version 18.1, last updated July 23, 2008. Accessed June 12, 2010
83. Kim H, Lee D, Kim H, et al: Life-threatening common carotid artery blowout: Rescue treatment with a newly designed self-expanding covered nitinol stent. Br J Radiol 79:226–231, 2006
84. Frawley T, Begley C: Caring for people with carotid artery rupture. Br J Nurs 15(1):24–28, 2006
85. Koutsimpelas D, Pitton M, Kulkens C, et al: Endovascular carotid reconstruction in palliative head and neck cancer patients with threatened carotid blowout presents a beneficial supportive care measure. J Palliative Med 11:784–789, 2008
86. McKoy JM, Angelotta C, Bennett CL, et al: Gemtuzumab ozogamicin-associated sinusoid obstructive syndrome (SOS): An overview from the research on adverse drug events and reports (RADAR) project. Leuk Res 31(5):599–604, 2007
87. Eisenberg S: Hepatic sinusoidal obstruction syndrome in patients undergoing hematopoietic stem cell transplant. Oncol Nurs Forum 35(3):385–397, 2008
88. Krimmel T, Williams LA: Hepatic sinusoidal obstruction syndrome following hematopoietic stem cell transplantation. Oncol Nurs Forum 35(1):37–39, 2008
89. Buchsel PC: Sinusoid occlusive syndrome. In Chernecky CC, Murphy-Ende K (eds): Acute Care Oncology Nursing, 2nd ed. St Louis, MO: Saunders Elsevier, 2009, pp 481–491
90. Cheuk DK, Wang P, Lee TL, et al: Risk factors and mortality predictors of hepatic veno-occlusive disease after pediatric hematopoietic stem cell transplantation. Bone Marrow Transplant 40(10):935–944, 2007
91. Schouten van der Velden AP, Punt CJ, Van Krieken JH, et al: Hepatic veno-occlusive disease after neoadjuvant treatment of colorectal liver metastases with oxaliplatin. Eur J Surg Oncol 34:353–355, 2008
92. Senzolo M, Burra P, Cholongitas E, et al: The transjugular route: The key hole to the liver world. Dig Liver Dis 39(2):105–116, 2007
93. Senzolo M, Germani G, Cholongitis E, et al: Veno occlusive disease: Update on clinical management. World J Gastroenterol 13(29):3918–3924, 2007
94. Matsumoto M, Kawa K, Uemura M, et al: Prophylactic fresh frozen plasma may prevent development of hepatic VOD after stem cell transplantation via ADAMTS13-mediated restoration of von Willebrand factor plasma levels. Bone Marrow Transplant 40(3):251–259, 2007
95. Sucak GT, Aki ZS, Yagci M, et al: Treatment of sinusoidal obstruction syndrome with defibrotide: A single-center experience. Transplant Proc 39(5):1558–1563, 2007
96. Ho VT, Linden E, Revta C, et al: Hepatic veno-occlusive disease after hematopoietic stem cell transplantation: Review and update on the use of defibrotide. Semin Thromb Hemost 33(4):373–388, 2007
97. Shah MS, Jeevangi NKS, Joshi A, et al: Late-onset hepatic veno-occlusive disease post autologous peripheral stem cell transplantation successfully treated with oral defibrotide. J Cancer Res Ther 5(4): 312–314, 2009
98. Dignan F, Gujral D, Ethell M, et al: Prophylactic defibrotide in allogeneic stem cell transplantation: Minimal morbidity and zero mortality from veno-occlusive disease. Bone Marrow Transplant 40(1):79–82, 2007
99. Batsis I, Yannaki E, Kaloyannidis P, et al: Veno-occlusive disease prophylaxis with fresh frozen plasma and heparin in bone marrow transplantation. Thrombo Res 118:611–618, 2006
100. Drews RE, Rabkin DJ: Malignancy-related Superior vena cava syndrome. UptoDate Online 18.1, last updated March 23, 2009. Retrieved June 30, 2010, from http://www.uptodate.com
101. Wan JF, Bezjak A: Superior vena cava syndrome. Emerg Med Clin North Am 27:243–255, 2009
102. Wilson LD, Detterbeck FC, Yahalom J: Clinical practice: Superior vena cava syndrome with malignant causes. N Engl J Med 356:18, 2007
103. Nunnelee JD: Superior vena cava syndrome. J Vasc Nurs 25:2–5, 2007
104. Bruno TF: Superior vena cava syndrome and telangiectasia in a man with lymphoma. CMAJ 177(10):1177–1179, 2007
105. Canon R, Shah M, Suydam E, et al. Early thrombosis of the superior vena cava in a patient with a central venous catheter and carcinoma of the ampulla of vater. Am Surg 74:1195–1197, 2008
106. Rice TW: Pleural effusion in SVCS: Prevalence, characteristics, and proposed pathophysiology. Curr Opin Pulm Med 13(4):324–327, 2007
107. Plekker D, Ellis T, Irusen EM, et al: Clinical and radiologic grading of superior vena cava obstruction. Respiration 32:585–589, 2008
108. Wilson P, Bezjak A, Asch M, et al: The difficulties of a randomized study in superior vena caval obstruction. J Thorac Oncol 2(6):514–519, 2007
109. Kostopoulou V, Tsiatas ML, Kelekis DA, et al: Endovascular stenting for the management of port-a-cath associated with superior vena cava syndrome. Emerg Radiol 16:143–146, 2009
110. Uberoi R: Quality assurance guidelines for superior vena cava stenting in malignant disease. Cardiovasc Intervent Radiol 29:319–322, 2006
111. Lanciego C, Pangua C, Chacon JI, et al: Endovascular stenting as the first step in the overall management of malignant superior vena cava syndrome. AJR Am J Roentgenol 193:549–558, 2009
112. Morales JP, Sabharwal T, Man-Hurun S, et al: Alleviation of severe compressive symptoms in a patient with advanced lung carcinoma using tracheal and superior vena cava stents. J Palliat Care 10:24–29, 2007
113. Ploegmakers MJM, Rutten MJCM: Fatal pericardial tamponade after superior vena cava stenting. Cardiovasc Intervent Radiol 32:585–589, 2008
114. Cope D. Pleural effusions: Malignant. In Chernecky CC, Murphy-Ende K (eds): Acute Care Oncology Nursing, 2nd ed. St Louis, MO: Saunders Elsevier, 2009, pp 435–441

115. Porcel JM: Pearls and myths in pleural fluid analysis. Respirology 16(1):44–52, 2011
116. Korczynski P, Krenke R, Safianowska A, et al: Diagnostic utility of pleural fluid and serum markers in differentiation between malignant and nonmalignant pleural effusions. Eur J Med Res 14(Suppl 4):128–133, 2009
117. Heffner JE: Management of malignant pleural effusion. UpToDate online version 18.1, last updated January 26, 2010. Accessed June 23, 2010
118. Held-Warmkessel J, Schieh L: Caring for the patient with malignant pleural effusion. Nursing 38(1):43–48, 2008
119. Barbetakis N, Asteriou C, Papadopoulou F, et al: Early and late morbidity and mortality and life expectancy following thoracoscopic talc insufflations for control of malignant pleural effusions: A review of 400 cases. J Cardiothorac Surg 5:27, 2010
120. Ozyurtkan MO, Balci AE, Cakmak M: Predictors of mortality within three months in the patients with malignant pleural effusions. Eur J Intern Med 21(1):30–34, 2010
121. Stark P: Imaging of pleural effusions in adults. UptoDate Online 18.1. Retrieved July 7, 2010, from http://www.uptodate.com
122. Heffner JE: Diagnostic evaluation of pleural effusion in adults. UptoDate Online 18.1. Retrieved July 7, 2010, from http://www.uptodate.com; last updated September 24, 2009
123. Moore AJ, Parker AJ, Wiggins J: Malignant mesothelioma. Orphanet J Rare Dis 3:34, 2008
124. Abouzgheib W, Bartter T, Dagher H, et al: A prospective study of the volume of pleural fluid required for accurate diagnosis of malignant pleural effusion. Chest 135:999–1001, 2008
125. James P, Gupta R, Christopher DJ, et al: Evaluation of the diagnostic yield and safety of closed pleural biopsy in the diagnosis of pleural effusion. Indian J Tuberc 57(1):19–24, 2010
126. Doelken P: Management of refractory nonmalignant pleural effusions. UpToDate online version 18.1, last updated June 16, 2009. Accessed July 7, 2010
127. Uzbeck MH, Almeida FA, Sarkiss MG, et al: Management of malignant pleural effusions. Adv Ther 27(6):334–347, 2010
128. Walker SJ, Bryden G. Managing pleural effusions: Nursing care of patients with a Tenckhoff catheter. Clin J Oncol Nurs 14(1):59–64, 2010
129. Varela G, Jimenez MF, Novoa N: Portable chest drainage systems and outpatient chest tube management. Thorac Surg Clin 20(3):421–426, 2010
130. Adeoye PO, Salami AK, Koledoye A: Early experience with outpatient tube drainage for management of pleural collections. West Afr J Med 28(6):364–367, 2009
131. Thornton RH, Miller Z, Covey AM, et al: Tunneled pleural catheters for treatment of recurrent malignant pleural effusion following failed pleurodesis. J Vasc Interv Radiol 21(5):696–700, 2010
132. Zhao WZ, Wang JK, Zhang XL: Clinical research on recombinant human Ad-p53 injection combined with cisplatin in treatment of malignant pleural effusion induced by lung cancer. Clin J Cancer 28(1):1324–1327, 2009
133. Lin YJ, Yu YH: Reexpansion pulmonary edema after large volume thoracentesis. Ann Thorac Surg 92(4):1550–1551, 2011
134. Bartels RH, van der Linden YM, van der Graaf WT: Spinal extradural metastasis: Review of current treatment options. CA Cancer J Clin 58(4):245–259, 2008
135. Cole J, Patchell R: Metastatic epidural spinal cord compression. Lancet Neurol 7(5):459–466, 2008
136. Coleman RE, Guise TA, Lipton A, et al: Advancing treatment for metastatic bone cancer: Consensus recommendations from the Second Cambridge Conference. Clin Cancer Res 14(20):6387–6395, 2008
137. James N, Brooks D: Managing patients with metastatic spinal cord compression. Cancer Nurs Pract 9(6):19–22, 2010
138. Miaskowski C: Spinal cord compression. In Chernecky CC, Murphy-Ende K (eds): Acute Care Oncology Nursing, 2nd ed. St Louis, MO: Saunders Elsevier, 2009, pp 492–498
139. Colen FN: Oncologic emergencies: superior vena cava syndrome, tumor lysis syndrome, and spinal cord compression. J Emerg Nurs 34(6):535–537, 2008
140. Wilkinson AN, Viola R, Brindage MD: Managing skeletal-related events resulting from bone metastases. BMJ 371:a2041, 2008
141. Elder JB, Lis E, Yamada Y, et al: Treatment of metastatic spinal disease. Curr Orthop Pract 21(4):348–355, 2010
142. Shuie K, Sahgal A, Chow E, et al: Management of metastatic spinal cord compression. Expert Rev Anticancer Ther 10(5):697–708, 2010
143. Hitron A, Adams V: The pharmacological management of skeletal-related events form metastatic tumors. Orthopedics 32(3):188–192, 2010
144. Gerber DE, Grossman SA: Does decompressive surgery improve outcome in patients with metastatic epidural spinal-cord compression? Nat Clin Pract Neurol 2(1):10–11, 2006
145. Sun H, Nemecek AN: Optimal management of malignant epidural spinal cord compression. Hematol Oncol Clin North Am 24(3):537–551, 2010
146. Saliou G, Kocheida el M, Lehmann P, et al: Percutaneous vertebroplasty for pain management in malignant fractures of the spine with epidural involvement. Radiology 254(3):882–890, 2010
147. Gofeld M, Bhatia A, Burton AW: Vertebroplasty in the management of painful bony metastases. Curr Pain Headache Rep 13(4):288–294, 2009
148. Liberman M: Bronchoscopic evaluation of the trachea and dilatation of the trachea. Semin Thorac Cardiovasc Surg 21(3):255–262, 2009
149. Theodore PR: Emergent management of malignancy-related acute airway obstruction. Emerg Med Clin North Am 27:310–312, 2008
150. Singer J, Henry S: Upper airway obstruction as the presenting manifestation of leukemia. Pediatr Emerg Care 24(5):231–241, 2008
151. Ernst A, Herth FJF, Becker HD: Diagnosis and management of central airway obstruction. UpToDate Version 19.3, January 2012, last updated March 20, 2010. Accessed January 25, 2012
152. Shin JH, Sing HY, Kim KR, et al: Radiologic and clinical outcomes with special reference to tumor involvement pattern after stent placement for malignant bronchial obstructions. Acta Radiol 50(9):1011–1018, 2009
153. Feller-Kopman DJ, O'Donnell C: Physiology and clinical use of heliox. UpToDate version 19.3, January 2012. Updated last January 2010. Accessed January 26, 2012
154. Beeson J: Palliation of tracheobronchial carcinoma: The role of cryosurgery. J Perioper Pract 17(7):332–339, 2007
155. Ernst A, LoCicero III J: Photodynamic therapy of lung cancer. UpToDate online version 18.1, last updated January 22, 2010. Accessed July 7, 2010
156. Colt HG: Endobronchial electrocautery. UpToDate online version 18.1, last updated October 6, 2009. Accessed July 7, 2010
157. Minnich DJ, Bryant AS, Dooley A, et al: Photodynamic laser therapy for lesions in the airway. Ann Thorac Surg 89(6):1744–1748, discussion 1748–1749, 2010
158. Fortunato M, Felijo S, Almeida T, et al: Endoluminal high dose rate brachytherapy in the treatment of primary and recurrent bronchogenic tree malignancies. Rev Port Pneumol 15(2):151–164, 2009
159. Colt HG: Airway stents. UpToDate online version 18.1, last updated June 13, 2008. Accessed July 7, 2010
160. Furukawa K, Ishida J, Yamaguchi G, et al: The role of airway stent placement in the management of tracheobronchial stenosis caused by inoperable advanced lung cancer. Surg Today 40:315–320, 2010
161. Kim H, Shin JH, Song H, et al: Palliative treatment of inoperable malignant tracheobronchial obstruction: Temporary stenting combined with radiation therapy and/or chemotherapy. AJR Am J Roentgenol 193:W38–W42, 2009
162. Oki M, Saka H, Kitagawa C, et al: Double Y-stent placement for tracheobronchial stenosis. Respiration 79:245–249, 2010
163. Horwitz MJ: Hypercalcemia of malignancy. UpToDate online version 18.1, last updated September 28, 2009. Accessed July 18, 2010
164. Santarpia L, Koch CA, Sarlis NJ: Hypercalcemia in cancer patients: Pathobiology and management. Horm Metab Res 42(3):153–164, 2010
165. Samphao S, Eremin JM, Eremin O: Oncological emergencies: Clinical importance and principles of management. Eur J Cancer Care 19(6):707–713, 2010
166. Siddiqui F, Weissman D: Fast facts and concepts: Hypercalcemia of malignancy #151. J Palliat Med 13(1):77–78, 2010
167. Shane E: Etiology of hypercalcemia. UpToDate online version 18.1, last updated January 13, 2010. Accessed July 12, 2010
168. Fitch M, Maxwell C, Ryan C, et al: Bone metastases from advanced cancers: Clinical implications and treatment options. Clin J Oncol Nurs 13(6):701–710, 2009

169. de Oliviera Filgueira PH, Vasconcelos LF, daSilva GB, et al: Paraneoplastic syndromes and the kidney. Saudi J Kidney Dis Transpl 21(2):222–231, 2010
170. Sargent JT, Smith OP: Haematological emergencies managing hypercalcemia in adults and children with haematological disorders. Br J Haematol 149(4):465–477, 2010
171. Desai HV, Gandhi K, Sharma M, et al: Thiazide-induced severe hypercalcemia: A case report and review of literature. Am J Ther 17(6):e234–e236, 2010
172. Kacprowicz RF, Lloyd JD: Electrolyte complications of malignancy. Hematol Oncol Clin North Am 24(3):553–565, 2010
173. Ijaz A, Mehmood T, Qureshi AH, et al: Estimation of ionized calcium and total calcium and albumin corrected calcium for the diagnosis of hypercalcemia of malignancy. J Coll Physician Surg Pak 16(1):49–52, 2006
174. Shane E: Diagnostic approach to hypercalcemia. UpToDate online version 18.1, last updated January 13, 2010. Accessed July 12, 2010
175. Shane E, Berenson JR: Treatment of hypercalcemia. UpToDate online version 19.3, January 2012; last updated December 2, 2011. Accessed January 26, 2012
176. Wang CC, Chen YC, Shiang JC, et al: Hypercalcemic crisis successfully treated with prompt calcium-free hemodialysis. Am J Emerg Med 27(9):1174e1–1174e3, 2009
177. Coleman R: Bisphosphonates and other osteoclast inhibitors in patients with metastatic cancer. UpToDate Version 19.3, January 2012; last updated January 6, 2012. Accessed January 25, 2012
178. Diskin CJ, Stokes TJ, Dansby L, et al: Malignancy-related hypercalcemia developing on a bisphosphonates but responding to calcitonin. Clin Lung Cancer 8(7):434–435, 2007
179. Pearson KE: Tumor induced hypercalcemia. In Chernecky CC, Murphy-Ende K (eds): Acute Care Oncology Nursing, 2nd ed. St Louis, MO: Saunders Elsevier, 2009, pp 284–297
180. Otto S, Abu-ld MH, Fedele S, et al: Osteoporosis and bisphosphonates-related osteonecrosis of the jaw: Not a sporadic coincidence- a multi-centre study. J Craniomaxillofac Surg 39(4):272–277, 2011
181. Rose BD: Pathophysiology and etiology of the syndrome of inappropriate antidiuretic hormone secretion (SIADH). UpToDate online version 18.1, last updated February 10, 2010. Accessed July 12, 2010
182. Ellison DH, Berl T: Clinical practice. The syndrome of inappropriate antidiuresis. N Engl J Med 356(20):2064–2072, 2007
183. Brown RR, Leitao MM Jr: Cisplatin-induced syndrome of inappropriate antidiuretic hormone (SIADH) in a patient with neuroendocrine tumor of the cervix: A case report and review of the literature. Eur J Gynaecol Oncol 31(1):107–108, 2010
184. Raftopoulos H: Diagnosis and management of hyponatremia in cancer patients. Support Care Cancer 15(12):1341–1317, 2007
185. Walji N, Chan AK, Peake DR: Common acute oncological emergencies: Diagnosis, investigation, and management. Postgrad Med J 84:418–427, 2008
186. Gleezerman IG: Successful treatment of Ifosphamide-induced hypotension with AVP receptor antagonist without interruption of hydration for prevention of hemorrhagic cystitis. Ann Oncol 20(7):1283–1285, 2009
187. Held-Warmkessel J: Syndrome of inappropriate antidiuretic hormone (SIADH). In Chernecky CC, Murphy-Ende K (eds): Acute Care Oncology Nursing, 2nd ed. St Louis, MO: Saunders Elsevier, 2009, pp 534–544
188. Liapisk K, Apostolidis J, Charitaki E, et al: Syndrome of inappropriate secretion of antidiuretic hormone associated with imatinib. Ann Pharmacother 42(12):1882–1886, 2008
189. Kuroda H, Kawamura M, Hato T, et al: Syndrome of inappropriate secretion of antidiuretic hormone after chemotherapy with vinorelbine. Cancer Chemother Pharmacol 62(2):331–333, 2008
190. Adam AK, Soubani AO: Outcome and prognostic factors of lung cancer patients admitted to the medical ICU. Eur Respir J 31:47–53, 2008
191. Yawar A, Jabbar A, Haque NU, et al: Hyponatremia: Etiology, management, and outcome. J Coll Physicians Surg Pak 18(8):467–471, 2008
192. Sterns RH: Treatment of hyponatremia: Syndrome of inappropriate antidiuretic hormone secretion (SIADH) and reset osmostat. UpToDate online version 18.1, last updated February 12, 2010. Accessed July 12, 2010
193. Benoit DD, Hoste EA: Acute kidney injury in critically ill patients with cancer. Crit Care Clin 26(1):151–180, 2010
194. Hochberg J, Cairo MS: Tumor lysis syndrome: Current perspective. Haematologica 93(1):9–13, 2008
195. Shelton BK: Tumor lysis syndrome. In Chernecky CC, Murphy-Ende K (eds): Acute Care Oncology Nursing, 2nd ed. St Louis, MO: Saunders Elsevier, 2009, pp 545–559
196. Tosi P, Barosi G, Lazzaro C, et al: Consensus conference on the management of tumor lysis syndrome. Haematologica 93(12):1877–1885, 2008
197. Larson RA, Pui C: Tumor lysis syndrome. UpToDate online version 18.1, last updated October 19, 2009. Accessed July 21, 2010
198. Maloney K, Denno M: Tumor lysis syndrome: Prevention and detection to enhance patient safety. Clin J Oncol Nurs 15(6):601–603, 2011
199. Ikeda AK, Sakamoto K, Krishnan K, et al: Tumor lysis syndrome. EMedicine. Available at: http://emedicine.medscape.com/article/989050-overview. Last updated September 26, 2008
200. Coiffer B, Altman A, Pui CH, et al: Guidelines for the management of pediatric and adult tumor lysis syndrome: An evidence-based review. J Clin Oncol 26(16):2767–2778, 2008
201. Hochberg J, Cairo MS: Rasburicase: Future directions in tumor lysis management. Expert Opin Biol Ther 8(10):1595–1604, 2008
202. Mayne N, Keady S, Thacker M: Rasburicase in the prevention and treatment of tumor lysis syndrome. Intensive Crit Care Nurs 24(1):59–62, 2008
203. Campara M, Shord SS, Haaf CM: Single-dose rasburicase for tumour lysis syndrome in adults: Weight-based approach. J Clin Pharm Ther 34(2):207–213, 2009

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

49 Common Hematological Disorders

Debby Greenlaw

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Describe the pathophysiology, assessment, and management of patients with disorders of red blood cells.
2. Discuss the pathophysiology, assessment, and management of patients with disorders of white blood cells.
3. Explain the pathophysiology, assessment, and management of patients with disorders of hemostasis.

Critically ill patients are at high risk for developing complications from a variety of hematological disorders. Anemia is common in the critical care unit; the causes are multifactorial and include preexisting disease, blood loss from multiple sources, and suppressed erythropoiesis. Critically ill patients are also highly susceptible to overwhelming infections from severe neutropenia as well as hemorrhagic complications secondary to severe thrombocytopenia and other clotting disorders.

This chapter presents an overview of the pathophysiology, assessment, and management of hematologic disorders in the critically ill patient. Red blood cell, white blood cell, platelet, and coagulation disorders are discussed.

▲ Disorders of Red Blood Cells

Polycythemia

Polycythemia Vera

Polycythemia vera is a myeloproliferative disorder of increased red blood cell (RBC) production resulting in a high hematocrit and an increased RBC mass. Increased RBC production causes decreased tissue oxygenation, increased blood viscosity, vascular insufficiency, and risk for thrombosis. As the disease progresses, some patients may develop bone marrow fibrosis, splenomegaly, and pancytopenia. Acute leukemia may develop in a small percentage of these patients.

Secondary Polycythemia

Secondary polycythemia is a disorder of increased RBC production that can develop as a normal response to chronic hypoxia. Conditions causing chronic hypoxia include living at high altitudes, cardiopulmonary disease, sleep apnea, obesity hypoventilation syndrome, and exposure to carbon monoxide. Secondary polycythemia may also result from an inappropriate increase in erythropoietin production as a result of renal disease or, in rare cases, hepatic disease.

Assessment

Arterial and venous thrombosis resulting from the blood's hyperviscosity is the major concern. The patient with polycythemia is at increased risk for thromboembolic events such as myocardial and cerebral infarction, deep venous thrombosis, and pulmonary embolism.

It is important to review the patient's medical history for cardiac or pulmonary disease. Also, any history of arterial or venous thrombosis is pertinent. Smoking history is relevant because cigarette smokers may have high carboxyhemoglobin levels. Patients with polycythemia often complain of itching after a hot shower or bath. Table 49-1 lists additional clinical findings in polycythemia vera.

Management

Serial phlebotomy is the first-line treatment for polycythemia vera. Generally 500 mL of blood is removed weekly until a hematocrit of less than 45% is achieved. Most patients managed by serial phlebotomy develop iron deficiency, which limits their ability to make RBCs and thus decreases the frequency of phlebotomy. The disadvantage of phlebotomy is that it stimulates bone marrow production, which leads to increased numbers of defective, sticky platelets. Antiplatelet aggregating agents, such as aspirin and dipyridamole, do not reduce thrombotic events and may increase the risk for bleeding.

Older patients with vascular disease are at high risk for thrombosis and require bone marrow suppression in addition to phlebotomy. Hydroxyurea is the medication of choice. Long-term therapy with bone marrow–suppressing agents has been associated with an increased risk for acute leukemia, so the potential benefits must be weighed against the anticipated duration of therapy. Measures to prevent thromboembolic complications, such as lower extremity compression

Table 49-1 Clinical Findings and Related Causes in Polycythemia Vera

Clinical Finding	Cause
Dizziness, headache	Increased blood viscosity
Thrombosis	Increased blood viscosity, thrombocytosis, platelet defects
Pruritus	Elevated blood levels of histamine and/or increased skin mast cells
Bleeding tendency	Increased red blood cell (RBC)/fibrin ratio; engorged capillaries and venules due to increased blood volume
Epigastric distress	Engorgement of gastric mucosa; increased blood histamine levels
Numbness and burning of toes	Peripheral vascular insufficiency
Cardiovascular insufficiency	Impaired tissue oxygenation due to increased blood viscosity

devices or facilitating ambulation if the patient's condition permits, should be instituted.

Treatment of secondary polycythemia focuses on correcting the underlying cause with long-term oxygen therapy, smoking cessation, weight loss, or surgical intervention as indicated. If these measures are ineffective, serial phlebotomy to maintain a hematocrit of 45% or less is required.

Anemia

Anemia may be seen in the intensive care unit (ICU) as an incidental condition in a patient admitted to the unit for another acute illness or as an acute condition requiring intensive monitoring and intervention. Typically, anemias are classified as blood loss, hemolytic (increased destruction of RBCs) or hypoproliferative (decreased production of RBCs). In addition, anemias can be classified by RBC size as microcytic, normocytic, or macrocytic.

Anemia is prevalent in critically ill patients. Acute blood loss, especially from intraoperative and gastrointestinal hemorrhage, is a frequent cause of anemia in critically ill patients. Phlebotomy for diagnostic tests has also been implicated as a cause for anemia cases in the ICU. It is estimated that for every 100 mL of blood drawn, there is an associated decrease in hemoglobin of 0.7 g/dL and in hematocrit of 1.9%. Even with a conservative measurement of blood loss of 100 mL/d from phlebotomy, the impact is significant, especially in the patient who is hospitalized for several weeks in the ICU.

In addition to blood loss, disseminated intravascular coagulation (DIC) and a variety of hemolytic disorders can decrease the survival of RBCs, causing anemia in the critically ill. Nutritional deficiencies, inflammation, and sepsis all contribute to anemia as well.

Types of Anemia

The following discussion gives a brief review of the different types of anemia. Because the workup and treatment of many of these anemias does not take place in the critical care setting, the discussion is limited. The main focus of the material presented is specific to anemia in the critically ill.

BLOOD LOSS ANEMIA. Blood loss anemia is probably the most common anemia requiring admission of the patient to the ICU. Blood loss should always be ruled out in any acute anemia. The primary focus of patient management is to identify and treat the underlying source of blood loss.

Stress gastritis can be the source of significant blood loss. This complication is easier to prevent than it is to treat. All critically ill patients should be considered at risk, and prophylactic therapy with H_2-blockers, proton pump inhibitors, or sucralfate should be initiated. Endoscopy may be performed to evaluate for a potential gastrointestinal source of blood loss.

HEMOLYTIC ANEMIAS. Hemolytic anemias result from the destruction of RBCs. They may be congenital or acquired and can vary greatly in the severity of the anemia.

Congenital Hemolytic Anemia. The most common types of congenital hemolytic anemias are caused by enzyme defects or RBC membrane defects (Table 49-2). Most congenital RBC enzymatic deficiencies are glucose-6-phosphate

Table 49-2 Congenital Hemolytic Anemias and Primary Interventions

Type of Defect	Primary Interventions
Enzyme Defects	
Glucose-6-phosphate dehydrogenase	Avoidance of agents that trigger hemolysis; hydration
Pyruvate kinase deficiency	Transfusion; splenectomy
Red Blood Cell Membrane Defects	
Hereditary spherocytosis	Splenectomy; folic acid supplements
Hereditary elliptocytosis	Usually no treatment required; folic acid supplements
Paroxysmal nocturnal hemoglobinuria	Corticosteroids, androgens, recombinant erythropoietin (rEPO), iron therapy; transfusion as needed; anticoagulation therapy if thrombotic events; possible bone marrow transplantation

BOX 49-1 Substances That May Cause Hemolytic Anemia in Susceptible Individuals

Congenital Hemolytic Anemia (glucose-6-phosphate dehydrogenase [G6PD] Deficiency)

- Norfloxacin
- Methylene blue
- Chloramphenicol
- Phenazopyridine
- Nitrofurantoin
- Sulfa drugs
- Mothballs
- Fava beans (Mediterranean variant of G6PD deficiency)

Acquired Hemolytic Anemia

- Wasp and bee stings
- Spider bites
- Snake bites
- Copper
- Lead
- Antineoplastic drugs, including mitomycin and cisplatin
- Primaquine
- Quinine
- Quinidine
- Methyldopa
- Procainamide
- Nonsteroidal anti-inflammatory drugs
- Penicillins
- Cephalosporins

dehydrogenase (G6PD) and pyruvate kinase deficiencies. Enzyme defects cause the RBCs to lyse when exposed to certain stressful conditions, such as drugs, chemicals, infections, surgery, or pregnancy. Substances to which people with G6PD deficiency may be susceptible are listed in Box 49-1.

Acquired Hemolytic Anemia. Acquired hemolytic anemias can be caused by several different factors (Table 49-3). In microangiopathic hemolytic anemia, RBCs are fragmented by vasculitis, collagen vascular disease, abnormal cardiac valves, arteriovenous (AV) malformations, thrombotic thrombocytopenic purpura (TTP), or DIC. Patients who experience hypothermia or cold cardioplegia with cardiac surgery may have RBCs with shortened life spans owing to membrane damage. Patients with RBCs recovered from the "cell saver" also experience significant membrane damage and hemolysis. Treatment focuses on removing the causative factor, such as replacing the abnormal heart valve or repairing the AV shunt. If this is not possible, the patient may be maintained on iron and folate supplements and periodic transfusions of RBCs.

Infectious agents may cause hemolytic anemia indirectly by causing splenomegaly or directly by invading the RBC and destroying its membrane. Malaria is an example of the latter. These patients are treated with transfusion support and anti-infective agents to address the underlying cause.

Abnormally shaped RBCs are frequently noticed in patients with liver disease. These patients may also have congestive splenomegaly, which causes sequestration and destruction of RBCs. In severe hemolysis, splenectomy and supportive RBC transfusions may be required.

Some patients can experience autoimmune hemolytic anemias. Warm autoimmune hemolytic anemia is the most common of these types. Approximately one half of all cases are idiopathic; known causative factors include collagen diseases, lymphoproliferative disorders, and drug reactions (see Box 49-1). Primary therapy is oral glucocorticoids to suppress the immune system. Additional treatments for patients who do not respond to glucocorticoids may include splenectomy, immunosuppressive agents, and intravenous immunoglobulin (IVIG).

Cold-reactive autoimmune hemolytic anemia is a disorder in which exposure to cold triggers complement-fixing immunoglobulin M antibodies to attach to RBCs in susceptible people, causing agglutination (clumping) and hemolysis (destruction). Often, these patients have an underlying lymphoproliferative disorder; others may have *Mycoplasma pneumoniae* infection, infectious mononucleosis, or hepatitis. If these patients require blood transfusion, use of a blood warmer and measures to keep the patient warm are recommended. Steroids and splenectomy are ineffective; intervention focuses on avoiding exposure to cold.

DEFICIENCY ANEMIAS. Deficiency anemias include iron deficiency anemia, megaloblastic anemia, anemia of chronic disease, and aplastic anemia. Table 49-4 lists common interventions for these anemias.

Table 49-3 Acquired Hemolytic Anemias and Potential Interventions

Acquired Hemolytic Anemia	Interventions
Microangiopathic	Removal of causative factor; iron and folate supplements; transfusion
Infectious agents	Treatment of underlying infection; transfusion
Liver disease	Splenectomy; transfusion
Autoimmune	
Warm antibody	Glucocorticoids; splenectomy; immunosuppressive agents; transfusion
Cold-reactive	Avoidance of exposure to cold; transfusion; plasma exchange
Drug-induced	Discontinuation of drug; transfusion

Table 49-4 Common Deficiency Anemias and Primary Interventions

Type of Anemia	Primary Interventions
Iron deficiency anemia	Iron supplements; correction of underlying stressor
Megaloblastic anemia	Vitamin B_{12} replacement; folic acid supplement
Anemia of chronic	Transfusion; rEPO; correction of underlying disorder
Aplastic anemia	Transfusion; immunosuppression; bone marrow transplantation

BOX 49-2 Drugs That Interfere With Folate Metabolism

- Alcohol
- Cholestyramine
- Metformin
- Phenobarbitol
- Phenytoin
- Pyrimethamine

Iron Deficiency Anemia. Iron deficiency is the most common cause of anemia in adults. It is usually caused by chronic blood loss, but it may be due to inadequate iron intake or absorption. It is imperative in chronic blood loss to search for the underlying cause and correct it. Oral iron replacement theory is typically administered. In patients with malabsorption disorders or poor tolerance of oral iron, however, parenteral iron may be considered.

Megaloblastic Anemias. Megaloblastic anemias are a group of anemias, most of which are caused by a deficiency of vitamin B_{12} (cobalamin), folate, or both. Drugs that interfere with folate metabolism are listed in Box 49-2. Treatment entails correcting the deficiency.

Vitamin B_{12} is poorly absorbed from the gut; therefore, intramuscular or subcutaneous injection is required. Most patients require maintenance injections monthly for the remainder of their lives. Body stores of folate can be restored with an oral folate supplement given daily for approximately 4 weeks. Once the deficiency is corrected, maintenance therapy is rarely necessary unless underlying factors, such as chronic alcoholism, are present.

Anemia of Chronic Disease. Finally, anemia is seen with a number of chronic disorders, such as renal failure, infections, malignancies, and connective tissue diseases including rheumatoid arthritis. Anemia of chronic renal failure generally starts to occur when the creatinine clearance is less than 45 mL/min and continues to worsen with increasing renal failure. Several mechanisms cause anemia of chronic disease. One factor is suppression of RBC production. Other factors are a decreased RBC survival time and low serum erythropoietin levels. Aspects of anemia in older patients are presented in Box 49-3. Treatment involves correcting the underlying cause, if possible. Transfusion may be of temporary benefit, although the survival of the transfused RBCs is reduced. Recombinant erythropoietin (rEPO) may be the treatment of choice for many people. rEPO is typically given as 50 to 100 units/kg intravenously or subcutaneously three times a week, with adjustments based on response. Complications of rEPO therapy are infrequent and are seen mostly in patients on renal dialysis. They may include hypertension, seizures, AV shunt thromboses, and increased blood viscosity.

Aplastic Anemia. In aplastic anemia, there is deficiency of RBCs as well as white blood cells (WBCs) and platelets (in other words, aplastic anemia is a condition of pancytopenia). In many cases, the cause of aplastic anemia is unknown. Possible factors include drugs, chemicals, viruses, and immunological and congenital disorders (Box 49-4). In some patients, aplastic anemia is thought to result from replacement of normal cells by clones of cells that are incapable of normal hematopoiesis. Clinical features of aplastic anemia are related to the underlying pancytopenia and include anemia, infections, and bleeding.

Severe aplastic anemia is treated with transfusion support, immunosuppressive agents, and bone marrow transplantation. Drugs that stimulate bone marrow function are of no benefit in this condition. Bone marrow transplantation, preferably from a human leukocyte antigen–matched sibling donor, should be considered for patients younger than age 60. These patients should be considered for immediate transplantation and should not receive any transfusions or drug therapy before transplantation, if possible. Immunosuppressive therapy with antithymocyte globulin, cyclosporine, or methylprednisolone

BOX 49-3 CONSIDERATIONS FOR THE OLDER PATIENT

Anemia

Anemia is common in the elderly, and its prevalence increases with age. As with other cells, the body's capacity for red cell replacement decreases with aging, typically with a greater decline seen in men than women. Although most elderly people are able to maintain their hemoglobin and hematocrit levels within a normal range, they are unable to replace their red cells as promptly in situations such as bleeding.

In the elderly, anemia is usually the result of bleeding, infection, malignancy, or chronic disease. Combined deficiencies are common in older adults. Undiagnosed and untreated anemia is associated with decreased functional and self-care abilities and depression. It can also cause neurologic and cognitive disorders, cardiovascular complications, and increased risk for mortality. Orally administered iron is poorly used in older adults.

BOX 49-4 Causative Factors in Aplastic Anemia

Congenital (20% of Cases)
- Fanconi's anemia
- Familial aplastic anemia

Acquired (80% of Cases)
- Idiopathic
- Irradiation
- Drugs
  - Chloramphenicol
  - Carbamazepine
  - Sulfonamides
  - Cimetidine
  - Gold salts
  - Acetazolamide
- Chemicals
  - Benzene and benzene derivatives
  - Insecticides
  - Cleaning solvents
- Infections
  - Non-A, non-B hepatitis
  - Epstein-Barr virus
  - Human immunodeficiency virus (HIV)
  - Mycobacteria
- Immunological
  - Graft-versus-host disease
  - Systemic lupus erythematosus
- Pregnancy

may be of benefit for older patients or those without a compatible donor for bone marrow transplantation.

Assessment

The history of the patient with anemia includes assessment of blood loss (including perioperatively, in the stool, and from the menses) as well as a thorough diet history. General symptoms of anemia include weakness, depressed mood, impaired cognitive function, and easy fatigability. Signs and symptoms indicative of decreased perfusion secondary to anemia are tachycardia, chest pain, dyspnea, and dizziness. Clinical consequences of anemia are impaired tissue oxygenation, impaired organ function, impaired susceptibility to thrombocytopenic bleeding, increased risk for postoperative mortality, increased probability of transfusion, and decreased survival.[1] Anemia in critically ill patients is similar in clinical presentation to the anemia of chronic disease, both of which lead to underproduction of RBCs.

Physical examination findings of anemia include pallor, tachycardia, hypotension, and signs of high-output heart failure. Patients with hemolytic anemia may have splenomegaly, jaundice, and dark urine owing to the excretion of bilirubin. It is necessary to review the intake and output records for fluid balance because hemodilution is common, particularly in the postoperative patient, because of aggressive intravenous hydration. All patients should have a stool guaiac test performed to look for occult gastrointestinal blood loss.

Typically, critically ill patients have low serum iron and total iron-binding capacity with elevated serum ferritin. When the iron studies are abnormal, serum erythropoietin levels are only mildly elevated, with little evidence of a reticulocyte response to endogenous erythropoietin. Further laboratory assessment to evaluate anemia is discussed in Chapter 46.

Management

The treatment of anemia starts with identifying the underlying cause. Iron sulfate, 325 mg by mouth two to three times per day, may be indicated. Parenteral iron may be administered when the patient is unable to take oral medications, or in the case of malabsorption or severe renal failure. Intramuscular iron injection may be painful and stain the patient's skin. Instead, intravenous injection is recommended. Close patient observation is necessary because severe anaphylactic reactions may occur with this treatment (Box 49-5). Vitamin C is often given to aid in the absorption of iron. Folic acid and vitamin B_{12}, if needed, should be considered.

BOX 49-5 PATIENT SAFETY

Parenteral Administration of Iron Dextran

- Anaphylactic reactions, including fatalities, have followed the parenteral administration of iron dextran injection.
- Have resuscitation equipment and personnel trained in the detection and treatment of anaphylactic reactions readily available.
- Administer a test dose prior to the first therapeutic dose.
- Use only in patients in an iron deficient state not amenable to oral iron therapy.

From FDA Warning/Alert, October 16, 2009, Iron dextran injection –938, 2008.

BLOOD TRANSFUSION. The risk versus benefit of blood transfusion in the critically ill patient continues to be an area of scrutiny and study. In the past, hemoglobin transfusion triggers varied between 7 and 10 g/dL, most often between 8 and 9 g/dL. However, this practice is changing because of a number of factors associated with worse clinical outcomes occurring in patients receiving blood transfusions.

Therapeutic Considerations. Currently, it is believed that transfusion of packed RBCs should be reserved for management of severe, active bleeding or for the patient who is experiencing serious symptoms from anemia. In other words, the practice should be to transfuse when the risks of decreased oxygen-carrying capacity outweigh the risks of transfusion, rather than relying on a specific hemoglobin or hematocrit trigger. Evidence-based guidelines have been developed to guide transfusion decisions in specific critical care populations, such as patients with sepsis, acute lung injury, and neurologic injury and diseases.[1]

The normal compensatory mechanisms in response to the decreased oxygen supply of anemia (increased heart rate, increased cardiac output and index, decreased systemic vascular resistance) may not work efficiently or at all in critically ill patients. This deficit in compensatory mechanisms is particularly relevant in patients with cardiac disease and in those at high risk for myocardial infarction. For example, a patient with coronary stenosis may not have the normal response of vasodilation as a compensatory mechanism to the anemia. Likewise, increased cardiac output may not be achievable in patients with cardiomyopathy or pulmonary edema. Other patients who may have a poor prognosis if they do not receive a transfusion include those with sepsis and higher APACHE II (disease severity) scores.

Complications. Complications of blood transfusion may be noninfectious, infectious, or immunologic. A common noninfectious complication of transfusion is volume overload, particularly in the patient with pulmonary edema or cardiomyopathy. Patients can have a transfusion-related acute lung injury resembling acute respiratory distress syndrome (ARDS) with dyspnea, hypoxia, and noncardiogenic pulmonary edema. Febrile reactions are associated with white cell antigens present in units of packed RBCs. Unfortunately, preventable fatal hemolytic reactions result from transfusion of ABO-incompatible blood.

Viral screening of blood products and careful selection of donors have certainly decreased the risk for transfusion transmission of viruses, such as human immunodeficiency virus (HIV) and hepatitis C. However, there is still a risk posed by donors who donate during the infectious period before seroconversion occurs. In addition to viral contaminants that can be tested, blood screening methods do not exclude other infectious agents, such as hepatitis A virus, human parvovirus B19, cytomegalovirus (CMV), malaria, and disease-causing bacteria.

Multiple published studies report an increased risk for infection and an increased mortality rate associated with transfusions. Exposure to the donor's leukocytes in transfusion may trigger an immune system response. Potential adverse outcomes include exacerbation of infections, earlier

recurrence of malignancy, impaired wound healing and other postoperative complications, and increased likelihood of mortality.

To avoid some of these complications, consideration may be given to leukoreduced blood transfusion for critically ill patients. Leukoreduction is the process of removing WBCs from the blood product by filtration. Leukoreduced blood is less likely to cause antibodies to develop against specific blood types and less likely to cause febrile transfusion reactions. Also, leukoreduced blood greatly reduces the chance of CMV transmission.

ERYTHROPOIETIC-STIMULATING PROTEINS. Administration of erythropoietic-stimulating proteins, such as epoetin and darbepoetin, may be a safer method of increasing hemoglobin and reducing the need for transfusion in many patients. Contraindications include uncontrolled hypertension and hypersensitivity to albumin. The optimal effective dose and timing of administering erythropoietic-stimulating proteins in critically ill patients have not yet been determined, and this is a topic of ongoing clinical trials.

Nursing Care of the Patient With Anemia

Nursing interventions in all anemias support treatment protocols and measures to identify the underlying cause. Other important actions include assessing for adverse effects of replacement therapy and for signs and symptoms indicative of decreased perfusion. If transfusion therapy is prescribed, vigilance in identifying the correct patient and ensuring ABO compatibility is of utmost importance to prevent adverse and potentially fatal outcomes. In addition, measures to decrease metabolic needs and reduce oxygen demand should be instituted, including promotion of a restful environment, adequate pain control, and minimization of agitation. Supplemental oxygen may be needed to assist in maintaining adequate oxygen supply to the tissues.

Critical care nurses are in an ideal position to identify when phlebotomy appears excessive or unnecessary and to question the clinical justification for testing. Strategies to reduce phlebotomy blood loss should be instituted. These include eliminating standing orders for laboratory tests, organizing blood draws to eliminate duplicate testing, consolidating multiple collections, using smaller collection tubes, and using devices that return waste blood to the patient. The frequency of arterial blood gas collection can often be decreased by using noninvasive monitoring techniques, such as pulse oximetry and capnography.

Sickle Cell Disease

Pathophysiology

Sickle cell disease (SCD) is a chronic hereditary hemolytic anemia that occurs almost exclusively in blacks; however, a variety of other ethnic groups can be affected. The sickle cell gene results in abnormal hemoglobin, usually hemoglobin S (HbS). When oxygen and pH levels fall, HbS RBCs become elongated, sickle or crescent shaped, and rigid. These sickled cells are unable to pass through small blood vessels, causing inflammation, obstruction of the vessels, and decreased delivery of oxygen that perpetuates the cycle with more sickling. The cells are hemolyzed or destroyed when the body recognizes their abnormal structure.

Clinical Presentation

The most common clinical picture in SCD is painful vaso-occlusive crisis. The crisis begins suddenly, sometimes as a consequence of infection or a change in temperature or for no identifiable reason. Severe deep pain is present in the long bones of the extremities. Sometimes, the abdomen is affected by severe pain resembling an acute abdomen. The pain may be accompanied by fever, malaise, and leukocytosis.

SCD also results in organ damage with microinfarctions of the heart, skeleton, spleen, and central nervous system (CNS). Repeated splenic infarctions result in splenic failure, predisposing the patient to overwhelming infection, especially Gram-negative sepsis. Other manifestations of SCD that may be seen in the critical care patient are stroke, cardiac chamber enlargement, pulmonary hypertension, renal failure, and chronic leg ulcers.

Acute chest syndrome is caused by pulmonary infarction from fat embolism; bacterial infections likely contribute to its development as well. Symptoms include chest pain, fever, tachypnea, leukocytosis, and pulmonary infiltrates. Acute chest syndrome is a medical emergency, and if not treated properly, complications, such as ARDS, may develop.

Management

Treatment of sickle cell crisis includes aggressive intravenous fluid hydration to decrease blood viscosity and maintain renal perfusion. The patient must be evaluated for infection, and if this is suspected, prompt treatment with broad-spectrum antibiotics is instituted until the causative organism is identified, and the therapy can be tailored to the patient's needs. Oxygen administration may be needed to maintain adequate tissue perfusion. RBC transfusions are not usually required. Patients with SCD take folic acid supplementation and may take hydroxyurea, which helps prevent sickling (see Evidence-Based Practice Highlight 49-1). Although activity is usually not restricted, the patient may not be able to tolerate exercise because of pain.

The pain experienced by patients in sickle cell crisis is intense. Patients require around-the-clock dosing with a strong narcotic, such as intravenous morphine. Frequent, lower doses of narcotics avoid the erratic levels of analgesia caused by infrequent doses of stronger narcotics. Time-release narcotics and patient-controlled analgesia are two methods

EVIDENCE-BASED PRACTICE HIGHLIGHT 49-1

Treatment of Sickle Cell Disease

Hydroxyurea is an important advance in the treatment of sickle cell disease. Strong evidence supports its use. It decreases severe painful episodes, hospitalizations, number of blood transfusions, and the acute chest syndrome, particularly in adults.

From Brawley OW, Cornelius LJ, Edwards LR, et al: National Institutes of Health consensus development conference statement: hydroxyurea treatment for sickle cell disease. Ann Intern Med 148(12):932–938, 2008.

of delivering steady doses of pain medication. Patients with sickle cell crisis may not display overt signs of pain, as is typical for patients who experience chronic pain. The patient's report of pain, as well as clinical indicators, should be used in assessing pain.

Nursing care of the patient with sickle cell crisis includes close monitoring for response to interventions to promote tissue perfusion, treat infection, and effectively manage pain. A multidisciplinary approach may include input from pain management experts, social workers, psychiatrists, physical and occupational therapists, and infectious disease specialists.

▲ Disorders of White Blood Cells

Leukopenia

Leukopenia refers to an abnormally low number of WBCs. The most common type of WBC deficiency is neutropenia, defined as a neutrophil count of less than 1,500 cells/mm^3. Severe neutropenia, in which the neutrophil count is less than 200 cells/mm^3, is referred to as agranulocytosis. Neutropenia can be seen as a result of a wide variety of conditions (Table 49-5). The most common cause of leukopenia is drug related.

Clinical Presentation

Because the neutrophil is essential to defense against bacterial and fungal infections, patients with neutropenia are susceptible to overwhelming infection and life-threatening sepsis. The risk for infection is related to the severity of the neutropenia. Untreated infections can be rapidly fatal, particularly if the neutrophil count is less than 250/mm^3. In severe neutropenia, the usual signs of the inflammatory response to infection may be absent.

Neutrophils provide the first line of defense against organisms that inhabit the skin and gastrointestinal tract. Thus, skin infections and ulcerative lesions of the mouth are common infections in the neutropenic patient. The most frequent site of serious infection is the respiratory tract, a result of bacteria or fungi that frequently colonize the airways.

Assessment

The history should include presence of viral infection or autoimmune diseases, such as systemic lupus erythematosus or rheumatoid arthritis. A thorough medication history, including over-the-counter preparations, should be obtained. Patients with neutropenia may present with mouth ulcers, fever, shaking chills, and systemic infection. Physical examination may reveal splenomegaly.

The presence of abnormal RBCs or WBCs suggests a primary bone marrow process. Laboratory studies should include viral serology for hepatitis and HIV as well as antinuclear antibody (ANA). Bone marrow aspiration and biopsy may be needed if the neutropenia is severe or the cause is not apparent.

Management

Neutropenia is treated by removing or managing the underlying cause if known. If the patient is febrile, appropriate cultures and a chest radiograph should be obtained, followed by immediate broad-spectrum intravenous antibiotic therapy to prevent progression to septic shock and death. Signs and symptoms of infection without fever should also prompt administration of antibiotics in patients with neutropenia. If evidence of infection persists despite adequate antibiotic therapy, antifungal agents may be added to provide coverage for potential *Candida* or *Aspergillus* infections.

If the neutropenia is severe, treatment may include a hematopoietic growth factor (filgrastim, pegfilgrastim, or sargramostim) that stimulates bone marrow production of new neutrophils and enhances the activity of already circulating neutrophils. Additionally, patients with severe neutropenia or those with recurrent or serious infections may benefit from steroids. IVIG may improve the neutrophil count. Transfusions are used cautiously in patients who may require bone marrow transplantation.

Table 49-5 Causes of Neutropenia

Cause	Mechanism
Accelerated removal (eg, inflammation and infection)	Removal of neutrophils from the circulation exceeds production
Drug-induced granulocytopenia	
Cytotoxic drugs used in cancer therapy	Depressed bone marrow function with decreased production of all blood cells.
Phenothiazines, propylthiouracil, and others	Toxic effect on bone marrow precursors
Aminopyrine, certain sulfonamides, phenylbutazone, and others	Immune-mediated destruction
Periodic or cyclic neutropenia (occurs during infancy and later)	Unknown
Neoplasms involving bone marrow (eg, leukemias and lymphomas)	Overgrowth of neoplastic cells, which crowd out granulopoietic precursors
Idiopathic neutropenia that occurs in the absence of other disease or provoking influence	Autoimmune reaction
Felty's syndrome	Intrasplenic destruction of neutrophils

From Porth CM: Essentials of Pathophysiology, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2007, p 183.

Neoplastic Disorders

Neoplastic disorders refer to those disorders characterized by new and abnormal growth of cells that may be benign or malignant. The clinical features of neoplastic disorders are determined largely by their site of origin. Lymphoproliferative disorders (disorders in which lymphoid tissue increases by reproducing) may originate in the bone marrow or in the lymph nodes and thymus. Lymphoproliferative disorders of the bone marrow are leukemias and multiple myeloma; lymphoproliferative disorders of the lymph nodes and thymus are lymphomas. Because blood cells circulate throughout the body, these neoplasms are systemically disseminated from the onset.

Lymphoma

HODGKIN'S LYMPHOMA. Hodgkin's lymphoma is a cancer of the lymphatic system, beginning as a malignancy in a single lymph node and then spreading to surrounding lymph nodes. A diagnosis of Hodgkin's disease is confirmed by the presence of abnormal Reed-Sternberg cells in biopsied tissue.

NON-HODGKIN'S LYMPHOMA. Non-Hodgkin's lymphoma (NHL) is a diverse group of malignancies that originate in the lymphoid cells. NHL can occur as a discreet mass, such as a single lymph node, or as a widespread disease that affects multiple organ systems, including the bone marrow.

CLINICAL PRESENTATION. Symptoms of both types of lymphoma are related to the body area affected by the rapid growth of abnormal lymphoid cells. Constitutional symptoms include fever, fatigue, and weight loss. In the advanced stages, patients may have extensive chest disease and experience increasingly severe dyspnea. Bulky chest disease can cause superior vena cava syndrome. Abdominal disease can cause obstruction of the bowel or the ureters. Bone marrow involvement can lead to decreased production of RBCs, WBCs, and platelets. Extensive involvement of the lymphatic system can lead to impaired immune function and frequent, severe infections. Lymphoma of the CNS can cause headaches, visual disturbances, motor dysfunction, and increased intracranial pressure.

MANAGEMENT. The diagnosis, staging, and treatment of lymphomas are typically performed entirely on an outpatient basis. The care of a critically ill patient with lymphoma is usually for the management of complications that arise from the disease or its treatment. Oncological complications and their management are discussed in detail in Chapters 47 and 48.

Leukemia

The leukemias are hematologic malignancies that affect the bone marrow and the lymph tissues. They are characterized by proliferation of hematopoietic stem cells, resulting in the accumulation of abnormal (leukemic) cells in the bone marrow and a decreased production of normal blood cells. These abnormal leukemic cells circulate in the blood stream and infiltrate many body organs. Leukemias commonly are classified according to their predominant cell type (lymphoblastic or myelogenous) and whether the condition is acute or chronic. The chronic leukemias are not discussed in this chapter.

ACUTE LYMPHOBLASTIC LEUKEMIA. Acute lymphoblastic leukemia (ALL) is a disorder in which a clone of immature lymphocytes proliferates and replaces the normal cells of the bone marrow. The leukemic clones proliferate and infiltrate other normal tissues, such as the liver, spleen, and lymph nodes. Anemia, thrombocytopenia, and granulocytopenia are common. The spleen, liver, thymus, and lymph nodes are usually enlarged.

ACUTE MYELOGENOUS LEUKEMIA. A malignant disorder of hematopoietic stem cells, acute myelogenous leukemia (AML) causes abnormal production of the myeloid cell lines (erythrocyte, neutrophil, megakaryocyte, and macrophage). The malignant clones proliferate but do not differentiate into mature, functional cells. The blood, bone marrow, or both contain more than 30% immature blast cells. The proliferation of immature cells and bone marrow infiltration results in anemia, neutropenia, and thrombocytopenia. The patient's symptoms are related to these conditions. Splenomegaly is present in approximately one third of patients. Patients may require immediate interventions at the time of diagnosis for infections or anemia or to achieve hemostasis.

CLINICAL PRESENTATION. Although ALL and AML are distinct disorders, they typically present with similar clinical features. These manifestations and their pathologic basis are summarized in Table 49-6.

Patients with leukocyte counts greater than 100,000 cells/mm^3 are at risk for leukostasis, a condition in which the high number of blasts increase blood viscosity, develop leukoblastic emboli, and aggregate in the capillaries. Manifestations include headache, confusion, CNS infarctions, acute respiratory insufficiency, and pulmonary infiltrates.

MANAGEMENT. Leukostasis requires immediate treatment to lower the blast count rapidly. Initial treatment involves emergent leukapheresis (removal of WBCs from the circulation) along with hydroxyurea. Chemotherapy should be initiated to stop leukemic cell production in the bone marrow.

A comprehensive discussion of diagnosis and treatment of leukemia is beyond the scope of this text. As in the patient with lymphoma, the patient with leukemia will typically be seen in the critical care unit as a result of complications of the disorder or its treatment. Refer to discussions of anemia, granulocytopenia, and thrombocytopenia in this chapter, as well as Chapters 47 and 48, for coverage of bone marrow or stem cell transplantation and oncological emergencies.

Nursing Care of the Patient With a White Blood Cell Disorder

The goals of nursing care of the patient with a WBC disorder are vigilant assessment for and prevention of infection, delivery of therapy, and management of disease and treatment-associated complications. Nursing interventions are guided

Table 49-6 Clinical Manifestations of Acute Leukemia and Their Pathologic Basis*

Clinical Manifestations	Pathologic Basis
Bone marrow depression	
Malaise, easy fatigability	Anemia
Fever	Infection or increased metabolism by neoplastic cells
Bleeding	Decreased thrombocytes
Petechiae	
Ecchymosis	
Gingival bleeding	
Epistaxis	
Bone pain and tenderness upon palpation	Subperiosteal bone infiltration, bone marrow expansion, and bone resorption
Headache, nausea, vomiting, papilledema, cranial nerve palsies, seizures, coma	Leukemic infiltration of central nervous system
Abdominal discomfort	Generalized lymphadenopathy, hepatomegaly, splenomegaly due to leukemic cell infiltration
Increased vulnerability to infections	Immaturity of the white cells and ineffective immune function
Hematologic abnormalities	Physical and metabolic encroachment of leukemia cells on RBC and thrombocyte precursors
Anemia	
Thrombocytopenia	
Hyperuricemia and other metabolic disorders	Abnormal proliferation and metabolism of leukemic cells

*Manifestations vary with the type of leukemia.

From Porth CM: Essentials of Pathophysiology, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2007, p 189.

by the treatment modality (eg, chemotherapy, radiation, bone marrow transplantation).

Meticulous attention to infection control procedures and vigilant surveillance of invasive therapeutic lines and equipment are mainstays of care. Attention must be paid during daily oral care to ensure that superinfection with *Candida* or herpesvirus has not developed. Antibacterial mouthwashes decrease the risk for infection. Severe mucositis may require opiates for pain control. Assessment for early indications of infection (eg, fever, chills, tachycardia, and tachypnea) may allow for prompt and aggressive initiation of pharmacological therapies to reduce morbidity and mortality associated with infection in patients with WBC disorders.

Diarrhea may be a side effect of chemotherapy, neutropenia, or secondary causes. *Clostridium difficile* infection must be considered and treated if present. Digital rectal examinations should not be performed on neutropenic patients.

▲ Disorders of Hemostasis

The term hemostasis refers to the stoppage of blood flow.[2] The hemostatic system is a finely balanced network of coagulation factors and endothelial and platelet components. Hemostasis may be impaired by disruption of numerous interactions in the coagulation pathways or deficiencies or dysfunction in the required components for coagulation. Disorders of hemostasis include hypercoagulable states and bleeding disorders, whether from platelet defects (thrombocytopenia) or coagulation defects. Disorders of hemostasis are often seen in critically ill patients.

Platelet Disorders

Normally, approximately two thirds of platelets are circulating in the blood, and one third are sequestered or stored in the spleen. The life span of circulating platelets is 7 to 10 days. Thrombocytopenia occurs when there is decreased production or increased destruction of platelets or increased sequestration of platelets in the spleen. Common causes of thrombocytopenia are listed in Table 49-7. The most common causes seen in critical care patients are discussed here. A complete etiological discussion is beyond the scope of this chapter.

Table 49-7 Causes of Thrombocytopenia

Decreased Platelet Production	Splenic Sequestration of Platelets	Increased Destruction of Platelets
Marrow infiltration	Splenic enlargement	Nonimmune
Malignancy	Tumor infiltration	Vascular prostheses
Myelofibrosis	Infection	Disseminated intravascular coagulation (DIC)
Granulomatous disease	Splenic congestion	Sepsis
Marrow failure	Portal HTN or liver disease	Vasculitis
Medication		Thrombotic thrombocytopenic purpura (TTP)
Chemotherapy		Immune
Aplastic anemia		Autoantibody (idiopathic thrombocytopenic purpura)
Severe iron deficiency		Drug-associated*
Infection (HIV, Epstein-Barr virus, TB)		Circulating immune complexes (systemic lupus erythematosus, viral, bacterial sepsis)
Alcohol use		Posttransfusion ab (PLA1)
Nutritional deficiency		
Iron, folate, vitamin B_{12}		

*One of most common causes of thrombocytopenia.

From Kwoh C, Buch E, Quartarolo J: The Washington Manual General Internal Medicine Consult. Philadelphia, PA: Lippincott Williams & Wilkins, 2004, p 167.

Types of Thrombocytopenia

DRUG-INDUCED THROMBOCYTOPENIA. The most common cause of thrombocytopenia in hospitalized patients is drug induced. The confirmation of this diagnosis occurs when the thrombocytopenia resolves after withdrawal of the causal medication. Medications commonly associated with thrombocytopenia include antineoplastic agents, heparin, histamine$_2$-blockers, trimethoprim-sulfamethoxazole, rifampin, vancomycin, amiodarone, and valproic acid; however, this list is not complete.

HEPARIN-INDUCED THROMBOCYTOPENIA. After antineoplastic agents, heparin is the next most common drug associated with thrombocytopenia. Heparin-induced thrombocytopenia (HIT) is interesting in that thrombosis, rather than bleeding, may accompany the low platelet count. HIT places patients at risk for life-and limb-threatening consequences, including deep venous thrombosis, arterial occlusion, ischemic stroke, limb gangrene, myocardial infarction, and pulmonary embolism.

It is important to be aware that subcutaneous unfractionated heparin (UFH) or low-molecular-weight heparin (LMWH) delivered at treatment or prophylaxis dosing, by arterial or venous line flushes, through heparin-coated catheters, and intermittent heparin administered during dialysis may lead to HIT. Although HIT typically occurs 4 to 14 days after initiation of heparin therapy, it can occur as early as 10 hours after administration if the patient has been exposed to heparin within the previous 100 days. For patients who are starting heparin therapy, whether UFH or LMWH, the recommendation is to obtain a baseline platelet count and then repeat a platelet count within 24 hours of starting heparin. Clinical guidelines also suggest platelet count monitoring at least every 2 or 3 days up to 14 days or until heparin therapy is stopped[3].

The hallmark of HIT is a decrease in platelet count to less than 50% of baseline or to fewer than 150,000/mm^3 or the occurrence of an unexplained thromboembolic event. Blood testing should be done for HIT antibodies.

For patients with confirmed or strongly suspected HIT, whether or not complicated by thrombosis, national guidelines recommend using a nonheparin anticoagulant (see Box 49-6).[3] Even if there is no clinical evidence of deep vein thrombosis, ultrasonography of the leg veins should be performed. Warfarin therapy should not be started until after the platelet count has substantially recovered, usually to at least 100,000/mm^3. Nonheparin anticoagulant should be continued until the platelet count has reached a stable plateau, the international normalized ratio (INR) has reached the intended target range, and after a minimum overlap of at least 5 days between nonheparin anticoagulation and warfarin therapy. Platelets are generally not transfused unless the patient is actively bleeding.

BOX 49-6 Nonheparin Anticoagulants

- lepirudin (renally adjust dose; APTT monitoring)
- argatroban (APTT monitoring)
- fondaparinux
- bivalirudin

THROMBOTIC THROMBOCYTOPENIC PURPURA. Thrombotic thrombocytopenic purpura (TTP) is an acute disorder with a mortality rate of 30% to 40%. Patients with TTP have absent or decreased levels of platelet-aggregating factor inhibitor, which is normally present in plasma. As a result, platelets become sensitized and clump in blood vessels, causing occlusion.

A presentation of five classic findings suggests TTP. Not every patient exhibits all five findings; however, the first two of the following conditions must be present for the diagnosis to be considered:

- Thrombocytopenia and bleeding from increased consumption of platelets
- Microangiopathic hemolytic anemia from rupture of RBCs as they try to pass through partially occluded blood vessels
- Fever, possibly from hemolysis or vascular infarction of the hypothalamus
- Neurologic abnormalities, including fluctuating and often bizarre neurological abnormalities, transient ischemic attacks, strokes, seizures, and coma from interrupted blood flow to the brain
- Renal dysfunction caused by obstruction of intraglomerular capillaries and infarction of the renal cortex

Clinical features of TTP are described further in Table 49-8. Initiation of the disease process may be related to endothelial damage, autoimmune disorders, viral and bacterial infections, toxic agents, and genetic predisposition.

IMMUNE THROMBOCYTOPENIC PURPURA. Immune thrombocytopenic purpura (ITP) is an immune-mediated disorder of platelet destruction. Historically, ITP stood for idiopathic thrombocytopenic purpura because the mechanism of thrombocytopenia was not understood. However, now the pathophysiology of the disease is clearer, and some have suggested that the name auto ITP might be more accurate.

There are two distinct forms of ITP. Acute ITP typically occurs in childhood and may resolve spontaneously in several

Table 49-8 Clinical Manifestations of Thrombotic Thrombocytopenic Purpura

Abnormality	Findings
Thrombocytopenia	Bleeding, ecchymosis, purpura at various sites
Hemolytic anemia	Schistocytes, reticulocytosis, elevated serum lactate dehydrogenase and bilirubin, jaundice, pallor, weakness
Neurological abnormalities	Headache, mental changes, confusion, visual problems, seizures, coma, aphasia, dysphasia, paresthesias
Renal dysfunction	Proteinuria, microscopic hematuria, elevated blood urea nitrogen (BUN) and creatinine, renal failure
Fever	Persistent elevation of temperature during acute phase
Other	Abdominal pain, malaise, nausea, vomiting, weakness, ECG changes

weeks. Autoimmune platelet destruction appears to be stimulated after a viral illness, even mild infections. Chronic ITP usually occurs in adults, more often in women than men. The platelet membrane is coated with an autoantibody (usually IgG), and the sensitized platelets are destroyed in the spleen and liver. Therefore, platelet survival in the circulation is decreased. In at least 50% of patients with ITP, no known causative agent is identified; other patients may have underlying autoimmune, rheumatic, or lymphoproliferative diseases or HIV infection. ITP is diagnosed when other disorders of platelet destruction are ruled out.

Assessment

The history, physical examination, and initial laboratory data help differentiate between the mechanisms of thrombocytopenia.

The patient history includes assessment of symptoms associated with any of the risk factors or associated disorders (see Table 49-8). A patient or family history of bleeding may help differentiate between acquired and congenital disorders. Critical to the assessment is an accurate medication and alcohol history. Fatigue, fever, weight loss, or night sweats may be associated with infection or malignancy. It is also important to note a history of platelet transfusion.

The physical examination includes a thorough examination of the skin for petechiae and bruising. It is important to also include the oropharynx in this examination as well as checking the stool for guaiac. Hypotension, tachycardia, and fever are suggestive of sepsis. A fever is also frequently present in TTP. An enlarged spleen suggests splenic sequestration of trapped blood from portal hypertension. In this setting, patients are likely to exhibit signs of liver disease, including jaundice, ascites, and extremity muscle wasting.

Laboratory examination is also significant. Signs and symptoms of low platelets vary depending on the platelet count. Platelet counts greater than 100,000/mm^3 should result in normal hemostasis. Platelet counts less than 50,000/mm^3 may lead to prolonged bleeding after procedures, easy bruising, and bleeding from the oral or gastrointestinal mucosa. Platelet counts less than 20,000/mm^3 are associated with an exponential increase in the risk for bleeding The nurse carefully assesses the patient for petechial rash or spontaneous bleeding such as epistaxis or bleeding gums. Platelet counts less than 10,000/mm^3 are associated with possible intracranial hemorrhage.

The complete blood count with differential is the most important first step to identifying the etiology of thrombocytopenia. It is important to check for accompanying anemia and leukopenia. If pancytopenia is present, bone marrow failure may be the cause of the problem. If this is suspected, a bone marrow biopsy may be performed. The presence of microcytosis or macrocytosis suggests vitamin deficiency. Severely decreased ferritin, folate, or vitamin B_{12} levels also support this as an etiology. The presence of schistocytes on the differential may indicate TTP or DIC.

It is important to rule out artifactual thrombocytopenia caused by clumping of the platelets in a test tube containing ethylenediamine tetraacetic acid (lavender top). A report of platelet clumping should raise this suspicion. If the platelets are clumped, the automatic counter may inaccurately measure the number of platelets resulting in a falsely low count. In this case, the sample should be redrawn in a citrate tube (blue top) or heparinized tube (green top).

Bleeding time is a test of platelet function. When the platelet count drops below 50,000/mm^3, the bleeding time is significantly prolonged and is not useful. The bleeding time is a helpful test when a patient has mucosal bleeding but a normal platelet count. Evaluating prothrombin time (PT), partial thromboplastin time (PTT), and fibrin degradation products (FDPs) may help identify DIC and distinguish a hemolytic anemia from TTP. Additional studies that may be ordered to assist in determining the underlying etiology include HIV and ANA screening as well as liver function tests.

Management

The initial step in management of thrombocytopenia is to review the patient's medication list, placing on hold any drugs that may be suspected to induce thrombocytopenia. In addition, medications that inhibit platelet function should be avoided, including aspirin, antiplatelet agents, and nonsteroidal antiinflammatory drugs. Avoidance of trauma is essential and may even preclude the placement of central venous catheters and other invasive procedures. Evaluation of continued blood loss and assessment of daily laboratory data for adequacy of the platelet count and other parameters of hemostasis are important measures.

Patients with mild to moderate thrombocytopenia without bleeding require no treatment. Platelet transfusions are indicated when the platelet count is below 20,000/mm^3 or in the case of spontaneous bleeding. A single donor unit of platelets is equivalent to six random donor platelets and should increase the platelet count by 30,000/mm^3. The patient must be monitored for allergic reactions, anaphylaxis, and volume overload.

In sequestration or destruction-mediated thrombocytopenia, the response to platelet transfusion is typically very poor. Transfused platelets are destroyed very quickly by the same mechanism that causes the disease. Platelet transfusion should be used only for major life-threatening bleeding. In these patients, because the platelets may not last long, platelets need to be administered shortly before performing any invasive procedures.

TTP is an emergency because of its extremely high mortality rate. Early recognition and prompt initiation of treatment are imperative to improve patient survival. Acutely ill patients require plasma exchange in which plasmapheresis is used to remove 2 to 3 L of the patient's plasma, with an equal amount of fresh plasma given as replacement. Plasmapheresis is initiated as quickly as possible and repeated daily until the platelet count is greater than 150,000/mm^3. This may take 5 to 10 days or more. Plasma exchange is superior to simple plasma infusion. However, because some patients do respond to simple plasma infusions, all patients should be infused immediately with fresh frozen plasma until plasma exchange can be arranged. Antiplatelet agents and prednisone may be used, although the effectiveness of these therapies is controversial. Platelet transfusion is contraindicated even in severe thrombocytopenia because the transfused platelets may aggregate, resulting in myocardial infarction, stroke, coma, or death. Patients with TTP may be "cured," may relapse years later or may have a chronic relapsing course.

Immunosuppression with corticosteroids is the initial therapy for ITP. Prednisone therapy usually takes a few days

before the count begins to rise. When the platelet count is critically low (5,000/mm^3 or less) or the patient shows signs of serious bleeding, prednisone alone may not raise the platelet count rapidly enough. When a rapid increase in the platelet count is needed, IVIG, in addition to steroids, is highly successful. A typical treatment regimen is IVIG at 1 g/kg/d and prednisone 1 mg/kg/d. Patients with chronic ITP who fail to respond to steroids or are steroid dependent may require splenectomy. These patients should receive pneumococcal, meningococcal, and *Haemophilus influenzae* type B vaccines.

Coagulation Disorders

Coagulation disorders can result from deficiencies or impairment of one or more of the clotting factors. These disorders of coagulation may be congenital or acquired. The most common congenital bleeding disorders are von Willebrand's disease and hemophilia A. These disorders are caused by a deficiency in a specific factor. Numerous complications can occur as a result of these bleeding disorders (Table 49-9).

Management involves correcting the coagulation factor deficiency and treating sequelae that occur as a result of abnormal bleeding. Patients with mild hemophilia A and mild von Willebrand's factor deficiency may respond to intravenous or nasal spray administration of desmopressin acetate, a hormone that temporarily stimulates the release of factor VIII to control bleeding. More severe cases of hemophilia A or active bleeding require intravenous infusion of factor VIII concentrate. More severe bleeding in von Willebrand's disease requires factor VIII concentrate that contains von Willebrand's factor. von Willebrand's factor is also found in cryoprecipitate, and factors VIII and IX are in fresh frozen plasma.

Disseminated Intravascular Coagulation

Pathophysiology

Disseminated intravascular coagulation (DIC) is defined as the inappropriate triggering of the coagulation cascade and a breakdown in the normal feedback mechanisms in the body that allow for the dissolution of clots. Instead of a localized response to tissue damage or vascular injury, there is systemic coagulation activity, resulting in diffuse intravascular fibrin formation and widespread intravascular clotting. Eventually, coagulation factors become depleted as the body attempts to dissolve the newly formed clots. Because of the rapidity of intravascular thrombin formation, clotting factors are used up at a rate exceeding factor replenishment. In essence, there is an imbalance between the natural procoagulant and anticoagulant systems in the body. The result is unregulated thrombin activity, microvasculature thrombi, platelet consumption, and microangiopathic hemolytic anemia.

Activation of coagulation mechanisms also activates the fibrinolytic system. The breakdown of fibrin and fibrinogen results in FDPs and d-dimers. These products interfere with platelet function and the formation of the fibrin clot. In addition, plasmin can activate the complement and kinin systems, leading to increased vascular permeability, hypotension, and shock. Thus, the patient has a simultaneous, self-perpetuating combination of diffuse clotting and simultaneous hemorrhage occurring in response to the precipitating event as well as the potential for hemodynamic instability (Fig. 49-1).

Table 49-9 Complications in Coagulation Disorders

Bleeding Site	Complication
Abdomen	Hypotension, hypovolemic shock
Muscle	Compartment syndrome
Joint	Hemarthrosis with destruction of bone and cartilage in joint capsule
Intracranial	Increased intracranial pressure
Retropharyngeal	Airway obstruction
Gastrointestinal	Anemia, melena
Urinary tract	Clots in ureters (especially after factor administration)

Etiology

DIC is a secondary complication that requires an underlying condition that serves as the initial triggering event for the inappropriate stimulation of clotting. This involves the activation of any of the coagulation pathways. The extrinsic pathway is activated by damage to the endothelial lining of blood vessels. Common causes include surgery, burns, heat stroke, bacterial endotoxin, and malignant tumors. The intrinsic pathway is activated when subendothelial tissue is exposed to the bloodstream and circulating factor XII comes in contact with the exposed tissue. The exposure may follow vascular injury or damage from immune complexes or bacterial endotoxins. The result is clot formation and activation of the coagulation cascade. Endotoxins released by Gram-negative bacteria and the resulting sepsis are significant triggers of DIC, accounting for approximately 20% of cases.

Shock or low-flow states can result in metabolic acidosis, tissue ischemia, and necrosis, which may also lead to clot formation. In cancer, a common etiology of DIC, the condition is caused by tumors eroding tissue with subsequent release of thromboplastin or stimulation of factor XII from vascular injury, as well as by the autolysis of tumor cells in rapidly proliferating tumors. In cancers that are able to autolyse (eg, acute leukemia, Burkitt's lymphoma, small cell lung cancer), the cell fragments that result from the lysis are seen as "foreign bodies" and stimulate clotting. Still other cancers release procoagulants that enhance clotting (eg, mucin-producing adenocarcinomas, prostate and renal cancer, promyelocytic leukemias, and brain tumors).[4] Box 49-7 outlines some malignant and nonmalignant states of physiological disequilibrium that can be precipitating factors for DIC.

Clinical Presentation

Clinical consequences include systemic ischemia from the thrombi formation as well as minor or major hemorrhage from ongoing fibrinolysis and depletion of clotting factors. A patient with DIC is vulnerable to a wide variety of complications resulting from the thrombotic or hemorrhagic disease processes. Thrombosis may result in ischemia or infarction in any organ, with concomitant loss of function. With depletion of clotting factors and platelets, bleeding into subcutaneous

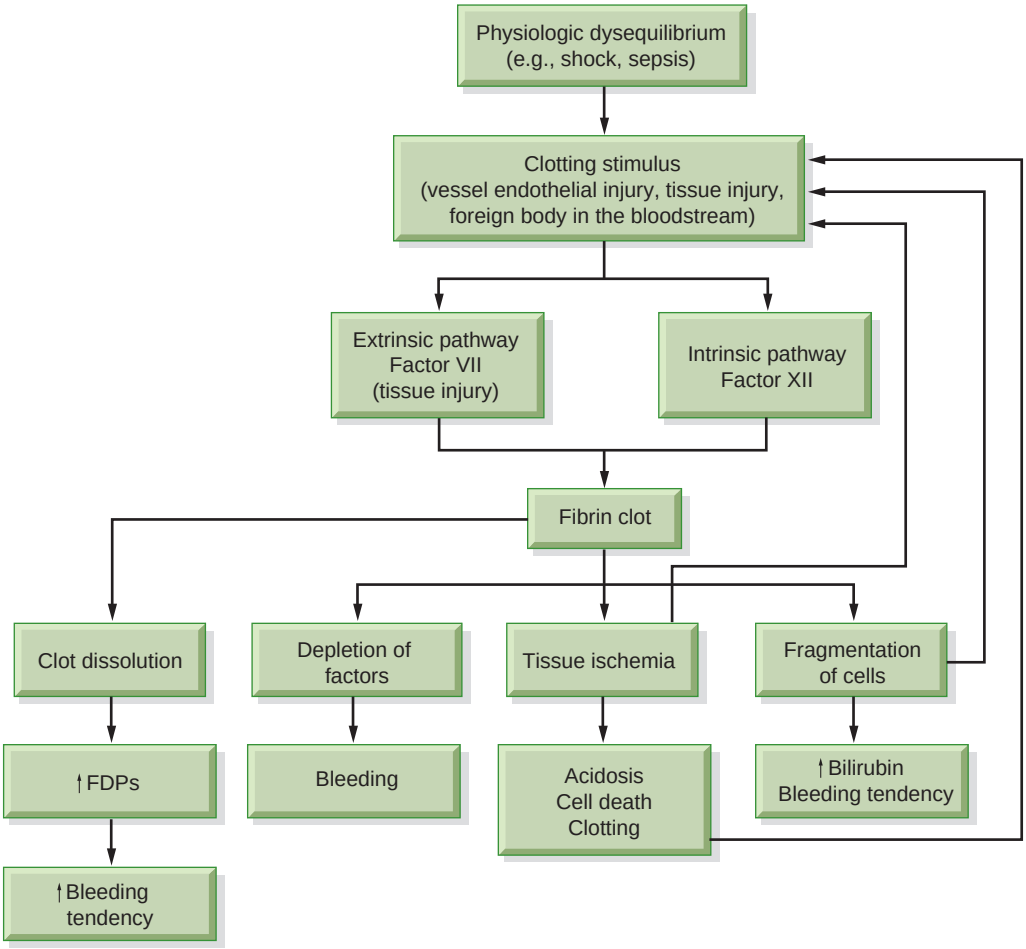


FIGURE 49-1 ▲ Self-perpetuating cycle of thrombosis and bleeding in disseminated intravascular coagulation (DIC).

tissues, skin, and mucous membranes, or more serious hemorrhage, may result.

Assessment

All critically ill patients are at risk for developing DIC because many are in the state of physiological disequilibrium characterized by hypovolemia, hypotension, hypoxia, and acidosis, all of which have procoagulant effects. In addition, the patient's critical illness may have been triggered by an injury that itself could result in the development of DIC. Increased awareness of DIC as a potentially catastrophic complication in the critically ill patient has resulted in earlier recognition and intervention. The critical care nurse who is armed with knowledge of physiological norms and who uses a systemic approach to assessment may be the first person to identify the early signs of coagulation dysfunction and its probable trigger.

BOX 49-7 Selected Disease States Associated With DIC

Nonmalignant

- Bacterial infections
- Viral infections (HIV, cytomegalovirus, hepatitis)
- Burns
- Heat stroke
- Brain injury
- Crush injuries and necrotic tissue
- Obstetrical complications (amniotic fluid embolism, missed abortion, eclampsia, retained dead fetus, abruptio placentae)
- Intravascular hemolysis (hemolytic transfusion reactions, massive blood replacement therapy)
- Acute liver disease
- Intravascular prosthetic devices
- Prolonged low-cardiac output states (cardiac failure, prolonged cardiopulmonary bypass, hemorrhagic shock, cardiopulmonary arrest)
- Vasculitis
- Immunological disorders (immune complex disorders, allograft reaction, incompatible blood transfusion)
- Surgery
- Other (snake venom, vascular malformations, fat embolism)

Malignant

- Leukemias
- Solid tumors (adenocarcinoma, lung cancer, breast cancer, hepatic cancer, colon cancer, prostate cancer, brain cancer)
- Chemoradiation therapy

HISTORY AND PHYSICAL EXAMINATION. DIC may be acute in presentation, evidenced by severe clinical deterioration, or chronic in presentation, evidenced by mildly abnormal laboratory values and minimal, varied clinical symptoms. In chronic DIC, thrombosis is the prevalent disorder, and the degree of symptomatology is related to the ability of the liver and bone marrow to compensate for the disorder. Affected patients may have low-grade bleeding if factors become depleted, unexpected thrombotic events (including large vessel thrombi), or both. Chronic DIC must be ruled out in patients with multiple thrombotic sites developing simultaneously, serial thromboses, superficial venous thromboses, or arterial thromboses, especially in the presence of malignancy.[4]

However, most critical care nurses care for patients with acute DIC. It is important to realize that the assessment varies as the disease state evolves. Clinical assessment is organized according to the basic pathological process of DIC: clot formation with resulting emboli and perfusion defects or unchecked clot dissolution with resultant bleeding. The nurse evaluates the patient for signs and symptoms of inappropriate clotting: cyanosis, gangrene, mental status changes, altered level of consciousness, cerebrovascular accident, pulmonary embolus, bowel ischemia and infarction, and renal insufficiency or failure. Thrombosis may involve both arteries and veins, and clinical examination may reveal demarcation cyanosis (total occlusion of microvessels, most common in digits but may be evident in earlobes). In addition, the nurse evaluates the patient for signs of bleeding: bleeding from the nose, gums, lungs, gastrointestinal tract, surgical sites, injection sites, and intravascular access sites; hematuria; petechial rashes; and purpura fulminans. Bleeding is a manifestation of later disease because it is evidence of depletion of clotting factors, bleeding diathesis, or both.

Care of the patient with possible DIC requires constant reassessment and interpretation of findings. For example, the patient with a dull headache is likely to have a thrombotic defect, whereas a sudden and acute headache is more likely to be hemorrhagic. Dyspnea can be from a thrombotic disorder (pulmonary embolism) or from a hemorrhagic disorder (bleeding in the lungs). Either condition may present with hemoptysis and blood with suctioning. Hypotension may result from myocardial infarction (a thrombotic disorder) or cardiac tamponade (a hemorrhagic disorder). Ischemic bowel is characterized by decreased bowel sounds and a crampy and painful abdomen, with potential gastrointestinal bleeding when the mucosal layer sloughs, whereas gastrointestinal bleeding presents with heme-positive to melanotic stool and hyperactive bowel sounds. Nurses must monitor patients with DIC closely for signs and symptoms of the onset of shock, either hypovolemic or ischemic in origin. When caring for patients at risk for DIC, constant assessment of all body systems and critical thinking are necessary.

LABORATORY STUDIES. Regardless of the inciting event, four basic diagnostic components of DIC can be appreciated: excessive rate of clot formation; increased rate of clot dissolution; consumption of essential clotting factors; and end-organ damage resulting from the excessive clotting process.

Table 49-10 outlines studies that are commonly used to assess DIC. These studies, unfortunately, are neither specific nor sensitive, and results vary throughout the course of the disease. For the average patient, thrombocytopenia occurs as platelets are consumed during the unchecked clotting, followed by hypofibrinogenemia. Normal fibrinogen levels do not preclude DIC because it is an acute-phase reactant. Serial measurements of platelet count, PT, PTT, and fibrinogen levels help gauge disease progression. Peripheral smears

Table 49-10 Laboratory Findings in Acute DIC

Test	Normal Value	Value in DIC
Massive Intravascular Clotting		
Platelet count	150,000–400,000/mm^3	Decreased
Fibrinogen level	200–400 mg/100 mL	Decreased
Thrombin time	7.0–12.0 s	Prolonged
Protein C level	4 mcg/mL	Decreased
Protein S level	23 mcg/mL	Decreased
Secondary Depletion of Essential Clotting Factors		
Prothrombin time	11–15 s	Prolonged
Activated partial thromboplastin time	30–40 s	Prolonged
International normalized ratio	1.0–1.2 times normal	Prolonged
Excessive/Accelerated Fibrinolysis		
Fibrin degradation products	<10 mg/mL	Increased
D-dimer assay	<50 mcg/dL	Increased
Antithrombin III level	89%–120%	Decreased
Clinical Effects of Microvascular Clotting/Cell Destruction		
Schistocytes on peripheral smear		Present
Bilirubin level	0.1–1.2 mg/dL	Increased
Blood urea nitrogen	8–20 mg/dL	Increased

may reveal the presence of schistocytes reflecting fragmentation of RBCs moving through clots or partially occluded vessels.

Management

The backbone of therapy for DIC is eliminating the causative agent. The culprit that activates the clotting factors must be eliminated, whether through antibiotic or antifungal therapy for sepsis, antineoplastic therapy, rehydration, increasing oxygenation, or resolution of low-flow states. Unfortunately, some causes (such as burns, crush injury, and brain injury) cannot be as easily eliminated. General treatment principles include avoiding vasoconstriction that may worsen perfusion defects, maintaining adequate fluid volume status, and screening for and eliminating all medications that may enhance bleeding. Attention is also directed to correcting hypotension, hypoxia, and acidosis, all of which have procoagulant effects (Box 49-8).

Patients *at risk for* DIC as a result of sepsis may be given activated protein C (drotrecogin alfa [activated], marketed as Xigris) to slow the development of uncontrolled clotting that may result from the septic process. There are stringent criteria for using activated protein C. The argument for use is that with aggressive treatment of the causative agent (sepsis), it may be possible to decrease the rate of clot formation, slow the clotting cascade, and reclaim the balance between clotting and fibrinolysis. This agent has not been investigated, however, for its potential benefit in patients with confirmed DIC.

Heparin therapy may be initiated to minimize further clotting by increasing neutralization of thrombin. However, the risk for increased bleeding is always a major concern. In acute DIC, few clinical studies have shown heparin's effectiveness in slowing the coagulation cascade.

Replacement therapy and repletion of clotting factors are the focus of the treatment for significant hemorrhage. Fresh frozen plasma contains components of both the coagulation

BOX 49–8 *COLLABORATIVE CARE GUIDE for the Patient With DIC*

Outcomes	Interventions
Oxygenation/Ventilation	
Arterial blood gases are within normal limits.	• Monitor pulse oximetry and/or arterial blood gases. • Transfuse as necessary to increase oxygen carrying capacity. • Maintain oxygen delivery whether noninvasive or mechanical ventilation.
Breath sounds are clear bilaterally.	• Suction oropharynx and trachea carefully when necessary (see Chapter 25, Box 25-7, p. 516) • Turn, cough, deep-breathe, and use incentive spirometer every 2 h.
Circulation/Perfusion	
Patient will achieve/maintain tissue perfusion.	• Monitor tissue perfusion: color, temperature, pulses, level of consciousness, urinary output, and SaO_2/PaO_2. • Monitor vital signs every 1–4 h based on clinical condition. • Monitor cardiac output, systemic vascular resistance, and pulmonary artery pressure every 4 h if pulmonary artery catheter in place. • Administer blood products, positive inotropic agents, intravenous (IV) infusions as ordered.
Hematological	
Patient will not experience bleeding related to coagulopathies.	• Monitor PT, PTT, and complete blood count daily; more frequently if monitoring for acute changes or response to therapy. • Assess every 2–4 h for thrombotic and hemorrhagic manifestations. • Quantify degree of bleeding (count dressing changes, measure bodily drainage; test stool, urine, and emesis for heme). • Assess organ systems for signs and symptoms: change in mental status with cerebral hemorrhage or thrombotic event; decreased SaO_2 and hemoptysis with pulmonary bleeding; visual changes (diplopia, blurred vision, visual field deficit) with retinal thrombosis/hemorrhage; back pain, flank pain, abdominal pain consistent with visceral organ bleeding. • Administer blood products and coagulation factors as indicated. • Maintain strict adherence to bleeding precautions. • Minimize invasive procedures and treatments • Avoid medications that inhibit coagulation or promote thrombosis.
Fluids/Electrolytes	
Patient is euvolemic.	• Trend daily weight. • Monitor intake and output; replace/diurese as required. • Maintain IV access and fluid replacement therapy.
Mineral and electrolyte levels are within normal limits.	• Monitor and replace electrolytes daily and PRN.

(*continued on page 1148*)

BOX 49–8 COLLABORATIVE CARE GUIDE for the Patient With DIC (continued)

Outcomes	Interventions
Mobility/Safety	
There is no evidence of bleeding due to preventable injury.	• Institute bleeding precautions (soft toothbrush, electric razor, no rectal temperatures). • Provide safe physical environment. • Apply pressure to puncture sites for 3–5 min, and then use pressure dressing.
Skin Integrity	
Skin will remain intact.	• Turn patient every 2 h and assess skin each time patient is repositioned for pressure areas, petechiae, and ecchymosis. • Assess injection sites and incisions for bleeding. • Consider pressure relief/reduction mattress, avoid shearing forces. • Use Braden scale to assess risk for skin breakdown.
Nutrition	
Caloric and nutrient intake meets metabolic requirements per calculation (eg, basal energy expenditure).	• Provide parenteral feeding if patient is NPO. • Assess for gastrointestinal bleeding and report. • Consult dietitian or nutritional support service.
Comfort/Pain Control	
Patient will be as comfortable as possible as evidenced by stable vital signs or cooperation with treatments or procedures.	• Objectively assess comfort/pain using a pain scale. • Correlate pain ratings with sites of potential ischemia or hemorrhage. • Provide analgesia and sedation as indicated by assessment. • Monitor patient response to medication.
Psychosocial	
Patient demonstrates decreased anxiety.	• Provide areas of control to patient and family as possible (eg, performance of ADLs, visitors) • Provide explanations and reassurance before procedures. • Consult social services and pastoral care as appropriate. • Provide for adequate rest and sleep.
Teaching/Discharge Planning	
Patient/significant others understand procedures and tests needed for treatment.	• Educate patient and family regarding disease process, need for intensive monitoring, and actions taken to correct disorder. • Prepare the patient and family for procedures, such as blood transfusions and laboratory studies. • Educate the patient and family regarding clinical parameters and patient presentation required for safe discharge from unit/hospital.

and fibrinolytic systems and can be given to normalize the INR. The recommended dose is 10 to 20 mL/kg. Platelet transfusions are usually used only for patients with active bleeding or a platelet count of less than 20,000/mm^3. Cryoprecipitate may be used for patients with plasma fibrinogen levels below 100 mg/dL. A single unit provides 200 mg of fibrinogen, as well as factor VIII, factor XII, and von Willebrand's factor. The usual adult dose is 5 to 10 units, with each unit raising the fibrinogen level by 5 to 10 mg/dL. Depleted antithrombin III (necessary to balance clot production) can be replaced using heat-treated pooled plasma concentrates; this has been shown to shorten the duration of DIC. RBC transfusions, although not useful for repleting coagulation factors, may be given to increase hemoglobin and oxygen-carrying capacity.

Localized bleeding can be minimized when possible. With venipuncture or removal of vascular access from compressible sites, pressure is applied for a minimum of 15 to 30 minutes or until bleeding has stopped. Sites are reassessed frequently for rebleeding because the initial clot may dissolve if the patient lacks the factors required to maintain hemostasis. Topical hemostatics may be used to provide superficial hemostasis.

▲ Clinical Applicability Challenges

CASE STUDY

Mrs. K. is a 63-year-old African American female with a known history of chronic kidney disease. She was hospitalized 6 weeks ago and had a left arm arteriovenous (AV) fistula placed in anticipation of eventual hemodialysis. The morning of this admission, she presents to her primary care physician's office complaining of worsening shortness of breath, dizziness, and generalized weakness. Mrs. K. is directly admitted to a telemetry unit on the hospitalist service. Physical examination findings include a markedly elevated blood pressure 222/118, generalized edema, but clear breath sounds. Initial laboratory work includes an elevated blood urea nitrogen, 141 mg/dL and creatinine, 15.9 mg/dL; hemoglobin, 7.1 mg/dL and platelet count, 306,000/mm^3. Admission orders include increasing her antihypertensive agents, transfusion of 2 units of packed red blood cells (PRBCs), a nephrology consult, and unfractionated heparin (UFH) 5,000 units subcutaneously every 8 hours for deep vein thrombosis (DVT) prophylaxis. A ventilation/perfusion scan is also performed with the finding of low probability for pulmonary embolism.

The nephrologist assesses that Mrs. K. has progressed to end-stage renal disease and recommends initiation of hemodialysis. Because Mrs. K's AV fistula is not mature, a Perma-Cath is placed. In addition to PRBC transfusion, the nephrologist recommends intravenous iron and erythropoietic-stimulating protein administration (eg, epogen, darbepoetin).

Mrs. K. gradually responds to treatment and on hospital day 12 is preparing for discharge. On rounds, the nurse practitioner notes that Mrs. K's last platelet count on hospital day 9 had dropped to 137,000/mm^3, and a complete blood count is ordered to be drawn by the dialysis nurse before the day's hemodialysis treatment. The dialysis nurse immediately pages the nurse practitioner noting that the platelet count is 13,000/mm^3. Heparin-induced thrombocytopenia (HIT) is strongly suspected. All heparin as well as any platelet-affecting medications are stopped, blood is drawn for a heparin-induced antibody (HITA) screen, and a hematology consult is requested. A nonheparin anticoagulant (see Box 49-6) is started. Even though Mrs. K. has no lower extremity edema or discomfort, lower extremity venous ultrasonography is performed, and a right common femoral vein thrombus is detected.

The following morning, laboratory results include a positive HITA and a platelet count of 36,000/mm^3. On hospital day 19, Mrs. K.'s platelet count is 102,000/mm^3. She is started on warfarin 5 mg each evening. On day 5 of warfarin therapy, her international normalized ratio (INR) is 1.36, and platelet count is 112,000/mm^3. Mrs. K. has been hospitalized a total of 23 days and is anxious to go home. She is taught how to self-administer the nonheparin anticoagulant injection.

Mrs. K. is discharged home and instructed to take warfarin 10 mg daily. She will have her INR checked with each dialysis treatment until her INR is stable between 2 and 3 at which time the nonheparin anticoagulant can be stopped. The nurse instructs Mrs. K. and her daughter to inform health care providers that she is not to be given UFH or low-molecular-weight heparin because of this episode of HIT. The nurse suggests Mrs. K. wear a medical identification bracelet with this information.

1. The nurse is concerned about Mrs. K.'s platelet count of 137,000/mm^3. Why is the platelet count important?

2. Mrs. K. is treated for HIT before the diagnosis is confirmed by laboratory testing. Why?

3. Despite Mrs. K. having no clinical evidence of DVT, bilateral lower extremity venous Doppler studies are ordered. What is the rationale?

References

1. Napolitano LM, Kurek S, Luchette FA, et al: Clinical practice guideline: Red blood cell transfusion in adult and critical care. Crit Care Med 37(12):3124–3157, 2009
2. Gaspard KJ: Alterations in homeostasis. In Porth CM (ed): Essentials of Pathophysiology. Philadelphia, PA: Lippincott Williams & Wilkins, 2007
3. Warkentin TE, Greinacher A: Heparin-induced thrombocytopenia: recognition, treatment, and prevention: the Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. Chest 126(3 Suppl): 311S–337S, 2004
4. Ziegfeld C, Shelton BK, Olsen M: Manual of Oncology Nursing. Philadelphia, PA: Lippincott Williams & Wilkins, 2005

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

PART 12

INTEGUMENTARY SYSTEM

50

Anatomy and Physiology of the Integumentary System

Joan M. Davenport

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Describe the features of the epidermis, dermis, and hypodermis.
2. Describe the appendages of the skin and state the purpose of each.
3. Discuss the homeostatic functions of the skin.
4. Explain the mechanism of infection resistance afforded by the integument.

The skin is described as protective, sensitive, reparative, and capable of maintaining a person's homeostasis. These physiological features are explored in this chapter as functions of the anatomy of the skin and its appendages. The skin, which covers 1.2 to 2.3 m^2 of area, is the largest organ of the body and is supplied with one third of the circulating blood volume.[1] The three layers of the skin are the outer epidermis, the middle dermis, and the underlying hypodermis, or subcutaneous tissue. The appendages include the hair, nail, eccrine and apocrine sweat glands, and sebaceous glands. Figure 50-1 pictures the structures and layers of the skin. The functions of the skin include protection, sensation, water balance, temperature regulation, and vitamin production.

▲ Epidermis

This outer layer of the skin serves to protect underlying structures from invasion by microbes and other foreign substances. The cornified, external layer of the epidermis helps in the body's regulation of water loss. The innermost sublayer bends into the dermis and serves as the basis for the glands, nails, and hair roots. The epidermis does not have vascular supply; it depends on the dermal level for its nourishment.

Melanin and keratin are formed in the inner cellular layer of the epidermis. Melanocytes provide melanin, a pigment for both the skin and hair. This pigment provides the color for the skin and, more important, protects the underlying structures from ultraviolet light exposure by absorbing and scattering the radiation.[2]

Keratin is a tough protein that makes up hair, nails, and the tough, outer epidermal surface. These flattened scales of the skin slough continually and are replaced every 2 to 4 weeks.[2] The epidermis is actually made up of five distinct layers; the keratinocytes move from inner to outer sublayers as they mature. At the top, outermost layer, the keratinocytes are dead and are arranged in various thicknesses depending on the area of the body. In parts of the face, there is a thin stratum made up of a layer 15 cells deep. This contrasts with the thicker soles of the feet and palms of the hands, with at least 100 layers of keratinized cells.[2] It is these tough protein cells that serve to protect the underlying structures of the body.

▲ Dermis

The middle layer of the skin, the dermis, provides support for the outer epidermal layer. It is a very vascular connective tissue, and the blood vessels are integral to regulation of body temperature and blood pressure. The arteriovenous anastomoses, under control of the sympathetic nervous system and found in the dermal layer, are able to dilate or constrict in

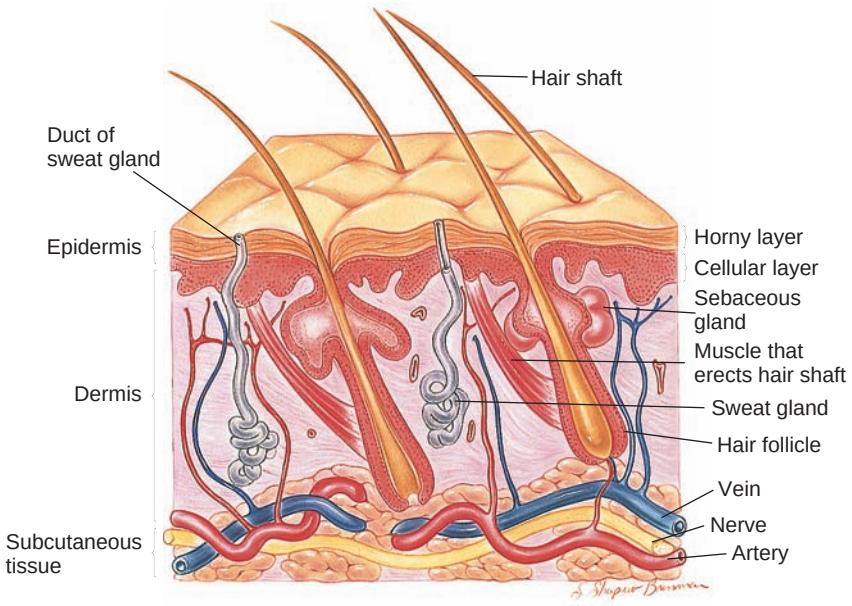


FIGURE 50-1 ▲ Layers of the skin. (From Bickley LS: Bates Guide to Physical Examination and history Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 163.)

response to environmental conditions of heat and cold and to internal stimulation from anxiety or blood volume loss.

The sensory function of the skin includes receptors for heat, cold, touch, pressure, and pain; these are located in the dermal layer. There is a great variety in the function of the nerve endings. Multiple stimuli are mediated centrally and result in patterned responses.[2]

The dermis is composed of two distinct layers. The papillary dermis is the more superficial of the two layers, lying just beneath the epidermis. This layer provides the attachment for the epidermis as the epidermal basal cells project into the papillary dermis.[3]

The thicker underlayer of the dermis is the reticular dermis. Collagen is organized in a three-dimensional mesh pattern in this portion of the dermis. It is this mesh arrangement that allows the dermis to stretch with movement. Immune system components of the skin are found in the dermal layer and include macrophages, mast cells, T cells, and fibroblasts.[2]

▲ Hypodermis

The hypodermis or subcutaneous skin layer consists of connective tissue interspersed with fat. The fat of the hypodermis has the protective functions of heat retention and cushioning the underlying structures. In addition, the fat of the subcutaneous skin layer serves as storage for calories.[2]

▲ Skin Appendages

The hair, nails, and sebaceous and sweat glands are considered a part of the skin. These structures arise from or extrude through the epidermal or dermal skin layers.

Sweat Glands

Eccrine sweat glands are distributed throughout the surface of the skin. These glands arise from the dermis and open at the skin surface. These specialized glands secrete sweat for the purpose of internal body temperature regulation.

Apocrine sweat glands are not as widespread as the eccrine glands, are larger than eccrine glands, and open through a hair follicle of the axillae, nipples, areolae, groin, eyelids, and external ears.[2] Another difference between the two types of sweat glands is that the larger, less abundant apocrine glands secrete an oily substance with a particular odor. This odor is used by animals to recognize the presence of other animals. In humans, the odor, known as body odor, is produced when the secretions come in contact with bacteria and when the fluid begins to decompose.[2]

Sebaceous Glands

The sebaceous glands secrete sebum, a combination of triglycerides, cholesterol, and wax, through the hair follicle. These glands are situated over the entire surface of the skin except for the palms and soles of the feet. Sebaceous glands are inactive until puberty. At this time, they enlarge and are stimulated to secrete sebum by a rise in sex hormones. The sebum serves to keep the skin and hair from drying out.[2] By protecting the outer layer of the epidermis from undue drying, the sebum helps to conserve body heat. Table 50-1 summarizes the location and function of the eccrine, apocrine, and sebaceous glands.[4]

Hair

Epidermal cells in the dermis form the hair. Together with the sebaceous gland, the hair follicle forms the pilosebaceous unit. Vellus hair is unobtrusive, soft, and less pigmented than terminal hair. Terminal hair is darker, coarser, and more obvious. The follicular bulb is the site of vascular papilla, which nourishes and maintains the hair follicle. Hair color is determined by the melanocytes also found at the bulb. Under the sebaceous gland, adjacent to the hair follicle, are the arrector pili muscles. Contraction of the arrector pili causes gooseflesh, a

Table 50-1 Sweat and Sebaceous Glands

Type of Gland	Location	Function
Eccrine sweat glands	All over body	Regulate body temperature
	Numerous in thick skin	Respond to emotional distress
	Extended from dermis to epidermis	Respond to physiological stimuli
Apocrine sweat glands	Axillae	Respond to hormonal influences
	Nipples, breasts	Respond to emotional distress
	Anogenital region	
	External ear canal	
	Eyelids	
Sebaceous glands	All over body except palms of hands, dorsum, and soles of feet	Produce sebum to lubricate hair and skin

From Allwood J, Curry K: Normal and altered functions of the skin. In Bullock BA, Henze RL (eds): Focus on Pathophysiology. Philadelphia, PA: Lippincott Williams & Wilkins, 2000, p 842.

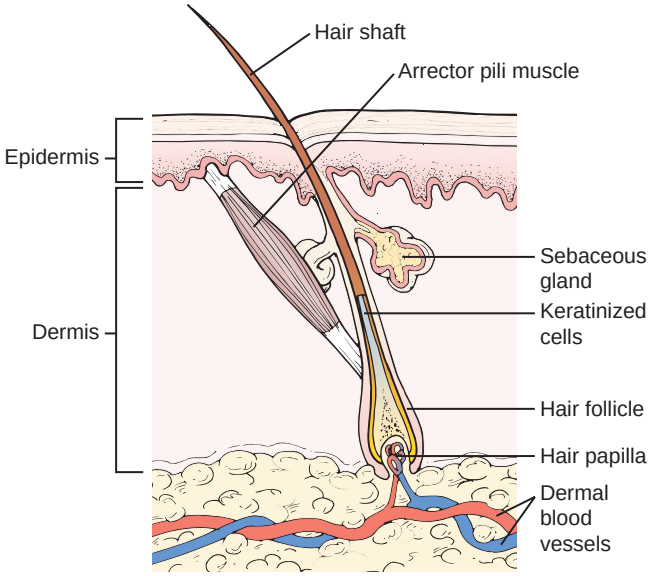


FIGURE 50-2 ▲ Structures of hair. (From Porth MP, Matfin G (eds): Pathophysiology, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 1551.)

reduction of skin surface area, and reduced surface area for heat loss (Fig. 50-2).

Nails

Hardened plates of epidermal keratin cells grow from a curved groove over the distal dorsal fingertips. These nails serve to protect the fingers and toes and increase physical dexterity. Approximately one fourth of the nail is covered by the proximal nail fold; the cuticle extends from the fold and serves to waterproof the space between the plate and the fold. The lunula is the white, "half-moon"–shaped edge distal to the cuticle. The angle between the proximal nail fold and the nail plate is expected to be less than 180 degrees (Fig. 50-3).

▲ Functions of the Skin

The epidermal layer of the integument provides protection against microbes, ultraviolet light exposure, and countless other threats. This tough, hardened outer layer also restricts water loss and thus helps to maintain organism homeostasis. The vascular dermis, with its rich supply of blood vessels, provides blood pressure and temperature-regulatory features. Immune functions performed by macrophages, mast cells, T cells, and fibroblasts are also housed in the dermis. Nerve endings here supply receptors for heat, cold, touch, pressure, and pain. Within this vascular dermal area, a potential space exists. This space, with its extracellular and extravascular fluid, may serve as a fluid reservoir to replace intravascular or intracellular fluid loss. This is also the place for fluid to migrate toward when intravascular hydrostatic pressure increases above the hydrostatic pressure with the dermal layer. This process produces the edema seen in patients. The skin's ability to stretch with movement is also provided by the dermal collagen's mesh formation. The underlying hypodermal layers of connective tissue and fat serve to retain heat and to cushion the underlying structures. The appendages of the skin, the hair, nails, and glands, contribute to the homeostatic function primarily by controlling heat loss with the hair's arrector pili muscles and secretions by the sebaceous and sweat glands. The integument is vital to an individual's survival.

During critical care hospitalizations, there are many insults to the skin. Surgical wounds, vascular catheter insertions,

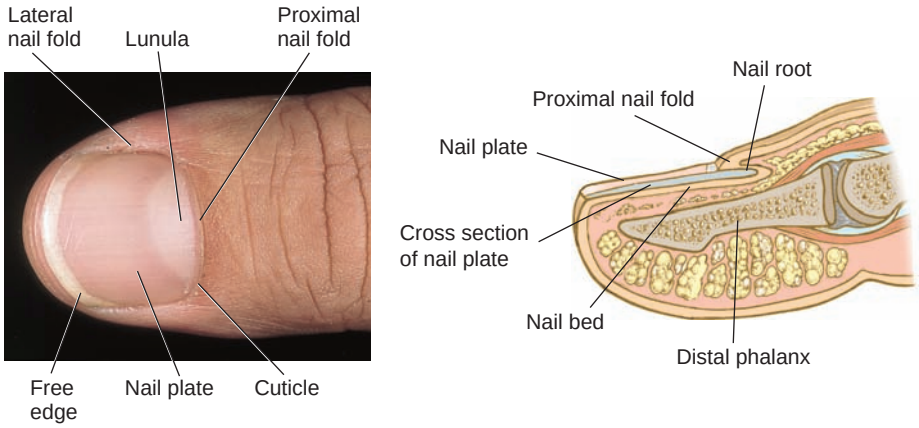


FIGURE 50-3 ▲ The normal nail. (From Bickley LS: Bates Guide to Physical Examination and history Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 164.)

BOX 50-1 Anatomical and Physiological Changes in the Integumentary System That Occur With Aging

- The skin becomes thinner, and there is a decrease in skin flexibility, placing the person at risk for epidermal tearing.
- The skin loses dermal elasticity, collagen, and mass, resulting in fine wrinkling, looseness, and sagging.
- The number of dermal blood vessels decreases; the vessels become thinner and more fragile, thus increasing the risk for bruising and hemorrhage.
- There is decreased density and activity of the eccrine and apocrine glands and decreased sebum production, resulting in dryness, itching, and decreased perspiration.
- Decreased peripheral circulation leads to slowed nail growth and brittle nails that split easily.
- Reduced hormone levels lead to thinning of the hair and transition from terminal to vellus hair.
- Decreased melanin leads to graying of the hair.
- Sun exposure over a long period of time leads to yellowing and thickening of the skin and the development of old age spots (solar lentigo).

opportunistic infections of the skin, nutritional compromise, and persistent pressure that leads to reduced blood flow are only a few of the challenges faced by the patient's integument. In older patients, age-related changes (Box 50-1; see also Chapters 12 and 51) leading to increased fragility of the skin and slower healing magnify the effects of these insults. Attention to the skin and appendages by the nurse maximizes this organ's functioning and results in protection of the patient.

▲ Clinical Applicability Challenges

SHORT ANSWER QUESTIONS

1. What does the integument system do to maintain physiological homeostasis?
2. What are the physiological changes that occur to cause the pitting edema seen often in the critically ill adult patient?
3. In what ways are the expected physiological changes of the skin of the older adult exacerbated or worsened during critical illness?

References

1. Bickley LS, Szilagyi PG: The skin, hair, and nails. In Bickley LS, Szilagyi PG (eds): Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009
2. Simandl G: Structure and function of the skin. In Porth CM (ed): Pathophysiology: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009
3. Jarvis C: Skin, Hair, and Nails. In Jarvis C (ed): Physical Examination and Health Assessment, 5th ed. St. Louis MO: Saunders, 2008
4. Cuzzell J, Workman ML: Assessment of the skin, hair, and nails. In Ignatavicius D, Workman ML (eds): Medical-Surgical Nursing: Patient–Centered Collaborative Care, 6th ed. St. Louis, MO: Saunders, 2010

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

51 Patient Assessment: Integumentary System

Joan M. Davenport and Janet A. Wulf

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Discuss the health history and physical assessment when evaluating a patient's skin.
2. Explain expected differences in skin color related to racial or skin tone characteristics.
3. Describe and recognize abnormal changes in skin color.
4. Explain and identify skin lesions resulting from increased vascularity.
5. Describe the significance of rashes related to infection or to allergic reaction.
6. Compare and contrast pitting and nonpitting edema.
7. Explain the cause of pressure ulcers and the Braden scale used to assess a patient for pressure ulcer development.
8. Discuss the features of malignant skin diseases.

The skin of a critically ill person is exposed to insults ranging from diminished blood flow and the resultant risk for pressure ulceration to rashes from hypersensitivity drug reactions and opportunistic infections. There is often ample opportunity for the critical care nurse to assess the skin—the intimacy involved in providing care to someone who is critically ill, the relative level of undress of the patient, and the attention to detail implicit in critical care nursing make integument assessment an ongoing and vital process.

▲ History

When caring for patients with skin disorders, it is important to obtain information from the health history (Box 51-1). The information is useful in guiding the physical examination and in determining appropriate interventions.

▲ Physical Examination

The assessment techniques necessary for an evaluation of the integument involve inspection and palpation.

Inspection

Inspection of the general appearance of the skin includes assessment of color; determination of the presence of lesions, rashes, or increased vascularity; and assessment of the condition of the nails and hair.

Color

Skin color is expected to be uniform over the body, except for the areas with greater degrees of vascularity. The genitalia, upper chest, and cheeks may appear pink or have a reddish tone in people with light skin. These same areas may appear darker in people with dark skin. Additional normal variations in skin color include those listed in Table 51-1.

Skin color is determined by the presence of four pigments: melanin, carotene, hemoglobin, and deoxyhemoglobin. The amount of melanin is genetically determined and produces varying degrees of dark skin tone. Carotene, a yellow pigment, is in subcutaneous fat and is most evident in those areas with the most keratin, the palms and soles of the feet. Skin color abnormalities, such as pallor, cyanosis, jaundice, and erythema, manifest differently depending on the person's normal skin tone (Table 51-2).

The degree of oxygenation affects skin color. Hemoglobin, attached to red blood cells, transports oxygen to the tissues. A diminished flow of oxyhemoglobin through the cutaneous circulation results in pallor. In people with light skin, the skin appears very pale, without the usual pink undertones. In people with darker skin, pallor manifests as a yellowish-brown or ashen appearance (again, because the usual pink undertones are lost).

As hemoglobin gives up its oxygen to the tissues, the hemoglobin changes to deoxyhemoglobin. When deoxyhemoglobin is present in the cutaneous circulation, the skin

BOX 51-1 HEALTH HISTORY for Integumentary Assessment

Chief Complaint
- Patient's description of the problem

History of the Present Illness
- Complete analysis of the following signs and symptoms (using the NOPQRST format; see Box 17-1, p. 207)
- Changes in skin color, pigmentation, temperature, or texture
- Changes in a mole
- Excess dryness or moisture
- Skin itching
- Excess bruising
- Delay in healing
- Skin rash or lesions
- Hair loss or increased growth
- Changes in hair texture
- Changes in nails

Past Health History
- Relevant childhood illnesses and immunizations: impetigo, scabies or lice exposure, measles, chickenpox, scarlet fever
- Past acute and chronic medical problems including treatments and hospitalizations: diabetes, peripheral vascular disease, Lyme disease, Parkinson's disease, immobility, malnutrition, trauma, skin cancers, radiation treatments, HIV/AIDS
- Risk factors: age, ultraviolet sun exposure, tanning beds, exposure to dyes, toxic chemicals, insect bites, contact with some poisonous plants, autoimmune disease, exposure to extremes of temperature
- Past surgeries: skin biopsy
- Past diagnostic tests and interventions: allergy testing
- Medications: aspirin, antibiotics, barbiturates, sulfonamides, thiazide diuretics, oral hypoglycemic agents, tetracycline, antimalarials, antineoplastic agents, hormones, metals, topical steroids
- Allergies and reactions: foods, medications, contrast dyes, latex, soaps
- Transfusions

Family History
- Health status or cause of death of parents and siblings: skin cancer, autoimmune diseases

Personal and Social History
- Tobacco, alcohol, and substance use
- Family composition
- Occupation and work environment: farmers, roofers, creosote or coal workers, furniture repair and refinishing, gardeners
- Living environment: ability for self-care and hygiene, exposure to insects and pests, availability of indoor sleeping in environmental temperature extremes
- Diet
- Sleep patterns
- Exercise
- Cultural beliefs
- Spiritual/religious beliefs
- Coping patterns and social support systems
- Leisure activities
- Sexual activity
- Recent travel

Review of Systems
- Psychiatric/emotional: increased anxiety, nervousness, sleeplessness
- Neurologic: loss or decrease in sensation, numbness, pain or neuropathy, stroke
- Cardiovascular: swelling of extremities, cold extremities, varicose veins
- Gastrointestinal: change in diet, recent weight loss or gain, loss of appetite
- Musculoskeletal: immobility, weakness
- Metabolic: altered glucose levels

takes on a blue cast, and the person is said to be cyanotic.[1] In light-skinned people, cyanosis may be seen as a grayish-blue color, especially in the palms and soles of the feet, the nail beds, the earlobes, the lips, and the mucous membranes. In those with darker skin, cyanosis appears as an ashen-gray color seen in the same areas.[2]

The yellowish hue of jaundice is indicative of liver disease or of hemolysis of red blood cells. In dark-skinned people, jaundice is seen as a yellowish-green color in the sclera, palms of the hands, and soles of the feet. In light-skinned people, jaundice is seen as a yellow coloration of the skin, sclera, lips, hard palate, and underside of the tongue. Bickley and Szilagyi[1] recommend using a transparent slide pressed against the lips to "blanch out the red color," making the yellow of jaundice more easily seen.

Table 51-1 Normal Variations in Skin Color

Normal Variation	Description
Moles (pigmented nevi)	Tan to dark brown; may be flat or raised
Stretch mark (striae)	Silver or pink; may be caused by weight gain or pregnancy
Freckles	Flat macules anywhere on the body
Vitiligo	Unpigmented skin area; more prevalent in people with dark skin
Birthmarks	Generally flat marks anywhere on the body; may be tan, red, or brown

Another skin color abnormality is erythema. Erythema manifests as a reddish tone in light-skinned people and a deeper brown or purple tone in dark-skinned people. It is indicative of increased skin temperature caused by inflammation. The process of inflammation increases vascularity of the tissues, and this, in turn, produces the color alteration seen with erythema. Erythema may be expected when associated with a surgical wound, due to the inflammatory process inherent in any tissue trauma. It is also seen in disease processes affecting the skin, such as cellulitis. In either case, the erythema is indicative of inflammation.

Lesions

Skin lesions are variously described by their color, shape, cause, or general appearance (Table 51-3, p. 1157; Table 51-4, p. 1160). They are considered abnormal conditions and arise from many factors (see Spotlight on Genetics 51-1, p. 1160). In general, it is important to note the anatomical location, distribution, color, size, and pattern of any abnormal skin lesion (Fig. 51-1). In addition, details about the lesion's borders or edges, as

Table 51-2 Skin Color Abnormalities

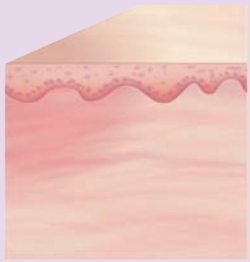
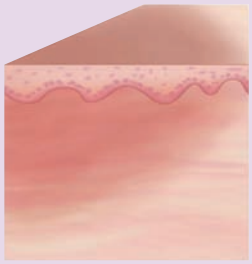
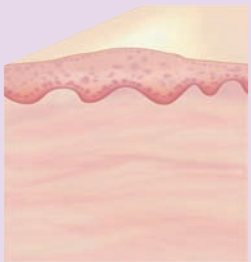
Skin Color Abnormality	Underlying Cause	Manifestation in Light-Skinned People	Manifestation in Dark-Skinned People
Pallor	Decreased blood flow (decreased oxyhemoglobin flow to tissues)	Excessively pale skin	Yellowish-brown or ashen color to the skin
Cyanosis	Increased deoxyhemoglobin in the cutaneous circulation	Grayish-blue color of the palms and soles of the feet, the nail beds, the lips, the earlobes, and the mucous membranes	Ashen-gray color of the conjunctiva, oral mucous membranes, and nail beds
Jaundice	Increased red blood cell hemolysis, liver disease	Yellow color of the sclera, lips, and hard palate	Yellow-green color of the sclera and palms and soles of the feet
Erythema	Inflammation	Reddish tone	Deeper brown or purple tone

well as whether the lesion is flat, raised, or sunken, should be noted. Finally, the length of time the lesion has been present and any environmental or medication exposure that may be considered contributory should also be noted.[3]

Vascular lesions can be either a normal variation or an abnormal finding. Vascular changes considered to be normal variants include nevus flammeus (port-wine stain), immature hemangioma (strawberry mark), telangiectasis, cherry angioma, and capillary hemangioma (Table 51-5, p. 1161). Abnormal vascular findings include petechiae, purpura, ecchymoses, spider angiomas, and urticaria (hives). These findings may indicate disease or injury and warrant further investigation by the critical care nurse.

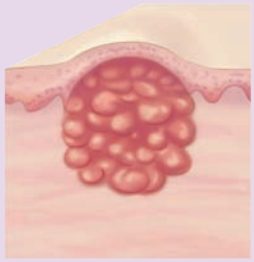
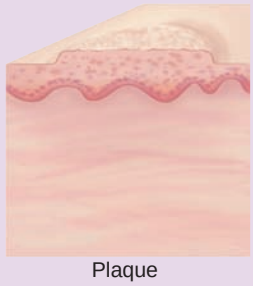
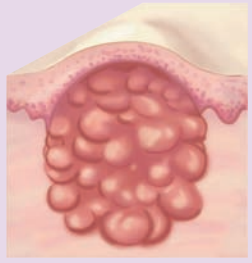
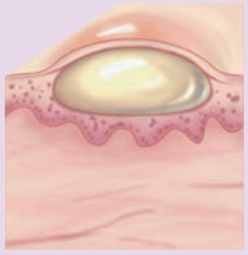
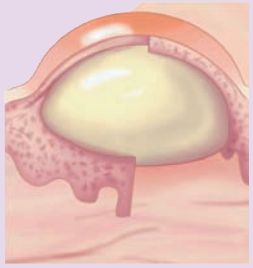
Petechiae are purple or red, small (1- to 3-mm) lesions easily seen on light-skinned people and more difficult to see on those with dark skin (Fig. 51-2A, p. 1161). These tiny

Table 51-3 Primary Skin Lesions

Type	Description	Examples	Illustration
Macule	<1 cm in diameter, flat, nonpalpable, circumscribed, discolored	Brown: freckle, junctional nevus, lentigo, melasma Blue: Mongolian spot, ochronosis Red: drug eruption, viral exanthema, secondary syphilis Hypopigmented: vitiligo, idiopathic guttate hypomelanosis	 Macule
Patch	>1 cm in diameter, flat, nonpalpable, irregular shape, discolored	Brown: larger freckle, junctional nevus, lentigo, melasma Blue: Mongolian spot, ochronosis Red: drug eruption viral exanthema, secondary syphilis Hypopigmented: vitiligo, idiopathic guttate hypomelanosis	 Patch
Papule	<1 cm in diameter, raised, palpable, firm	Flesh, white or yellow: flat wart, milium, sebaceous hyperplasia, skin tag Blue or violaceous: venous lake, lichen planus, melanoma Brown: seborrheic keratosis, melanoma, dermatofibroma, nevi Red: acne, cherry angioma, early folliculitis, psoriasis, urticaria, and eczema	 Papule

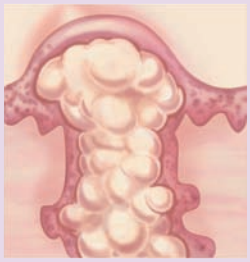
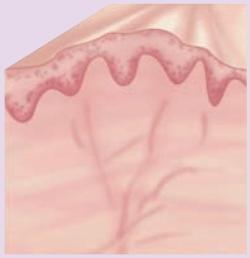
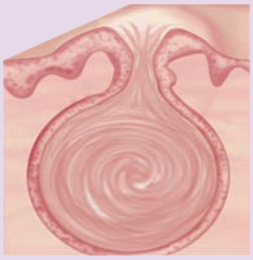
(continued on page 1158)

Table 51-3 Primary Skin Lesions (continued)

Type	Description	Examples	Illustration
Nodule	>1 cm, raised, solid	Wart, xanthoma, prurigo nodularis, neurofibromatosis	 Nodule
Plaque	>1 cm, raised, superficial, flat-topped, rough	Psoriasis, discoid lupus, tinea corporis, eczema, seborrheic dermatitis	 Plaque
Tumor	Large nodule	Metastatic carcinoma, sporotrichosis	 Tumor
Vesicle	<1 cm, superficially raised, filled with serous fluid	Herpes simplex, herpes zoster, erythema multiforme, impetigo	 Vesicle
Bulla	>1-cm vesicle	Pemphigus, herpes gestationis, fixed drug eruption	 Bulla

(continued on page 1159)

Table 51-3 Primary Skin Lesions (continued)

Type	Description	Examples	Illustration
Pustule	Raised, superficial, filled with cloudy, purulent fluid	Acne, candidiasis, rosacea, impetigo, folliculitis	 Pustule
Wheal	Raised, irregular area of edema, solid, transient, variable size	Hives, cholinergic urticaria, angioedema, dermatographism	 Wheal
Cyst	Raised, circumscribed, encapsulated with a wall and lumen, filled with liquid or semisolid	Digital mucus, epidermal inclusion, pilar	 Cyst

From Rhoads J: Advanced Health Assessment and Diagnostic Reasoning. Philadelphia, PA: Lippincott Williams & Wilkins, 2006, pp 81–83.

hemorrhages in the dermal or submucosal layers may be seen anywhere on the body including oral mucosa and conjunctiva. They are caused by extravasated blood and do not disappear when pressure is applied to them.[3] Purpura are very similar to petechiae, only larger. Purpura may appear brownish-red.

Ecchymoses are bruises. They may appear as purple to yellowish-green rounded or irregular lesions and are more easily seen in people with light skin (see Fig. 51-2B). Ecchymoses occur as a result of trauma, when blood leaks from damaged blood vessels into the surrounding tissue.

Spider angiomas are fiery red lesions that are most often located on the face, neck, arms, or upper trunk (see Fig. 51-2C). Spider angiomas are seldom seen below the waist. They have a central body that is sometimes "raised and surrounded by erythema and radiating legs."[1] These lesions are most often associated with liver disease and vitamin B deficiency but also occur normally in some people.[1]

Urticaria is a reddened or white, raised, nonpitting plaque that often occurs as a result of an allergic reaction. The lesion often changes shape and size during the course of the reaction. The edema associated with urticaria is a result of local vasodilation and inflammation, which is followed by transudation of serous vascular fluid into the surrounding tissue.

Rashes

Rashes identified during inspection may indicate infection or a reaction to drug therapy. Some of these rashes are identified by the names listed in Table 51-3. Identifying the type of lesion may help in identifying the cause of the rash. Attention to the development of a rash in association with a change in pharmacotherapy is essential to help identify the occurrence of an allergic hypersensitivity reaction.[4] The development of urticaria is often associated with food or drug reactions. Urticaria usually resolves completely over days to several weeks as the excess local fluid is reabsorbed. These lesions are often pruritic, and patient scratching may precipitate secondary skin abrasions, which can place the patient at risk for localized skin infections.

Skin infections are most often caused by fungi or yeasts and may range from superficial tinea pedis (athlete's foot)

Table 51-4 Secondary Skin Lesions

Type	Description
Crust	Dried exudates over a damaged epithelium; may be associated with vesicles, bullae, or pustules. Large adherent crust is a scab.
Erosion	Loss of superficial epidermis; does not extend to the dermis; may be associated with vesicles, bullae, or pustules.
Fissure	Crack in the epidermis usually extending into the dermis.
Keloid	Hypertrophied scar tissue; secondary to collagen formation during healing; elevated irregular and red; more common in African Americans.
Lichenification	Thickening and roughening of the skin; accentuated skin markings; may be secondary to repeated rubbing irritation and scratching.
Scale	Skin debris on the surface of the epidermis secondary to desquamated, dead epithelium. Color and texture vary.
Scar	Skin mark left after healing of wound or lesion that represents replacement by connective tissue of the injured tissue. Young scars are red or purple. Mature scars are white or glistening.
Ulceration	Loss of epidermis, extending into dermis or deeper. Bleeding and scarring are possible.

Adapted from Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 199.

to intermediate yeast infections (eg, moniliasis resulting from *Candida albicans* infection) to deep fungal infections (eg, aspergillosis) that invade the underlying tissues. Most often in the critical care setting, fungal and yeast infections are of the intermediate type and are the result of an opportunistic infection by normal flora. Antibiotics, corticosteroids, poor nutrition, and diabetes mellitus place the patient at risk for these infections. Candidiasis presents in the groin and under the breasts of female patients with erythema, a whitish pseudomembrane, and macropapular satellite lesions.[5] Oral candidiasis, also known as thrush, manifests as a whitish coating of the oral mucosa, especially the tongue. This painful condition may produce fissures on the tongue and often restricts a patient's oral intake, further compromising the patient from a nutritional perspective.

SPOTLIGHT ON GENETICS 51-1

Focal Dermal Hypoplasia

- Focal dermal hypoplasia is characterized by skin abnormalities present from birth, such as streaks of very thin skin with 90% of affected individuals being female.
- Mutations in the *PORCN* gene cause focal dermal hypoplasia. This gene provides instructions for making a protein that is responsible for modifying other proteins.
- Mutations in the *PORCN* gene appear to prevent the production of any functional PORCN protein. Researchers believe Wnt proteins cannot be released from the cell without the PORCN protein. When Wnt proteins are unable to leave the cell, they cannot participate in the chemical signaling pathways that are critical for normal development
- Genetic testing via sequence analysis of the entire coding region is available for the diagnosis of focal dermal hypoplasia.

Genetic Home Reference-http://ghr.nlm.nih.gov, accessed July 14, 2011.

Garavelli L, Wischmeijer A, Rosato S, et al: Focal dermal hypoplasia (Goltz syndrome): A new case with a novel mutation in the PORCN gene and unusual spinal anomaly. Genetic Counseling 21(1):126–128, 2010.

Condition of the Hair

The patient's terminal hair is inspected daily, noting the hair's quantity, distribution, and texture. Scalp hair should be resilient and evenly distributed.

Alopecia refers to hair loss and can be diffuse, patchy, or complete. Hair loss in the critical care setting can be associated with pharmacotherapy. Chemotherapy used in oncology treatment produces alopecia. Other drugs, such as heparin, used for a prolonged time may also be responsible for hair loss.[6,7] Hirsutism or increased facial, body, or pubic hair growth is an abnormal finding in the examination of women and children. Hirsutism has a familial pattern and is associated with menopause, endocrine disorders, and certain pharmacotherapies (eg, corticosteroids and androgenic medications).[2]

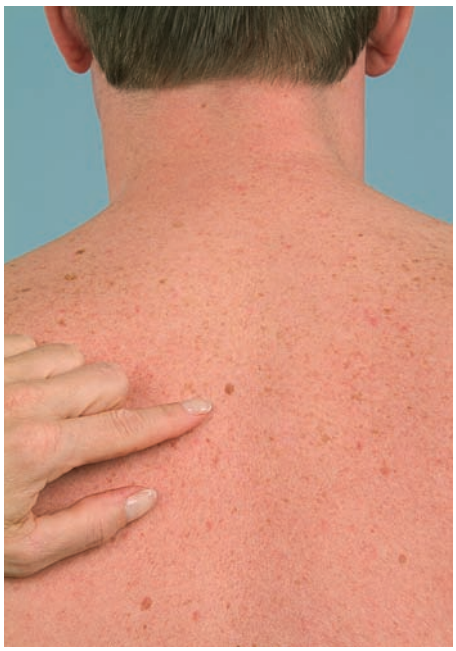


FIGURE 51-1 ▲ Inspecting for lesions. (From Rhoads J: Advanced Health Assessment and Diagnostic Reasoning. Philadelphia, PA: Lippincott Williams & Wilkins, 2006, p 76.)

Table 51-5 Vascular Lesions: Normal Variations

Normal Variation	Description
Nevus flammeus (port-wine stain), immature hemangioma (strawberry mark)	Range from dark red to pale pink and are considered birthmarks
Cherry angioma	Small, slightly raised, bright red lesions on the face, neck, and trunk; increase in size and number with advancing age
Capillary hemangioma	Red, irregular patch caused by capillary dilation in the dermis of the skin
Telangiectasis	Irregular, fine red lines caused by permanent dilation of a group of superficial vessels

A change in the hair's texture may indicate ongoing health concerns. Hair that is thin and brittle occurs in hypothyroidism. In those with severe protein malnutrition, the hair color may appear reddish or bleached, and the hair texture is described as brittle and dry.[7]

Also not to be overlooked is the presence of infection or infestation of the scalp and hair. The patient's scalp and body hair are inspected regularly for evidence of flaking, sores, lice, louse eggs, scabies, and ringworm. During the inspection, the hair is parted in several areas to reveal the underlying scalp (Fig. 51-3).

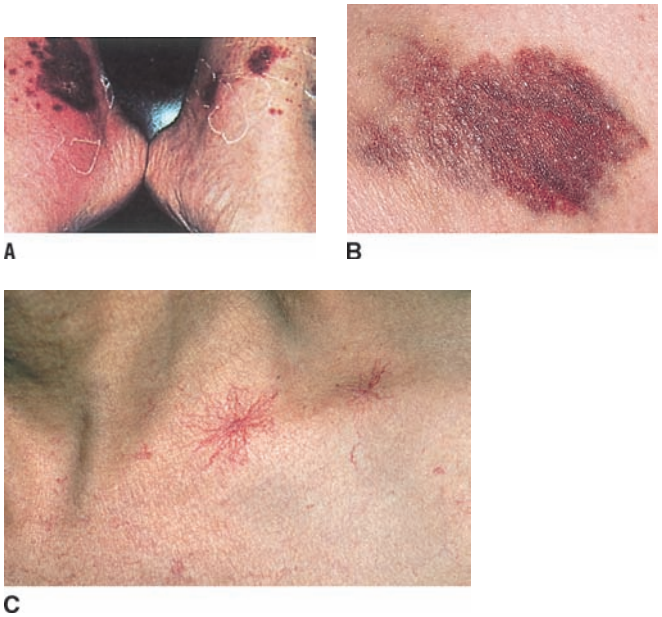


FIGURE 51-2 ▲ A–C: Abnormal vascular lesions. (**A**, from Smeltzer SC, Bare BG. Textbook of Medical-Surgical Nursing, 9th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2000. **B**, from Bickley LS: Bates' Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 184. **C**, from Goodheart HP. Goodheart's Photoguide of Common Skin Disorders, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2003.)

FIGURE 51-3 ▲ Inspecting the scalp and hair. (From Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 191.)

Condition of the Nails

Nails, like hair, can be overlooked in the rush of critical care nursing; however, a careful inspection as part of the routine assessment can reveal information about the patient's general state of health. The nail bed is very vascular and is an excellent location for assessing the adequacy of the patient's peripheral circulation. The capillary refill test, done by blanching the nail beds and then releasing the pressure, should indicate a return of the pink tones in less than 3 seconds. Nail beds that are tinted bluish or purplish may indicate cyanosis; nail beds that are pale may indicate reduced arterial blood flow.

When the angle of the nail is 180 degrees or greater, clubbing is said to be present (see Chapter 24, Fig. 24-2). Clubbing is attributed to chronic hypoxemia. Other shapes that the nail takes on may provide clues to deficient nutritional states of the patient (Fig. 51-4). A spoon-shaped nail, called koilonychias, is associated with iron deficiency anemia.

Chronic disease states, such as cirrhosis, heart failure, and type II diabetes mellitus, may affect the nails by producing Terry's nails.[1] These nails are whitish with a distal band of dark reddish-brown color, and the lunulae may not be visible (Fig. 51-5). Bands across the nails, especially in the older adult, may indicate protein deficiency. However, in dark-skinned individuals, lengthwise, linear, dark stripes or diffuse brown, blue, or black pigmentation may be a normal finding.[5] Hyperkeratotic, dull, discolored, and distorted nails may be onychomycosis, a fungal infection of the nails seen frequently in critically ill patients. It is more common in toenails than fingernails. Risk factors include diabetes, poor venous and lymphatic drainage, poorly fitting shoes, history of athlete's foot, and increasing age.[8]

Palpation

The skin is palpated for texture, moisture, temperature, mobility and turgor, and edema (Fig 51-6). In addition, any evidence of discomfort arising from the areas palpated is noteworthy.

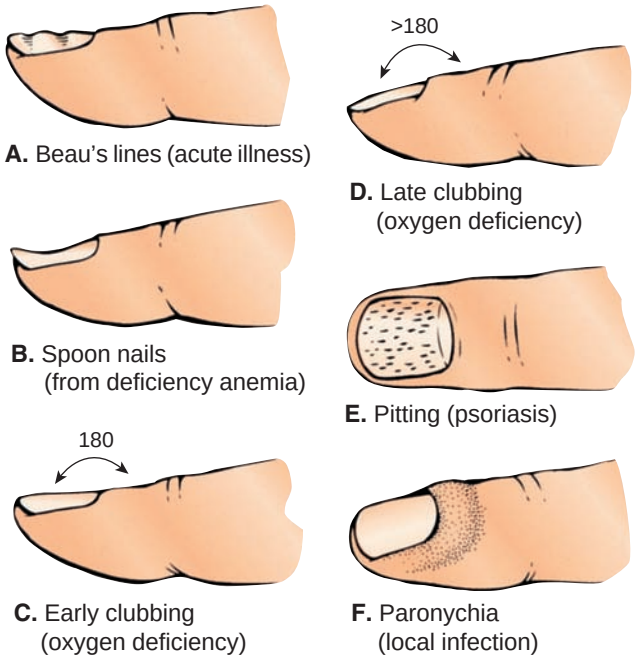


FIGURE 51-4 ▲ **A–F:** Common nail disorders. (From Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 203.)

Texture

Texture refers to the smoothness of the skin surface. It requires gentle palpation to assess. Rough skin occurs in patients with hypothyroidism.

Moisture

The skin may be described as dry, oily, diaphoretic, or clammy. Dry skin may be seen in the patient with hypothyroidism. Skin is oily with acne and with increased activity of the sebaceous glands, as in Parkinson's disease. Diaphoresis may be a response to increased temperature or increased metabolic rate. Hyperhidrosis is the term given to excessive perspiration. Bromhidrosis refers to foul-smelling perspiration. Low cardiac output states may produce skin that is referred to as clammy.

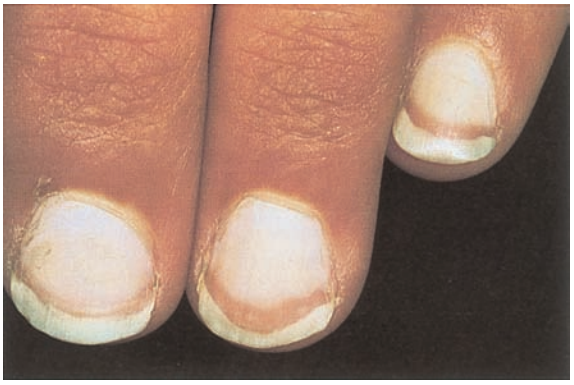


FIGURE 51-5 ▲ Terry's nails, seen in people with chronic diseases such as cirrhosis, congestive heart failure, and type II diabetes mellitus. (From Bickley LS: Bates' Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 193.)

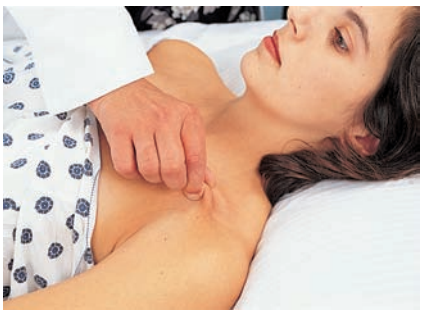


FIGURE 51-6 ▲ Palpating to assess skin turgor and mobility. (From Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Lippincott Williams &Wilkins, 2010, p 190.)

Temperature

Temperature is usually assessed with the dorsal surface of the hand to identify the general skin temperature as warm or cool. The skin's temperature can also be used to assess the possibility of reduced blood flow from an arterial insufficiency. In this case, the skin may be noticeably cooler distal to an occluding lesion.

Mobility and Turgor

Mobility and turgor provide information about the health of the skin and may yield information about the patient's fluid volume balance. When assessed centrally, over the clavicles, the skin is expected to lift up easily and quickly return into place. Skin mobility may be decreased in scleroderma or in a patient with increased edema. Skin turgor is decreased in the patient with dehydration.[1]

Edema

Edema is classified as either nonpitting or pitting. Nonpitting edema is that which does not depress with palpation. Nonpitting edema is seen in patients with a local inflammatory response and is caused by capillary endothelial damage. In addition to the edema, the skin is usually red, tender, and warm. Pitting edema is usually in the skin of the extremities and in dependent body parts. Pitting edema is identified as edema that retains the depression made when palpated. This type of edema can be further classified by the depth of the depression and, occasionally, by the amount of time it takes the pit to rebound (Table 51-6).

Table 51-6 Pitting Edema Scale

Scale	Description	Depth of Indentation (mm)	Return to Baseline
4+	Severe	8	2–5 min
3+	Moderate	6	1–2 min
2+	Mild	4	10–15 s
1+	Trace	2	Disappears rapidly

From Rhoads J: Advanced Health Assessment and Diagnostic Reasoning. Philadelphia, PA: Lippincott Williams & Wilkins, 2006, p 253.

▲ Assessment of Pressure Ulcers

The development of pressure ulcers in the critically ill patient is a preventable complication. The difficulty arises in the patient with multiple-system dysfunction with concomitant fluid, electrolyte, and nutritional deficiencies. Common pressure ulcer points include the occiput, scapula, sacrum, buttocks, ischium, heels, and toes. Pressure applied by the weight of the body causes a reduction in arterial and capillary blood flow, leading to these ischemic events. Therefore, frequent position changes are required to prevent the development of pressure ulcers. Pressure ulceration on the toes occurs as a result of the pressure of the bed linen on the feet. Dressing devices and wound appliances can place pressure on underlying skin, resulting in reduced blood flow. The back of the neck of the patient with a tracheostomy tube must be assessed because the tube holder may be applied too tightly. The tape securing a nasogastric tube must be regularly removed and the condition of the tip of the nose and nares assessed for changes resulting from pressure from the tube.

Assisting the patient with frequent position changes is crucial in preventing pressure ulcers from developing. In addition, keeping the skin clean and dry is requisite in preventing pressure ulceration. Moisture increases the risk for maceration of the skin and promotes its breakdown. Infectious matter in wound drainage or feces increases the risk that an ulcer will progress and become a major source of sepsis.

Patients with decreased sensation or awareness (eg, from brain or spinal cord injury or from a peripheral neuropathy, such as that caused by diabetes) are at greater risk for ulceration because they do not recognize the discomfort from being in one position for extended periods. Similarly, patients with sedation or frequent analgesic dosing are at increased risk for problems related to their immobility. Patients with poor circulation, such as that caused by hypotension, heart failure, or peripheral vascular insufficiency, are also at higher risk because of the underlying possibility of tissue hypoxia. Lack of movement then serves only to accelerate the process of pressure ulcer development.

Identifying those people most at risk for pressure ulcer development is a focus of assessment. The National Pressure Ulcer Advisory Panel recommends assessment on admission or change of status and at least every 24 hours in the intensive care unit.[10] Recognizing that there are certain features that increase a patient's risk for development of pressure ulcers allows the critical care nurse to increase surveillance and implement preventive treatment modalities. Problems with sensory perception, moisture, incontinence, activity, mobility, nutrition, and friction and shearing forces increase the patient's risk of developing pressure ulcers, which are debilitating and expensive to treat. Critically ill patients are among those with the most significant limitations of these parameters, and therefore are at very high risk for the development of pressure ulcers.

Many tools for assessing pressure ulcer risk use a point system.[9,10] The Braden Scale for Predicting Pressure Sore Risk, recommended in the guidelines set forth by the U.S. Agency for Health Care Policy and Research and widely used in hospital settings, requires the daily assessment of six parameters and provides a numerical score ranging from a very high-risk score of 6 to a very limited risk or minimal-risk score of 23[9] (Fig. 51-7). Adults with a score below 16 (18 for older adults) are considered at risk, and specific interventions to prevent the development of ulceration are recommended.

Studies suggest that stage 1 pressure ulcers are less likely to be identified in dark-skinned people, leading to higher incidence of stage 2 pressure ulcers in black patients than in white patients.[11,12] Bates, McCreath, and Pongquan[12] propose that difficulties with accurate visual assessment may contribute to this disparity when "lack of early recognition results in failure to institute preventive interventions." They further suggest that a nonvisual assessment technique, such as subepidermal moisture assessment, may offer a more objective method for assessing skin integrity.

During assessment of the skin, the nurse must be vigilant for signs of skin breakdown. The formation of pressure ulcers is illustrated in Figure 51-8. See Chapter 52 for the management of skin integrity.

▲ Assessment of Skin Tumors

Benign nevus and seborrheic keratosis are common, benign skin lesions. The benign nevus or mole appears in the first two to three decades, and its appearance remains unchanged over time. These lesions have clearly defined borders, are uniform in color, and are round or oval. The nevus is periodically assessed for changes because a change may indicate dysplasia of the tissue and the risk for melanoma. Seborrheic keratoses are common, yellow to brown lesions that are described as velvety when touched (Fig. 51-9A, p. 1166). These lesions are often multiple and often symmetrically distributed on the trunk and face. Precancerous lesions (actinic keratoses) are thick, rough patches that develop on sun-exposed areas of the skin, especially in fair-skinned people (see Fig. 51-9B, p. 1166). They are described as dry, scaly, and rough textured; however, not all actinic keratoses look alike.[13] The color may vary from brown to red to yellowish-black, or they may appear as red bumps or scaly patches. They are often described as feeling like sandpaper. These lesions require attention because there is a risk for development of squamous cell carcinoma.[13]

Skin cancer is the most common type of cancer in the United States. It is estimated that one in five people will be diagnosed with skin cancer in their lifetimes.[14] Basal cell and squamous cell cancers are often grouped as nonmelanoma skin cancers. Basal cell carcinomas are found exclusively in light-skinned people and arise from the hair follicles on the head and neck. Prolonged and cumulative exposure to the sun is recognized as the cause of basal cell carcinoma. These tumors are slow growing and rarely metastasize but do cause local skin destruction and disfigurement. Basal cell carcinomas appear with pearly, raised borders and depressed centers[1] (see Fig. 51-9C, p. 1166).

Squamous cell carcinomas affect the skin and the mucous membranes. Like basal cell cancers, the primary cause is exposure to ultraviolet light. Radiation and tissue damage from scars, ulcers, and fistulas may give rise to squamous cell carcinomas. These cancers can be invasive and are more malignant than basal cell cancers if not treated promptly. As it develops, the carcinoma takes on a hyperkeratotic appearance and may ulcerate and bleed[14] (see Fig. 51-9D, p. 1166).

Malignant melanomas are highly metastatic lesions that come from the melanin-producing cells of the body. The

Braden Scale

FOR PREDICTING PRESSURE SORE RISK

Patient's Name_____ Evaluator's Name_____ Date of Assessment

SENSORY PERCEPTION Ability to respond meaningfully to pressure-related discomfort	**1. Completely Limited:** Unresponsive (does not moan, flinch, or grasp) to painful stimuli, due to diminished level of consciousness or sedation. OR limited ability to feel pain over most of body surface.	**2. Very Limited:** Responds only to painful stimuli. Cannot communicate discomfort except by moaning or restlessness. OR has a sensory impairment which limits the ability to feel pain or discomfort over 1/2 of body	**3. Slightly Limited:** Responds to verbal commands, but cannot always communicate discomfort or need to be turned. OR has some sensory impairment which limits ability to feel pain or discomfort in 1 or 2 extremities.	**4. No Impairment:** Responds to verbal commands. Has no sensory deficit which would limit ability to feel or voice pain or discomfort.	
MOISTURE Degree to which skin is exposed to moisture	**1. Constantly Moist:** Skin is kept moist almost constantly by perspiration, urine, etc. Dampness is detected every time patient is moved or turned.	**2. Very Moist:** Skin is often, but not always, moist. Linen must be changed at least once a shift.	**3. Occasionally Moist:** Skin is occasionally moist, requiring an extra linen change approximately once a day.	**4. Rarely Moist:** Skin is usually dry, linen only requires changing at routine intervals.	
ACTIVITY Degree of physical activity	**1. Bedfast:** Confined to bed	**2. Chairfast:** Ability to walk severely limited or nonexistent. Cannot bear own weight and/or must be assisted into chair or wheelchair.	**3. Walks Occasionally:** Walks occasionally during day, but for very short distances, with or without assistance. Spends majority of each shift in bed or chair.	**4. Walks Frequently:** Walks outside the room at least twice a day and inside room at least once every 2 hours during waking hours.	
MOBILITY Ability to change and control body position	**1. Completely Immobile:** Does not make even slight changes in body or extremity position without assistance.	**2. Very Limited:** Makes occasional slight changes in body or extremity position but unable to make frequent or significant changes independently.	**3. Slightly Limited:** Makes frequent though slight changes in body or extremity position independently.	**4. No Limitations:** Makes major and frequent changes in position without assistance.	
NUTRITION Usual food intake pattern	**1. Very Poor:** Never eats a complete meal. Rarely eats more than 1/3 of any food offered. Eats 2 servings or less of protein (meat or dairy products) per day. Takes fluids poorly. Does not take a liquid dietary supplement. OR is NPO and/or maintained on clear liquids or IVs for more than 5 days.	**2. Probably Inadequate:** Rarely eats a complete meal and generally eats only about 1/2 of any food offered. Protein intake includes only 3 servings of meat or dairy products per day. Occasionally will take a dietary supplement. OR receives less than optimum amount of liquid diet or tube feeding.	**3. Adequate:** Eats over half of most meals. Eats a total of 4 servings of protein (meat, dairy products) each day. Occasionally will refuse a meal, but will usually take a supplement if offered. OR is on a tube feeding or TPN regimen which probably meets most of nutritional needs.	**4. Excellent:** Eats most of every meal. Never refuses a meal. Usually eats a total of 4 or more servings of meat and dairy products. Occasionally eats between meals. Does not require supplementation.	
FRICTION AND SHEAR	**1. Problem:** Requires moderate to maximum assistance in moving. Complete lifting without sliding against sheets is impossible. Frequently slides down in bed or chair, requiring frequent repositioning with maximum assistance. Spasticity, contractures or agitation leads to almost constant friction.	**2. Potential Problem:** Moves feebly or requires minimum assistance. During a move skin probably slides to some extent against sheets, chair, restraints, or other devices. Maintains relatively good position in chair or bed most of the time but occasionally slides down.	**3. No Apparent Problem:** Moves in bed and in chair independently and has sufficient muscle strength to lift up completely during move. Maintains good position in bed or chair at all times.		
					Total Score

Braden Scale Scores
1 = Highly Impaired
3 or 4 = Moderate to Low Impairment
Total Points Possible: 23
Risk Predicting Score: 16 or Less

NPO: Nothing by Mouth
IV: Intravenously
TPN: Total parenteral nutrition

FIGURE 51-7 ▲ The Braden Scale is a widely used screening tool to identify people at risk for pressure ulcers. (Courtesy of Barbara Braden and Nancy Bergstrom. Copyright, 1988. Reprinted with permission.)

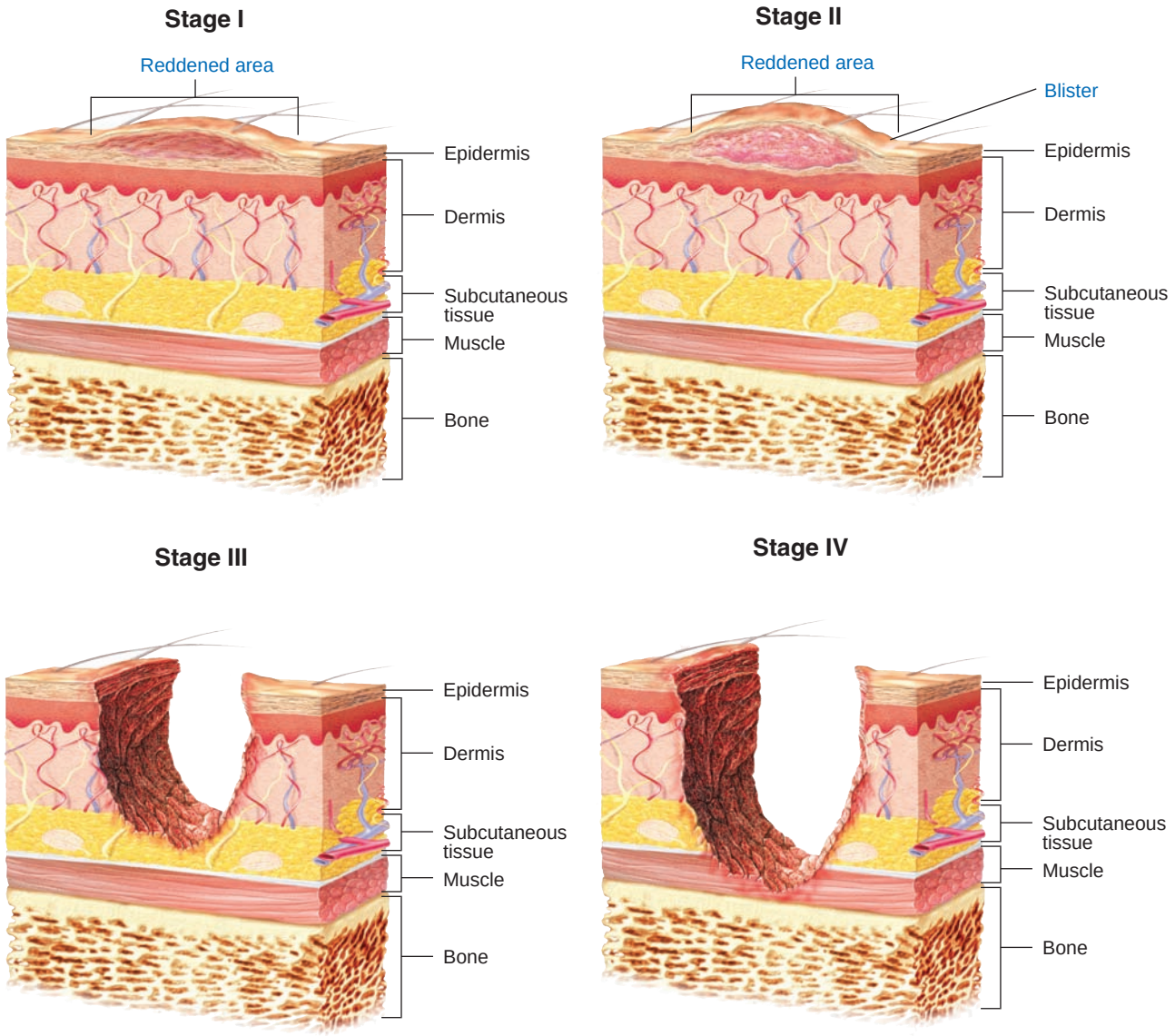
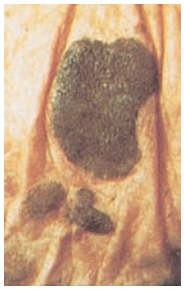
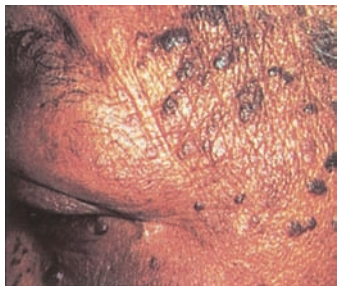


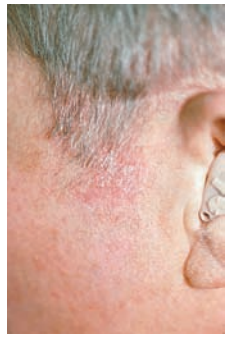
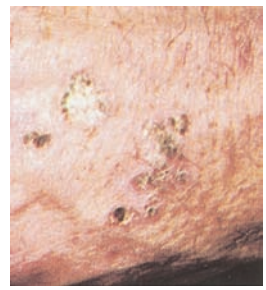
FIGURE 51-8 ▲ Pressure ulcers: identifying stage. (From Weber J, Kelley J: Health Assessment in Nursing, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 194–195.)

worldwide frequency of malignant melanomas is growing more rapidly than for any other cancer except lung cancer. Those at highest risk include those with fair complexions, those prone to sunburn, and those with a family history of melanoma.[14] The most common location for the development of these lesions is on the trunk in men and on the legs in women. The tumors have irregular borders, are dark brown or black, and are usually larger than 6 mm (see Fig 51-9E). The American Cancer Society[14] recommends a monthly self-assessment for melanoma using the "ABCDs." A is for asymmetry; B is for borders (are they irregular, ragged, notched, or blurred?); C is for color (dark brown or black, red, white, or blue?); and D is for diameter.

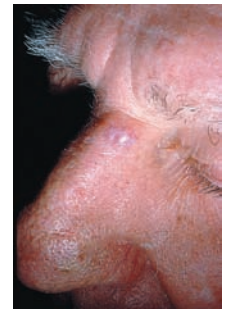
Figure 51-9 provides pictures and descriptions of these benign, premalignant, and malignant lesions. While in a critical care setting, it is possible to perform a thorough assessment for suspect skin lesions that may be cancerous, refer the patient to a dermatologist or oncologist, and have treatment initiated much sooner than would otherwise be the case.

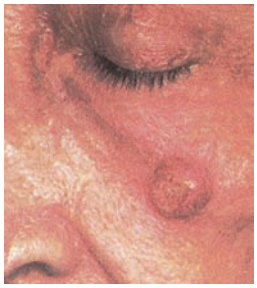
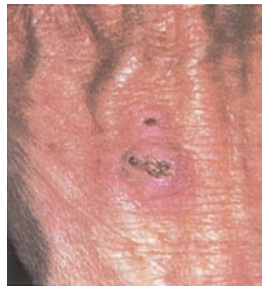
A. Seborrheic keratosis

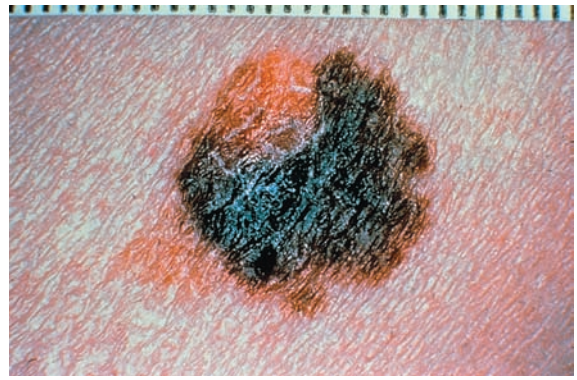
B. Actinic keratosis



C. Basal cell carcinoma

D. Squamous cell carcinoma



E. Malignant melanoma

FIGURE 51-9 ▲ Benign, premalignant, and malignant skin lesions. (**A, B,** and **D** from Hall JC: Sauer's Manual of Skin Diseases, 9th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2007; **C,** from Bickley LS: Bates' Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 185; **E,** Courtesy of the American Cancer Society, Inc. Atlanta, GA.)

▲ Clinical Applicability Challenges

CASE STUDY

Mr. P., a 74-year-old man, came in for coronary artery bypass graft 4 weeks ago and was unable to be weaned from the ventilator. He was moved to surgical intensive care unit during a 2-week period of hemodynamic instability that required intravenous norepinephrine (Levophed) to support his blood pressure, and during that time he was sedated. Mr. P. subsequently developed acute renal failure and pneumonia. His medical history includes chronic obstructive pulmonary disease, obesity, chronic renal insufficiency, and diabetes mellitus. He also has a history of alcohol abuse. Mr. P. is oriented to person reliably and sometimes to place. He was on continuous enteral feedings through a nasogastric tube until a week ago when a percutaneous endoscopic gastrostomy tube was placed. He has a right-arm peripherally inserted central catheter line, through which he is receiving antibiotics for pneumonia.

Mr. P. is scheduled for a tracheostomy tomorrow. He has a continuous bladder catheter and an incontinence fecal bag in place draining liquid stool. Physical therapy consultation was made on day 3, and once each day, he is transferred by three care givers to a stretcher chair for 4 to 6 hours. He cannot hold a pencil to write and has difficulty communicating.

1. What factors place Mr. P. at increased risk for developing pressure ulcers?
2. What are Mr. P.'s risk factors related to compromised integument that may lead to infection?
3. What nursing interventions would you use to reduce the risk to integument?

References

1. Bickley LS, Szilagyi PG: The skin. In Bickley LS (ed): Guide to Physical Examination and History Taking, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009
2. Weber J, Kelley JH: Skin, hair and nails. In Weber J, Kelley JH (eds): Health Assessment in Nursing, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010
3. Jarvis C: Skin, hair and nails. In Jarvis C (ed): Physical Examination and Health Assessment, 5th ed. St. Louis, MO: Saunders Elsevier, 2008
4. Dermnet NZ: Drug hypersensitivity syndrome (2010). Retrieved April 20, 2010 from http://dermnetnz.org/reactions/drug-hypersensitivity-syndrome.html
5. Simandl G: Disorders of skin integrity and function. In Porth C, Matfin G (eds): Pathophysiology, 8th ed. Philadelphia, PA: Wolters Kluwer Health/ Lippincott Williams & Wilkins, 2009, pp 1557–1602
6. MedlinePlus. Heparin (Injection). 2010. Retrieved April 20, 2010, from http://www.nlm.nih.gov/medlineplus/druginfomeds/a682826.html
7. Thrombophilia Awareness Project: Hair loss and Coumadin. 2009. Retrieved April 20, 2010, from http://www.fvleiden.org/ask/22.html
8. American Podiatric Medical Association: Nail problems. 2010. Retrieved April 20, 2010, from http://www.apma.org/MainMenu/Foothealth/FootHealthBrochures/GeneralFootHealthBrochures/Nailproblems.aspx
9. Bergstrom N, Braden BJ, Laguzza A, et al: The Braden scale for predicting pressure sore risk. Nurs Res 36:205–210, 1987
10. European Pressure Ulcer Advisory Panel and National Pressure Advisory Panel: Prevention and treatment of pressure ulcers: quick reference guide. 2009. Retrieved April 20, 2010, from http://npuap.org
11. Bates-Jensen B, McCreath H, Pongquan V: Subepidermal moisture is associated with early pressure ulcer damage in nursing home residents with darker skin tones. J Wound Ostomy Continence Nurs 36(3):277–284, 2009
12. Baumgarten M, Margolis D, van Doorn C, et al: Black/white differences in pressure ulcer incidence in nursing home residents. J Am Geriatr Soc 52:1293–1298, 2004
13. National Cancer Institute: What you need to know about skin cancer? 2009. Retrieved April 21, 2010, from http://www. cancer.gov/cancertopics/wyntk/skin
14. American Cancer Society: Skin cancer prevention and early detection. 2010. Retrieved April 19, 2010, from http://www.cancer.org

52 Patient Management: Integumentary System

Susan Luchka

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Define specific terms related to wounds: *acute wound, chronic wound, partial thickness, full thickness, stages of wound healing*.
2. Explain the normal healing process.
3. Describe what is meant by *primary intention*, *secondary intention*, and *tertiary intention*.
4. Describe nursing care for the patient with a wound.
5. Discuss the influence of nutrition and pharmacotherapy on wound healing.

Nursing management of wounds is both challenging and rewarding. Effective wound care requires a sound knowledge base and meticulous method. This chapter discusses wounds, such as pressure ulcers and skin tears, the healing process, and nursing assessment, management, and patient teaching related to caring for serious wounds that need ongoing care.

▲ Types of Wounds

A wound is simply a break in skin integrity. Wounds may be acute or chronic. An acute wound is a wound that follows an orderly, sequential healing process, resulting in an area that has anatomical and functional integrity.[1,2] Acute wounds are caused by surgery or trauma. Conversely, a chronic wound fails to yield an area that has anatomical and functional integrity. Chronic wounds fail to follow an orderly, sequential process because of precipitating factors, such as diabetes, pressure, malnutrition, peripheral vascular disease, immune deficiencies, and infection.[1,2] An acute wound may become a chronic wound at any time.

Acute and chronic wounds may be defined as partial- or full-thickness wounds. Partial-thickness wounds involve the epidermis and may involve the dermis. A partial-thickness wound is a shallow wound that is usually moist and painful (because the loss of the epidermis exposes the nerve endings). Full-thickness wounds involve the loss of the epidermis, dermis, and subcutaneous tissue, and they may involve muscle, tendons, ligaments, and bone. A full-thickness wound involves a large amount of tissue loss and appears as a crater or crevice.

Pressure ulcers and leg ulcers are two specific types of wounds that may be seen in the critical care setting. Critically ill patients are at risk for developing pressure ulcers related to hemodynamic factors, disease processes, immobility, and nutritional deficits. Leg ulcers are due to specific disease processes. Both pressure ulcers and leg ulcers may complicate the critically ill patient's overall recovery.

Pressure Ulcers

Pressure ulcers are wounds caused by pressure, shearing, and friction. Pressure ulcers start as acute wounds but become chronic in patients with other risk factors. Risk factors for the development of pressure ulcers include prolonged and impaired mobility, incontinence, malnutrition, diabetes, spinal cord injuries, metastatic cancers, decreased level of consciousness, impaired mental status, and peripheral vascular disease.[1–3] A patient teaching guide for pressure ulcers is shown in Box 52-1.

Pressure ulcers are the only type of wound that is staged. Staging occurs when the wound is assessed and documented.

- Stage I is defined as nonblanchable erythema of intact skin. In patients with darker skin, the stage I pressure ulcer may be red, blue, or purple. It may be accompanied by hardness, induration, and edema.
- Stage II involves partial-thickness tissue loss and presents as a fluid-filled blistered or denuded area (a shallow open wound).
- Stage III is a full-thickness wound involving the subcutaneous tissue and presents as a crater.
- Stage IV is also a full-thickness wound involving a large amount of tissue loss. A stage IV wound extends through the subcutaneous tissue and deep into the fascia, involving muscle, bone, ligament, or tendon.
- Unstageable is a pressure ulcer covered by eschar (black, brown, tan) or slough (yellow, brown, grey, green, or tan), which prevents assessment of the wound bed. The wound must be débrided prior to staging.
- Suspected deep tissue injury is a localized maroon or purple discolored area of intact skin or a blood-filled blister. The area is usually tender, mushy, or boggy.[3]

BOX 52-1 TEACHING GUIDE Pressure Ulcers

- Pressure ulcers are also known as *pressure sores* or *bed sores*.
- Pressure ulcers occur in people who have trouble moving around easily.
- At first, a pressure ulcer is just a reddened, tender area. If pressure is not relieved, the skin in this area may break down (open up or pull off, forming a blistered area). Pressure ulcers can destroy the underlying muscles, bone, ligaments, and tendons if they are not treated.
- Risk factors for pressure ulcers include difficulty moving around, medical problems (such as diabetes), spinal cord injury, incontinence of urine and stool, surgeries that limit mobility for an extended period (such as hip or knee replacement surgery), poor nutrition, and poor hydration (decreased fluid intake).
- Pressure ulcers occur most frequently over bony prominences (eg, heels, sacral area, hips, and shoulder blades), but they can occur anywhere on the body where there is constant pressure that is unrelieved.
- Many times, pressure ulcers can be prevented by turning the person in bed at least every 2 hours and by placing a pillow under the person's ankles to keep the heels off the bed, thus relieving pressure. A specialty bed may also be used to decrease pressure.
- *Not all pressure ulcers can be prevented*. The person's medical condition, nutrition and hydration status, immune status, and overall health status are all factors that affect the person's risk for developing pressure ulcers.
- Treatment depends on the type of pressure ulcer and the person's health status.

Reverse staging is inappropriate. The tissue that fills in the wound bed is not the same as the tissue that has been lost. Lost muscle or subcutaneous tissue cannot be replaced. Therefore, it is appropriate to document "healing stage IV wound," but it is not appropriate to document "stage IV wound now stage III."

Pressure ulcers covered by eschar or slough are considered unstageable. Eschar prevents the assessment of the wound bed. Documentation is "unstageable, wound covered by eschar/slough." If the wound is débrided, it may then be staged.

The standards of care for pressure ulcers are established by the Agency for Healthcare Research and Quality (AHRQ), the Wound Ostomy and Continence Nurses (WOCN), the National Pressure Ulcer Advisory Panel (NPUAP), and the European Pressure Ulcer Advisory Panel (EPUAP).

The most recent standards of care were set by the NPUAP and the EPUAP in 2009 and are considered the gold standard at this time. These standards guide institution policy and procedure and are supported by evidence-based practice.

The Wound Ostomy and Continence Nurses Society (WOCN) have expanded the AHRQ standards through evidence-based practice. The WOCN issues clinical practice guidelines that apply evidence-based practice, new research and drugs, and the AHRQ standards. Guidelines specific to wound care are:

- Guidelines for Management of Wounds in Patients with Lower Extremity Arterial Disease (2002)
- Guideline for the Prevention and Management of Pressure Ulcers (2003)
- Management of Wounds in Patients with Lower-Extremity Neuropathic Disease (2004)
- The V.A.C. Therapy Clinical Guidelines (2007)
- NPUAP-EPUAP Guidelines for Pressure Ulcer Prevention and Treatment (2009)

Leg Ulcers

Leg ulcers are chronic wounds seen frequently in critically ill patients with underlying health problems, such as venous stasis ulcers, arterial ulcers, and diabetic foot ulcers. Although patients with leg ulcers may have a high risk for pressure ulcers, leg ulcers are not pressure ulcers and are not staged.

Venous Stasis Ulcers

Venous stasis ulcers are usually found on the medial aspect of the lower leg, superior to the medial malleolus.[1,2] The wound margins are irregular and present as shallow craters, and the wound margins and lower leg may have a ruddy appearance or hemosiderin staining.[1,2] The drainage from venous stasis ulcers may vary from mild to heavy. The primary treatment for venous stasis ulcers is compression therapy using an Unna boot or a multiple-wrap dressing.[1,2] Multiple-wrap dressings have the advantage of continuous compression, which may not be achieved with the Unna boot. The affected leg is elevated above heart level to decrease edema (edema impedes the healing process).

Arterial Ulcers

Arterial ulcers (ischemic ulcers) are usually found on the distal leg, medial malleoli, and dorsal aspect of the foot and toes.[1,2] The wound margins of arterial ulcers are round, smooth (*not* irregular), and frequently described as having a punched-out appearance.[1,2] Arterial ulcers have pale wound beds and may be shallow or deep. The affected leg may be cool to the touch, cyanotic, and pale with minimal hair distribution. The patient experiences increased pain to the affected area if the leg is elevated.[1,2] The primary dressing for arterial leg ulcers is an occlusive dressing. Healing does not occur unless the vascular deficit is addressed surgically.

Diabetic Foot Ulcers

Diabetic foot ulcers are found in patients with diabetes and are frequently not recognized early, owing to the patient's accompanying neuropathy. The primary locations for diabetic foot ulcers are the plantar aspect of the foot, heels, and metatarsals.[1,2] To promote wound healing, a dressing that provides a moist environment is used most often. The ulcer area usually needs débridement and must be assessed carefully for infection. Other treatment modalities include off-loading the patient's weight using special shoes. Osteomyelitis is always a concern in patients with diabetic foot ulcers. Healing is prolonged because of the diabetes. Therefore, it is important to aggressively manage the diabetes to promote an optimal healing environment.

Skin Tears

Skin tears (partial-thickness wounds) are acute wounds secondary to the removal of tape or transparent occlusive dressings. Skin tears occur when the skin is thin and fragile. Fragile skin may be due to age, disease process, nutritional status, drug therapy (ie, steroids), or a combination of these factors. The skin is so fragile that it literally tears as tape or plastic film dressings are removed.

It is a common misconception that plastic film dressings, wound closure strips (Steri-Strips), or wound adhesives should

be applied to skin tears. Plastic film dressings and Steri-Strips potentiate more skin tears as they are removed or become dislodged. Because it is difficult to approximate the wound margins in a skin tear, wound adhesive frequently drips into the wound bed, prolonging healing and promoting infection.

Skin tears are cleansed gently with normal saline solution (or other institution-approved cleanser). Care is taken not to create a larger skin tear. After the wound area is cleansed, a hydrogel is applied to the wound and covered with a nonadherent dressing. Then the wound is wrapped with a self-adherent wrap, such as Kling or Coban, to hold the dressing in place without using tape on the skin. It is important to minimize the use of adhesives in all forms for patients prone to skin tears.

▲ Wound Healing

Optimal wound healing occurs in a moist (not extremely wet or dry) environment. The wound-healing process consists of three phases (Fig. 52-1).

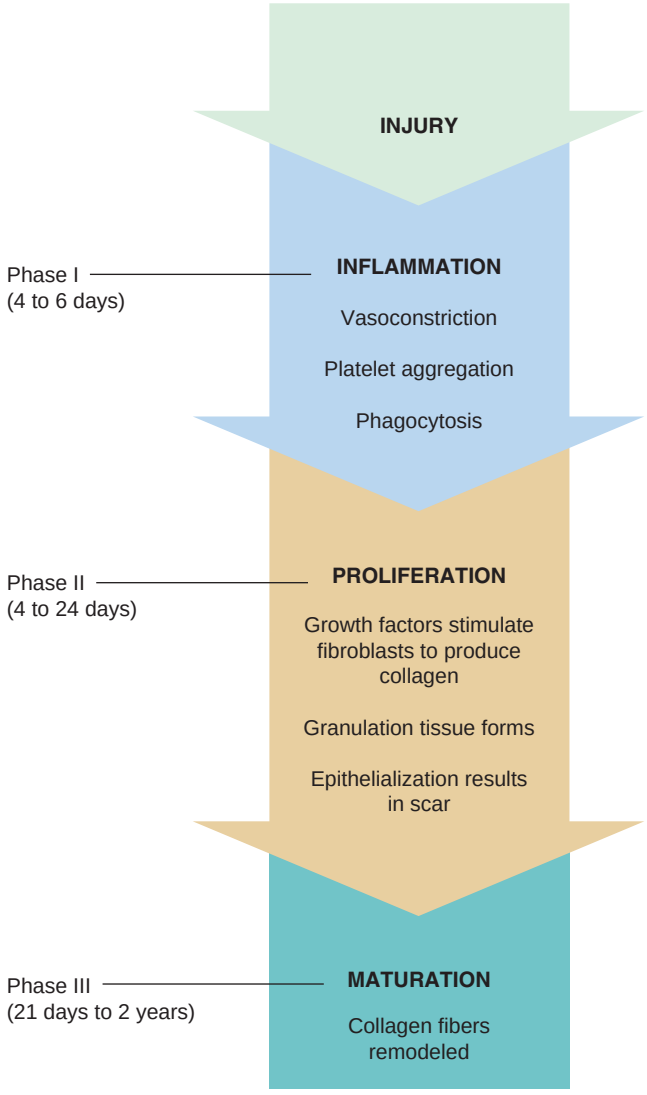


FIGURE 52-1 ▲ The stages of wound healing.

Phases of Wound Healing

The first phase is the *inflammatory phase*, which occurs immediately after the wound occurs. At the time of injury, there is immediate vasoconstriction; this is the body's way of controlling bleeding. Once vasoconstriction occurs, platelets collect at the site and deposit fibrin to form a clot. The vasoconstriction holds the wound together, and the platelets with their fibrin clot formation essentially "plug the hole." Phagocytosis also occurs during the inflammatory phase. Phagocytosis is the release of macrophages at the site of injury to destroy any bacteria that may be present and to remove the wound's cellular debris. This is the body's way of providing the optimal environment for wound healing (ie, a clean wound bed). It is at this time that growth factors are also present at the site of the injury. Overall, the inflammatory phase is estimated to last between 4 and 6 days. Visual assessment of the wound during the inflammatory phase reveals a wound with erythema, edema, and pain.

The second phase of wound healing is the *proliferation phase*. Growth factors stimulate the fibroblast to produce collagen. Collagen, along with new blood vessels and connective tissue, creates granulation tissue. Visual assessment of the wound at this point reveals a wound that is beefy red and shiny with a grainy or bumpy appearance. The appearance of granulation tissue prompts the wound margins to contract. Pulling together of the wound edges decreases the overall size of the wound. The last step of the proliferation phase is *epithelialization* or *reepithelialization*. Epithelialization results in a scar. The estimated overall duration of the proliferation phase is anywhere from 4 to 24 days.

The final and third phase of wound healing is the *maturation phase*. During the maturation phase, the collagen fibers are remodeled. The goal is to increase the tensile strength of the scar tissue. It has been estimated that only 70% to 80% of the skin's original strength is attained when the wound is healed. The maturation phase can extend from 21 days to 2 years. The outcome is always an area of tissue that is at greater risk for breakdown and more fragile than undamaged tissue.

If the wound becomes extremely wet or dry, the phases of wound healing occur, but at a slower rate. This may affect the final quality of the scar tissue with respect to anatomical and functional integrity as well as tensile strength. The patient's age and physical status also have an impact on the healing process (Box 52-2). Other factors that affect wound healing are listed in Table 52-1.

BOX 52-2 CONSIDERATIONS FOR THE OLDER PATIENT

Factors That Affect Wound Healing

- Less subcutaneous tissue
- More fragile skin secondary to age and drug therapy
- Increased number of precipitating risk factors for pressure ulcers
- Increased number of precipitating risk factors for chronic wounds
- Nutrition: less than or more than body requirements
- Decreased ability to care for self with age
- Decreased immune system function
- Decreased pulmonary and cardiovascular function
- Increased potential for incontinence (urine and stool)

Table 52-1 Factors Affecting Wound Healing

Factors	Rationale	Nursing Interventions
Age of patient	The older the patient, the less resilient the tissues.	Handle all tissues gently.
Handling of tissues	Rough handling causes injury and delayed healing.	Handle tissues carefully and evenly.
Hemorrhage	Accumulation of blood creates dead spaces as well as dead cells that must be removed. The area becomes a growth medium for organisms.	Monitor vital signs. Observe incision site for evidence of bleeding and infection.
Hypovolemia	Insufficient blood volume leads to vasoconstriction and reduced oxygen and nutrients available for wound healing.	Monitor for volume deficit (circulatory impairment). Correct by fluid replacement as prescribed.
Local Factors		
Edema	Reduces blood supply by exerting increased interstitial pressure on vessels.	Elevate edematous part; apply cool compresses.
Inadequate Dressing Technique Too small Too tight	 Permits bacterial invasion and contamination Reduces blood supply carrying nutrients and oxygen	 Follow guidelines for proper dressing technique.
Nutritional deficits	Protein–calorie depletion may occur. Insulin secretion may be inhibited, causing blood glucose level to rise.	Correct deficits; this may require parenteral nutritional therapy. Monitor blood glucose levels. Administer vitamin supplements as prescribed.
Foreign bodies	Foreign bodies retard healing.	Keep wounds free of dressing threads and talcum and powder from gloves.
Oxygen deficit (tissue oxygenation insufficient)	Insufficient oxygen may be due to inadequate lung and cardiovascular function as well as localized vasoconstriction.	Encourage deep breathing, turning, controlled coughing.
Drainage accumulation	Accumulated secretions hamper healing process.	Monitor closed drainage systems for proper functioning. Institute measures to remove accumulated secretions.
Medications		
Corticosteroids	May mask presence of infection by impairing normal inflammatory response	Be aware of action and effect of medications patient is receiving.
Anticoagulants	May cause hemorrhage	
Broad-spectrum and specific antibiotics	Effective if administered immediately before surgery for specific pathology or bacterial contamination. If administered after wound is closed, ineffective because of intravascular coagulation.	
Patient overactivity	Prevents approximation of wound edges. Resting favors healing.	Use measures to keep wound edges approximated: taping, bandaging, splints. Encourage rest.
Systemic disorders Hemorrhagic shock Acidosis Hypoxia Renal failure Hepatic disease Sepsis	These depress cell functions that directly affect wound healing.	Be familiar with the nature of the specific disorder. Administer prescribed treatment. Cultures may be indicated to determine appropriate antibiotic.
Immunosuppressed state	Patient is more vulnerable to bacterial and viral invasion; defense mechanisms are impaired.	Provide maximum protection to prevent infection. Restrict visitors with colds; institute mandatory hand hygiene by all staff.
Wound stressors Vomiting Valsalva maneuver Heavy coughing Straining	Produce tension on wounds, particularly of the torso.	Encourage frequent turning and ambulation and administer antiemetic medications as prescribed. Assist patient in splinting incision.

From Smeltzer SC, Bare BG, Hinkle JL, et al: Brunner & Suddarth's Textbook of Medical–Surgical Nursing, 12th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 475.

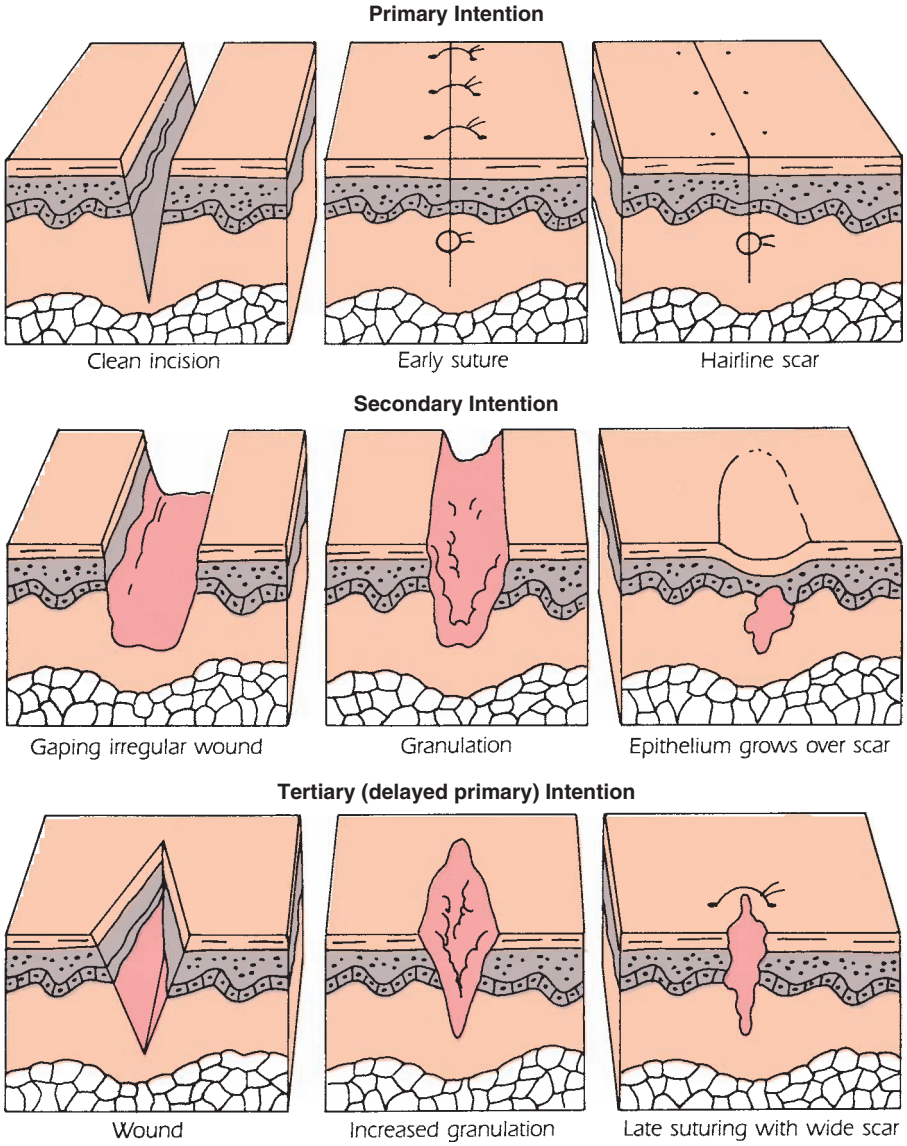


FIGURE 52-2 ▲ Types of wound healing: primary intention healing, secondary intention healing, and tertiary intention healing. (Adapted from Smeltzer SC, Bare BG, Hinkle JL, et al: Brunner & Suddarth's Textbook of Medical–Surgical Nursing, 12th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, p 474.)

Methods of Wound Healing

Wounds can heal through primary intention, secondary intention, or tertiary intention (Fig. 52-2). *Intention* is used for acute or surgical wounds. The edges of the wound are drawn together (approximated), shortening the time required for the wound to heal to about 4 to 14 days overall. Primary intention is associated with a decreased risk for infection and minimal scarring. With primary intention, not only is scarring minimal and the risk for infection decreased, but the amount of tissue loss is decreased.

Secondary intention is seen most frequently in chronic wounds but can occur in acute wounds, when the wound edges cannot be approximated to each other because of a significant tissue loss. An example of secondary intention is a pressure ulcer or a venous stasis ulcer. The potential for infection is increased because of the inability to approximate the edges, thus leaving the area open to bacteria. Scarring may also be significant, depending on the amount of tissue loss.

The last form of wound repair is *tertiary intention*, which may also be called *delayed primary intention*. Tertiary (delayed primary)intention should not be confused with primary intention. With this type of wound healing, the wound is not closed for a period (usually 3 to 5 days) to allow infection, edema, or both, to resolve. During this time, the wound is packed or irrigated to remove exudate and cellular debris. When the edema and risk for infection have decreased, the wound edges are approximated, and the wound is closed as it is in primary intention. Scarring is usually greater than that seen with primary intention but less than that seen with secondary intention.

▲ Wound Assessment

Wound assessment is performed in an orderly, sequential manner (Box 52-3). The location of the wound is defined as precisely as possibly using anatomical terminology

BOX 52-3 Wound Assessment

Location: Document the location, using anatomical positions.
Size: Document the size, in centimeters or millimeters. Measure the length (by clock positions) from the 12- to 6-o' clock position. Measure the width from the 9- to 3-o' clock position.
Depth: Use a sterile swab to determine the depth of the wound (see Fig. 52-4).
Undermining or tunneling: Document the presence or absence of undermining or tunneling (see Fig. 52-5).
Tissue type: Describe the wound bed. If the wound bed is not visible, document the presence and condition of the eschar (scab), sutures, staples, or other wound closure.
Drainage: Note the presence or absence of drainage. If drainage is present, describe its odor, color, amount, and consistency.
Wound margins: Describe the wound margins (approximation, condition, and appearance of surrounding tissue).
Drains and tubes: Note the type of drain or tubing and its location (using anatomical or clock positions).
Condition of dressing: Describe the amount and type of drainage on the dressing, as well as the ease with which the dressing was removed.
Pain: Evaluate on a 0 to 10 scale (or other institution-approved assessment scale). Provide pain relief as needed before, during, and after wound assessment or dressing change.

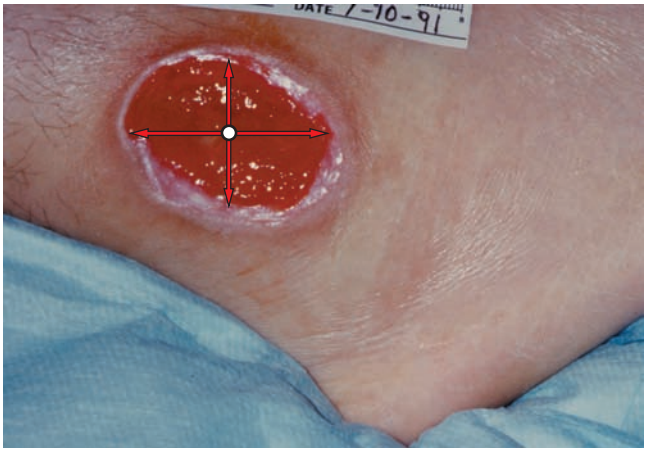


FIGURE 52-3 ▲ Wound measurement. Linear measurements of a wound should be taken at the greatest length and width perpendicular to each other, as shown. (From Baranoski S, Ayello EA: Wound Care and Essentials Practice Principles, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, p 84.)

(eg, "medial aspect of the left lower leg, 10 cm distal to the knee"). Using correct anatomical terminology allows other health care professionals to visualize the location of the wound. Correct location is especially important if the patient has more than one wound. Photography may be used, more frequently in chronic wounds than in acute wounds. Factors to consider when using photography are lighting consistency and distance. To portray the wound accurately, room lighting and distance from the wound must be as identical as possible from one photograph to another.

The size of the wound should always be measured in centimeters, millimeters, or both.[2,3] Terminology such as "the size of a half dollar" should be avoided. This leads to inconsistent and inaccurate documentation. Linear measurements of a wound are taken at the greatest length and width perpendicular to each other (Fig. 52-3).

Depth of the wound is measured by placing a sterile swab in the deepest area of the wound and marking the location of the wound's margin on the swab.[2] The sterile swab is dipped in normal saline solution before inserting it into the wound to minimize the potential of leaving cotton fibers in the wound. After removing the swab, measure from the distal tip of the swab to the area that was marked. Documentation includes the depth in centimeters and also the location where the assessment was made (eg, "depth 5.8 cm in the distal wound bed in the 9-o' clock position") (Fig. 52-4). Clear, concise documentation allows other health care professionals to reassess the wound depth in the same area with each reevaluation.

Undermining and tunneling do not occur in acute wounds, but the nurse always assesses the wound for their presence. Undermining occurs when there is the loss of tissue along the wound margins, the "lip of tissue."[2] Tunneling is exactly what it implies, a tunnel opening somewhere in the wound bed.

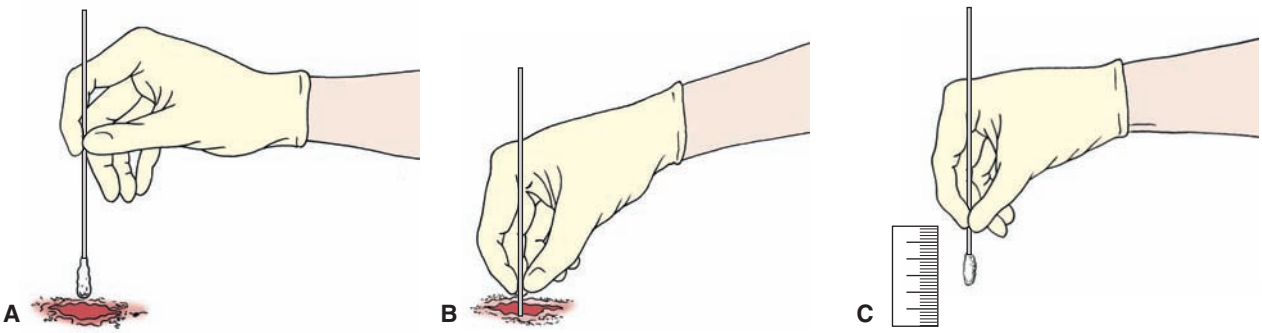


FIGURE 52-4 ▲ Procedure for measuring wound depth. **A:** Put on gloves. Gently insert the swab into the deepest portion of the wound that you can see. **B:** Grasp the swab with your thumb and forefinger at the point corresponding to the wound margin. **C:** Carefully withdraw the swab while maintaining the position of your thumb and forefinger. Measure from the tip of the swab to that position. (From Thomas Hess C: Clinical Guide: Wound Care, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, p 25.)

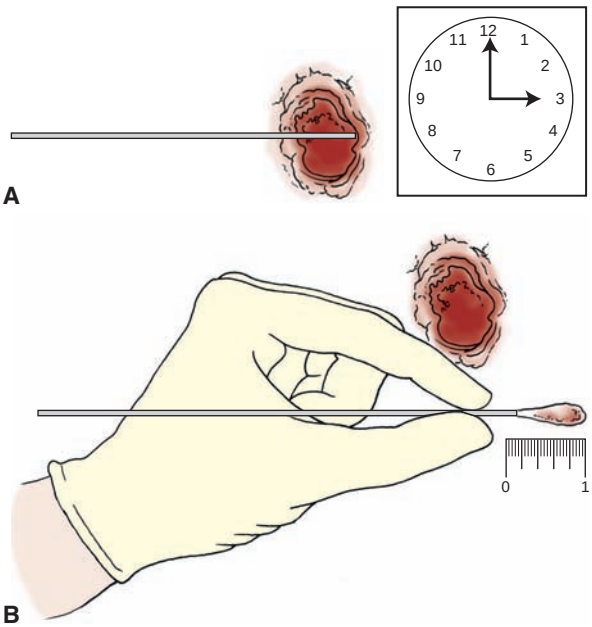


FIGURE 52-5 ▲ Procedure for determining the direction and depth tunneling. **A:** To assess the direction of tunneling, put on gloves and insert the swab into the sites where tunneling occurs. Progressing in a clockwise direction, document the deepest sites where the wound tunnels. (Twelve o'clock points in the direction of the patient's head, so in this example, tunneling occurs at three o'clock.) **B:** To assess the depth of tunneling, insert the swab into the tunneling areas, and grasp the swab where it meets the wound margin. Remove the swab, place it next to a measuring guide, and document the measurement centimeters. (From Thomas Hess C: Clinical Guide: Wound Care, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, p 27.)

Tunneling can begin in acute wounds in which there are drains. (Note that if tunneling occurs, the wound is not an acute wound but has become a chronic wound.) A sinus tract is an opening somewhere in the wound bed that extends into the tissue ending with a "small pocket" of open area. The process for assessing the direction and depth of tunneling is shown in Figure 52-5.

Determining the tissue type entails visual assessment of the wound bed. The tissue in the wound bed should be beefy red (as opposed to pale). The presence or absence of granulation tissue (shiny red, grainy, or bumpy tissue) is noted. The nurse assesses for necrotic tissue, which presents as black or brown tissue. Slough may also be present in the wound bed. Slough is yellow and stringy. If the wound bed is not visible, the presence or absence of eschar (scab), sutures, staples, Steri-Strips, wound adhesives, or negative pressure dressings is documented.

The presence or absence of drainage is also important to note, along with the location of the drainage or exudate (eg, "drainage/exudate noted at the proximal end of the wound"). The drainage or exudate needs to be assessed for odor, color, consistency, and amount (eg, "abdominal dressing of ten 4″ × 4″ gauze pads is saturated with serosanguineous drainage every 2 hours").

The wound margins are also assessed when performing a wound assessment. Are the edges well approximated? Is the surrounding tissue clean, dry, reddened, edematous, pale, intact, or blistered? Again, it is important to be as exact as possible to paint an accurate picture of the wound margins for the next health care professional.

Drains or tubes may be present in or near the wound bed. Drains or tubes are assessed for location, the appearance of the surrounding tissue, and the characteristics of the drainage. Consider the insertion site of a drain or tube as an acute wound in itself.

The dressing is assessed after it is removed. The soiled dressing's condition (eg, "saturated"), the ease with which the dressing was removed (eg, "sticking"), and the location and type of drainage on the dressing are described. If the dressing came off without being removed by the nurse, this is noted as well (eg, "dressing found lying in bed—wound uncovered").

Pain is assessed using an institution-approved standardized scale, such as the 0 to 10 scale. The patient should never be in pain while a wound is being assessed. If the patient experiences pain during wound assessment, the assessment should be stopped, and the patient should be medicated before continuing. Management of pain as it relates to wound assessment and care is discussed in more detail later in this chapter.

Wound documentation includes all descriptions and measurements, the presence and absence of pain during the procedure, and the type of dressing applied.[1–3] Many institutions use special wound measurement tools, wound assessment tools, and documentation tools (eg, flow sheets or computerized documentation) for wound documentation. The presence or absence of wounds must be documented upon admission per Medicare guidelines by both the physician and the nurse. Pressure ulcers are considered preventable; therefore, if the patient develops a pressure ulcer during hospitalization, the hospital will not be reimbursed for the care.

▲ Wound Care

Nursing diagnosis for patients with wounds revolves around a few basic themes[4] (Box 52-4). Wound care seeks to address these problems.

Wound Cleansing

The goal of cleansing the wound is to remove bacteria and cellular debris without damaging the wound bed or granulating tissue. The periwound area must also be cleansed

BOX 52-4 NURSING DIAGNOSES

For the Patient With an Acute or Chronic Wound

- Acute Pain related to acute débridement and wound care
- Ineffective Peripheral Tissue Perfusion related to immobility, edema, infection decreased cardiac output
- Impaired Tissue Integrity related to etiology/contributing factors of wound development
- Imbalanced Nutrition: Less Than Body Requirements
- Risk for Infection related to altered skin integrity
- Disturbed Body Image related to dysfunctional open wound, scarring, or amputation
- Risk for Imbalanced Fluid Volume related to metabolic changes
- Risk for Electrolyte Imbalance related to metabolic changes
- Impaired Physical Mobility related to wound

to prevent bacteria from migrating into it. All wounds are cleansed before reapplying the dressing. Normal saline solution is the safest wound cleanser. Some commercial cleansers are also safe for the wound bed. Solutions to be avoided when cleansing wounds are povidone-iodine, acetic acid, sodium hypochlorite (Dakin's solution), and hydrogen peroxide. These solutions are toxic to epithelial cells and thereby impede granulation and wound healing.

Open wounds are cleansed starting in the middle and moving outward in a circular motion to include the periwound area. Incisions are cleansed from top to bottom, again starting in the middle and moving outward to include the periwound area.

Wound Closure

The goal of all wound care is ultimately the closure of the wound and restoration of skin integrity. Wound closure is usually promoted by various types of treatments and dressings.

Vacuum-Assisted Wound Closure (Negative Pressure Therapy)

Vacuum-assisted wound closure (VAC) is a system that assists wound closure by providing localized negative pressure to the wound bed and wound margins. The occlusive dressing promotes a moist environment for healing, and the negative pressure removes excessive wound drainage, which assists in pulling the wound margins together (Fig. 52-6).[5]

Tubing, similar to that of suction tubing, is placed into a special foam dressing. The foam dressing is shaped in wedges that are cut to fit the wound. The sponge wedge and tubing are then covered with an occlusive transparent dressing. The tube is then connected to the vacuum unit at low suction levels (as directed by the manufacturer). The negative pressure draws the wound edges together by collapsing the foam dressing and removing wound fluids while maintaining a moist wound environment that promotes healing. If the dressing is not collapsed, there is a leak in the system, and the dressing must be replaced with attention given to the transparent occlusive dressing application. The transparent occlusive dressing must be securely in place to maintain negative pressure in the wound. Dressings have the appearance of being "vacuum packed" when the dressing is secure and occlusive.

With the VAC system, granulation tissue is stimulated, infection and bacterial colonization are decreased, and wound closure occurs in a moist "vacuum" environment. In addition, the VAC system decreases the frequency of dressing changes, thus decreasing patient discomfort and nursing time.[5]

The VAC system can be used in both acute and chronic wounds.[5] The VAC system may be indicated for chronic wounds (including diabetic and nonhealing stage III and IV pressure ulcers); flaps and grafts (both acute surgical wounds); dehisced incisions; acute and traumatic wounds; and burns. Dehisced incisions are ones that split open along natural or sutured lines. The VAC system should be used with extreme caution in patients with active bleeding, those who are on anticoagulant therapy, or patients with a history of uncontrolled bleeding.[5] The VAC system is contraindicated for any patient with untreated osteomyelitis, necrotic tissue with eschar, malignancies of the wound, or nonenteric and unexplored fistulas. The foam wedge dressing is not to be placed in direct contact with exposed blood vessels, organs, or nerves. VAC sponges must be positioned on viable tissue; therefore, if necrotic tissue or devitalized tissue is present, the wound needs to be débrided before the VAC sponges can be placed. The VAC system may be used in an infected wound but only with appropriate antibiotic therapy.[5] Patients at risk for bleeding must be monitored carefully, and if bleeding occurs, the VAC therapy must be discontinued.[5]

The use of the VAC system continues to increase as clinical case studies show positive patient outcomes in grafts, flaps, and orthopedic surgeries. The use of wound irrigations or instillations (antibiotics or anesthetic agents) in conjunction with this type of wound therapy is another promising area.

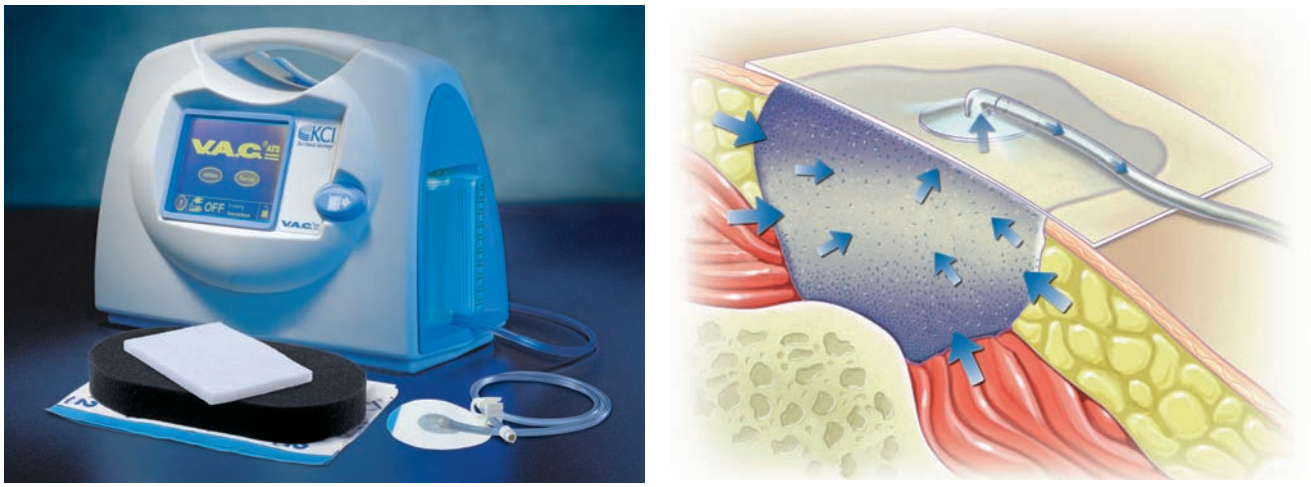


FIGURE 52-6 ▲ A vacuum-assisted wound closure device, such as the V.A.C. ATS device shown here, assists wound closure by providing localized negative pressure to the wound bed and wound margins. (Courtesy of KCI Licensing, Inc., 2007.)

VAC therapy also has made an economic impact. It decreases the length of wound healing, nursing labor, supplies, length of stay, complications, and hospital readmission, while it promotes the salvage of limbs.[5] In addition, this therapy has an emotional impact that can be directly related to the nursing diagnosis of Disturbed Body Image related to dysfunctional open wounds, scarring, or amputation.[4]

It is the nurse's responsibility to be familiar with the operation and maintenance of the VAC system. Nursing responsibilities include wound assessment and documentation, along with placing the patient on the VAC system, changing the canister, and maintaining the system. The wound should demonstrate progressive healing. If documentation fails to demonstrate progressive wound healing within 30 days, then alternative therapies must be considered.

Sutures, Staples, and Wound Adhesives

Sutures or staples must be cleaned with sterile normal saline solution or a wound cleanser. Immediately after surgery, the wound needs to be covered with a dry sterile dressing. Frequently after the initial postoperative period, the staples or sutures are left open to air.

Wound adhesives may be used on surgical or traumatic wounds to approximate the wound margins, in which case sutures are used to close the underlying tissue, and the wound adhesive is applied topically to the wound margins as they are drawn together. Wound adhesives are not to be placed in the wound bed (only on the margins) because this may lead to delayed wound healing or infection. The wound adhesive appears like a shiny, clear coating over the incision. Caution is needed when applying wound adhesives because of their liquid status. Wound adhesives may inadvertently spread to other areas. Extreme caution must be exercised when using wound adhesives near the eyes. Incisions in which wound adhesives are used are not rubbed or soaked with any wound cleaner. They may be gently rinsed. Steri-Strips should not be used in conjunction with wound adhesives. Wounds in which a wound adhesive has been used are left uncovered.

Wound Drainage

Often, a drain is inserted in the wound to prevent the pooling of exudate in the wound bed. Pooling of exudate in the wound bed decreases healing and increases the potential of infection or tunneling. The most common types of drains are Hemovac drains, Penrose drains, Jackson-Pratt drains, and chest tubes. Basic care of all drains and chest tubes includes cleansing with sterile normal saline solution and applying a dressing. The dressing stabilizes the drain and prevents the drain insertion site from coming in contact with drainage and other potentially infectious surfaces. Drain and tube insertion sites are never left open to air because of the risk for infection. If drainage from another source may potentially saturate the dressing (over the drain site), the dressing also needs to be occlusive. Drain tubing is stabilized with tape to decrease the potential for inadvertent dislodgment, removal, and pain. Inadvertent removal of a drain potentiates pain and infection, and an acute wound may become a chronic one.

Antibiotic preparations (eg, bacitracin or Neosporin) may be applied, although hydrogen peroxide and povidone-iodine (Betadine) ointment are always avoided because they destroy granulation tissue and prolong the healing process. Normal saline solution causes no damage to the wound bed and is physiologically normal and cost-effective. Some institutions may use prepared wound cleansers. Most wound cleansers have some potential to destroy granulating tissue (compared with normal saline solution) but are less toxic to granulating tissue than hydrogen peroxide or Betadine. Prepared wound cleansers are improving rapidly, becoming less cytotoxic and more time-efficient and cost-effective.

Impregnated gauzes for packing and various solutions (eg, Betadine and Dakin's solution) may be used in the event that the wound is infected; however, they should not be used as a routine wound treatment for a prolonged time because they destroy granulating tissue and inhibit the normal healing process. Remember that the use of these products signals that the wound is not an acute wound but has become a chronic wound.

Wound Dressings

The goal of wound dressings is to protect the wound from infection and promote a moist environment. There are hundreds of dressing products available. The dressing of choice depends on the wound.

Wet-to-Dry Dressings

A wound healing by secondary or tertiary intention is frequently packed with wet-to-dry dressings. The use of wet-to-dry dressings is not recommended. Although wet-to-dry dressings are frequently used in clinical practice, evidence-based practice has shown they are actually detrimental to the wound. Wounds need a moist environment to heal without impediment. Changing a wet-to-dry dressing every 8 or 12 hours leads to the dressing becoming exceptionally dry. Thus, when it is removed, indiscriminate débridement of both necrotic and granulating tissue occurs. This constant débriding of the wound increases the patient's discomfort, promotes infection (caused by frequent dressing changes), slows the healing process, and may enlarge the wound. Wet-to-dry dressings affect not only wound healing but also health care costs by prolonging the healing time and increasing nursing labor and supply expenses.

Calcium Alginates and Foam Dressings

Wound healing by secondary or tertiary intention may also be promoted with calcium alginates or foam dressings (as opposed to wet-to-dry dressings). Calcium alginates are made from brown seaweed. They come in ropelike or flat pieces that must be "fluffed" and packed into the wound bed. Calcium alginates have an absorptive quality and can hold up to 20 times or more their weight in wound drainage. As the calcium alginate absorbs the wound drainage, its appearance changes from dry, fluffed strands to that of a gel that is easily removed from the wound. Calcium alginates may be covered with a hydrocolloid or a transparent dressing.

Foam dressings have the advantage of being highly absorptive. They are available in various shapes and sizes and are placed over the wounds. When it is time to remove the foam dressing, it is simply lifted off the wound. Minimal trauma occurs to the wound bed and surrounding tissue. Foam dressings, like calcium alginates, provide a moist wound environment.

Contraindications to calcium alginates and foam dressings vary according to the manufacturer. Caution should always be used if the wound is infected.

Hydrocolloids

Hydrocolloids are most frequently used in the care and treatment of stages I and II pressure ulcers. Hydrocolloids are occlusive, self-adhesive, and absorptive, although their absorptive capacity is not as great as that of calcium alginates or foam dressings. The advantage of hydrocolloids is that they need changing only every 3 to 5 days or if they are inadvertently removed. Contraindications to hydrocolloids depend on the manufacturer's recommendations. Again, caution is always used if the wound is infected.

Hydrogels

Hydrogels are most frequently used for dry wounds. They help maintain a moist wound environment, promoting granulation, epithelialization, and autolytic débridement. Hydrogels have a water or glycerin base.

Absorptive Wound Dressings

Absorptive dressings vary in construction; they are composed of hydrofibers, cellulose, rayon, or cotton. Absorptive dressings can contain larger amounts of exudates than calcium alginate dressings. Absorptive dressings may be used as primary or secondary dressings.

Silver (Ag) Dressings

Silver dressings are dressings that have been impregnated with silver. They may be topical dressings, such as Acticoat, which has the appearance of a 4″ × 4″ gauze pad and is placed against the wound bed, or hydrofiber dressings, such as Aquacel Ag, which is highly absorptive and can be packed into the wound. Many silver-impregnated dressings may be left in place for prolonged periods, which is advantageous for nursing care and patient teaching.

The silver has a bactericidal effect. Before the advent of antibiotics, silver was the treatment of choice for wound infections. The potential for bacteria to develop a resistance to silver is negligible; to date, only one possible case has been reported. Silver dressings work well in conjunction with other medical and pharmacotherapeutic treatments.

Bilayered Dressings

Bilayered dressings are engineered dressings that are applied as "grafts" to wounds that fail to progress with other treatment plans. These graft materials may be composed of fibroblast, collagen, and growth factors depending on the type and brand. They act by giving the (noninfective) chronic wound a "jump start." Examples of these dressings are Apligraf, Integra, and Oasis. They are frequently used on venous stasis ulcers and diabetic foot ulcers or on exposed bone, tendon, or joints. The cost of these graft materials is much more than conventional treatments; however, by "jump-starting" the process of wound healing, they may actually be more cost-effective in difficult cases.

Wound Débridement

At times, both acute (ie, grafts) and chronic wounds need to be débrided. Débridement is the removal of necrotic (dead) or devitalized tissue. Necrotic or devitalized tissue presents as dark brown, black, yellow, pale, cyanotic, or crusty eschar. To promote optimal wound healing, this tissue needs to be removed from the wound. Débridement creates an "acute wound," and the three phases of wound healing are initiated. Débridement may be performed in several ways: autolytic, chemical, mechanical, or laser. Occasionally, a combination of débridement methods may be used throughout the healing process. Combination therapy depends on the type of wound and its location, the patient's status, and physician preference.

Autolytic Débridement

In autolytic débridement, the body breaks down necrotic or devitalized tissue. Hydrocolloid dressings are frequently used to promote autolytic débridement. Autolytic débridement is not the optimal choice in wounds that have large amounts of necrotic tissue. Autolytic débridement takes time for the body to use its own ability to lyse and dissolve necrotic tissue.

Chemical Débridement

Chemical débridement is accomplished using collagen-based drugs applied topically to the wound. An example of a chemical débridement medication is Collagenase Santyl. Chemical débridement agents dissolve nonviable tissue. Product instructions must be reviewed before use.

Mechanical Débridement

Mechanical débridement can be accomplished by wet-to-dry dressings (which are not recommended), whirlpool procedures, or ultrasound treatment. Although wet-to-dry dressings are an effective method of débridement, care must be taken to change to another method of wound care when the wound bed is débrided. Use of the whirlpool procedure is controversial because although it does débride (but not effectively), the potential for infection is increased with multiple patients using a static number of whirlpools (even though they are cleaned between patients). Use of whirlpool procedures also leaves the wound margins macerated, which increases tissue loss, impeding wound closure. The use of ultrasound treatment for débridement requires more research/evidence-based practice to determine its effectiveness.

Sharps Débridement

In sharps débridement, using a scalpel or scissors, the wound bed is cleared of all necrotic and devitalized tissue surgically. This surgical procedure may require anesthesia, intravenous

conscious sedation, a local anesthetic, or a combination of the three.

Laser Débridement

Laser débridement may also be used to provide a clean wound bed. Currently, laser débridement is not performed as frequently as autolytic, chemical, and mechanical débridement. As technology advances, the use of laser débridement will become more common.

Biosurgical Débridement

Biosurgical débridement is the instillation of sterile larvae (maggots) into a wound. These sterile larvae selectively digest necrotic or devitalized tissue. However, they may not be accepted well by the patient.

Wound Cultures

Routine wound cultures are not recommended unless there are signs and symptoms of infection, such as fever, erythema, edema, induration, foul odor, purulent exudates, increased amount of exudate, abscess, cellulitis, discoloration of granulation tissue, friable granulation tissue (bleeds easily), increased or unexpected pain or tenderness, or an elevated white blood cell count. All wounds are considered contaminated and have the potential to become infected. Several methods may be used to culture a wound, including fluid biopsy, wound (tissue) biopsy, and surface culture (culture swab).

A surface culture is usually done first. The wound is cleansed or irrigated with sterile normal saline solution before swabbing the wound. Exudate and necrotic tissue are not cultured—doing so provides invalid results. After the wound is cleansed, the swab is gently rolled or rotated, starting at the 12-o'clock position and moving in a zigzag pattern from side to side down the wound to the 6-o'clock position.[2] Optimally, there should be 10 points of contact (Fig. 52-7).[2] A colony count of 100,000 organisms/mL indicates an infection that needs to be treated with the appropriate antibiotic.[6] At colony counts of greater than 100,000 organisms/mL, normal wound healing is inhibited, and the wound becomes a chronic wound.[2] Wounds that do not respond to antibiotic treatment need to be recultured. The most appropriate form of culture in this scenario is a wound biopsy. Wounds that contain necrotic tissue or tunneling need both aerobic and anaerobic cultures.

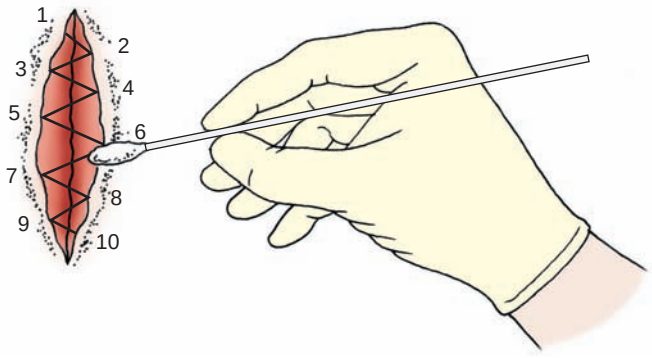


FIGURE 52-7 ▲ Procedure for collecting a wound culture. The wound edges are swabbed using 10-point coverage. (From Thomas Hess C: Clinical Guide: Wound Care, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, p 101.)

Use of Pressure-Relieving Devices

Pressure relief is a major component of wound care. A variety of methods may be used, ranging from low to high technology.[1–3,7] Programs that use both high- and low-technology solutions and adapt to meet the patient's needs at various stages in the recovery process tend to be the most successful.

The easiest and most effective treatment for pressure ulcers on the heels is to keep the heels off the bed by placing a pillow under the lower legs (a low-technology, cost-effective treatment). A schedule for turning and positioning the patient is an effective, easily implemented, and cost-effective intervention for relief of pressure. Critically ill patients may require specialty beds that are designed to reduce pressure. Many specialty beds inflate, deflate, alternate pressures, and laterally rotate. To prevent the occurrence of additional pressure ulcers, it is necessary to follow manufacturer's recommendations when positioning patients on specialty beds. Although these beds do relieve some pressure, they do not eliminate all pressure, as turning the patient from side to side does. Another pressure-relieving device is the Vollman-Turner device, which places the patient in the prone position (thus relieving all pressure on the patient's back). The Vollman-Turner device is not a specialty bed; rather, it is a device that is attached to the bed frame. The advantage of the Vollman-Turner device is that only a minimal number of people are needed to turn the patient.

Pain Management

In all areas of wound care (assessment, cleansing, dressing changes, and positioning), the nurse needs to focus on pain assessment and control. No procedure should occur without assessing for pain and then medicating the patient as needed. Once the pain is controlled, the nurse may proceed with the wound care. The choice of pain medication and the delivery method used (eg, continuous drip, epidural, patient-controlled analgesia pump, local anesthesia) depends on the patient's status.

Pharmacotherapy

Pharmacotherapy in wound care entails the use of pain medications and, in some cases, growth hormones and steroids. Pain medications are used to control pain during wound assessment, cleansing, and dressing changes. Growth hormones, for example, becaplermin (Regranex Gel 0.01%), may be used to stimulate wound healing. Regranex is applied topically to the wound in measured doses that are recalculated weekly or biweekly. The gel is spread evenly over the wound and covered with gauze moistened with saline solution. Topical steroid creams, such as clocortolone pivalate (Cloderm) and doxepin hydrochloride (Prudoxin), may be prescribed for wound care to relieve surface

inflammation and pruritus of the wound margins. Chemical débridement agents (discussed earlier) are also considered pharmacotherapy.

Although silver-impregnated dressings are referred to as antimicrobials, they are considered a dressing and not a pharmacotherapeutic agent. Silver gels such as SilvaSorb can be used instead of a silver-impregnated dressing. SilvaSorb gel is a combination of silver and a hydrogel applied into the wound and is a controlled-release formula.

Xenaderm is a protective ointment composed of balsam Peru, castor oil, and trypsin. This ointment increases blood flow to partial-thickness wounds while acting as a barrier in incontinent patients.

▲ Care of Specific Wounds

Pressure Ulcers

Pressure ulcer treatment depends on the stage of the wound. Stage I and II pressure ulcers are usually treated with hydrocolloid dressings. Stage III and IV pressure ulcers may be dressed using absorptive hydrofiber dressings in the wound bed or with calcium alginates fluffed and placed into the wound bed and then covered with hydrocolloid or occlusive transparent dressings. Although they are used, wet-to-dry dressings are not optimal, as previously discussed. Other options for stage III and IV pressure ulcers are foam dressings and the VAC system.

Burns

Burns are acute wounds, graded as first, second, or third degree and described as partial or full thickness. Wound care goals in burns are a clean wound, free from infection. Burns are cleaned with sterile normal saline solution or a mild soap and water. Topical ointments, such as bacitracin, polymyxin, or silver sulfadiazine, may be applied. After cleansing the wound, a dressing is applied. The type of dressing depends on the type of burn, the amount of tissue involved, institutional policy, and physician preference. Care of the patient with burns is discussed thoroughly in Chapter 53.

High-Volume Draining Wounds

Some wounds may have high volumes of exudate (drainage). Exudate is the response of the body to the inflammatory phase. Wound drainage is composed of neutrophils, macrophages, cellular debris, proteins, and toxins. High-volume draining wounds generate more drainage than traditional gauze pad dressings can manage, in which case hydrocolloids, calcium alginates, hydrogels, or foam dressings may be used. A composite dressing may also be used. These dressings combine the physical attributes of two or more dressings to enhance the absorptive capability.

If the exudate cannot be controlled with these measures, alternatives must be considered. The goal of wound care in this instance is to contain the drainage and protect the surrounding tissue from breakdown. Frequently, the wound maybe "pouched" or "bagged." The same supplies used for ostomy pouching are used for pouching a wound, or a product designed specifically for pouching high-volume draining wounds may be used. Pouching the high-volume draining wound allows for accurate measurement of output from the wound and protects the surrounding wound margins.

To pouch a wound, the skin is first cleaned with saline solution or an antibacterial soap and water, then dried. The skin may then be prepared with a protective skin barrier wipe, which protects the skin and enhances the adherence of the wafer. The wound is measured or traced, and a wafer is cut to fit. Stoma paste is applied around the cut-out area to prevent leakage of the drainage onto the skin. A one- or two-piece pouching system may be used. With a one-piece pouching system, the wafer with pouch is applied to the wound. With a two-piece system, the wafer is applied first, and then the pouch is attached. Both systems need a closure device at the end of the pouch. A benefit of a two-piece system is that it allows for pouch removal so that the wound can be assessed without disturbing the wafer. The wound margins need to be assessed for skin breakdown when the system is changed and may be protected by a variety of skin wipes or protective ointments.

▲ Nutrition and Wound Healing

In critical care, monitoring nutritional status is as important as monitoring hemodynamics. Nutrition needs to be addressed early in the patient's admission to promote the optimal opportunity and environment for healing. One study showed that patients who receive a nutritional assessment within the first 48 hours of admission to the hospital setting have a lower incidence of pressure ulcer development during their hospital stay.[8] Nutrition is paramount in critically ill patients or patients with wounds, whether the wounds are acute or chronic. To heal properly, the body needs adequate carbohydrates, fats, proteins, minerals, calories, vitamins, and hydration (Table 52-2).[1,2,8]

Protein is a basic and key component of all cellular activity. Without proteins, the inflammatory process is impaired, and the risk for infection increases. Proteins also affect oncotic pressure, which predisposes the patient to edema. Wound edema decreases the diffusion of oxygen and nutrients, impeding the healing process even more.

Although protein is a key component in the healing process, other nutrients play major roles. Carbohydrates are the body's fuel source and spare the proteins, so they can be used in cellular construction. Fats maintain cell membrane function and assist with the movement of minerals and fat-soluble vitamins in and out of the cell. Vitamins act as catalysts in the body's chemical reactions and are also needed for protein and cellular replication. Minerals are needed in the body's biochemical reactions and control the movement of fluids into and out of the cell, through the process of osmosis.

An adequate caloric intake is required for a wound to heal. Normal adult caloric intake is 25 to 30–40 kcal/kg/d, and the normal adult protein intake is 0.8 g/kg/d.[1,2] In a critically ill or critically injured patient, caloric and protein intakes must be dramatically increased. Caloric intake requirements

Table 52-2 Necessary Nutrients for Wound Healing

Nutrient	Function	Results of Deficiency
Proteins	• Wound repair • Clotting factor production • White blood cell production and migration • Cell-mediated phagocytosis • Fibroblast proliferation • Neovascularization • Collagen synthesis • Epithelial cell proliferation • Wound remodeling	• Poor wound healing • Hypoalbuminemia and generalized edema, which slows oxygen diffusion and metabolic transport mechanisms from the capillaries and cell membranes • Lymphopenia • Impaired cellular immunity
Carbohydrates	• Supply cellular energy • Spare protein	• Body uses visceral and muscle proteins for energy
Fats	• Supply cellular energy • Supply essential fatty acids • Cell membrane structure • Prostaglandin production	• Inhibited tissue repair • Use of visceral and muscle proteins for energy
Vitamin A	• Collagen synthesis • Epithelialization	• Poor wound healing • Impaired immunity
Vitamin C	• Membrane integrity • Antioxidant	• Impaired immunity • Poor wound healing • Capillary fragility
Vitamin K	• Normal blood clotting	• Increased risk for hemorrhage and hematoma formation
Iron	• Collagen synthesis • Enhances leukocytic bacterial activity • Hemoglobin synthesis	• Anemia, leading to increased risk for local tissue ischemia • Impaired tensile strength
Zinc	• Cell proliferation • Cofactor for enzymes • Vitamin A utilization	• Impaired collagen cross-linkage • Slow healing • Alteration in taste • Anorexia • Impaired immunity
Copper	• Collagen cross-linkage • Red blood cell synthesis	• Decreased collagen synthesis • Anemia
Pyridoxine, riboflavin, and thiamine	• Energy production • Cellular immunity • Red blood cell synthesis	• Decreased resistance to infection • Impaired wound healing
Arginine	• Increases local wound immune system • Nitrogen-rich (32% nitrogen, whereas the average amino acid is 16% nitrogen) • Precursor to proline, which is converted to hydroxyproline and then to collagen	• Decreased local wound immune system
Glutamine	• Primary fuel for fibroblasts • Preservation of lean body mass	• Less fuel for fibroblasts

From Hess CT: Clinical Guide: Wound Care, 5th ed. Ambler, PA: Lippincott Williams & Wilkins, 2005, p 28.

increase to 35 to 40 kcal/kg/d, and protein requirements increase to 1.5 to 2 g/kg/d (Table 52-3).[2] Optimal nutritional care for the patient with wounds can be achieved by consulting a dietitian and monitoring laboratory test results along with the patient's basic intake, output, daily weights, anthropometrics, calorie count, and social history.

Serum albumin and prealbumin levels are of particular interest in that they are a key indicator of protein available for cellular construction and replication. Table 52-4 demonstrates the various albumin requirements at specific ages.[6] In each case, if the albumin level is less than the minimal parameter, replacement therapy is needed to provide the optimal wound-healing environment. Normal serum albumin is defined as 3.8 to 5 g/dL. In an adult, a serum albumin level of less than 3.5 g/dL necessitates replacement therapy.

Serum total protein levels are also monitored. Normal levels for serum total protein are 6 to 8 g/dL (see Table 52-4).[6] As stated earlier, protein also affects the oncotic pressure, and levels less than 6 g/dL lead to edema. Note that the serum total protein level, like the albumin level, varies according to age.

Table 52-3 Nutrient Needs Based on Body Weight

Nutrient	Requirements
Calories	
Normal	25–30 kcal/kg/d
Protein–calorie malnutrition (PCM)*	30–35 kcal/kg/d
Critically ill or injured*	35–40 kcal/kg/d
Protein	
Recommended daily allowance (RDA)	0.8 g/kg/d
PCM	1.5 g/kg/d
Critically ill or injured*	1.5–2.0 g/kg/d
Fat	<30% kcal
Water	30 mL/kg body weight or 1 L/1,000 kcal

*Nutrient supplementation required.

From Hess CT: Clinical Guide: Wound Care, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008, p 31.

Along with electrolytes, complete blood count (CBC), serum albumin, and serum total protein, two other laboratory tests that may be assessed are serum transferrin and the total lymphocyte count (TLC). The serum transferrin level is an indicator of the body's ability to transfer iron through the plasma. The normal serum transferrin level is 180 to 260 mg/dL.[6,7] Decreased serum transferrin levels lead to anemia, as demonstrated by the CBC. TLC normal parameters are 1,500 to 3,000 cells/mcL.[6] A TLC assists in the assessment of the patient's immune status and may be decreased in states of malnutrition.

Micronutrients, vitamins, and minerals also affect wound healing. Zinc is needed in the structure of collagen and production of protein. Ascorbic acid is also a component of collagen synthesis. Vitamin A plays a role in cellular proliferation and increases the tensile strength of healing wound tissue. Therefore, use of a high-potency daily multivitamin and mineral supplement is recommended for patients with altered skin integrity.[8]

Patients who are on nothing by mouth (NPO) status for longer than 24 to 48 hours are at risk for slowed healing owing to the lack of an adequate supply of protein, carbohydrates, and other nutrients. Nutritional management includes monitoring laboratory test results; documenting intake and output and daily weights; having a nutritional assessment performed by a dietitian; feeding by total parental or peripheral parental nutrition or enteral means; and making calorie counts.

Adequate hydration is paramount to ensure oxygen delivery to the tissues. If the patient is hypovolemic, oxygen transport to the peripheral tissues is impaired. The optimal goal is to maintain hemodynamic stability. (For a thorough discussion of hemodynamic assessment, see Chapter 17.) In the critically ill patient, tissue perfusion must be addressed based on the symptom and cause. For example, if the cardiac output is decreased, systolic blood pressure is decreased, the heart rate is tachycardic, and the pulmonary artery wedge pressure is decreased, the patient is hypovolemic. To improve tissue perfusion for this critically ill patient, fluids are given. The hemoglobin and hematocrit must also be assessed, and if values are low, the patient should receive a blood transfusion. By improving hydration and correcting the anemia, the circulating volume and the oxygen-carrying capacity of the blood are increased, thus improving tissue perfusion. This enhances the environment for wound healing.

▲ Patient Teaching and Discharge Planning

Patient teaching and discharge planning are ongoing processes that occur throughout the patient's hospital stay. Discharge planning for patients with wounds is a multidisciplinary challenge. An important part of discharge planning is ensuring that the patient or a family member knows how to care for the wound after the patient leaves the hospital. Examples of patient teaching guides for wound care are given in Boxes 52-5 and 52-6.

Table 52-4 Normal Values for Serum Total Protein and Serum Albumin

Age of Patient	Total Protein	Albumin
Adult	6.0–8.0 g/dL or 60–80 g/L	3.5–5.0 g/dL or 38–50 g/L
10–19 y	6.3–8.6 g/dL or 68–86 g/L	3.7–5.6 g/dL or 37–56 g/L
7–9 y	6.2–8.1 g/dL or 62–81 g/L	3.7–5.6 g/dL or 37–56 g/L
4–6 y	5.9–7.8 g/dL or 59–78 g/L	3.5–5.2 g/dL or 35–52 g/L
1–3 y	5.9–7.0 g/dL or 59–70 g/L	3.4–4.2 g/dL or 34–42 g/L
<5 d	5.4–7.0 g/dL or 54–70 g/L	2.6–3.6 g/dL or 26–36 g/L

From Fischbach FT: A Manual of Laboratory and Diagnostic Tests, 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2004, p 576.

BOX 52-5 TEACHING GUIDE Wound Care (Sutures, Staples, and Wound Adhesives)

Wound Closure
- Sutures are thread-like. They are placed using a needle to pull the skin together. You may see "knots."
- Staples are special surgical staples. They are placed using a special staple gun to pull the skin together.
- Wound adhesives are a type of glue that holds the edges of the skin together.

Patient Activity
- Keep your sutures/staples/wound adhesive clean and dry.
- Wash your hands before you start.
- Do not rub or pull the area.
- Clean the wound gently with mild soap and water and rinse, or use a wound cleanser as directed by your physician. Do not "soak" the sutures/staples/wound adhesive. Gently pat the area dry.
- A dry gauze dressing may be applied to keep your wound clean or pad the area if your clothes rub.
- Wash your hands when you are done.

When To Call Your Physician
- Call if you find any redness, tenderness, pus-type drainage, swelling, missing sutures or staples, or increased pain, or if the area is warm or hot to the touch.
- Call if you have a fever of more than 101°F.
- Call immediately and go to the emergency department if your incision "pulls open."

Medications
- Take your medications as prescribed.
- If you are taking an antibiotic, take all pills prescribed. Do not stop taking them when you feel better.

Safety
- You may drive, climb stairs, work, begin sexual activity, and lift when instructed by the physician.

BOX 52-6 TEACHING GUIDE Wound Care (Dry Dressing, Calcium Alginate, Hydrocolloid, or Hydrofiber)

Patient Activity
Always wash your hands before and after changing your dressing.

Dry Dressing
- Change your dressing every day.
- Clean your wound with normal saline solution, mild soap and water, or a wound cleanser as directed.
- Cover the wound with a dry gauze dressing.

Calcium Alginate
Calcium alginate is a dressing that can be packed (lightly) into your wound. It is made of a special type of seaweed that has healing properties. Calcium alginate looks similar to "angel hair" when "fluffed."

- Change your dressing every 3 days unless otherwise directed by your physician.
- Remove the old dressing (it will appear to be a gelatinous mass, not the fibrous "angel hair" you put in.)
- Clean your wound with normal saline solution or with a wound cleanser as directed.
- Take the calcium alginate out of the package and gently fluff it (pull it apart slightly so that it has a light, fluffy look).
- Place the fluffed calcium alginate into the wound.
- Cover the wound and calcium alginate with the type of covering you were directed to use.

Hydrocolloid
Hydrocolloid is a thicker type of wound covering that can be placed over open wounds, such as bedsores (pressure ulcers). Hydrocolloid may also be placed over a wound in which calcium alginate has been packed. Hydrocolloid comes in various shapes and sizes. It has an adhesive (sticky) side. The adhesive side goes over the wound.

- Change your dressing every 3 to 7 days unless otherwise directed by your physician, or when it comes off.
- Gently peel the old hydrocolloid off, being careful not to peel too quickly or too roughly.
- Clean your wound with normal saline or with a wound cleanser as directed.
- If you are packing the wound with calcium alginate, do it at this time.
- Peel the paper off the adhesive side of the hydrocolloid.
- Place the hydrocolloid adhesive side down over the wound.
- Press gently and smooth the hydrocolloid over the wound.
- Place your hand on top of the hydrocolloid for about 1 minute. This helps the adhesive to stick better.

Hydrofiber
Hydrofiber is a dressing that is absorbent, interacting with wound drainage by forming a soft gel that is easily removed from the wound while maintaining a healing environment. Hydrofiber may or may not have silver added to the dressing. Silver has an antimicrobial effect that helps to keep the wound free of bacteria.

- Change your dressing when it is saturated with drainage, or after 7 days.
- Remove the old dressing (it will appear to be a gelatinous mass).
- Clean your wound with normal saline solution or with a wound cleanser as directed.
- Take the hydrofiber out of the package and place it in the wound.
- Cover the wound and hydrofiber with the type of covering you were directed to use.

When to Call Your Physician
- Call if you have any redness, tenderness, pus-type drainage, swelling, missing sutures or staples, or increased pain, or if the area is warm or hot to the touch.
- Call if you have a temperature higher than 101°F.

Medications
- Take your medications as prescribed.
- If you are taking an antibiotic, take all pills prescribed. *Do not stop taking them* when you feel better.

Safety
- You may drive, climb stairs, work, begin sexual activity, and lift when instructed by the physician.

▲ Clinical Applicability Challenges

CASE STUDY

Mr. B. is a 75-year-old Japanese American with a history of diabetes mellitus, chronic obstructive pulmonary disease, hypertension, and smoking. He was active and still working 4 days per week when he suffered a cerebral vascular accident. He was found lying on the kitchen floor; estimated time down was 24 hours or slightly more. He is intubated on FIO_2 (fraction of inspired oxygen) 70%, tidal volume of 700 ml, assist control of 16, and pressure support of 5 cm H_2O. He is nonresponsive, except to pain, and no movement on his right side is noted. Vital signs are blood pressure 140/82 mm Hg; temperature 100°F; heart rate 110 bpm; and respiratory rate 16 bpm (not assisting the ventilator, in that the patient is unable to initiate or attempt any spontaneous triggering of the ventilator). Glascow Coma Scale is 9. He has an 8-cm maroon-purple area on his right hip, a blood-filled blister on the right heel, and a fluid-filled blister on the left heel present upon admission to the intensive care unit.

1. Compare and contrast the fluid-filled blister and the blood-filled blister.
2. Discuss the staging/classification of the right hip wound.
3. Explain the importance of "present on admission."

References

1. Baranoski S, Ayello EA: Wound Care Essentials: Practice Principles, 2nd ed. Philadelphia, PA: Lippincott, Williams & Wilkins, 2008
2. Thomas Hess C: Clinical Guide: Wound Care, 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2007
3. National Pressure Ulcer Advisory Panel & European Pressure Ulcer Advisory Panel 2009: Pressure Ulcer Prevention Recommendations, 21–23, 2009
4. Carpenito-Moyet LJ: Handbook of Nursing Diagnosis, 13th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010
5. KCI Licensing, Inc., V.A.C.® Therapy Clinical Guidelines, May 2007
6. Fischbach FT, Dunning MB III: Nurse's Quick Reference to Common Laboratory and Diagnostic Tests, 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2006
7. Junkin J: Failure to thrive in wounds: Prevention and early intervention. Infect Control 1(2):1–8, 2002
8. Stefanski JL, Smith KJ: The role of nutrition intervention in wound healing. Home Health Care Manag Pract 18(4):293–299, 2006

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

53 Burns and Common Integumentary Disorders

Louis R. Stout and Lisa M. Johnson

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Discuss the classifications of a burn injury.
2. Describe the pathophysiology of a burn injury.
3. Review the physiological changes associated with each organ system in relation to a burn injury.
4. Discuss the initial priorities of caring for a patient with a burn injury.
5. Formulate a plan of care for a patient who has sustained a burn injury.
6. Discuss other types of injured patients who are cared for in the burn unit.

The past two decades have witnessed a significant decline in the number of burn injuries, hospitalizations, and deaths. Total burn injuries in the United States have decreased more than 50%, from 2.5 million to approximately 1 million per year. Burn injuries account for 500,000 emergency department (ED) visits per year, with about 40,000 patients being hospitalized; nearly 70% of burn patients are men.[1] The average size of a burn is about 10% of the total body surface area (TBSA).[2]

Of the patients with burns who are hospitalized, about half are treated at specialized burn centers.[1] Burn treatment centers are composed of nurses, physicians, physical therapists, occupational therapists, recreational therapists, nutritionists, psychologists, social workers, and spiritual support staff. The American Burn Association (ABA) has established guidelines for transfer and referral of these patients to burn centers.

Great strides have been made in the technological and pharmacological care of the burned patient. In the 1940s and 1950s, a patient with a 30% to 40% TBSA burn had a 50% chance of survival (LD_{50}).[3] Over the decades, survivability increased as the result of penicillin and broad-spectrum antibiotics, the burn center concept, and aggressive nutrition and excision. Today, a patient with a 78% TBSA burn has a 50% chance of survival. The trend toward outpatient management has contributed to the decreased number of hospitalized patients. Acute hospitalization resulting from a burn injury has declined 50% since the 1970s. From 1995 to 2005, the average length of stay for inpatient hospitalization declined from 13 to 8 days, and the mortality rate decreased from 6.2% to 4.7%.[1,2] Most recent mortality rates have declined further to 3.7%.[2]

Despite the dramatic reductions in incidence, an acute burn injury remains the third leading cause of death in children between the ages of 1 and 9 years, although it has decreased to the sixth leading cause of death in the remainder of the population. The advocacy of several national organizations has helped introduce increased fire-resistant products, fire prevention programs, and legislation (eg, flame-retardant children's sleepwear, smoke alarms, fire suppression systems).[4] Nevertheless, there are an estimated 4,000 fire and associated burn deaths per year.[1,2] Measures for safely preventing burns are given in Box 53-1.

▲ Classification of Burn Injuries

Burn injuries are described in terms of causative agent, depth, and severity.

Causative Agent

A burn injury usually results from energy transfer from a heat source to the body. The heat source may be thermal, chemical, electrical, or radiation producing. Skin is a great resistor; injury occurs when the agent exceeds the threshold for resistance.

Thermal Burns

Thermal burns account for 70% of all burn injuries.[2] They may be caused by a flame source such as a house fire, a cooking accident, or a fiery explosion. Scald burns from steam or contact with a hot object, such as a cooking pan or hot steel, may also cause thermal injury.

Chemical Burns

Chemical injuries are commonly encountered after exposure to acids and alkali, including hydrofluoric acid, formic acid, anhydrous ammonia, and organic compounds. Other specific chemical agents that cause chemical burns include

BOX 53-1 TEACHING GUIDE Preventing Burns

Prevent Accidents in the Home

- Install a smoke detector on each floor of your house; change the batteries twice a year.
- Plan an exit route in your house in the event of a fire and have routine fire drills once a month.
- Exercise caution with cooking. Avoid wearing clothes with sleeves that may dangle and accidentally ignite clothing.
- Exercise caution with foods that are cooked in a microwave.
- Keep pot and skillet handles turned inward on the stove.
- Never allow children to stand on an open oven door, as this may cause the entire stove to collapse.
- Never leave children unattended in a bathtub.
- Set your hot water heater no higher than 120°F.
- Never leave candles unattended and always be sure candles are fully extinguished.
- Have your furnace serviced once a year.
- Install a carbon monoxide detector.
- Never use the oven or barbeque grill as a heating source.

Prevent Accidents Outside the Home

- Only a responsible adult should handle fireworks. Never leave fireworks out where children can have access.
- Exercise caution with campfires and grills.
- Do not pour accelerant (gasoline, lighter fluid) on a lighted fire.
- If an electrical wire is found in a tree, do not touch! Call the local electric company and police/fire company as soon as possible.
- Use sunscreens! Choose a sunscreen with ultraviolet A and ultraviolet B protection and a sun protection factor of 30. Apply every 2 to 3 hours.

Should a Burn Occur

- Stop the burning process by removing the source (refer to Box 53-2 for immediate treatment based on burn depth and size).

white phosphorus, certain elemental metals, nitrates, and hydrocarbons.

Contact time is a critical element in determining the severity of injury. Removal of contaminated clothing and water irrigation is crucial to limit the effects of the chemical. Regardless of the causative agent, the irrigation must continue once the patient arrives at the ED. Forego identification of a specific agent to neutralize the acid or alkali agents because a neutralizing agent will also cause a chemical reaction in the neutralizing process thereby risking further burn injury. For all chemical burns, hydrotherapy treatment should continue until the pain resolves, taking 1 to 3 hours or longer. Chemicals to the eyes should be flushed continuously until a full evaluation can be completed by an ophthalmologist. Some agents such as hydrofluoric acid require specific treatments to stop tissue destruction. Calcium chloride topical gels or dermal injections are used.[5,6]

Electrical Burns

The effects of electricity on the body are determined by the type of current (alternating or direct), the pathway of the current, the duration of contact, the resistance of the body tissue, and the amount of voltage. Because of their highly developed nervous system, humans are sensitive to very small electric currents. Electricity travels the path of least resistance; therefore, tissue, nerves, and muscle are easily damaged, whereas bone is not.

Low-voltage injuries are considered to be caused by 1,000 V or less. Low-voltage injuries tend to occur at home and involve the hands and oral cavities. The most common cause of low-voltage electric burns of the hand is contact with an extension cord in which the insulating material has worn off, either from wear or misuse. A low-voltage burn of the hand usually consists of a small, deep burn that may involve vessels, tendons, and nerves. Although these burns involve a small area of the hand, they may be severe enough to require amputation of a finger. Low-voltage electricity can also damage the oral cavity, leaving a permanent scar. These injuries occur most frequently in children between the ages of 1 and 2 years.[6] Most are caused by sucking on, or biting, an extension cord socket. Whereas low-voltage current usually follows the path of least resistance (nerves, blood vessels), high-voltage current takes a direct path between entrance and ground. Current is concentrated at its entrance to the body, then diverges centrally, and finally converges before exiting. Unfortunately, the most severe damage to tissue occurs at the sites of contact, which are commonly referred to as entrance and exit wounds. High-voltage electric entry wounds are charred, centrally depressed, leathery in appearance, and local muscle flexion (ie, contact with the hand is likely to present with the hand and forearm in a fixed, almost fully flexed position, often described as a "claw-like" position). Exit wounds are more likely to "explode" as the charge exits. They are usually caused by lightning.

Depth

Many factors alter the response of body tissues to heat. The degree or depth of burn depends on (1) the temperature of the injuring agent, (2) the duration of exposure to the injuring agent, and (3) the areas of the body that are exposed to the injuring agent. Whereas the body can sustain prolonged exposure to moderate temperatures such as a hot tub of water (110°F or 43°C), significant damage can occur in as little as 1 second when the temperature exceeds 150°F (68°C). Hot water heaters are often installed with the setting at 140°F (60°C).[1,5,6] A safer setting would be 120°F (49°C) especially in homes with children or elderly family members. Damage to the skin is frequently described according to the depth of injury and is defined in terms of superficial, partial-thickness, and full-thickness injuries, which correspond to the various layers of the skin (Table 53-1; Fig. 53-1).

Superficial Burns

Superficial burn injuries are commonly known as first-degree burns. Superficial burns affect the epidermal layer and heal with minimal intervention. Sunburn is a familiar example of a first-degree superficial burn injury; others may be very brief exposure to hot liquid, flash, flame, or chemical agent. The burned skin is painful at first and later itches because of the stimulation

Table 53-1 Characteristics of Burns of Various Depths

Depth	Tissues Involved	Usual Cause	Characteristics	Pain	Healing
Superficial (first-degree)	Epidermis	Sun Scalds	Dry Pinkish red Blanches Typically don't blister	Painful	About 3–5 days No scarring
Superficial partial-thickness (second-degree)	Epidermis into papillary dermis	Flash Scalds	Moist, blisters Pinkish to redden Blanches	Hypersensitive Very painful	2–3 weeks Minimal scarring
Deep partial-thickness (second-degree)	Epidermis into reticular dermis (epidermal-lined hair and sweat glands intact)	Above plus hot solids, flame, and intense radiant injury, chemical	Slightly moist to dry Mottled, pink and white Slow to no blanching	Discomfort to pressure	3 or more weeks Late hypertrophic scarring; marked contracture formation
Full-thickness (third-degree)	Entire dermis into subcutaneous fat; may involve connective tissue, muscle, bone	Sustained flame, electrical, chemical, and steam	Dry, leathery, cracked White, cherry red, or black, thrombosed vessels No blanching	Typically no pain, pressure with palpation	Cannot self-regenerate; needs grafting

of sensory receptors. Because of the continuous replacement of epidermal epithelial cells, this type of injury heals spontaneously without scarring in 3 to 5 days. Care of superficial burns is minimally supportive and is summarized in Box 53-2.

Partial-Thickness Burns

Partial-thickness burns (second-degree burns) are differentiated into superficial and deep partial-thickness burns. Superficial partial-thickness burns affect the epidermal and superficial dermal layers and usually heal with minimal intervention in 10 to 14 days (see Box 53-2). Deep partial-thickness burns affect the entire epidermal layer and more deep dermal layers. Fluid resuscitation, nutritional status, and premorbid conditions may affect the healing potential of a deep partial-thickness burn injury. Deep partial-thickness burn injuries may have some spontaneous healing in 3 to 4 weeks but require surgical intervention (excision and skin grafting) if the burn is of any significant size. Delayed healing may result in scarring and loss of function.

Full-Thickness Burns

Prolonged exposure to flame, a hot object, a chemical agent or contact with high-voltage electricity can result in full-thickness burns (third-degree burns). These burns extend into the poorly vascularized adipose tissue. All epidermal and dermal elements, sweat glands and hair follicles, are

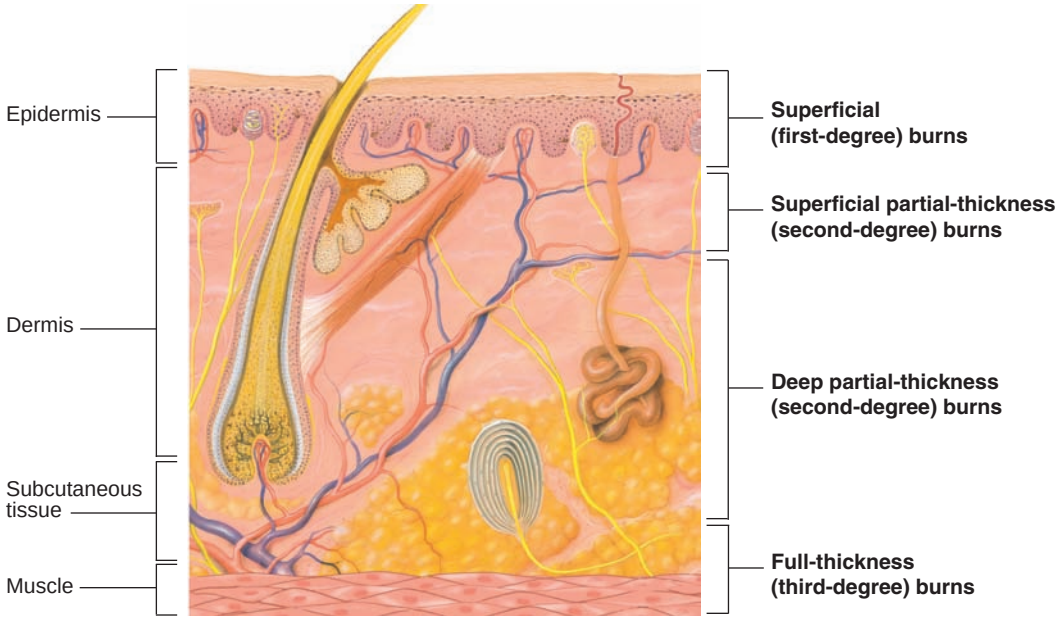


FIGURE 53-1 ▲ Classification of burns by depth of injury. (Adapted from Anatomical Chart Company: Atlas of Pathophysiology. Springhouse, PA: Springhouse, 2010, p 385.)

BOX 53-2 Care of Superficial (First-Degree) Burns and Superficial Partial-Thickness (Second-Degree) Burns

Superficial (First-Degree) Burns

- Apply ice packs or cold compresses.
- No dressing is required.
- Aloe gel with lidocaine can be applied topically as necessary for localized relief.
- Acetaminophen, aspirin, or ibuprofen can be taken as necessary for generalized discomfort.

Superficial Partial-Thickness (Second-Degree) Burns

- If the skin or blister is broken, wash the area with water and mild antiseptic soap.
- Apply a layer of silver sulfadiazine or bacitracin.
- Apply a layer of nonadherent gauze and secure with a gauze roll.
- Dressings should be changed twice a day.
- Wrap fingers and toes individually to prevent "webbing" of healing granulation tissue.
- The patient may continue his or her usual activity depending on the burned site.
- Dependent extremities should be elevated above the level of the heart to prevent excessive edema and promote venous return.
- The patient should be aware of signs and symptoms of infection, including fever, marked tenderness and erythema surrounding the burn wound, purulent drainage of pus, red streaks radiating from the wound, or pain that cannot be controlled with analgesics.
- The patient should follow up in 2 days with a primary care provider.

destroyed. These burns may appear white, red, brown, or black. Reddened areas do not blanch in response to pressure because the underlying blood supply has been interrupted. Thrombosed blood vessels and capillaries may be visualized.[6,7] These burns are insensate because the sensory receptors have been completely destroyed, and the patient may feel deep pressure only. In addition, the burns may appear sunken because of the destruction of underlying fat and muscle.

The loss of the hair follicle eliminates the ability of the skin to regenerate. A small wound (<4 cm) may be allowed to heal by granulation and migration of healthy epithelium from the wound margins. However, extensive, open full-thickness wounds leave the patient highly susceptible to overwhelming infection and malnutrition. Wound closure by skin grafting restores the integrity of the skin.

Severity

Burn severity is determined by the extent and depth of the burn and the causative agent, time, and circumstances surrounding the burn injury. To assess the severity of the burn, several factors must be considered:

- The percentage of body surface area burned
- The depth of the burn
- The anatomical location of the burn
- The person's age (Box 53-3)
- The person's medical history
- The presence of concomitant injury
- The presence of inhalation injury

BOX 53-3 CONSIDERATIONS FOR THE OLDER PATIENT

Burns

Older patients respond differently to burn injuries because of age-related changes and diminished physiological reserve. Preexisting medical conditions and complications as a result of injury are significant factors leading to mortality of the older burn patient. The adverse effects of trauma, including burns, can persist for an extended period after injury. Once injured, elderly patients may never regain their preinjury level of health. Postdischarge destination and care are challenging obstacles in discharge planning. Family interest in assuming caregiver responsibility, ability of the patient to render self-care, insurance, and financial limitations must be considered in planning for discharge. Many independent elderly patients will no longer be able to return home alone after a burn injury. Rehabilitation and long-term care facilities can create an emotional and financial burden on the family. In addition, acute rehabilitation requires the patient to participate in 3 hours of therapy each day. Many elderly patients are unable to meet this requirement, and thus may not be eligible for acute rehabilitation.

Several methods using percentages of TBSA may be used to estimate the extent of a burn. The "rule of nines" or "rule of palms" allows for quick estimation until a detailed Lund and Browder assessment can be done. The rule of nines divides the body into parts in multiples of 9% (see Fig. 53-2). When estimating burn size, burns may involve only one surface of a body part or they may be circumferential. For example, if only the anterior surface of the arm is burned, then the TBSA is estimated to be 4.5%. However, if the burn circles the entire arm, then the value is 9%. The rule of palms can be used for estimating small scattered burns (eg, scald or grease burns). The patient's palmar surface (including the fingers) equals 1% of the patient's TBSA.

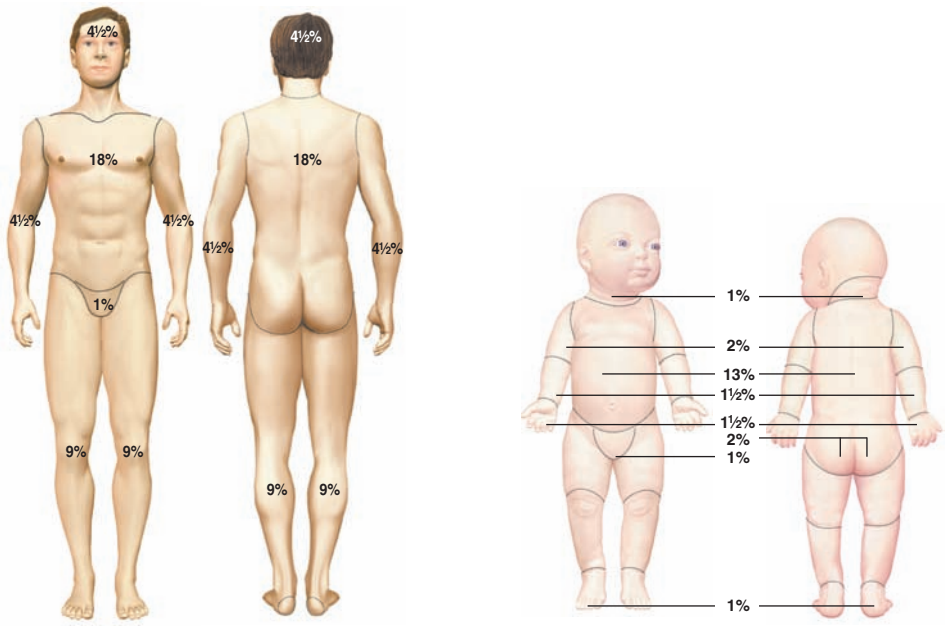
The Lund and Browder method (see Fig. 53-2) is highly recommended because it corrects for the large head/body ratio of infants and children. Surface measurements are assigned to each body part in terms of the age of the patient. However, because this method of measuring burn size is time consuming, it should be done after resuscitation efforts are well established.

A burn injury may range from a small blister to a massive full-thickness burn. Recognizing the need for a clear description of terms, the ABA developed the Injury Severity Grading System, which is used to determine the magnitude of the burn injury and to provide optimal criteria for hospital resources for patient care. The severity of burn injury is categorized into minor, moderate, and major, as outlined in Box 53-4. Minor burn injuries can be treated in the ED with outpatient follow-up every 48 hours, until the risk for infection is reduced and wound healing is underway. Patients with moderate, uncomplicated burn injuries or major burn injuries should be referred to a regional burn center and, if appropriate, transferred for specialized care.

▲ Pathophysiology

Localized Tissue Response

Cellular injury starts when tissues are exposed to an energy source (thermal, chemical, electrical, or radiation). The depth of the thermal injury is demonstrated by the extent of injury



A. Rule of Nines

AREA	PERCENT OF BURN					SEVERITY OF BURN		TOTAL PERCENT
	0-1 Year	1-4 Years	5-9 Years	10-15 Years	Adult	2	3	
Head	19	17	13	10	7			
Neck	2	2	2	2	2			
Ant. Trunk	13	13	13	13	13			
Post. Trunk	13	13	13	13	13			
R. Buttock	2½	2½	2½	2½	2½			
L. Buttock	2½	2½	2½	2½	2½			
Genitalia	1	1	1	1	1			
R. U. Arm	4	4	4	4	4			
L. U. Arm	4	4	4	4	4			
R. L. Arm	3	3	3	3	3			
L. L. Arm	3	3	3	3	3			
R. Hand	2½	2½	2½	2½	2½			
L. Hand	2½	2½	2½	2½	2½			
R. Thigh	5½	6½	8½	8½	9½			
L. Thigh	5½	6½	8½	8½	9½			
R. Leg	5	5	5½	6	7			
L. Leg	5	5	5½	6	7			
R. Foot	3½	3½	3½	3½	3½			
L. Foot	3½	3½	3½	3½	3½			
Total	**Blue areas indicate 2 Red areas indicate 3**			**Total**				

B. Lund and Browder chart

FIGURE 53-2 ▲ **A:** The "rule of nines" method for determining percentage of body area with burn injury. **B:** Lund and Browder method for determining percentage of body area with burn injury. (**A,** Adapted from Anatomical Chart Company: Atlas of Pathophysiology. Springhouse, PA: Springhouse, 2010, p 385.)

BOX 53-4 Classification of Severity of Burn Injury

Minor Burn Injury
- Second-degree burn of less than 15% TBSA burn in adults or less than 10% TBSA in children
- Third-degree burn of less than 2% TBSA not involving special care areas (eyes, ears, face, hands, feet, perineum, joints)
- Excludes all patients with electrical injury, inhalation injury, or concurrent trauma; all poor-risk patients (ie, extremes of age, intercurrent disease)

Moderate, Uncomplicated Burn Injury
- Second-degree burns of 15% to 25% TBSA in adults or 10% to 20% in children
- Third-degree burns of less than 10% TBSA not involving special care areas
- Excludes all patients with electrical injury, inhalation injury, or concurrent trauma; all poor-risk patients (ie, extremes of age, intercurrent disease)

Major Burn Injury
- Second-degree burns of more than 25% TBSA in adults or 20% in children
- All third-degree burns of 10% or more TBSA
- All burns involving eyes, ears, face, hands, feet, perineum, joints
- All patients with inhalation injury, electrical injury, or concurrent trauma; all poor-risk patients

From Pham TN, Gibran NS, Heimbach DM: Evaluation of the burn wound: management decisions. In Herndon DN (ed): Total Burn Care, 3rd ed. Philadelphia, PA: Saunders, 2007, pp 119–126.

down through the layers of skin. Figure 53-3 represents the concentric zones of a burn injury.[6] The zone of coagulation is the area where the most damage has been sustained; temperatures have reached 113°F (45°C). The tissues are black, gray, khaki, or white and have undergone protein coagulation and cell death. This area has lost the ability to recover and requires surgical intervention. The zone of stasis immediately surrounds the zone of coagulation. This area contains cells that are at the most risk during burn resuscitation. They can recover or become necrotic in the initial 24 to 72 hours, depending on the conditions and course of resuscitation. The zone of hyperemia is the area of increased blood flow in an effort to bring the needed nutrients to the tissue for recovery (*active hyperemia*) and to remove the metabolic waste products (*reactive hyperemia*). This area heals rapidly and has no cell death.

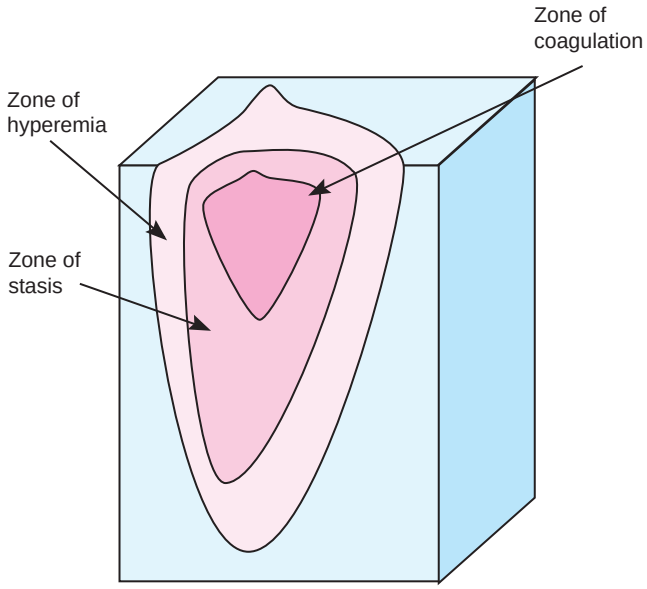


FIGURE 53-3 ▲ The concentric zones of a burn injury.

Systemic Response

Major changes at the cellular level are responsible for the tremendous systemic response noted in a patient with burns. The localized response causes a coagulation of cellular proteins, leading to irreversible cell injury with local production of complement, histamine, and oxygen free radicals (ie, byproducts of oxidative processes). Oxygen free radicals alter cell lipids and proteins, affecting the integrity of the cell membrane. This is particularly problematic in the endothelium of the microvascular circulation because disruption of the cell membrane leads to increased vascular permeability.[8] Increased vascular permeability leads to loss of plasma proteins into the interstitium and results in a marked decrease in circulating volume. Complement activation and histamine release contribute to the increased vascular permeability by increasing production of oxygen free radicals.[8,9] Increased vascular permeability leads to the formation of interstitial edema, which usually peaks within 24 to 48 hours of injury. It is hypothesized that the microvasculature takes weeks to restore itself completely to its premorbid state. The pulmonary vasculature is not spared, and pulmonary interstitial edema forms, with intraalveolar hemorrhages; this initial pulmonary insult is thought to be a precursor to the development of acute respiratory distress syndrome (ARDS).[8]

Systemically, a burn injury causes a release of vasoactive substances, such as histamine, prostaglandins, interleukins (ILs), and arachidonic acid metabolites. These substances initiate the systemic inflammatory response syndrome (SIRS). The potent mediators and cytokines (nitric oxide, platelet-activating factor [PAF], serotonin, thromboxane A_2, and tumor necrosis factor [TNF]) deplete the intravascular volume, decreasing blood flow to the kidneys and the gastrointestinal (GI) tract. If left uncorrected, hypovolemic shock, metabolic acidosis, and hyperkalemia may occur. Intestinal mucosal permeability also markedly increases and can become the primary source of bacterial infection. Early enteral feeding is one step to help prevent the translocation of bacteria.[10,11]

Nitric oxide relaxes smooth muscle and produces vasodilation and hypotension. It may also depress myocardial function and block platelet aggregation and adhesion. PAF initiates neutrophil and white blood cell (WBC) activation and produces tissue inflammation. PAF increases permeability of vessels, thereby decreasing myocardial contractility, causing vasodilation and hypotension. Some prostaglandins, and its activation, cause vasoconstriction, increased blood flow, and fever. Serotonin causes vasodilation, hypotension, and increased vessel permeability. TNF is responsible for numerous cellular responses, including increased formation of oxygen free radicals, which leads to injury of the lungs, GI tract, and kidneys; increased cytokine production; initial hyperglycemia followed by hypoglycemia; hypotension; metabolic acidosis; coagulopathy; and activation of the coagulation cascade.[8]

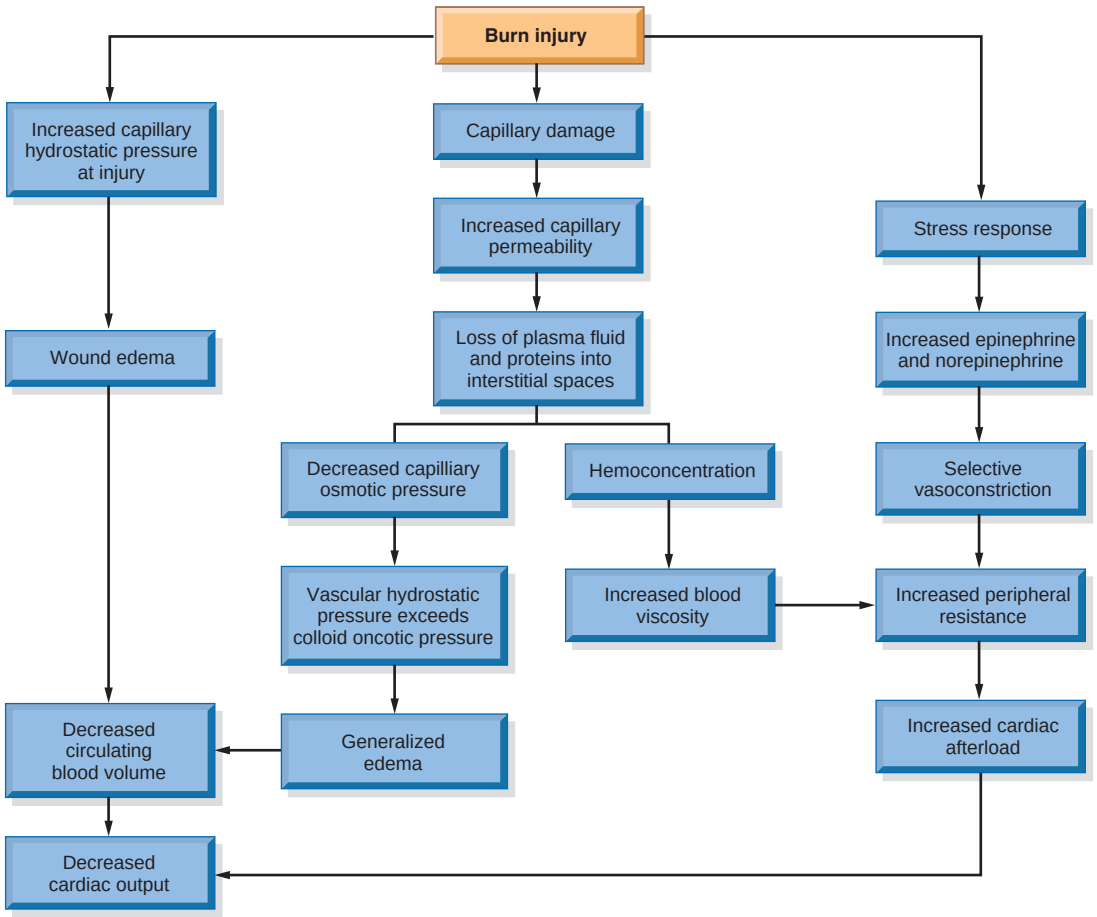


FIGURE 53-4 ▲ Fluid shifts in burn shock.

The end results of the local and systemic responses are dramatic if the burn covers more than 20% of the TBSA. The person with a major burn injury experiences a form of hypovolemic shock known as burn shock (Fig. 53-4). Within minutes of thermal injury, a marked increase in capillary hydrostatic pressure occurs in the injured tissue, accompanied by an increase in capillary permeability. This results in a rapid shift of plasma fluid from the intravascular compartment across heat-damaged capillaries, into interstitial areas (resulting in edema), and to the burn wound itself. The loss of plasma fluid and proteins results in a decreased colloid osmotic pressure in the vascular compartment. As a result, fluid and electrolytes continue to leak from the vascular compartment, resulting in additional edema formation in the burned tissue and throughout the body.

This "leak," which consists of sodium, water, and plasma proteins, is followed by a decrease in cardiac output, hemoconcentration of red blood cells, diminished perfusion to major organs, and generalized body edema. The pathophysiological response after burn injury is biphasic. In the early postinjury (ebb) phase, generalized organ hypofunction develops as a consequence of decreased cardiac output. Peripheral vascular resistance increases as a result of the neurohumoral stress response after trauma. This increases cardiac afterload, resulting in a further decrease in cardiac output. The increase in peripheral vascular resistance (selective vasoconstriction) and the hemoconcentration resulting from plasma fluid loss may cause the blood pressure to appear normal at first. However, if fluid replacement is inadequate, and plasma protein loss continues, hypovolemic shock soon occurs.

In patients receiving adequate fluid resuscitation, the cardiac output usually returns to normal in the latter part of the first 24 hours after burn injury. As plasma volume is replenished during the second 24 hours, the cardiac output increases to hypermetabolic levels (hyperfunction phase) and slowly returns to more normal levels as the burn wounds are closed.[8,10,12]

In some instances, with burns exceeding 60% of the TBSA, depressed cardiac output does not respond to aggressive volume resuscitation. A myocardial depressant factor capable of depressing ventricular contractility by 60% has been identified. Myocardial depression in the early postburn period may also be the result of reduced coronary blood flow.[8,10]

The response of the pulmonary vasculature is similar to that of the peripheral circulation. However, pulmonary vascular resistance is greater and lasts longer. Immediately after burn injury, the patient may experience a mild, transient pulmonary hypertension. A decrease in oxygen tension and lung compliance may also be evident.

The loss of fluid throughout the body's intravascular space results in a thickened, sluggish flow of the remaining circulatory blood volume. The effects reach all body systems. This slowing of circulation permits bacteria and cellular material to settle in the lower portions of blood vessels, especially in the capillaries, which results in sludging.

The antigen–antibody reaction to burned tissue adds to circulatory congestion by the clumping or agglutination of cells. Coagulation problems occur as a result of the release of

thromboplastin by the injury itself and the release of fibrinogen from injured platelets. If thrombi occur, they may cause ischemia of the affected part and lead to necrosis. Although of limited incidence in patients with burns, the increased coagulation process may develop into disseminated intravascular coagulation.

▲ Concomitant Problems

Pulmonary Injury

Pulmonary damage usually occurs within 24 to 48 hours of the injury and is secondary to the inhalation of combustible products or may be the result of inhaled superheated air. In an incident involving large amounts of steam, the risk for injury is far greater because water has a heat-carrying capacity 4,000 times greater than air and has the ability to be inhaled deeply in the pulmonary system.[1,6] Pulmonary injury may also be the result of a systemic process related to SIRS.

Fiberoptic bronchoscopy permits direct visualization of the airways (facilitating evaluation of erythema, edema, ulceration, and enlarged vessels) as well as the removal of debris by lavage. Fiberoptic bronchoscopy should be completed when feasible and repeated as indicated during the acute phase to document the extent of the pulmonary injury and complete direct lavage when necessary.

Carbon Monoxide Toxicity

Carbon monoxide is a nonirritating, odorless, colorless gas that is formed as a result of incomplete combustion of any carbon fuel. This gas is found in a variety of sources, including exhaust from vehicles, hot water heaters, furnaces and tobacco smoke. Carbon monoxide poisoning produces its effect on the body by competing with oxygen for uptake on hemoglobin, thereby acting as an asphyxiant. Because carbon monoxide affinity for hemoglobin is 200 to 300 times higher than oxygen, carbon monoxide readily displaces the oxygen, leading to the formation of carboxyhemoglobin and a reduction in systemic arterial oxygen content.[13–15] Carboxyhemoglobin shifts the oxyhemoglobin dissociation curve to the left, further decreasing the ability of the red blood cells to release oxygen to body tissues.[15] This may lead to severe anoxia and related brain injury.

The patient with a clear history of exposure to carbon monoxide is usually found in a closed environment in the presence of combusted gases, such as smoke, automobile exhaust, or fumes from a faulty furnace.[6] The signs of carbon monoxide poisoning depend on the carboxyhemoglobin present in the patient's blood (Table 53-2).[13]

When carbon monoxide poisoning is suspected, 100% high-flow oxygen is administered. Carbon monoxide has a half-life of 4 hours if the patient breathes room air and 45 minutes if the patient is breathing 100% oxygen. Hundred percent oxygen should be continued until the carboxyhemoglobin level is less than ten percent and neurological symptoms resolve.[6,13,14] Serial carboxyhemoglobin level measurements are the most accurate way to assess responsiveness to oxygen therapy.[13] Pulse oximetry is inaccurate when the carboxyhemoglobin level is elevated because pulse oximetry cannot distinguish between oxygen and carbon monoxide on the hemoglobin. The analysis and trending of arterial blood gas (ABG) levels for acid–base balance, lactate levels, and bicarbonate are helpful to manage carbon monoxide poisoning with lactic or metabolic acidosis.[13]

Table 53-2 Signs and Symptoms of Carbon Monoxide Poisoning

Carboxyhemoglobin Saturation (%)	Clinical Presentation
10	No symptoms
20	Headache, nausea/vomiting, dyspnea on exertion
30	Confusion, lethargy, tachypnea
40–60	Seizure, coma, changes on electrocardiogram
More than 60	Death

Inhalation Injury

Besides carbon monoxide poisoning, smoke inhalation can result in thermal injury to the airway. Pulmonary damage, primarily as a result of inhalation injury, is historically seen in less than 10% of the total cases but accounts for 20% to 84% of burn mortality and is a significant factor in increasing the hospital length of stay.[16] Three stages of injury have been described:

1. Acute pulmonary insufficiency may occur during the first 36 hours.
2. Pulmonary edema occurs in 5% to 30% of patients with burns between 6 and 72 hours after injury.
3. Bronchopneumonia appears in 15% to 60% of patients with burns 3 to 10 days after injury.

Upper airway injury is the result of inhalation of superheated air, which may cause blisters and edema in the supraglottic area around the vocal cords. This situation may cause airway obstruction and edema. Hoarseness, stridor, dyspnea, carbonaceous sputum, and tachypnea indicate airway compromise, which must be addressed immediately. Early intubation may thwart such disastrous occurrences.

Tracheobronchial and parenchymal lung injuries are usually a result of incomplete combustion of chemicals (eg, aldehyde, acrolein) or noxious gases which in a chemical pneumonitis. Pathophysiologic changes associated with lower lung injuries include impaired ciliary activity, hypersecretion, edema, inflammation, and bronchospasm.[6,14,16] Inflammatory changes in the trachea and alveoli occur within 24 hours of injury. The pulmonary tree becomes irritated and edematous. The alveoli may collapse, causing a decreased compliance, which leads to atelectasis. ARDS may develop rapidly. However, changes may not become apparent until the second 24 hours. Pulmonary edema is a possibility any time from the first few hours to 7 days after the injury. Subtle changes in the patient's sensorium may indicate hypoxia.

History and physical assessment findings that should alert the nurse to the potential for inhalation injury are given in Box 53-5. Serial ABG evaluations show a decreasing arterial oxygen tension (PaO_2). Usually, the admission chest film appears normal because changes are not reflected until 24 to 48 hours after the burn. A sputum specimen is obtained for culture and sensitivity studies. Laryngoscopy and

BOX 53-5 PATIENT SAFETY

History and Physical Examination Findings Suggestive of Inhalation Injury

- History of incident occurring in a confined area
- Singed nasal or facial hairs
- Burns of the oral or pharyngeal mucous membranes
- Burns in the perioral area or neck
- Carbonaceous sputum
- Change in voice
- Change in level of consciousness

bronchoscopy may be of value in determining the presence of extramucosal carbonaceous material (the most reliable sign of inhalation injury) and the state of the mucosa (blistering, edema, erythema). More specific confirmation of inhalation injury is achieved with the use of fiberoptic bronchoscopy, which permits direct examination of the proximal airway, and xenon-133 scintigraphy (ventilation–perfusion scanning).[6,14] Xenon-133 scintigraphy is helpful in establishing a diagnosis of injury to small airways and lung parenchyma.

Infection

There is no greater problem for the patient with burns than infection. Infection is the most common cause of death in patients with burns after the first 7 days.[17] Loss of the mechanical barrier between the human body and the environment is the first step in the weakening of defenses. All aspects of the immune system, including phagocytosis, soluble mediators of innate immunity such as complement, antibody production, and cellular (T-cell) defense systems, are compromised by severe burn injury.

Actions of the health care team can compromise patient survival. All catheters invading the body, including endotracheal tubes, central venous catheters, and bladder catheters, must be handled with as clean a technique as possible. Although the skin and gut are the source of endogenous bacteria, a greater threat to the patient is colonization with antibiotic-resistant pathogens carried by the burn team from other patients. The hands must be washed without fail before and after handling the patient, the patient's bed, or equipment. When dressings are removed and wounds exposed, sterile gloves must be worn. Frequent and meticulous hand washing alone probably prevents infection more than any other single action. Infection control policies vary from burn center to burn center, but the philosophy remains the same: Make every effort to minimize the transmission of bacteria from patient to patient.

Diagnosis of invasive infection in the patients with burns is unusually difficult. Most patients with burns meet two or more of the SIRS criteria because they are constantly and chronically exposed to the environment thus releasing inflammatory mediators. The ABA Consensus developed *triggers* that should prompt the health care team to look for an infection in the burn patient (see Table 53-3). Manifestations of multisystem organ dysfunction, such as hypotension, hypoxia, decreased pulmonary compliance, renal failure, or hepatic dysfunction, are almost certain signs of septic shock.

Qualitative wound cultures done by swabbing the wound yield no new information other than the nature of the bacterial species colonizing the surface of the wound. A biopsy of the burn wound permits a quantitative assay of the number of colony-forming units (CFUs) of bacteria per gram of tissue. Burn wound sepsis is likely if the colony count is greater than 105 CFU/g, and the quantitative culture also allows isolation and identification of the invading organism.

Table 53-3 American Burn Association (ABA) Consensus: Sepsis and Infections in Burns Versus Systemic Inflammatory Response Syndrome (SIRS)

ABA Consensus	SIRS
Temperature > 39°C or <36.5°C	Temperature > 38°C or <36°C
Heart rate > 110 bpm	Heart rate > 90 bpm
Respiratory rate > 25 bpm or minute ventilation > 12 L/min ventilated	Respiratory rate > 20 bpm or $PaCO_2$ < 32 mm Hg
Thrombocytopenia < 100,000/mcl	WBC > 12,000/mm^3 or <4,000/mm^3 or left shift > 10% bands
Hyperglycemia (no preexisting diabetes) or insulin resistance	
Enteral feeding intolerance	

Concern for infection if at least three of the above.

bpm, breaths per minute.

From Greenhalgh DG, Saffle JR, Holmes JH, et al: American Burn Association consensus conference to define sepsis and infection in burns. J Burn Care Res 28(6):776–790, 2007.

Trauma

Concomitant injuries such as fractures and head trauma pose significant risk for the patient with burns. Ensuring adequate airway, breathing, and circulation takes precedence over caring for specific injuries. Cervical spine injuries should be stabilized and cleared. If head trauma is suspected, a computed tomography scan is obtained. The history of the burn injury is critical to assisting with the evaluation of the patient. The burn wounds may mask some of the classic signs of underlying injuries, such as ecchymosis or swelling. The burn event may include events, such as an explosion, the patient being thrown or falling, or a motor vehicle crash. Patients with electrical injuries must also be evaluated for fractures secondary to the violent muscular contraction after exposure; special focus should be placed on the cervical spinous process and long bones.

▲ Assessment and Management

The initial assessment of the patient with burns is like that of any trauma patient. The ABA has identified criteria for referral to a burn center (Box 53-6). Patients with these burns should be treated in a specialized burn facility after initial assessment and treatment at an ED. Whether the patient

BOX 53-6 Criteria for Referral to a Burn Center

- Partial-thickness burns more than 10% of TBSA
- Burns that involve the face, hands, feet, genitalia, perineum, or major joints
- Third-degree burns in any age group
- Electrical burns, including lightning injury
- Chemical burns
- Inhalation injury
- Burn injury in patients with preexisting medical disorders that could complicate management, prolong recovery, or affect mortality
- Concomitant trauma, in which the burn injury poses the greatest risk for morbidity or mortality*
- Children with burns in hospitals without qualified personnel or equipment for the care of children
- Patients with burns who will require special social, emotional, or long-term rehabilitative intervention

*In such cases, if the trauma poses the greater immediate risk, the patient may be initially stabilized in a trauma center before being transferred to a burn unit. Physician judgment is necessary in such situations and should be in concert with the regional medical control plan and triage protocols.

Data from the American Burn Association Burn Center Referral Criteria.

stays at the initial hospital or is transferred to a burn center facility, the resuscitation phase begins immediately after the burn insult has occurred. The primary and secondary surveys are completed before transfer. Proper stabilization of the patient is crucial for successful transfer. As with any major trauma, the first hour is crucial, and the next 24 to 36 hours are also important. The management of fluid balance, the respiratory system, and nutrition is vital, and all systems have a major impact on the patient's survival.

Resuscitative Phase

Primary Survey

The following parameters are assessed in the primary survey:

- Airway maintenance with cervical spine protection
- Breathing and ventilation
- Circulation with hemorrhage control
- Disability (assess neurological deficit)
- Exposure (completely undress the patient, but maintain temperature)

Airway

On initial assessment of the patient with burns, the airway must be assessed immediately. The compromised airway may be controlled by a chin lift, jaw thrust, insertion of an oropharyngeal airway in an unconscious patient, or endotracheal intubation. It is crucial not to hyperextend the neck in patients with suspected cervical spine injuries.

Breathing and Ventilation

Ventilation requires adequate functioning of the lungs, chest wall, and diaphragm. To assess for breathing and ventilation, the nurse must listen to the chest and verify breath sounds in each lung, assess adequacy of rate and depth of respiration, administer high-flow oxygen at 15 L/min using a nonrebreathing mask, and assess for circumferential full-thickness burns of the chest that may impair ventilation.

Circulation

Assessment of the circulation includes a measurement of blood pressure and heart rate. Special attention should be paid to the distal pulses of any extremity with circumferential burns. Intravenous (IV) cannulation is performed by inserting two large-bore catheters into the skin that is unburned, if possible. A central venous catheter should be inserted when indicated. Doppler ultrasonography can be used to assess for pulses. Box 53-7 lists risk factors for impaired circulation.

Disability

Typically, the patient with burns is alert and oriented. If not, associated injuries, such as inhalation injury, head trauma, substance abuse, or preexisting medical conditions, should be considered. The assessment is initiated by determining the patient's level of consciousness using the AVPU (Alert, responds to Verbal stimuli, responds to Painful stimuli, Unresponsive) method.

Exposure

All the patient's clothing and jewelry are removed to complete the primary and secondary survey. Ensure the environment is warmed as the burn patient is poikilothermic. After examination, the patient is covered with a clean, dry sheet and warm blankets to prevent evaporative cooling. If possible, IV fluids are warmed at 98.6°F (37°C) to 104°F (40°C).

Secondary Survey

The secondary survey is completed after resuscitative efforts are well established and consists of a detailed history and physical examination of the patient as well as a complete history of the accident. Every attempt is made to determine exactly what happened (Box 53-8). A detailed neurological examination is completed, and initial radiographic and laboratory studies are done. Resuscitative measures are ongoing and constantly evaluated.

A complete history and physical examination are the hallmarks of the secondary survey. It is not uncommon for patients to have comorbid diseases. Preexisting diseases, such as diabetes, hypertension, asthma, cancer, and stroke, should be documented. A medication list is obtained from the patient if possible, or a family member is asked to provide the information. In addition, any allergies, the person's tetanus immunization history, and the time of the person's last meal should be documented. Burn depth and burn size are assessed.

BOX 53-7 *PATIENT SAFETY*

Risk Factors for Impaired Circulation

- Progressive diminution of pulses despite adequate resuscitation
- Decreased capillary refill
- Decreased sensation
- Progressive worsening of pain
- Paresthesias
- Pallor of extremity

BOX 53-8 Questions to Ask During a Secondary Survey

Thermal Burns
- How did the burn occur?
- Did the burn occur inside or outside?
- Did the clothes catch fire?
- How long did it take to extinguish the fire?
- Were there any explosions?
- Was the patient found in a smoke-filled room?
- How did the patient escape?
- Did the patient jump out of a window?
- Were there other people injured or killed at the scene?
- Was the patient unconscious at the scene?
- Was there a motor vehicle crash?
- Was the car severely damaged?
- Was there a car fire?
- Are the purported circumstances of the injury consistent with the burn characteristics (is there possibility of abuse)?

Scald Injuries
- How did the burn occur?
- What was the temperature of the liquid?
- What was the liquid; how much liquid was involved?
- What was the burn cooled with?
- Who was present when the burn took place?
- Where did the burn take place? Is there possibility of abuse?

Chemical Burns
- What was the agent?
- How did the exposure occur?
- What was the duration of contact?
- Did contamination take place?

Electrical Burns
- What kind of electricity was involved?
- Did the patient lose consciousness?
- Did the patient fall?
- What was the estimated voltage?
- Was cardiopulmonary resuscitation administered at the scene?

Burn injuries require a global assessment. The following laboratory and diagnostic studies are indicated for patients with burns:

- Complete blood count (CBC)
- Comprehensive chemistry panel, including blood urea nitrogen
- Creatinine level
- Urinalysis
- ABG values to include carboxyhemoglobin determination
- Electrocardiogram
- Chest radiograph

After the primary and secondary surveys are complete, the burned area is usually covered with a dry sheet. This reduces the risk for infection and keeps the patient warm. A cool compress can be applied to small superficial burns. If the patient has a high-voltage electrical burn or cardiac changes are noted, continuous cardiac monitoring is provided. If the patient has a chemical burn, the area is immediately flushed with large amounts of water to remove the chemical, and all contaminated clothing is removed and bagged. If the patient is going to be transferred to a burn center, initiation of fluid resuscitation, insertion of a nasogastric tube (NGT), and insertion of an indwelling urinary catheter may be carried out during the secondary assessment.

Providing Hemodynamic Support

Therapy for burn shock is aimed at supporting the patient through the period of hypovolemic shock until capillary integrity is restored. Fluid resuscitation is the primary intervention in the resuscitative phase in the intensive care unit (ICU) to maintain tissue perfusion and end organ function. Goals in fluid resuscitation are as follows:

- Correct fluid, electrolyte, and protein deficits.
- Replace continuing losses and maintain fluid balance.
- Prevent excessive edema formation.
- Maintain an hourly urinary output in adults of 30 to 50 mL/h (approximately 0.5 mL/kg/h).[6]

Formulas for Fluid Administration

Numerous formulas have been developed for fluid resuscitation (Box 53-9). Each has advantages and disadvantages. They differ primarily in terms of recommended volume administration and salt content. In general, lost crystalloid and colloid solutions must be replaced rigorously. Free water, given as dextrose 5% in water (D_5W) with or without added electrolytes, is regulated so that insensible fluid loss is covered. Lactated Ringer's solution is the crystalloid of choice because it is a balanced salt solution that closely approximates the composition of extracellular fluid.

The ABA recommends the use of the ABA consensus formula for the resuscitation of patients with burns. The formula is a combination of the modified Brooke formula and the Baxter (commonly called Parkland) formula. The ABA consensus formula requires 2 to 4 mL of lactated Ringer's solution per kilogram of body weight per percentage TBSA burn. The total amount calculated is administered in the first 24 hours after injury. One half is given in the first 8 hours *from the time of the burn*, one fourth is given during the next 8 hours, and the remaining one fourth is given over the next 8 hours. The ABA consensus formula and the other fluid resuscitation formulas are guidelines, and individual patients may require more or less than 2 to 4 mL/kg per percentage TBSA during the first 24 hours. Patients who often require more fluid than the formula predicted include those with electrical injuries, inhalation injuries, delayed resuscitation, prior dehydration at time of injury, and concomitant trauma.

Other formulas contain various amounts of hypertonic saline or colloid. Hypertonic saline resuscitation lowers the amount of fluid that needs to be given to selected patients; however, it can cause severe hypernatremia and must be used cautiously. The argument against colloid administration within 12 hours of injury is that during this time, the diffuse postburn capillary leak allows colloids to extravasate through endothelial junctions. Therefore, colloid administration does not produce any demonstrable oncotic benefit over administration of a crystalloid while the capillary leak is present. The postinjury time at which capillary integrity is restored varies among people but usually is between 12 and 14 hours. Many physicians administer colloids at this point to restore albumin levels to 2.0 to 3.0 mg/dL. Controversy exists over the type of colloid to be administered, with some centers using salt-poor albumin, and others using fresh frozen plasma.

BOX 53-9 Fluid Resuscitation Formulas

Baxter (Parkland) Formula
- First 24 hours: Lactated Ringer's solution (4 mL/kg/% TBSA); half given over first 8 hours, remaining half given over next 16 hours
- Second 24 hours: Dextrose in water, plus potassium- and colloid-containing fluid (0.3 to 0.5 mL/kg/%TBSA)

Brooke Formula
- First 24 hours: Lactated Ringer's solution (1.5 mL/kg/% TBSA) plus colloid solution (0.5 mL/kg/%TBSA); half given over first 8 hours, remaining half given over next 16 hours
- Second 24 hours: Lactated Ringer's solution (0.5 to 0.75 mL/ kg/%TBSA), plus 5% dextrose in water (D_5W) (2 L)

Modified Brooke Formula
- First 24 hours: Lactated Ringer's solution (2 mL/kg/% TBSA); half given over first 8 hours, remaining half given over next 16 hours
- Second 24 hours: Colloid solution (0.3 to 0.5 mL/kg/% TBSA), plus D_5W to maintain adequate urine output

Consensus Formula
- First 24 hours: Lactated Ringer's solution (2 to 4 mL/ kg/% TBSA in adults; 3 to 4 mL/kg/%TBSA in children); half given over first 8 hours, remaining half given over next 16 hours
- Second 24 hours: Colloid-containing fluid (0.3 to 0.5 mL/ kg/%TBSA), plus electrolyte-free fluid (in adults) or half-normal saline solution (in children) to maintain adequate urine output

Dextran Formula
- First 8 hours: Dextran 40 in saline (2 mL/kg/h), plus lactated Ringer's solution infused to maintain urine output at 30 mL/h
- Second 8 hours: Fresh-frozen plasma (0.5 mL/kg/h) for 18 hours, plus additional crystalloid to maintain adequate urine output

Evans Formula
- First 24 hours: 0.9% normal saline solution (1 mL/kg/%TBSA), plus colloid solution (1 mL/kg/%TBSA); half given over first 8 hours, remaining half given over next 16 hours
- Second 24 hours: 0.9% normal saline solution (0.5 mL/kg/% TBSA), plus D_5W (2 L)

The challenge is to balance resuscitation since under or over resuscitation have significant consequences. Care must be taken to avoid fluid overload and pulmonary edema. This is often difficult because large amounts of fluids are given over a short period during fluid resuscitation immediately after the burn. For example, using the high range of the ABA consensus formula, a male patient weighing 75 kg who received burns over 50% of his body would require up to 15,000 mL of fluid (4 mL × 75 kg × 50% TBSA = 15,000 mL). Of this, 7,500 mL is to be administered during the first 8 hours, and 3,750 mL is to be administered in the second and third 8-hour periods. It is extremely difficult to avoid fluid overload and pulmonary edema when it is necessary to infuse large amounts of fluids so rapidly.

After the first 24 hours after injury, replacing the massive evaporative water loss is a major consideration in fluid management. The primary solution given at this time is D_5W, with the goal of keeping the patient's sodium concentration at 140 mEq/L. The fluid volume depends on the severity of injury, the age of the patient, the physiological status of the patient, and any associated injuries. Consequently, the volume recommended by a resuscitation formula must be modified according to the person's response to therapy (Fig. 53-5).

Urine output is the single best indicator of fluid resuscitation in patients with previously normal renal function. The onset of spontaneous diuresis is a hallmark indicating the end of the resuscitative phase. Infusion rates can be decreased by 20% to 30% for 1 hour if the urine output is satisfactory and can be maintained for 2 hours; the reduction may then be repeated. It is essential that urinary outputs be maintained within normal limits of 30 to 50 mL/h (0.5 mL/kg/h) in the adult. Other indications of adequate fluid replacement are listed in Box 53-10.

Patients are usually weighed daily. A gain of 15% of admission weight may be expected with large fluid resuscitation. Intake and output must be monitored meticulously. Patients who sustain deep muscle injury (ie, second- or third-degree burns or electrical injuries) are at risk for development of acute renal insufficiency. This renal dysfunction may be the result of inadequate fluid resuscitation, or it may be the consequence of the liberation of the myoglobin and hemoglobin from damaged cells. These compounds, sometimes called hemochromogens, may precipitate in renal tubules, resulting in acute tubular necrosis. Hemochromogens produce a clear reddish-brown color in the urine. Should hemochromogens appear in the urine, acidosis should be corrected promptly and IV fluids increased to maintain a brisk urine output (75 to 100 mL urine/h) until the urine returns to its normal clear yellow and there is no urinary myoglobin.

Providing Pulmonary Support

Inhalation injury is the leading cause of death in the first 24 hours after burn injury. It increases the mortality rate by 20% alone and by as much as 60% when combined with pneumonia.[16,18] Goals for the successful treatment of inhalation injury include improving oxygenation and decreasing interstitial edema and airway occlusion.

The conventional treatment for inhalation injury is largely supportive because direct intervention is difficult. Humidified oxygen is administered to prevent drying and sloughing of the mucosa. Upper airway edema peaks 24 to 48 hours after injury. If the injury is mild or moderately severe, placing the patient in a high Fowler's position and administering aerosolized racemic epinephrine may be sufficient to limit further edema formation. Severe upper airway obstruction may require endotracheal intubation to protect the airway until the edema subsides.

In patients with mild tracheobronchial injury, atelectasis may be prevented by frequent pulmonary toilet, using a high Fowler's position, coughing and deep breathing, chest physiotherapy, repositioning, frequent tracheal suctioning, and incentive spirometry.[13,14] In patients with more severe inhalation injury, more frequent suctioning may be necessary, and bronchoscopic removal of debris may be appropriate. These patients usually require endotracheal intubation and mechanical ventilatory support. The objective of ventilatory support is to provide adequate gas exchange at the lowest possible inspired oxygen concentration and airway pressure, in an attempt to reduce the incidence of oxygen toxicity and pulmonary barotrauma. The use of volumetric diffusive

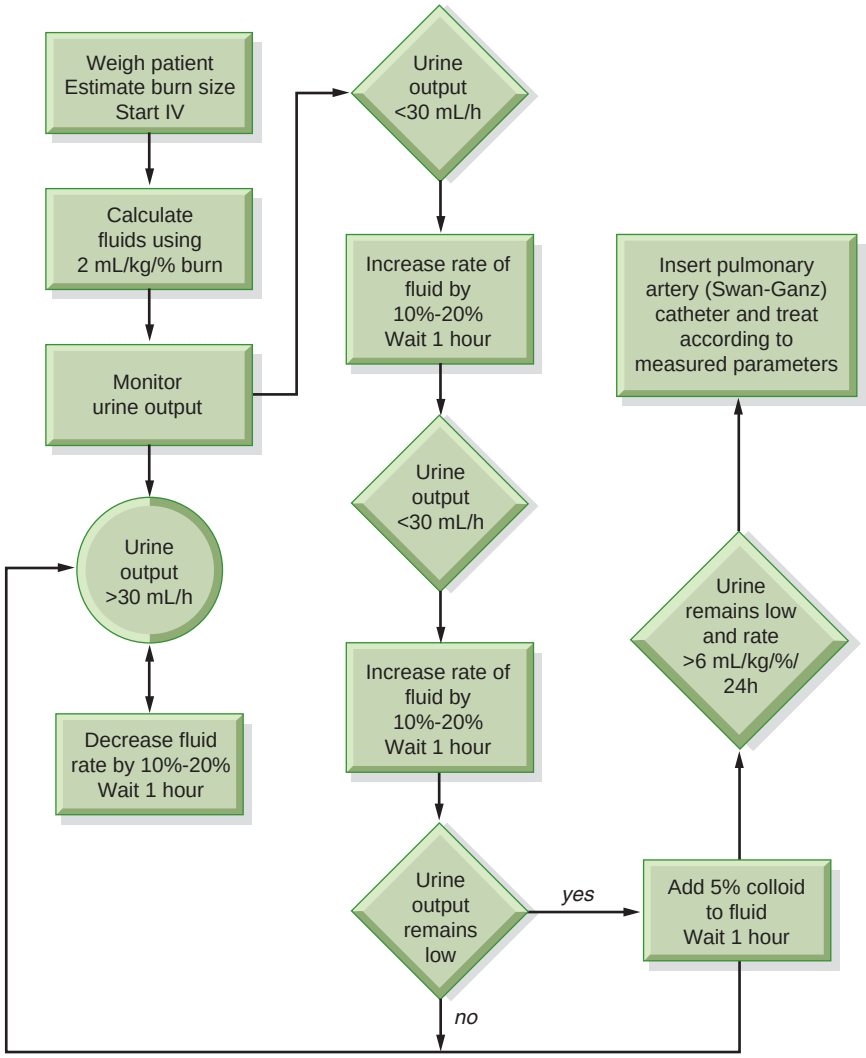


FIGURE 53-5 ▲ Initial 24-hour fluid management. (From Rue LW, Cioffi WG: Resuscitation of thermally injured patients. Crit Care Nurs Clin N Am 3(2):186, 1991.)

respiration (VDR) appears to offer advantages over conventional mechanical ventilation.[14,18] In VDR, subtidal volume breaths accumulate and build to a set airway pressure, which is then followed by passive exhalation. Throughout the ventilatory cycle, high-frequency pulsations of air are continuously administered. This method of inspiration appears to aid in ventilation and recruitment of partially obstructed alveoli.

Patients with bronchospasm are treated with aerosolized or intravenously administered bronchodilators. Respiratory parameters are monitored closely, and constant attention is paid to breath sounds and vital signs to detect fluid overload as early as possible.

Bronchopneumonia may be superimposed on other respiratory problems at any time and may be hematogenous or airborne. Airborne bronchopneumonia is most common, with onset occurring soon after injury. It is often associated with a lower airway injury or aspiration. Hematogenous, or miliary, pneumonia begins as a bacterial abscess secondary to another septic source, usually the burn wound. The time of onset usually is 2 weeks after injury.

Prophylactic antibiotics and steroids have not been demonstrated to prevent the common complications of infection encountered in patients with inhalation injury. New methods to decrease the incidence of nosocomial pneumonia in critically ill patients that are currently under investigation include selective decontamination of the orodigestive tract.

Escharotomy

Any circumferential burn to an arm or leg may mimic compartment syndrome. Edema formation in the tissues under the tight, unyielding eschar of a circumferential burn of a deep partial-thickness or full-thickness injury produces significant vascular compromise in the affected limb.

To minimize the risk for circulatory compromise, the patient's rings, watch, and other jewelry are removed during the initial examination. Elevation and range of motion of the injured extremity may alleviate minimal degrees of circulatory distress. Skin color, sensation, capillary refill, and peripheral pulses are assessed and documented hourly in an extremity with a circumferential burn. Doppler ultrasonography is the most reliable means of assessing arterial blood flow and the need for an escharotomy. In the upper extremity, the radial, ulnar, and palmar arch pulses are checked hourly. In the lower extremity, the posterior tibial and dorsalis pedis pulses are checked hourly. Loss—or a progressive diminution (decrease)—of the ultrasonic signal is an indication for escharotomy.[19] In circumferential chest burns, respiration and ventilatory effort needs

BOX 53-10 Indications of Adequate Fluid Replacement*

Urinary output	30–70 mL/h (0.5 mL/kg/h)
Pulse rate	100–120 bpm
Central venous pressure (CVP)	<12 cm H_2O
Pulmonary artery occlusion pressure (PAOP)	<18 mm Hg
Lungs	Clear
Sensorium	Clear
Gastrointestinal tract	Absence of nausea and adynamic ileus
Arterial base deficit and lactate	Normalizing values

*Central lines and Swan-Ganz catheters are not inserted routinely because of the danger of sepsis; however, they are used in selected instances.

to be continuously assessed. Escharotomy may be necessary to relieve chest wall restriction and improve ventilation.[19]

The escharotomy is carried out as a bedside procedure, using a sterile field and scalpel, an electrocautery device, or both. Taking the patient to the operating room is not necessary and causes unacceptable delay. Local anesthesia is rarely needed because full-thickness injuries are insensate. However, small doses of narcotics and benzodiazepines assist in patient comfort. Patients with this severity of injury are often already intubated and therefore receiving sedatives and analgesics.

The incision should be placed along both the mid-medial and mid-lateral aspect of the extremity and should extend through the eschar down to the subcutaneous fat to permit adequate separation of the cut edges for decompression (Fig. 53-6). The incisions should be made from an area of unburned tissue and extended to unburned tissue if possible.

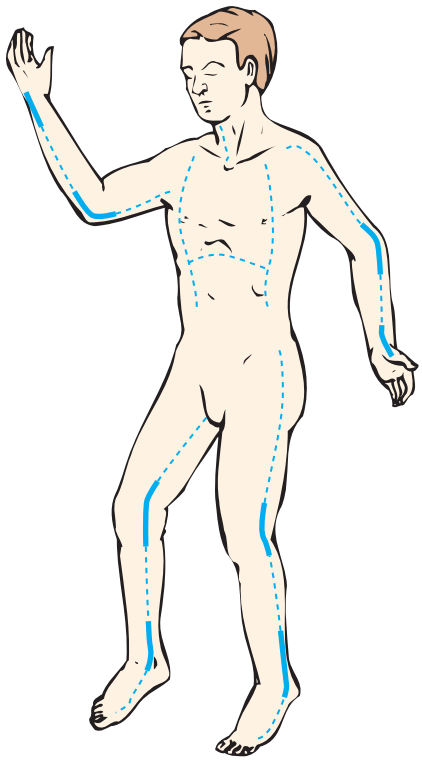


FIGURE 53-6 ▲ Preferred sites of escharotomy incisions.

The procedure should be completed with the patient in the anatomically correct position to minimize the risk for damage to major blood vessels and nerve bundles, especially when incisions cross joints.

Reparative Phase

Once the patient stabilizes, measures are taken to promote healing and prevent infection. As noted earlier, burn wounds can have profound effects on nearly every organ system. Box 53-11 lists nursing diagnoses for patients with burns, and Box 53-12 contains a collaborative care guide for the patient with burns.

Ensuring Optimal Nutrition

Before the unique nutritional needs of patients with burns were fully recognized in the late 1970s, those who had severe burn injuries, and who survived, languished in a hospital ward with minimal oral intake until they became severely cachectic. It is currently clear that appropriate nutrition plays a significant role in improving outcome for patients with severe burn injuries.

Although early parenteral feeding has been associated with increased mortality because of an increase in the risk for infection, early enteral feeding has been proposed because it may reduce the translocation of bacteria from the intestinal lumen.[10,11] Passage of bacteria from the gut into the intestinal lymphatics or portal venous system probably occurs in all healthy people. However, the intestinal edema that accompanies the burn resuscitation period and the

BOX 53-11 EXAMPLES OF NURSING DIAGNOSES

For the Burn Patient

- Ineffective Airway Clearance related to impaired cough, oropharyngeal and tracheal swelling, or artificial airway
- Impaired Gas Exchange related to inhalation injury, atelectasis, ARDS, or carbon monoxide poisoning
- Ineffective Breathing Pattern related to circumferential chest burn, upper airway obstruction, or ARDS
- Impaired Peripheral Tissue Perfusion related to edema or circumferential burn (eschar syndrome)
- Deficient Fluid Volume related to altered capillary permeability, insensible, and third-spacing losses
- Fluid Volume Excess related to fluid resuscitation and subsequent fluid mobilization 3 to 5 days postburn
- Impaired Skin Integrity related to burn injury or surgical interventions
- Hypothermia related to impaired integument
- Imbalanced Nutrition: Less Than Body Requirements related to hypermetabolic response to burn injury, paralytic ileus
- Impaired Urinary Elimination related to indwelling urinary catheter
- Risk for Infection related to loss of integument, invasive procedures, and immunocompromise
- Acute Pain related to exposure of nerve endings, invasive procedures, surgical procedures, and dressing changes
- Ineffective Coping related to altered body image and fear
- Anxiety related to traumatic injury, fear of dying, fear of disfigurement, change in body image, and change in role relationships

BOX 53-12 COLLABORATIVE CARE GUIDE for the Patient With a Burn

Outcomes	Interventions
Oxygenation/Ventilation	
Patent airway is maintained. Lung is clear on auscultation.	• Auscultate breath sounds every 2–4 h and as needed (PRN). • Assess for inhalation injury, and anticipate intubation. • Assess quantity and color of tracheal secretions. • Suction endotracheal airway when appropriate (see Chapter 25, Box 25-16, pp. 538–539). • Hyperoxygenate and hyperventilate before and after each suction pass.
Peak, mean, and plateau pressures are within normal limits for a patient on a ventilator.	• Monitor airway pressures every 1–2 h. • Monitor lung compliance every 8 h (see Chapter 24). • Administer bronchodilators and mucolytics. • Perform chest physiotherapy every 4 h. • Monitor airway pressures and lung compliance for improvement after interventions. • Calculate PaO_2/FiO_2 ratio and oxygen index, monitoring trends.
There is no evidence of atelectasis or infiltrates.	• Turn side to side every 2 h. • Consider kinetic therapy or prone positioning. • Daily chest x-ray.
Arterial blood gases (ABGs) are within normal limits.	• Initial and serial carboxyhemoglobin (carbon monoxide) levels until <10% • Monitor ABGs monitor acid-base balance, lactate and bicarbonate levels. (Pulse oximeter and calculated SaO_2 are inaccurate measures in the presence of carbon monoxide.) • Provide humidified oxygen. • Consider hyperbaric therapy.
Circulation/Perfusion	
Blood pressure, heart rate, CVP, and pulmonary artery (PA) pressures are within normal limits.	• Assess vital signs every 1 h. • Assess hemodynamic pressures every 1 h if patient has PA catheter. • Administer intravascular volume as ordered to maintain preload (see below).
Temperature is within normal limits.	• Monitor temperature every 1 h. • Maintain a warm environment, and use warming lights or blankets to prevent hypothermia. • Treat fever by cooling the environment, antipyretics and cooling blankets.
Perfusion to extremities is maintained; pulses are intact.	• Monitor perfusion using Doppler and palpation every 1 h. • Elevate burned extremities. • Prepare for escharotomy or fasciotomy.
Fluids/Electrolytes	
Restore and maintain fluid balance: Urine output 30–70 mL/h or 0.5 mL/kg/h. CVP 8–12 mm Hg; PAOP 12–18 mm Hg; blood pressure, within normal limits; heart rate 100–120 bpm.	• Assess intake and output every 1 h. • Give lactated Ringer's 2–4 mL/kg/TBSA%, divided into first 24-h postburn. • Monitor for spontaneous diuresis, and reduce intravenous infusion rate as indicated. • Take daily weight.
Electrolytes, mineral, and renal function values are within normal limits.	• Monitor and replace minerals and electrolytes. • Monitor blood urea nitrogen, creatinine, myoglobin, and urine electrolytes and glucose. • Monitor neurological status. • Monitor and treat dysrhythmias. • Monitor and limit water consumption, burn patients have extreme thirst and if allowed drastically decrease sodium level.
Mobility/Safety	
Patient is free of joint contractures.	• Provide passive and active range-of-motion exercises every 1–2 h. • Apply positioning splints as needed.
There is no evidence of complications related to immobility.	• Turn and reposition every 2 h. • Consider kinetic therapy. • Consider deep venous thrombosis prophylaxis.

(*continued on page 1199*)

BOX 53-12 *COLLABORATIVE CARE GUIDE for the Patient With a Burn (continued)*

Outcomes	Interventions
There is no evidence of infection.	• Maintain strict sterile technique, and monitor technique of others. • Maintain sterility of invasive catheters and tubes. • Per hospital protocol, change dressings and invasive catheters. Culture wounds, blood, urine, as necessary. • Monitor for changes in sputum or wounds. • Monitor SIRS/ sepsis using burn criteria: temperature, tachycardia, tachypnea or ↑minute ventilation, feeding intolerance, elevated glucose/insulin resistance.
Skin Integrity	
Unburned skin will remain intact.	• Assess skin every 4 h and each time patient is repositioned. • Turn every 2 h. • Consider pressure relief/reduction mattress and devices.
Burns begin healing without complications.	• Treat burns per hospital protocol; apply topical medications and débride as indicated. • Monitor skin graft viability. • Protect grafted areas (eg, bed cradle, dressings). • Consider air fluidized bed to enhance healing and relieve pressure from burned surface.
Nutrition	
Caloric and nutrient intake meets metabolic requirements per calculation (eg, Basal Energy Expenditure).	• Provide enteral or parenteral nutrition within 24 h of injury. • Consult dietitian or nutritional support service to assess nutritional requirements with team. • Monitor protein and calorie intake. • Monitor albumin, prealbumin, transferrin, cholesterol, triglycerides, glucose, nitrogen balance.
Comfort/Pain Control	
Patient will have minimal pain (<5 on pain scale) and discomfort.	• Assess pain and discomfort using objective pain scale every 4 h, PRN, and following administration of pain medication. • Administer analgesics before procedures and monitor patient response. • Use nonpharmacological pain management techniques (eg, music, distraction, touch).
Psychosocial	
Patient demonstrates decreased anxiety.	• Assess vital signs during treatments, discussions, and so forth. • Administer anxiolytics before treatments/procedures. • Consult social services, clergy, and so forth as appropriate. • Provide for adequate rest and sleep. • Encourage discussion regarding long-term effects of burns, available resources, and coping strategies.
Teaching/Discharge Planning	
Patient/significant others understand procedures and tests needed for treatment.	• Prepare patient/significant others for procedures, such as débridement, escharotomy, fasciotomy, intubation, and mechanical ventilation.
Significant others understand the severity of the illness, ask appropriate questions, and anticipate potential complications.	• Explain the potential effects of burns and the potential for complications, such as infection, respiratory or renal failure. • Encourage significant others to ask questions related to the management of burns, disfigurement, coping, and so forth.

immunosuppression that follows make it difficult for the body to clear these microorganisms effectively. Microbial products—either live organisms or cell wall fragments—disseminate through the body, prompting the release of cytokines such as TNF, interleukin-1 (IL-1), and interleukin-6 (IL-6). These cytokines exacerbate the hypermetabolic response and may initiate SIRS.

The rationale for enteral feeding within the first 24 hours of injury is that the presence of food in the gut lumen reduces the rate of microbial translocation. Although not proved definitively in the clinical setting of patients with burns, safety and simplicity of early feeding have been demonstrated. One approach is to slowly infuse tube feedings through the NGT at a rate of 10 to 20 mL/h. Although this

clearly does not meet the nutritional needs of adult patients, it is enough to protect the gut mucosa. Long feeding tubes can be placed into the small bowel using endoscopy or fluoroscopy and the rate steadily increased to meet the estimated calculated caloric requirements. The advantages of such tubes are higher and earlier rates of infusion and continuous feeding of patients during surgical procedures requiring general anesthesia. Patients with minor burns may be able to satisfy their caloric needs and fluid resuscitation through oral intake only.

Despite the theoretical advantages and need for the calories provided by enteral feeding, difficulties exist, and the technique cannot be used in all patients. Patients receive, on average, only 80% of the goal rate for enteral feedings because of frequent interruptions for patient care, including radiological procedures and surgery. This deficit increases when patients develop intestinal ileus, as typically occurs with major infection. Osmotic diarrhea is troublesome, particularly when the patient's feces soil the burn dressings. A variety of techniques combat diarrhea, including replacement of intestinal flora with lactobacillus granules and nonpasteurized yogurt, as well as retardation of small bowel motility with diphenoxylate hydrochloride.

The estimated caloric and protein needs of a patient may be met more reliably with parenteral than enteral feeding. The central venous catheter, which predisposes the patient to invasive infections (particularly infections with *Candida* species), is a disadvantage. Reports suggest that the rate of bacterial translocation is increased with the use of parenteral nutrition compared with enteral nutrition and that infection rates are higher.[10,20] Long-term use of parenteral nutrition alone is associated with hepatobiliary dysfunction, including cholestatic hepatitis and acalculous cholecystitis. Nevertheless, parenteral nutrition can be used for patients who do not tolerate enteral feedings because of paralytic ileus of the intestine or prolonged diarrhea.

Burn injury results in an increase in the metabolic expenditure. Initial investigative work performed in the 1970s demonstrated that some patients with burns needed as many as 7,000 or 8,000 kcal/d to maintain weight. Although patients with burns still become hypercatabolic after injury, they do not become so to the same degree because of changes in management. Because of the effect of earlier enteral feeding and the introduction of procedures that promote early wound closure (early and aggressive excision and grafting and the use of biological dressings), the increase in metabolic rate has diminished; healing does not fully begin until the wounds are closed. Indirect calorimetry has shown that the most severe injuries require no more calories than twice the resting energy expenditure (REE) as described in the Harrison-Benedict formula. The REE calculated by the Harrison-Benedict formula is multiplied by a stress factor in direct proportion to the size of the burn (Box 53-13). The stress factor is judged conservatively to avoid overfeeding, which is associated with increased susceptibility to infection. Although indirect calorimetry prevents gross underestimation or overestimation of the patient's caloric needs, in most patients, it is probably not superior to estimating their needs from a formula (such as the Harrison-Benedict formula) alone.

Wound repair depends on amino acids, which are the building blocks of proteins. The type of amino acids used in enteral feedings varies. The amino acids arginine and glutamine have immune-enhancing properties, improve nitrogen retention, and maintain lean body mass. Formulas containing arginine supplements have been reported to reduce infections in trauma patients and to reduce length of stay of critically ill patients.

BOX 53-13 Stress Factors for Energy Expenditure Related to Burn Size (Harrison-Benedict Equations)

Women:REE = 655 + [4.3 × Wt(lb)] + [4.3 × Ht(in)] – [4.7 × age]
Men:REE = 65 + [6.2 × Wt(lb)] + [12.7 × Ht(in)] – [6.8 × age]

TBSA (%)	Stress Factor
0–10	1.4
11–20	1.5
21–30	1.6
31–40	1.7
41–50	1.8
51–60	1.9
>60	2.0

REE, resting energy expenditure.

Judging the amount of protein necessary for recovery from burn injuries is difficult. Massive and unquantified loss of protein from the burn wound exudates precludes nitrogen balance studies based on urine excretion alone. Sequential measurements of serum proteins, such as transferrin and prealbumin, are a better index of the body's response to the amount and type of dietary protein given; however, few clinical studies show a correlation between an increase in serum proteins and improved clinical outcome. It is important to avoid overfeeding of protein because it predisposes patients to sepsis. Amounts of protein greater than 3 g/kg/d in adults are usually not tolerated because of azotemia. Dietary protein should be started at an administration rate of 1.2 g/kg/d and should be increased if there is not a subsequent increase in serum protein markers. A patient's diet can also be supplemented with vitamins A and C, and with the trace element zinc, all of which improve wound healing.

Successful weaning of patients from nutritional supplements sometimes occurs earlier than expected. A regular diet with liquid supplements is offered within 24 hours of extubation. The increased thirst of patients with burns is used to encourage the intake of protein-containing solutions, either soy- or milk-based supplements, or protein-containing fruit drinks. Using supplements, patients can take up to 2,000 kcal each day. It is preferable to feed patients or allow them to feed themselves because of the inherent risks of feeding tubes and central lines.

Providing Musculoskeletal Support

Physical and occupational therapy begins on day 1 of a burn injury. Independent of the patient's general condition, injured upper and lower extremities can be elevated to allow adequate venous drainage and reduce edema. Passive exercises are initiated and, if alert and cooperative, the patient should participate in these exercises. Active and passive exercises to maintain joint range of motion are continued throughout hospitalization and the outpatient rehabilitation period.

Two important axioms influence rehabilitation. First, the burn wound will shorten by contraction until it meets an opposing force. Across a flexor surface, this may result in a contracture. Second, the position of comfort is the position of contracture. Range-of-motion exercises prevent tendon shortening and restriction of joint motion by burn scar contractures. As patients begin to recover and participate actively in therapy, exercises are designed to increase muscle strength and endurance. A return to activities of daily living frequently takes months.

An unfortunate consequence of contractures and immobility is heterotopic ossification. Heterotopic ossification develops when there is an abnormal deposition of calcium phosphate crystals in joint spaces or along tendons. Heterotopic ossification restricts the motion of joints, particularly in elbows and knees. Unlike the heterotopic ossification seen in patients with spinal cord injuries, the heterotopic ossification seen in patients with burns does not respond to treatment with etidronate disodium, and early surgical removal is not indicated. Resolution occurs with time in most patients, and few need surgical removal of the ossified crystals in the joints.

Managing Pain

The pain associated with burns is managed aggressively. All narcotics are given intravenously because absorption of the drug is unpredictable when given intramuscularly or subcutaneously secondary to the hypermetabolic response and the fluid shifts.[6,19,21] Patients are given anxiolytics for anxiety related to appearance, procedures, and fear.[22] Patient-controlled analgesia (PCA) is ideal for patients who are awake, sufficiently oriented, and physically able to use the pump. PCA pumps can provide a continuous pain medication, with a "dose" available every 6 to 8 minutes for intermittent pain. The nurse can give the patient a "bolus" dose before procedures, such as dressing changes and physical therapy. Recommended narcotics include morphine, fentanyl, and hydromorphone.[21,22]

Caring for the Wound

CLEANSING. The wound protocols of all burn centers and hospitals vary, but the most common wound cleansing involves water and chlorhexidine or saline solution and povidone-iodine (Betadine). Wounds are cleansed at each dressing change and are observed for signs of infection and rate of healing.

Hydrotherapy is the preferred approach of most burn centers because the warm, flowing water is beneficial to help loosen exudates, clean and assess the wound, and provide range-of-motion exercise. The solutions used vary and may contain salt, povidone-iodine, and bleach. Because the procedure is usually painful, patients should receive an analgesic 20 to 30 minutes before beginning and small, frequent doses throughout as needed. In addition, the patient should receive a complete explanation of, and assistance with, pain-controlling techniques (eg, imagery, music therapy). Additional support should be offered by providing ongoing explanations of what is to be done and why and by permitting the patient to participate in care as much as possible. Limiting the time the procedure takes is important to the patient's pain tolerance and temperature control. Hydrotherapy should be limited to 20 minutes to prevent extreme chilling, which increases metabolic demand.

Care must be taken to avoid cross-contamination of wounds during bathing procedures. For this reason, many centers no longer immerse patients in Hubbard tanks. Portable shower trolleys with disposable liners can provide hydrotherapy without the risk for contamination. These may be used in a central shower room or in facilities so equipped, or used directly in the patient's room, reducing the risk for transporting the critically ill. Clean or healing wounds should be cleaned separately from contaminated ones.

APPLICATION OF TOPICAL ANTIMICROBIAL MEDICATIONS. The choice of topical antimicrobial medications depends on the wound depth, location, and condition and on the presence of specific organisms. Common antimicrobial drugs used from time of admission to a burn unit include silver sulfadiazine (Silvadene), mafenide acetate (Sulfamylon), 0.5% silver nitrate, nitrofurazone, povidone-iodine, bacitracin, gentamicin, and nystatin (Table 53-4). No single drug is totally effective against all burn wound infections. Treatment is guided by in vitro testing or in vivo results. Eschar and granulating wound surfaces may be cultured three times weekly to identify contaminating organisms and determine antibiotic sensitivity.

Silver sulfadiazine is the primary topical drug of choice on admission. The most common adverse reaction is transient leukopenia; therefore, serial CBCs must be monitored. If the WBC count falls below 3,000 cells/mm^3, the physician will probably prescribe another topical agent. When the leukocyte count returns to normal (4,000 to 5,000 cells/mm^3), silver sulfadiazine therapy may be reinstituted.[23]

If the colony counts increase, the topical agent of choice is usually mafenide acetate cream, an effective broad-spectrum bacteriostatic agent. Mafenide acetate diffuses through third-degree eschar to the burn wound margin within 3 hours of application. Patient discomfort is common because mafenide acetate may cause a burning sensation as it penetrates the eschar tissue, lasting 20 to 30 minutes after application. This agent inhibits carbonic anhydrase, resulting in metabolic acidosis. This acidosis initially is compensated for by hyperventilation. Oral administration of sodium citrate dihydrate (Bicitra) or IV sodium bicarbonate usually corrects this acid–base imbalance.

The application of topical antimicrobial drugs inhibits the rate of wound epithelialization and may increase the metabolic rate. Electrolyte imbalances (eg, sodium leaching by silver nitrate) and acid–base abnormalities may occur. The best topical drugs are water soluble because they do not hold in heat and macerate the wound. With the application of any topical drug, it is important to use sterile technique. Antimicrobial creams should be applied to a thickness recommended by the manufacturer and reapplied at the necessary frequency to maintain consistent coverage.

DÉBRIDEMENT. Eschar covers the burn wound until it is excised or has separated spontaneously. Small burn wounds may be allowed to separate on their own if the wound shows no signs or symptoms of infection, the patient is hemodynamically stable, or the situation does not allow for excision. In theory, burn wound management is simple. It calls for débridement of the eschar and skin graft closure before the eschar becomes infected. However, the sometimes serious

Table 53-4 Medications for Burn Wound Management

Agent	Advantages	Disadvantages	Nursing Considerations
Mafenide acetate	Broad-spectrum, penetrates eschar	Painful application, acid–base imbalances	Apply twice a day, leave open to air
Silver nitrate	Painless application, broad-spectrum, rare sensitivity	No eschar penetration, discolors wound and environmental surfaces, must be kept moist	Wet-to-wet dressing with nonadhering layer, followed by a gauze layer every 24 h
Silver sulfadiazene	Painless application, broad-spectrum, easy application	May cause transient leukopenia, minimal eschar penetration	Apply a moderate layer and wrap in a gauze dressing every 12 h
Bacitracin	Painless application, nonirritating	No eschar penetration, antimicrobial spectrum not as wide as above medications	Apply a thin layer and nonadhering dressing; if used on face, leave open to air
Mupirocin	Antimicrobial spectrum broader than bacitracin	Expensive	Apply a thin layer and nonadhering dressing; if used on face, leave open to air
Neomycin	Painless application	Antimicrobial spectrum not as wide as above medications	Apply a thin layer and nonadhering dressing; if used on face, leave open to air

systemic complications of burn injury, such as hypovolemia and sepsis, may delay this course of action significantly.

Mechanical Débridement. Mechanical débridement may be accomplished using forceps and scissors to gently lift and trim loose necrotic tissue. Another form of mechanical débridement is dressing the wound with coarse gauze in the form of wet-to-dry or wet-to-wet dressings. Wet-to-dry dressings consist of layers of moistened coarse mesh gauze. As the inner layer dries, it adheres to the wound, entrapping exudate and wound debris. The dressing should be removed at a 90-degree angle, and every effort should be made to avoid damaging fragile, newly granulating tissue. As the wound forms increasing amounts of granulation tissue, wet-to-wet dressings may be used to prevent desiccation and trauma. These dressings remain moist until the next dressing change. The dressing should be removed by first gently lifting from the edges toward the center of the wound and then removing the dressing at a 180-degree angle. This procedure prevents detachment of newly formed epithelial tissue.

Enzymatic Débridement. Enzymatic débridement involves the application of a proteolytic substance to burn wounds to shorten the time of eschar separation. Travase and Elase are the most commonly used agents. The wound is first cleaned and débrided of any loose necrotic material. The agent is then applied directly to the wound bed and covered with a layer of fine-mesh gauze. A topical antimicrobial agent is applied next, and the entire area is covered with saline-soaked gauze. The dressing is changed two to four times per day.

Enzymatic débridement has the advantage of eliminating the need for surgical excision; however, certain complications must be considered. Hypovolemia may occur as a result of excessive fluid loss through the wound. Hence, no more than 20% TBSA should be treated in this manner. Cellulitis and maceration of normal skin may occur around the wound periphery, and patients often complain of a burning sensation lasting 30 to 60 minutes after enzyme application.

Surgical Débridement. In surgical excision, the wound is excised to viable bleeding points while minimizing the loss of viable tissue. Early excision has contributed significantly to the survival of people with major burns. The open burn causes hypermetabolism and a stress response that is not corrected until wound closure occurs. Surgical excision should be done as soon as the patient is hemodynamically stable, usually within 72 hours.[24]

After excision is complete, hemostasis must be achieved. This may be accomplished by topical thrombin sprayed on the wound or application sponges soaked in a 1:10,000 epinephrine solution.[24] After removal of necrotic tissue, the exposed underlying structures must be dressed with a temporary or permanent covering to provide protection and prevent infection.

GRAFTS. The ideal substitute for lost skin is an autograft of similar color, texture, and thickness from a close location on the body. Sheets of the patient's epidermis and a partial layer of the dermis are harvested from unburned locations using a dermatome. These grafts, referred to as split-thickness skin grafts, can be applied to the wound as a sheet graft or mesh grafts.

In a sheet graft, the harvested skin is applied to the surgically excised area. It is usually covered with a petrolatum-based gauze dressing. Overexposed areas such as the face and hands, a sheet graft gives a more natural appearance than a mesh graft.

Grafts must be inspected frequently to ensure that fluid is not collecting underneath them. Fluid accumulation is prevented by rolling a cotton-tipped applicator over the graft to express any trapped fluid. The sheet graft should be "pie-crusted" to allow for the expression of fluid through the closest opening; this avoids rolling the fluid to the edge of the graft, increasing the risk for dislodging the grafted tissue. After adherence has begun, usually after 24 hours, the fluid may be removed with a very small-gauge needle (26-gauge) to avoid disrupting the adherence of graft.

In a mesh graft, the harvested skin is slit, and the graft is then placed on the burn site. The slits (or interstices) allow the skin to expand, providing for greater coverage and drainage and facilitating draping over uneven surfaces. Mesh grafts frequently have to be expanded to obtain maximal coverage from each piece of autograft. An expansion ratio of 1:2 or 1:4 is often practical. Sometimes ratios such as 1:6 or 1:7 are used to cover large burns when donor tissue is limited. With these larger ratios, the expanded autograft is covered with either cadaver skin allografts, synthetic skin (Biobrane, Winthrop Pharmaceuticals), or negative pressure dressings.[25] In addition to physically stabilizing the fragile mesh, the cover decreases evaporation, heat loss, and bacterial contamination.

Dressings are used after surgery to immobilize the grafted area and prevent shearing and dislodging of the graft. Postoperative dressings also provide a degree of compression to minimize hematoma and seroma formation, but they may be a source of vascular compression in the extremities. Pulse checks distal to the dressings are documented every 4 hours for 24 hours after surgery. The dressings are usually left in place until the third postoperative day. Until that time, the dressings are moistened every 6 hours with a solution containing normal saline and polymyxin. The antibiotic solution keeps the fragile meshed grafts moist and protects against infection. On postoperative day 3, the dressings are removed and evaluated by the physician, who determines the success of the grafting. This is expressed in terms of percentages. The grafted area is then covered with a nonadherent dressing and a gauze layer, which are secured with a gauze roll. All components of the dressing are moistened with the antibiotic solution.

Donor site care can vary with a variety of products used at burn centers and sometimes between burn surgeons. The donor site is covered during surgery with a single layer of fine-mesh gauze (eg, Scarlet Red, Biobrane, Acticoat).[25] Most products are kept in place until separation from the donor site begins. Positioning to prevent pressure on the site and to allow for drying is important. Daily inspection of the donor site is essential to detect early signs of infection or cellulitis.

A new technique that involves the growth and subsequent graft placement of cultured epithelial autografts has become an important adjunct to permanent coverage of extensive burn wounds. Biopsies are taken from unburned skin, and cells are cultured in the laboratory. Sheets of cultured epithelial cells are attached to petrolatum-impregnated gauze and applied to the wound. The cultured cells are highly fragile, and the surgeon may elect to place the patient in traction for increased protection of the grafted tissue. After 7 to 10 days, the gauze application is removed, and a nonadherent dressing is applied to prevent mechanical trauma.

Providing Psychological and Familial Support

Providing psychological support for the newly admitted patient with burns and the family is not the least of the many tasks facing the critical care nurse. The patient most often is awake and alert, although anxious and overwhelmed by the suddenness and magnitude of injuries. With high anxiety levels and lack of knowledge pertaining to burns, the family approaches the burn unit with fear, hesitancy, and sometimes hysteria. The physical appearance of the patient and the high-technology atmosphere of the burn unit are frightening. Preparing the family for the initial visit by explaining what to expect and escorting them to the bedside is extremely important. Visitors often are overwhelmed on the first visit and stand silently with increasing feelings of anxiety and hopelessness. Burn injuries are dramatic and are psychologically traumatic for the patient and for those who witnessed the accident. Counseling for the patient and the family begins on the day of admission. Families require constant support, and the burn team should plan weekly family meetings to discuss the patient's care plan and progress. This is often the all-important basis for establishing a trusting relationship for the long months of rehabilitation ahead. The trusting relationship that is established initially provides a strong base for patient and family teaching and rehabilitation in the months to follow. Critically ill patients with burns are likely to experience a series of small gains combined with intermittent setbacks, and this pattern does not stop until the burn wounds are closed, which can be 2 to 3 months after injury. The family needs to be informed and provided with the means to care for their own physical and psychological needs. Families of patients who were transferred from a great distance and who lack nearby support systems find the entire situation particularly stressful. The need to provide support for families cannot be overemphasized.

Patients with burns often become depressed and withdrawn, asking to be left alone and not to be made uncomfortable. The nurse should respond by making certain expectations of the patient clear. That is, the nurse should make it clear to the patient that he or she is to feed himself or herself, go to the bathroom, or do as much as his or her physical condition permits, while communicating to the patient that the situation is not hopeless and that recovery is expected.

The best way to handle regression in a patient with burns is to acknowledge it. First, the nurse must accept the fact that the patient may be unable to cope on an adult level and that the patient may be unstable emotionally and physically. Second, the nurse must devise ways to help the patient cope on an appropriate level. Interventions that usually help include following a regular schedule so that the patient knows what is expected, rewarding the patient for adult behavior, and permitting the patient as much control and choice as possible.

It is not uncommon for severely burned patients, and sometimes family members, to transfer their fears to a specific caregiver (physician, nurse, therapist) and to complain that they are being treated unjustly or unkindly. Working with a psychiatric liaison nurse may help the burn survivor recognize and deal with his or her fears more effectively and help the caregiver support the patient by responding therapeutically.

Hallucinations, confusion, and combativeness are common in severely burned patients for physical and mental reasons. Exhaustion, pain, and medications may distort reality and produce psychotic-like behavior.

Although the patient tends to concentrate on the present, the family members look to the future and want to know what to expect. Information about the patient's condition and treatments should be shared with them using an honest and open approach.

Rehabilitative Phase

Patients with extensive burns require many months for recovery and rehabilitation. Physical and psychological rehabilitation measures are begun in the ICU and continued throughout the recovery period.

Physical Rehabilitation

The diet of the patient with burns should remain high in protein until all wounds have healed. As healing takes place, the diet should be tapered to meet normal caloric requirements. The patient with burns may become accustomed to eating frequently and in large amounts. After healing is complete, metabolism returns to normal, and weight will be gained if eating habits are not controlled properly.

Prevention of Scarring and Contractures

Once regarded as inevitable, hypertrophic scarring and joint contractures are now largely preventable. Preventive measures start when the person is admitted to the hospital and continue for at least 12 months or until the scar is fully mature.

These preventive measures (ie, positioning the body and helping the patient perform range-of-motion exercises) are not new to the nurse. Positioning the body with the extremities extended is extremely important. Although tightly flexed positions are preferred by patients for comfort, they result in severe contractures. The range-of-motion exercises should be carried out with each dressing change or more often if indicated. Special splints are used to maintain arm, legs, and hands in extended, yet functional, positions. Later, when the wounds have healed sufficiently, the person is custom-fitted for a special pressure garment. By applying continuous uniform pressure over the entire area of the burn, the garment prevents hypertrophic scarring. The garment must be worn almost 24 hours a day for approximately 1 year. The smooth elastic garment forms a shield that permits the person to wear normal clothing and resume ordinary activities much sooner.

Healed and grafted skin is dry and tight. Itching is a major patient complaint as healing occurs. Massaging a mild, nonirritating lotion into the healed skin provides lubrication, aids in range of motion, and promotes circulation.

Psychological Rehabilitation

The burn survivor may have many psychological issues once discharge is nearing. The patient may have posttraumatic stress disorder, anxiety, depression, or a combination of these. To ensure that these issues are effectively managed, the multidisciplinary team caring for the patient with burns must include mental health professionals.

▲ Other Types of Injuries Treated in Burn Centers

Burn center referrals are not limited to the traditional burn injuries (thermal, chemical, electrical, or radiation). Patients with disease processes that primarily involve the integumentary system have had better outcomes in burn centers because the unit is structured with the high levels of necessary resources for patient care. Patients who are referred to burn centers may have such conditions as toxic epidermal necrolysis syndrome (TENS), erythema multiforme minor, and extensive Stevens-Johnson syndrome, as well as diseases that may result in the loss of large amounts of tissue, such as necrotizing fasciitis, staphylococcal scalded skin syndrome, bullous pemphigoid, or pemphigus vulgaris. Patients with significant cold injuries may also be routinely referred to regional burn centers in some areas of the country.

Toxic Epidermal Necrolysis Syndrome

Toxic epidermal necrolysis syndrome (TENS) is a superficial, exfoliative dermatitis that has been related to multiple sources, the most common being an adverse reaction to medications, the staphylococcal toxin, or viral infections.[26] The source may be undeterminable. TENS can involve the entire mucosal surface of the body to include the oral mucosa and conjunctiva as well as the vaginal or urethral linings. The immediate concern is the oral lesions and the possible occlusion of the respiratory system; intubation may be indicated as a supportive measure. The definitive diagnosis is made by sending a biopsy from the involved tissue for microscopic examination where an epidermal split is observed at the junction of the epidermis and dermis.

Patients with TENS are often described by the percentage of their body surface area that is currently open, requiring fluid and electrolyte replacement for evaporative water loss through the open wounds. This disease is similar in its effects to the body as a partial-thickness injury seen with a thermal burn. The lesions in TENS tend to be hypersensitive and extremely painful, with the nerve endings exposed to the environment. If the soles of the feet are involved, ambulation is extremely painful and may lead to debilitation if physical activity is not maintained. Nutritional support is also important to assist with the healing process; the insertion of a small-bore feeding tube may be indicated if caloric intake cannot be maintained secondary to oral lesions.

To increase patient survival, patients with TENS should be admitted to a burn center because of the complexity of wound care and the need for meticulous infection control standards. Historically, these patients have been treated with silver nitrate dressings, but more recently, a variety of products, such as Acticoat or biological dressings, have been used. The primary concern of wound care is that the dressings remain moist, not wet, at all times to decrease the desiccation of the dermal layer. Steroids are not indicated, and antibiotics used only for specific infections.

Necrotizing Fasciitis

Necrotizing fasciitis is a rapid and progressive inflammatory infection of the soft tissue that presents a diagnostic and therapeutic challenge. The pathogens enter the tissue through an open wound and spread rapidly in the extracellular space between the subcutaneous tissue and the fascia. It is classified into two types based on the culture findings. Type I is polymicrobial and accounts for 90% of the cases. Risk factors include postoperative status, obesity, diabetes, or older adulthood.[26] Type II accounts for 10% of all cases and typically affects the upper and lower extremities. The infecting agent is Group A beta-hemolytic streptococcus, with or without *Staphylococcus aureus*.[26,27] Diagnosis may be difficult initially because the external cutaneous damage may not be readily appreciated and the signs and symptoms may be diffuse. Early diagnosis is paramount, with timely and radical surgery being the definitive intervention. Necrotic tissue

must be completely excised and explored to clean tissue borders. Broad-spectrum antibiotics are started preoperatively, and culture and Gram stain are used to provide guidance for the antibiotic regimen. Local and systemic infection must be completely controlled before wound closure is addressed.[11]

As in burn injuries, infection control is the primary component of wound management in necrotizing fasciitis. The need for extensive excision of tissue to the depth of the fascial compartments can result in extreme contractures and the loss of the protective mechanisms of the subcutaneous tissue (eg, protection from shearing and blunt forces, fat storage, temperature regulation).[26]

Cold Injuries

Environmental exposure without appropriate protection may result in a cold injury. Although these injuries are more common in temperatures below freezing, their onset may occur also at relatively moderate temperatures depending on the length of the exposure and the person's condition at the time. Injuries may range from a localized frostbite to systemic lowering of the body's core temperature with hypothermia.

Frostbite is most often seen of the fingers, toes, and nose because they tend to be the most exposed to the environment with outdoor activity, and circulation is the most difficult to maintain in the microvasculature. With prolonged exposure, the intracellular and extracellular fluids of the body tissue become chilled and may eventually form ice crystals, impeding the blood flow to the area and leading to tissue destruction.[6]

Mild cases involve only the skin and subcutaneous tissues, whereas the more severe cases involve deeper structures. The symptoms range from numbness and itching to paresthesia and decreased motion.[6]

Tissue should not be rewarmed until the patient is in a controlled environment where the warmth can then be maintained. Rewarming the tissue also contains risks because this may release a shower of microemboli. In addition, it is also extremely painful. Purplish or bluish blisters are left intact, whereas clear or white blisters are débrided, similar to burn blisters. To preserve as much length as possible for functionality, amputation is discouraged until definitive demarcation occurs, which can take weeks to months.[6,9]

▲ Clinical Applicability Challenges

CASE STUDY

Mr. R. is a 35-year-old man who sustained burns while making repairs to his car. While parked in the garage during the repairs, the car engine caught fire. Mr. R. was able to get out from under the car and out of the garage unassisted. A neighbor witnessed the event and used the garden hose to extinguish the fire on the patient. The neighbor drove Mr. R. to the hospital. They arrived 25 minutes after the fire.

Mr. R. walked into the emergency department (ED) and the ED staff noted burns to chest, abdomen, bilateral circumferential arms, face, neck, and upper back. They estimated 55% total body surface area (TBSA) burned using the Rule of Nines. Mr. R. is a well-nourished, well-developed man in relatively good health. His preburn weight is 154 pounds (70 kg). He denies allergies and reports no significant past medical or surgical history. His only significant family medical history is a mother with type 2 diabetes mellitus. Mr. R denies using tobacco products or recreational drugs but reports drinking one case of beer per week for the past 5 years. He is unsure of having a tetanus vaccine. He is married with two young children.

The ED staff conducts a primary and secondary survey in a warmed trauma room and places 100% humidified oxygen by nonrebreather mask on the patient. Since the accident Mr. R. complains of severe pain to the skin of his face and arms. It is noted he is becoming hoarse and has burnt facial hair.

Based on his examination (facial burns, singed facial hair) and airway protection, Mr. R. is intubated. Two large-bore intravenous (IV) catheters are placed in bilateral upper extremities and an indwelling catheter is inserted. Fluid resuscitation is started, tetanus immunization is given, and analgesics are given in frequent small IV doses for pain.

The regional burn center is contacted and transportation arranged. Mr. R. is wrapped in clean, dry dressings with additional blankets to help insulate and maintain body temperature during the transport. Copies of all documents are sent with Mr. R. who arrives 3 hours after the burn occurred and he is directly admitted to the burn ICU. The airway is confirmed. All pulses are immediately assessed and present. The upper extremities are of concern due to circumferential burns and they are slightly diminished.

Mr. R.'s vital signs are heart rate 138 bpm, blood pressure 105/56 mm Hg, respirations (assisted by ventilator) 12 breaths/min, temperature 96.8°F (36.0°C). Breath sounds are auscultated and coarse rhonchi are heard. Heart sounds are normal and rapid. The monitor displays sinus tachycardia without ectopy. At admission, samples are obtained and sent to the laboratory for study. A chest

(continued on page 1206)

CASE STUDY (Continued)

x-ray is taken. Distal pulses on extremities with circumferential burns are assessed at least every hour. The upper extremities are checked more frequently because the pulses are diminished, and these are confirmed with Doppler ultrasound when not palpable. Bronchoscopy is performed, revealing erythema to the bilateral upper airways and no carbonaceous material; the bilateral lower airways appear to be uninjured. A fentanyl infusion and scheduled doses of lorazepam (Ativan) are given for analgesic relief and sedation, with additional dosing ordered for wound care and breakthrough pain.

Once stabilized, Mr. R. is taken to the warm shower room, and all wounds are manually débrided. Shower time is minimized to reduce the risk for hypothermia, a significant contributor to the mortality of trauma patients; a nasogastric tube is inserted and arterial lines and central lines are placed.

When resuscitation is well underway, the Lund-Browder chart is completed, and a burn size of 40% TBSA is calculated. Full-thickness burns (third-degree) account for 24% of the injuries; the burn area texture is leathery, and the surface is dry with thrombosed vessels. Prior to intubation, Mr. R. denied pain in these areas when touched or exposed to the air. The remaining partial-thickness burns are reddened, moist, and weeping serous fluid where thin-walled blisters have ruptured. When these are touched, Mr. R. has facial grimacing and blood pressure rises. To determine estimated fluid requirements in the first 24 hours from the time of the burn, the burn resuscitation formula is calculated:

$$2\text{mL} \times 70\text{kg} \times 40\%\,\text{TBSA} = 5{,}600 \text{ in first 24 hours;}$$
$$2{,}800 \text{ mL in first 8 hours}$$

Mr. R. received 900 mL fluid at the transferring facility and 560 mL fluid while in transport and arrival to the burn center, which means that he needs an additional 1,350 mL of fluid in the first 8 hours from the time of the burn. It is now 4 hours since the time of the burn, so the fluid is divided for the remaining 4 hours, or 340 mL/h. Since the placement of the urinary catheter at the transferring facility, urine output has been 180 mL, or an average of 45 mL/h. Although this is currently adequate, it will be closely monitored because it has been trending down.

After Mr. R. returns from the shower, the following topical agents are applied: silver sulfadiazine (Silvadene) cream to all partial-thickness and full-thickness burns, bacitracin ophthalmic ointment around eyes and bacitracin ointment to the rest of the face, and mafenide acetate (Sulfamylon) cream to the ears. The patient is placed on a pressure relief mattress to decrease the risk for pressure ulcers, with particular concern for the occiput, sacrum, and heels. The head of the bed is elevated to 30 degrees, and the bilateral upper extremities are placed in airplane slings to elevate hands and abduct the axilla.

Selected initial laboratory results are as follows: sodium, 146 mEq/L; potassium, 3.5 mEq/L; chloride, 107 mEq/L; blood urea nitrogen, 19.0 mg/dL; creatinine, 0.9 mg/dL; WBC count, 10,100 cells/mm^3; and hematocrit, 53.6%. Urine outputs for hours 6 and 7 from the time of the burn are 23 and 17 mL, respectively, and the IV rate of fluid administration is increased by 20% to 408 mL/h. Urine output for hour 8 is 25 mL and the IV rate of fluid infusion is increased by 20% to 490 mL/h.

Twelve hours after the burn, Mr. R.'s forearms are tight and edematous, and pulses in the upper extremities are faint and confirmed with a Doppler. Fluid resuscitation appears controlled; the patient is not hypotensive, with a blood pressure of 112/63 mm Hg. The heart rate has decreased within the expected range (122 bpm), and urinary output is adequate (43 mL in the last hour). Escharotomies are performed to the medial and lateral aspects of both arms and the dorsum of the hands. Pulses are immediately palpable at the conclusion of this procedure. A small-bore feeding tube is placed, and a nutrient-dense tube feeding is started and increased slowly as tolerated.

Albumin 5% is added to Mr. R.'s fluid resuscitation during the second 24 hours at 39 mL/h (0.3 mL/70 kg/44% TBSA for 30%–49% TBSA burn). The burn wounds are to be cleansed twice a day with 4% chlorhexidine gluconate (Hibiclens) soap. Alternating solutions are applied to all burn wounds except the face, with mafenide acetate (Sulfamylon) in the morning and silver sulfadiazine in the evening. Mafenide acetate is always applied to the ears, with bacitracin to the face and bacitracin ophthalmic around the eyes. Nursing and rehabilitative personnel provide passive range of motion to all major joints frequently, with particular attentiveness to the hands.

Mr. R. is taken to the operating room on postburn day 3 for a full excision and grafting. The donor sites are the areas of bilateral thighs that were not burned and the bilateral calves. These are covered with fine mesh (Xeroform) gauze. The arms, right thigh, and posterior torso are covered with 3:1 autograft and wrapped with a veil dressing and 5% mafenide acetate solution-moistened gauze. The posterior torso is bolstered to protect from shearing forces. The dressings are moistened with mafenide acetate solution every 6 hours and whenever necessary to avoid tissue desiccation. To maximize donor tissue, the anterior torso is excised and covered with the template Integra to close the wounds. The dorsa of the hands are covered with a split-thickness sheet graft and left open, and the palms are covered with Integra. The hands are splinted in a position of function. The sheet graft is observed hourly for the first 24 hours postoperatively; blebs are rolled to ensure that grafts adhere. After this time, small hematomas are aspirated via a tuberculin

(continued on page 1207)

CASE STUDY *(Continued)*

syringe. Donor sites are elevated on leg nets for exposure to the air. Heat lamps are used at low settings to assist with drying.

Mr. R. is weaned from the ventilator postoperatively and extubated the morning of postburn day 5. Aggressive pulmonary toileting is initiated to reduce the risk for pneumonia. He is started on vancomycin for burn wound cellulitis, which is stopped after 5 days when the cellulitis resolves. He is assisted to the bedside chair on postoperative day 5 (postburn day 8), and diet is advanced as tolerated. He is assisted to walk on postoperative day 6 (postburn day 9). When he tolerates 50% of his daily caloric needs orally, enteral feedings are changed to only night feedings and stopped when he tolerates more than 75%. He begins ambulating on postoperative day 11 (postburn day 14) and is transferred to the ward.

Mr. R. returns to the operating room on two more occasions for additional excision and grafting procedures and to cover the Integra templates when donor sites are ready for reharvesting. His wounds are healing with minimal graft loss and without difficulty. On postburn day 37, he is discharged to an inpatient rehabilitation facility with compression garments, for continued strengthening and stretching of contracted tissue. He is discharged from rehabilitation on postburn day 50. He is seen in the outpatient burn clinic for periodic followup and is readmitted to the burn center 6 months after the burn for surgical intervention of contracted tissue.

1. Discuss the pathophysiology of why fluid boluses are contraindicated and therefore not used during the resuscitation phase to treat marginally low hourly urine outputs. If the hourly urine output is exceeding the 0.5 mL/kg/h, how would you adjust the resuscitation infusion?

2. Discuss the different types of pain Mr. R. may be experiencing and how they may be treated.

3. Discuss how you would secure Mr. R.'s medical devices: endotracheal tube, NGT, and peripheral IVs inserted through partial thickness burns.

4. Discuss the signs and symptoms of ventilator compromise that would indicate the need for a chest escharotomy.

References

1. American Burn Association: Available at: http://www. ameriburn.org; accessed April 2010
2. American Burn Association: National burn repository 2010. Available at: http://www.ameriburn.org; accessed April 2010
3. Kochanek KD, Murphy SL, Anderson RN, et al: Deaths: Final data for 2002. Natl Vital Stat Rep 53(5):1–116, 2004
4. Hunt JL, Arnoldo BD, Purdue GF: Prevention of burn injuries. In Herndon DN (ed): Total Burn Care, 3rd ed. Philadelphia, PA: Saunders, 2007, pp 33–42
5. Pham TN, Gibran NS, Heimbach DM: Evaluation of the burn wound: management decisions. In Herndon DN (ed): Total Burn Care, 3rd ed. Philadelphia, PA: Saunders, 2007, pp 119–126
6. American Burn Association: Advanced Burn Life Support Course. Chicago, IL: Author, 2005.
7. Kagan RJ, Peck MD, Ahrenholz DH, et al: Surgical management of burn wound and use of skin substitutes. In American Burn Association: White Paper, 2009. Available at http://ww.ameriburn.org; accessed April 2010
8. Kramer GC, Lund T, Beckum OK: Pathophysiology of burn shock and burn edema. In Herndon DN (ed): Total Burn Care, 3rd ed. Philadelphia, PA: Saunders, 2007, pp 93–106
9. Cohen R, Moelleken BRW: Disorders due to physical agents. In Tierney LM, Papadakis MA, McPhee SJ (eds): Current Medical Diagnosis and Treatment, 44th ed. New York, NY: McGraw-Hill, 2005
10. LaBorde PJ: Management of patients with burn injury. In Smeltzer SC, Bare BG (eds): Brunner and Suddarth's Textbook of Medical-Surgical Nursing, 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2004, pp 1703–1745
11. Hedman TL, Quick CD, Richard RL, et al: Rehabilitation of burn casualties. In Pasquina PF, Copper RA (ed): Care of the Combat Amputee. District of Columbia: Borden Institute, 2009, pp 277–379
12. Warden GD: Fluid resuscitation and early management. In Herndon DN (ed): Total Burn Care, 3rd ed. Philadelphia, PA: Saunders, 2007, pp 107–118
13. Traber D, Herndon D, Enkhbaatar P, et al: The pathophysiology of inhalation injury. In Herndon DN (ed): Total Burn Care, 3rd ed. Philadelphia, PA: Saunders, 2007, pp 248–261
14. Cancio LC: Airway management and smoke inhalation injury in the burn patient. Clin Plastic Surg 36(4): 555–567, 2009
15. Van Meter KW: Carbon monoxide poisoning. In Tintinalli JE, Kelen GD, Stapcznski JS (eds): Emergency Medicine: A Comprehensive Study Guide, 6th ed. New York, NY: McGraw-Hill, 2004
16. Sterner JB, Zanders TB, Morris MJ, et al: Inflammatory mediators in smoke inhalation injury. Inflamm Allergy Drug Targets 8(1): 63–69, 2009
17. Greenhalgh DG, Saffle JR, Holmes JH, et al: American Burn Association consensus conference to define sepsis and infection in burns. J Burn Care Res 28(6):776–790, 2007
18. Cancio LC, Pruitt BA: Inhalation injury. In Tsokos GC, Atkins JL (eds): Combat Medicine: Basic and Clinical Research in Military, Trauma, and Emergency Medicine. Totowa, NJ: Humana Press, 2002, pp 325–349
19. Mlcak RP, Buffalo MC: Pre-hospital management, transportation, and emergency care. In Herndon DN (ed): Total Burn Care, 3rd ed. Philadelphia, PA: Saunders, 2007, pp 81–92
20. Hart DW, Wolf SE, Chinkes DL, et al: Effects of early excision and aggressive enteral feeding on hypermetabolism, catabolism, and sepsis after severe burn. J Trauma 54(4):755–764, 2003
21. Connor-Ballard PA: Understanding and managing burn pain: Part 1. Am J Nurs 109(4):48–56, 2009
22. Connor-Ballard PA: Understanding and managing burn pain: Part 2. Am J Nurs 109(5):54–62, 2009
23. Bessey PQ: Wound care. In Herndon DN (ed): Total Burn Care, 3rd ed. Philadelphia, PA: Saunders, 2007, pp 127–135
24. Orgill D: Excision and skin grafting of thermal burns. N Engl J Med 360(9):893–901, 2009

25. Muller M, Gahankari D, Herndon D: Operative wound management. In Herndon DN (ed): Total Burn Care, 3rd ed. Philadelphia, PA: Saunders, 2007, pp 177–195
26. Fagan S, Spies M, Hollyoak M, et al: Exfoliative and necrotizing diseases of the skin. In Herndon DN (ed): Total Burn Care, 3rd ed. Philadelphia, PA: Saunders, 2007, pp 554–565
27. Astorino T, Genrich I, MacGregor L et al: Necrotizing fasciitis early detection may save your patient's limb. Orthop Nurs 28(2): 70–76, 2009.

PART 13

MULTISYSTEM DYSFUNCTION

54 Shock, Systemic Inflammatory Response Syndrome, and Multiple Organ Dysfunction Syndrome

Kathryn T. Von Rueden, Emily Smith Des Champs, and Karen L. Johnson

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Describe common pathophysiological processes involved in the generalized shock response.
2. Compare and contrast the etiology and clinical manifestations of the major types of shock.
3. Explain the anticipated management and rationale for treatment of the various shock states.
4. Describe patients at risk for development of shock and complications associated with the various shock states.
5. Discuss nursing management principles for patients with shock, systemic inflammatory response syndrome, and multiple organ dysfunction syndrome.

Under normal conditions, oxygen delivery to the cells is sufficient to meet metabolic needs. Under stress, oxygen requirements of cells, tissues, and organs increase. Oxygen is consumed more rapidly, and compensatory mechanisms are initiated to meet the increased oxygen demands and restore perfusion to cells. The compensatory mechanisms are the same, regardless of the clinical condition causing the cellular hypoperfusion. Clinical conditions that result in cellular hypoperfusion are often referred to as shock states.

▲ Pathophysiology of Shock

Although shock states have different causes and different clinical presentations, some features, such as hypoperfusion, hypercoagulability, and activation of the inflammatory response, are common to all shock states. Once a shock state develops, the subsequent course may have more to do with the physiological response to shock, including activation of the sympathetic nervous system, the inflammatory response, and the immune system, rather than with the initial cause of the shock. Thus, shock can be considered as a derangement of compensatory mechanisms that results in further circulatory and respiratory dysfunction with subsequent multiple organ damage.

Tissue Oxygenation and Perfusion

Oxygenation of all organs and tissues is directly related to the cellular oxygen demands, adequacy of oxygen supply to

meet demand, cellular extraction of oxygen from the blood, and the ability of the cells to use oxygen. The pulmonary system functions to provide for the diffusion of oxygen into the blood. Oxygen binds with hemoglobin in the pulmonary capillaries to form oxyhemoglobin to carry oxygen to tissues. This is measured as arterial oxygen saturation (SaO_2). The cardiovascular system transports oxygenated blood to the cells for metabolism. Typically, cells consume about 25% of the oxygen delivered; this utilization of oxygen is referred to as oxygen consumption (VO_2). Oxygen delivery (DaO_2) is the amount of oxygen transported to the cells every minute. Chapter 17 reviews how these oxygen parameters are calculated.

Under normal conditions, VO_2 is independent of DaO_2. This means that when cells need to consume additional oxygen to produce energy, they can extract the necessary amount required to produce energy in the form of adenosine triphosphate (ATP). However, during times of physiological stress, VO_2 becomes dependent on DaO_2.[1,2]

Initial respiratory, endocrine, and circulatory compensatory mechanisms respond to the cells' need for oxygen by increasing DaO_2 (eg, by increasing respiratory rate, cardiac output (CO), antidiuretic hormone (ADH) release, and renin–angiotensin aldosterone activity). If additional oxygen is required, and the cells cannot extract the oxygen, they must use anaerobic metabolism to produce ATP. Anaerobic metabolism is not an efficient method of energy production, and the ATP produced is insufficient to meet cellular demands. Moreover, anaerobic metabolism produces lactic acid as a by-product and can result in metabolic acidosis. If oxygen continues to be insufficient to meet cellular demands for energy, cell death ensues. As more cells die, tissues and organs become progressively dysfunctional.[2]

During shock states, oxygen is consumed at a much greater rate than it is delivered. Oxygen supply is insufficient to meet oxygen demand, resulting in cellular hypoxia and dysfunction. To meet the increased need for cellular VO_2, the DaO_2 must be increased. Although it is not possible to manipulate cellular VO_2 directly, many interventions can be implemented to manipulate and increase DaO_2. In shock states, the primary goal is to maximize DaO_2 to meet cellular oxygen requirements in an ongoing effort to prevent tissue and cell death and maintain end-organ perfusion.

Compensatory Mechanisms

Cellular perfusion depends on the synergy of multiple physiological processes. The pulmonary, endocrine, and circulatory systems maintain an intricate balance of oxygen exchange and delivery to the cells by generation of an adequate oxygenated blood supply and CO (Fig. 54-1). The autonomic nervous system assists in the orchestration of this delicate balance.

During states of hypoxia, activated compensatory mechanisms increase the depth and rate of respiration. The

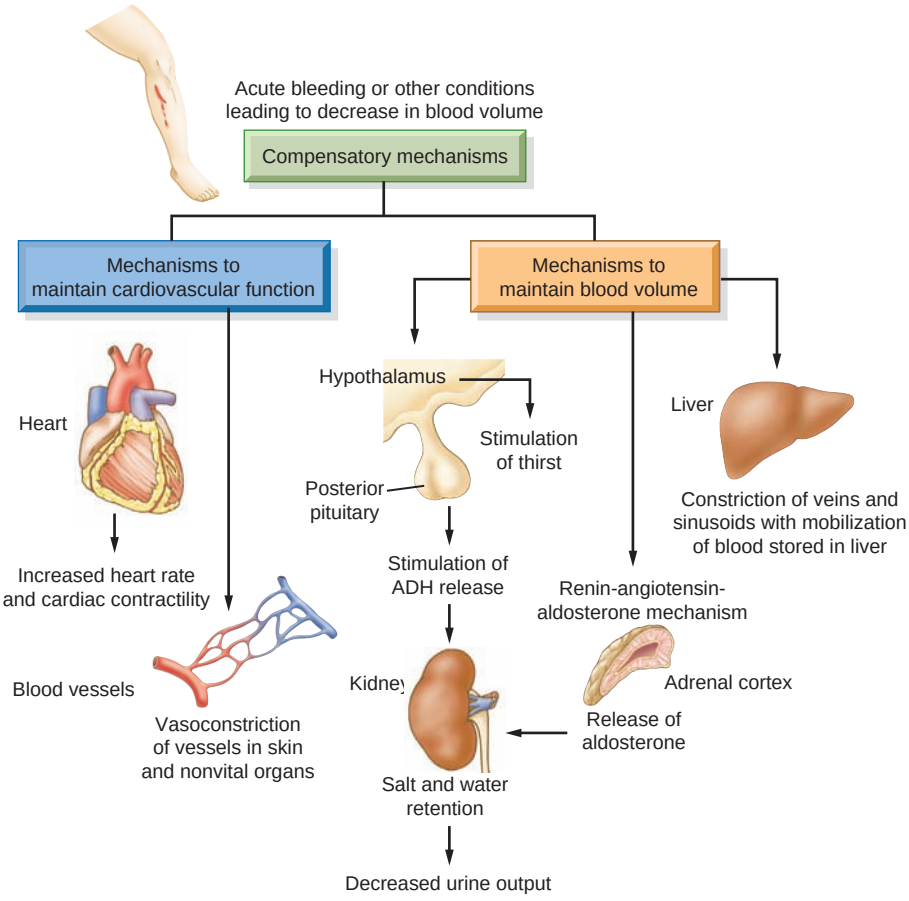


FIGURE 54-1 ▲ Compensatory mechanisms used to maintain circulatory function and blood volume in hypovolemic shock. ADH, antidiuretic hormone. (From Porth CM: Essentials of Pathophysiology: Concepts of Altered Health States, 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2011, p 503.)

cardiovascular system increases CO to increase oxygen delivery to the cells. During states of low perfusion (low blood pressure), compensatory mechanisms are initiated and result in increases in heart rate, systemic vascular resistance (SVR), preload, and cardiac contractility in an effort to restore appropriate circulatory volume. Chapters 16 and 17 provide a discussion of these terms. The fall in systemic blood pressure activates a series of neurohormonal responses aimed at reestablishing sufficient CO and perfusion to vital organs. The fall in blood pressure results in decreased stimulation of baroreceptors and eventually an increase in sympathetic response.

Continued sympathetic stimulation causes an increased heart rate and contractile force, increasing the CO. Arteriolar vasoconstriction (increased SVR) increases blood pressure and also shunts blood from less vital organs such as the stomach and intestines to vital organs, such as the heart, lungs, and brain. Preload and subsequently stroke volume and CO are increased by venoconstriction. The kidneys respond to sympathetic stimulation and local hypoperfusion by activating the renin–angiotensin system, which increases vasoconstriction of the arterioles and veins, increasing SVR and blood pressure. Activation of the renin–angiotensin system also stimulates the adrenal cortex to release aldosterone, which acts on the kidney to conserve sodium and water, increasing circulating volume. A drop in blood pressure also causes the pituitary gland to release ADH. ADH stimulates water and sodium retention by the kidney, further increasing preload. An increase of preload (from multiple sources) increases stroke volume, thereby increasing CO and blood pressure. Collective compensatory responses act together to increase the body's circulating volume, blood pressure, and CO to provide perfusion and oxygen to the cells[2] (Fig. 54-2).

The goal in treating patients in shock states is to reestablish perfusion to provide adequate oxygen to meet the needs to the cells as quickly as possible. Early recognition of signs of shock and ongoing assessments guide therapeutic interventions. The nurse plays a key role in the ongoing assessment of shock. The patient's clinical presentation depends on the cause of the shock state and degree of compensation as discussed later in this chapter. Clinical assessment parameters should be evaluated frequently to monitor the progression of shock state and the effectiveness of interventions. Traditional assessment parameters include altered level of consciousness, tachypnea, arterial blood gases (PaO_2, $PaCO_2$, SaO_2), tachycardia, hypotension, decreased urine output, and metabolic acidosis (base deficit and serum lactate levels), which are commonly found in all states of hypoperfusion. Recent advances in technology provide methods for earlier assessment of the patient's tissue oxygenation and perfusion using measures of gastric pH, sublingual end-tidal carbon dioxide, and central mixed venous oxygen saturation ($ScvO_2$) (refer to Chapter 17). These technologies allow for earlier assessment and detection of shock and metabolic acidosis.[3]

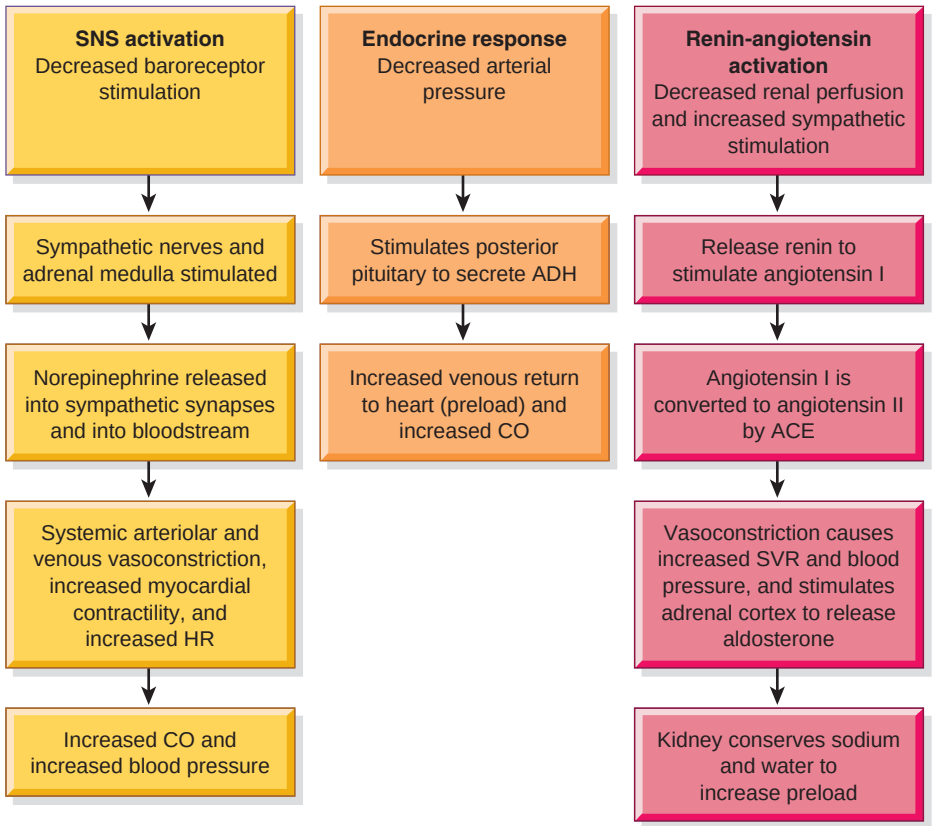


FIGURE 54-2 ▲ Compensatory mechanisms in shock. ACE, angiotensin-converting enzyme; ADH, antidiuretic hormone; CO, cardiac output; HR, heart rate; SNS, sympathetic nervous system.

▲ Systemic Inflammatory Response Syndrome

The progression of shock states involves systemic activation of the inflammatory response. The inflammatory response has both protective and potentially detrimental effects. The latter results in damage to tissues and organs. The term systemic inflammatory response syndrome (SIRS) is used to describe patients in whom the inflammatory response is fully and systemically activated. Efforts have been made to identify patients in whom this systemic reaction is occurring, with the thought that prompt, effective intervention might prevent progression of the shock to the irreversible stage. SIRS is manifested by two or more conditions listed in Box 54-1.[4] The SIRS criteria are sometimes used as the triggers for initiating rapid response teams. (Refer to Chapter 14.)

Etiology

SIRS may be caused by any type of shock or other insults such as massive blood transfusion, traumatic injury, brain injury, surgery, burns, and pancreatitis and typically precedes septic shock.[5] Thus, SIRS criteria should be evaluated in any patient with shock or any condition that might lead to shock. Normally, the inflammatory response is an essential, tightly regulated, and controlled protective mechanism of local response to invasion by microorganisms or to local tissue damage. However, in SIRS, this usually local inflammatory response becomes a systemic response that results in an unregulated inflammatory response with widespread involvement of endothelial cells and a generalized activation of inflammation and coagulation.

BOX 54-1 Clinical Terminology: SIRS, Sepsis, and Organ Failure

Bacteremia: The presence of viable bacteria in the blood

Hypotension: A systolic blood pressure (SBP) of less than 90 mm Hg or a reduction of more than 40 mm Hg from baseline in the absence of other causes for hypotension

Infection: Pathological process caused by the invasion of normally sterile tissue, fluid, or body cavity by pathogenic or potentially pathogenic organisms.

Systemic inflammatory response syndrome: The systemic inflammatory response that can be triggered by a variety of infectious and noninfectious conditions. The response is manifested by two or more of the following conditions:

- Temperature greater than 38°C or less than 36°C (>100.4°F or <96.8°F)
- Heart rate more than 90 beats/minute
- Respiratory rate more than 20 breaths/min or $PaCO_2$ less than 32 mm Hg (<4.3 kPa)
- WBC count greater than 12,000 cell/mm^3, less than 4,000 cells/mm^3, or greater than 10% immature (band) forms

Sepsis: SIRS plus a known or suspected infection

Severe sepsis: Sepsis associated with organ dysfunction, hypoperfusion, or hypotension. Hypoperfusion and perfusion abnormalities may include, but are not limited to, lactic acidosis, oliguria, or an acute alteration in mental status.

Septic shock: Sepsis with hypotension, despite adequate fluid resuscitation, along with the presence of perfusion abnormalities that may include, but are not limited to, lactic acidosis, oliguria, or an acute alteration in mental status. Patients who are on inotropic or vasopressor agents may not be hypotensive at the time that perfusion abnormalities are measured.

Multiple organ dysfunction syndrome: Presence of altered organ function in an acutely ill patient such that homeostasis cannot be maintained without intervention.

From Levy MM, Fink MP, Marshall JC, et al: 2001 SCCM/ESICM/AACP/ATS/SIS International sepsis definitions conference. Crit Care Med 31: 1250–1256, 2003.

Pathophysiology

Activation of the inflammatory response causes the release of various cytokines from macrophages. Examples of proinflammatory cytokines include tumor necrosis factor-α (TNF) and interleukin-1 (IL-1). Under normal circumstances, there are tight junctions between the endothelial cells that line blood vessels. However during proinflammatory states, these cytokines break these tight junctions, causing endothelial cells to separate. This increases capillary permeability and allows plasma to leak into the interstitial spaces. As a result of endothelial cell separation and exposure of the subbasement membrane, coagulation processes are stimulated. Platelets aggregate and adhere to endothelial cells and the subbasement membrane to form platelet plugs. The coagulation cascade is also activated. Fibrin, the end product of the coagulation cascade, forms strands around the clot to give it stability and strength. Proinflammatory cytokines also attract phagocytic white blood cells (WBCs) to the area and activate the complement cascade. The combination of activity of WBCs and complement proteins may result in elimination of the invading microorganism.[2]

Endothelial cells that line blood vessels are central to the development of a local inflammatory response. Important functions of endothelial cells include providing an anticoagulant surface and controlling permeability of vessels.[6] In a local inflammatory response, endothelial cells near the site of inflammation become activated as a result of mediators released by injured tissue cells. The activated endothelial cells express cell surface proteins that attract platelets and neutrophils. A procoagulant endothelial surface is formed in the area. Microthrombi form in the capillaries and obstruct blood flow.[6]

WBCs, platelets, and activated endothelial cells release vasodilating substances such as nitric oxide (NO), histamine, and bradykinin. These substances also promote additional capillary leak from blood vessels, resulting in additional extravasation of plasma and coagulation factors.

In SIRS, the inflammatory response is systemic; it occurs throughout the body. The result is an overwhelming, unregulated inflammation with uncontrolled coagulation, disruption of capillaries and intravascular volume loss, maldistribution of circulating volume, and oxygen supply and demand imbalance.[2] Endothelial cells are activated in many vessels throughout the body, causing widespread extravasation of fluid into the interstitial compartment and systemic activation of the immune system and coagulation cascade (Fig. 54-3).

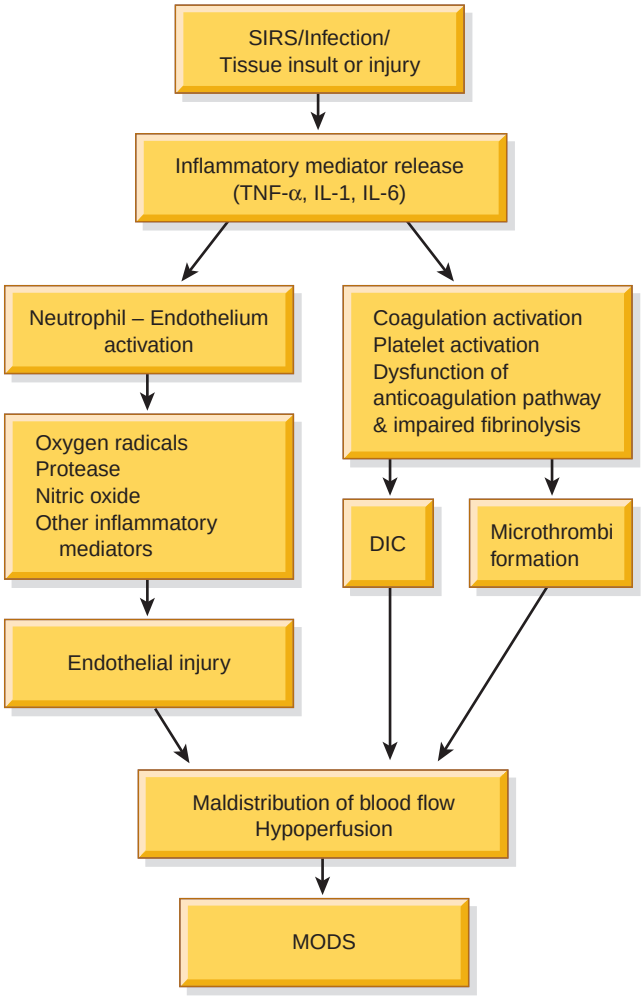


FIGURE 54-3 ▲ Inflammation, coagulation, and impaired fibrinolysis in systemic inflammatory response syndrome and multiple organ dysfunction syndrome. TNF-α, tumor necrosis factor-alpha; IL, interleukin; DIC, disseminated intravascular coagulation; MODS, multiple organ dysfunction syndrome.

There are substantial extravascular fluid accumulation and microthrombi formation in capillaries and in the interstitium. The combination of intravascular coagulation and decreased circulating blood volume results in reduced perfusion of vital organs, increasing the likelihood of multiple organ dysfunction syndrome (MODS) and death.

Events surrounding the complex interactions of the mediators of SIRS remain an active area of clinical research. Several mediators are believed to play a key role in the maldistribution of blood flow and oxygen delivery and consumption imbalance associated with SIRS and sepsis. Table 54-1 lists the key mediators of SIRS and summarizes their activity.

▲ Stages of Shock

Shock is believed to progress through three increasingly severe stages, the last of which cannot be reversed by known means. It is difficult to determine the stage of shock in a particular person at a particular time for three reasons: (1) shock has diverse causes, (2) the exact time of onset is unknown in many cases, and (3) diagnostic tests that give a clear-cut measurement of the extent of shock at a given time are lacking. Nevertheless, the stages are useful because they allow shock to be viewed as a progressive, rather than a static, process. Timely reversal of the shock state can prevent development of multiple organ failure and death.[2]

In the initial, nonprogressive stage (stage 1), the previously described compensatory mechanisms are effective in maintaining relatively normal vital signs and tissue perfusion. During stage 1, shock is poorly diagnosed and frequently goes unrecognized. However, if the cause of the shock is successfully recognized and treated, the patient may make a full recovery.

In the intermediate, progressive phase (stage 2), compensatory mechanisms that maintain normal perfusion begin to fail, metabolic and circulatory derangements become more pronounced, and activation of the inflammatory and immune responses may fully develop. At this point, interventions that target both the cause of the shock and the resultant metabolic, circulatory, and inflammatory responses may result in salvage of the patient. At this time, signs of failure in one or more organs may become apparent.

In the final, irreversible stage (stage 3), cellular and tissue injury is so severe that correction of metabolic, circulatory, and inflammatory derangements is difficult or impossible, and cellular hypoxia and death ensue. MODS develops, often resulting in the demise of the patient. This is discussed in the latter section of this chapter.

As previously mentioned, any shock state can trigger SIRS and if left unrecognized and untreated, can cause MODS. Understanding the classification, etiology, and clinical presentation of shock enables clinicians to more rapidly identify and manage shock, thus improving the patient's likelihood of survival.

▲ Classification of Shock

The causes of shock can be classified as hypovolemic, cardiogenic, and distributive. Hypovolemic and distributive shock result in inadequate venous return to the heart, while cardiogenic shock is caused by the failure of the heart to pump effectively. Inadequate venous return may result from hypovolemia (dehydration, hemorrhage) or widespread vasodilation (sepsis, anaphylaxis, or loss of sympathetic tone with a spinal cord injury), which cause a relative hypovolemia. Pump failure may result from myocardial infarction, abnormal heart rate or rhythm, or impaired diastolic filling.[7]

Hypovolemic Shock

Etiology

Hypovolemic shock is a result of inadequate circulating volume. Most commonly, hypovolemic shock is caused by sudden blood loss or severe dehydration. Some injuries, such as burns, cause significant fluid shifts from the intravascular space to the interstitial space, resulting in hypovolemia. See Chapter 53 for a discussion of care for patients with burns. Volume disorders in critically ill patients may be classified as either depletion or expansion disorders and involve both

Table 54-1 Mediators of the Inflammatory/Immune Responses

Mediator	Description of Activity	Clinical Response
Endotoxin	• Activates complement system and coagulation cascades • Activates macrophages, which release TNF and IL-1	• Increased microvascular permeability, vasodilation, third spacing, microthrombi formation • Inflammatory response
Tumor necrosis factor (TNF)	• Released by monocyte–macrophages • Multiple effects locally and systemically • Stimulates other mediator activity	• Hypotension, tachycardia, myocardial depression, tachypnea, hyperglycemia, metabolic acidosis, third spacing, fever, microvascular vasoconstriction
Interleukin-1 (IL-1)	• Released by monocyte–macrophages • Stimulates leukocytosis • Triggers production of acute phase proteins and release of amino acids from skeletal muscle • Activates procoagulant activity • Decreases vascular responsiveness to catecholamines	• Increased white blood cells (WBCs) • High urinary nitrogen excretion and muscle wasting • Elevated coagulation laboratory values • Decreased systemic vascular resistance, which is not as responsive to low dosages of vasopressor or synthetic catecholamine agents
Interleukin-6 (IL-6)	• Released by monocytes, helper T cells, and macrophages • Increases inflammatory response • B-cell stimulation and differentiation • Synergistic with IL-1	• Fever • Antibody secretion
Complement cascade	• Inflammatory process • Opsonization and lysis of foreign particles and cells • Stimulates neutrophils (and oxygen radicals) and IL-1 • Degranulation of mast cells and basophils	• Edema formation, vasodilation, vascular permeability, third spacing • All effects of IL-1
Platelet aggregating factor	• Released by mast cells, basophils, macrophages, neutrophils, platelets, and damaged endothelium • Increases platelet aggregation • Increases neutrophil adhesion • Increases vascular permeability and bronchoconstriction • Negative inotropic effects on the heart	• Microthrombi formation interfering with perfusion • Third spacing • Bronchoconstriction, rhonchi and wheezes, increased pulmonary airway pressures • Decreased heart contractility and force, which is not as responsive to low dosages of vasopressor and inotropic agents
Arachidonic acid (AA) metabolites	• Stimulation of AA causes the release of metabolites prostaglandins (PG), thromboxanes (TX), and leukotrienes (LT). • PGF and TXA2 cause pulmonary hypertension, vasoconstriction, and platelet activation and aggregation. • PGE, PGD, and prostacyclin cause vasodilation and decreased platelet aggregation. • Leukotrienes increase neutrophil chemotaxis, vascular constriction, and vascular permeability. • Increase gastric permeability to Gram-negative bacteria • Inhibits leukocyte adhesion and platelets	• Oxygenation and ventilation difficulties, increased airway resistance, wheezing • Third spacing and edema formation • Vasodilation, increased capillary permeability, and hypotension
Oxygen radicals	• Generate metabolites (O_2-, H_2O_2, OH-) during the respiratory burst of the neutrophils • Damage cell structure and interfere with cell activities • Damage endothelial cells, which stimulate the coagulation system • Increase permeability	• Inflammatory response, edema formation, fever • Microthrombi formation • Third spacing

intracellular and extracellular compartments. Acute fluid volume loss does not allow the normal compensatory mechanisms to restore an appropriate circulating volume rapidly enough. If left untreated, hypovolemia may lead to a variety of secondary complications, such as hypotension, electrolyte, and acid–base disturbances, and organ dysfunction resulting from hypoperfusion (Fig. 54-4).

Pathophysiology

A sudden loss of intravascular volume decreases venous return to the heart and results in reduced CO. Compensatory mechanisms are initiated to increase the circulating volume through the activation of the sympathetic nervous system and neurohormonal responses (see Fig. 54-1, p. 1210). If the condition persists, existing blood volume is shunted to the vital organs (heart, lungs, and brain), causing hypoperfusion to such organs as the liver, stomach, and kidneys. If volume is not replaced, compensatory mechanisms eventually become ineffective. The failure of the compensatory mechanisms to restore adequate circulating volume causes cellular hypoperfusion and inability to meet cellular oxygen requirements for metabolism. The cells must use anaerobic metabolism in an effort to meet their ATP requirements; this results in lactic acidosis.

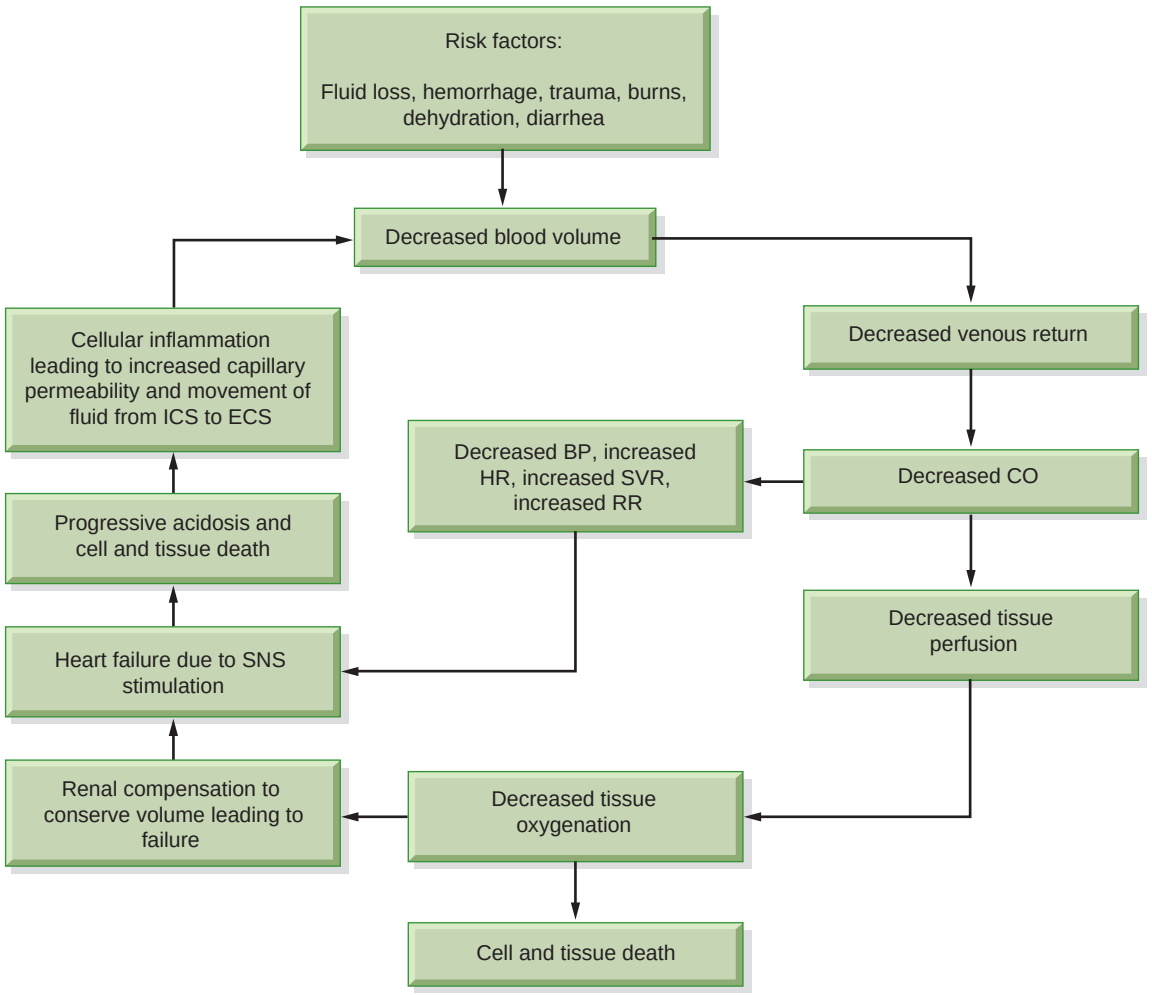


FIGURE 54-4 ▲ Hypovolemic shock. BP, blood pressure; CO, cardiac output; ECS, extracellular space; HR, heart rate; ICS, intracellular space; RR, respiratory rate; SNS, sympathetic nervous system; SVR, systemic vascular resistance.

Failed compensatory mechanisms, which were initiated to restore CO, eventually cause the myocardium to fatigue. Sympathetic stimulation to increase heart rate, contractility, and SVR escalates the workload of the heart. Ejection of a higher volume of blood against a higher SVR requires utilization of more oxygen and energy. Such stress on the heart causes an increase in myocardial metabolism and myocardial oxygen consumption ($M\dot{v}O_2$). The continued lack of circulating volume prevents appropriate oxygen delivery to the heart, creating a vicious cycle. Inability of the circulatory system to provide end-organ perfusion with enough oxygen forces conversion to anaerobic metabolism to meet cellular energy needs. Anaerobic metabolism cannot provide enough ATP to meet energy demands; thus, ischemic damage may ensue. If the situation continues, end-organ failure may occur (see Fig. 54-4).

Assessment

Clinical findings are directly related to the severity and acuity of volume loss (Table 54-2). Some patients, especially older patients or those who have chronic diseases, have more subtle compensatory responses, which may be overlooked. Box 54-2 lists considerations in older patients. Serial assessments of physical and laboratory findings may uncover trends that guide treatment and prevent vascular collapse.

HISTORY. A thorough history of the patient's presenting problem may reveal risk factors for hypovolemic shock. Patients experiencing significant blood loss because of gastric hemorrhage or liver or splenic rupture from trauma require a rapid replacement of circulating volume to prevent the consequences of hypovolemia.

PHYSICAL FINDINGS. Patients with hypovolemic shock have the following signs and symptoms caused by poor organ perfusion:

- Altered mentation, ranging from lethargy to unresponsiveness
- Rapid and deep respirations, which gradually become labored and shallower as the patient's condition deteriorates
- Cool and clammy skin, with weak and thready pulses
- Tachycardia from activation of the sympathetic nervous system
- Hypotension
- Decreased urine output; urine is dark and concentrated because the kidneys are conserving fluid.

Table 54-2 Correlation of Clinical Findings Associated With Volume Loss in Hypovolemic Shock

Estimated Blood Loss	Clinical Findings
<500 mL	None
500–1,000 mL	• Tachycardia (↑HR > 20% of patient's baseline) • Hypotension (↓SBP > 10% of patient's baseline) • ↓Urine output • Pulses weaker • Skin and extremities cool to touch • Hemodynamics: within normal limits CO, ↑SVR • Mild acidosis (↑base deficit, ↑lactic acid, ↓gastric pH)
1,000–2,000 mL	• Tachycardia (↑HR > 20%–30% of patient's baseline) • Hypotension (↓SBP > 10%–20% of patient's baseline) • Tachypnea (↑RR > 10% of patient's baseline) • Oxygen saturation may not be altered dependent on the percentage of exogenous oxygen the patient is receiving • SvO_2 < 60% • ↓Urine output (<30 mL/h) • Altered level of consciousness: restlessness, agitation, confusion, or obtunded • Cool, diaphoretic skin • Poor peripheral pulses • Hemodynamics: ↓CO, ↑SVR • Progressive acidosis (↑base deficit, ↑lactic acid, ↓gastric pH)
2,000–3,000 mL	• Tachycardia (↑HR > 20%–30% of patient's baseline) • Hypotension (↓SBP > 10%–20% of patient's baseline) • Tachypnea (↑RR > 10%–20% of patient's baseline) • ↓Oxygen saturation • SvO_2 < 55%–60% • Oliguria → anuria • Mental stupor • Marked peripheral vasoconstriction: cold extremities, poor peripheral pulses, pallor • Hemodynamics: ↓CO, ↑SVR • Severe acidosis (↑base deficit, ↑lactic acid, ↓gastric pH)

SBP, systolic blood pressure; SVR, systemic vascular resistance; CO, cardiac output; RR, respirations; HR, heart rate.

LABORATORY STUDIES. Useful laboratory studies include determinations of serum lactate, arterial pH, and base deficit to assess the presence of anaerobic metabolism. Test results can be used to measure the effectiveness of fluid replacement therapy. A serum lactate level that remains elevated after initial resuscitation is a poor prognostic indicator.[8,9] Metabolic laboratory studies and electrolyte determinations assist with adjustment of fluid and electrolytes. Serial hemoglobin and hematocrit determinations and coagulation panels may be drawn to assess the need for blood product replacement. However, the hemoglobin and hematocrit may not directly reflect the severity of blood loss from either hemoconcentration caused by dehydration or hemodilution caused by IV fluid therapy.

Management

Management focuses on restoring circulating volume and resolving the cause of volume loss. Composition of volume replacement therapy depends on what was lost. Crystalloid solutions are used primarily as first-line therapy. Isotonic solutions, such as lactated Ringer's solution or 0.9% normal saline solution, are preferred over hypotonic solutions (5% dextrose solution). Blood products and other colloid solutions (albumin and synthetic volume expanders) may be used to assist in the resuscitation process, especially if blood loss is the primary cause. Packed red blood cells are administered to maximize oxygen-carrying capacity.

The use of colloids in the early phase of fluid replacement is controversial. Because colloids stay within the intravascular space better than crystalloids, patients generally require smaller volumes of colloids for resuscitation. However, capillary membrane permeability is increased in shock, which allows colloids to "leak" from the blood vessels into the extravascular space, further shifting fluids from the intravascular space to interstitial tissues and thereby worsening the hypovolemia. Current research suggests that colloids are not superior to crystalloids in treating hypovolemia in critically ill patients.[10–12] Table 54-3 summarizes some of the known complications of fluid resuscitation.

Nursing management of hypovolemic shock focuses on the restoration of circulating volume through volume administration. Obtaining and maintaining adequate IV access is essential. Ideally, large-bore (16-gauge or larger) IV catheters are inserted in the antecubital space or central venous system to assist with the rapid infusion of fluids. Care must be taken to administer fluids as rapidly as possible without compromising the pulmonary system. Fluids given too rapidly may cause pulmonary congestion and inhibit adequate oxygenation, further compromising oxygen delivery

BOX 54-2 CONSIDERATIONS FOR THE OLDER PATIENT

Response to Shock States

As a person ages, normal physiological changes may limit the ability of the body to respond efficiently to shock states. The nurse should be aware of physiological changes of aging and monitor closely for changes in the older patient's baseline assessment(s). The patient's medical history may reveal other chronic diseases or conditions that further compromise normal physiological changes seen with aging. (See also Chapter 12, The Critically Ill Older Patient)

Cardiovascular system: Increased dysrhythmias, increased atrial size and irritability, left ventricular myocardial thickening leading to decreased compliance and lower ejection fraction; thickened heart valves that interfere with forward flow; decreased response to sympathetic nervous system; decreased sensitivity of baroreceptors; generalized stiffening of arterial vessels, including aorta

Pulmonary system: Decreased tidal volume and respiratory muscle strength, decreased alveolar surface area, increased dead space at end expiration, decreased elastic recoil of lungs, increased resting respiratory rate, increased risk for infection as a result of decreased number of cilia, blunted response to hypoxemia, decreased gag and cough reflex leading to increased risk for infection, aspiration

Hematological system: Decreased ability of bone marrow to produce cells (red blood cells, white blood cells [WBCs], platelets), increased anemia, decreased immune function (decreased production of T and B lymphocytes) leading to increased infections, lower baseline temperature, gradual changes in temperature in the elderly versus spikes (101.3°F [38.5°C]), increased risk for adverse drug reactions

to the tissues. Fluids should also be warmed during infusion to limit the negative effects of hypothermia. Frequent documentation of vital signs, heart rate, respiratory rate and depth, oxygen saturation, urine output, and mentation, as well as laboratory results and interventions, is essential.

Cardiogenic Shock

Etiology

Cardiogenic shock is actually extreme congestive heart failure and therefore results from loss of critical contractile function of the heart. Usually, cardiogenic shock is diagnosed by the presence of systemic and pulmonary hemodynamic alterations, which result from inadequate CO and tissue perfusion. Typically, this occurs when greater than 40% of ventricular mass is damaged. The most common cause of cardiogenic shock is an extensive left ventricular myocardial infarction. In-hospital mortality rates are 40% to 60%, having declined somewhat after the introduction of early revascularization procedures such as cardiac catheterization, percutaneous coronary intervention, and open heart surgery.[7] Although cardiogenic shock may develop within a few hours after the onset of myocardial infarction symptoms, it often occurs after hospitalization. Other causes of cardiogenic shock include papillary muscle rupture, ventricular septal rupture, cardiomyopathy, acute myocarditis, valvular disease, and dysrhythmias.

Box 54-3 shows independent predictors for development of cardiogenic shock. Patients with all five risk factors have a greater than 50% chance of developing cardiogenic shock. Identifying patients at risk for development of cardiogenic shock and formulating strategies for prevention is extremely important.

Table 54-3 Complications of Volume Resuscitation

Fluid Type	Complications
Crystalloid and colloid	Dilutional coagulopathy Dilutional thrombocytopenia Hypothermia Increased hemorrhage Decreased blood viscosity Pulmonary edema Intracranial hypertension (patients with traumatic brain injury)
Packed red blood cells	Acidosis (banked blood has pH 6.9–7.1) Left shift on the oxyhemoglobin dissociation curve (banked blood is deficient in 2,3-DPG) Hyperkalemia Immunological and infectious complications Dilutional coagulopathy Dilutional thrombocytopenia Hypothermia

Pathophysiology

Cardiogenic shock is caused by loss of ventricular contractile force, which results in decreased stroke volume and decreased CO (Fig. 54-5). Neuroendocrine compensatory mechanisms, which are discussed in detail in the section on hypovolemic shock, are activated to increase preload through retention of sodium and water (see Fig. 54-1, p. 1210). Vasoconstriction also increases afterload (SVR). Ventricular filling pressures increase because of the increased preload, but lack of contractility prevents complete ejection. The ventricle becomes distended, further impairing effective contraction, and CO continues to decrease. Compensatory mechanisms continue the vicious cycle of elevated ventricular filling pressures and SVR in combination with an inability of the heart to eject an adequate volume of blood into circulation. Blood pools in the pulmonary circulation, resulting in pulmonary congestion. Pulmonary capillaries are under increased pressure and leak fluid into the interstitium and alveoli, preventing the diffusion of oxygen from alveoli into the pulmonary capillaries and reducing oxygen tension in the blood.

BOX 54-3 PATIENT SAFETY

Risk Factors for Inpatient Development of Cardiogenic Shock

- Increased age (elderly)
- Left ventricular ejection fraction less than 35% on hospital admission
- Large myocardial infarction
- History of diabetes mellitus
- Previous myocardial infarction

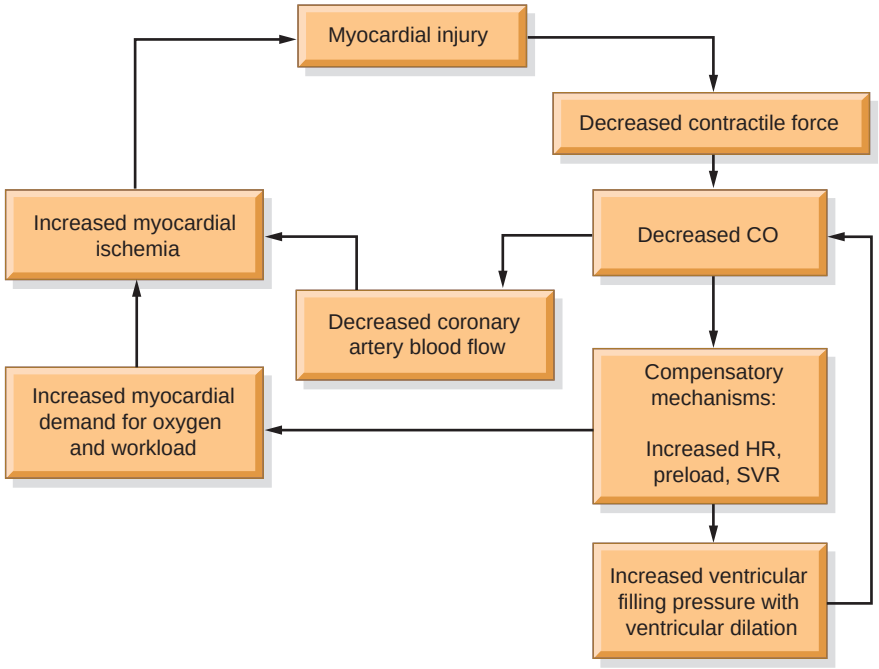


FIGURE 54-5 ▲ Cardiogenic shock. CO, cardiac output; HR, heart rate; SVR, systemic vascular resistance.

Cells become ischemic because of the decreased CO, adding to an already tenuous state of myocardial functioning by further stimulating compensatory mechanisms to increase perfusion to the cells. Increased sympathetic stimulation increases the heart rate even more, further escalating myocardial oxygen demands and compounding the crisis. Associated hypotension prevents adequate oxygenation of myocardial tissue, exacerbating the anaerobic metabolism of the myocardial tissue and further decreasing the contractile state of the heart. These stressors placed on the failing heart may result in extension of a myocardial infarction.

Assessment

Patients who are at high risk of cardiogenic shock require close monitoring. Assessment parameters are similar to the signs and symptoms of congestive heart failure but are more extreme. Assessment findings should be followed over time to allow the nurse to perceive the subtle changes that signal the beginning of cardiogenic shock.

HISTORY. A thorough history provides the information necessary to predict patients at risk for developing cardiogenic shock. Cardiogenic shock frequently occurs in people who have suffered an extensive myocardial infarction, have an admission ejection fraction of less than 35%, have diabetes mellitus, or are elderly (see Boxes 54-2 and 54-3). The existence of these predisposing factors should alert the clinician to assess for the initial phases of shock, allowing for rapid, life-saving intervention. It is important to rule out other causes of decreased CO before initiating therapy. Patients with acute myocardial infarction may require rapid revascularization with thrombolytics (see Chapter 21), percutaneous coronary intervention (see Chapter 18), or cardiac surgery (see Chapter 22).

PHYSICAL FINDINGS. Clinical manifestations associated with cardiogenic shock are outlined in Box 54-4. In addition to the signs and symptoms listed in the box, patients with cardiogenic shock often experience recurrent chest pain, which may indicate extension of the infarction. Other clinical findings are directly related to the decrease in CO.

LABORATORY STUDIES. Presence of elevated myocardial tissue markers, accompanied by progressive hemodynamic compromise and clinical deterioration, is often a hallmark of extensive myocardial necrosis, which may precipitate

BOX 54-4 Clinical Manifestations of Cardiogenic Shock

Hemodynamic Findings
SBP less than 90 mm Hg
Mean arterial pressure (MAP) less than 70 mm Hg
Cardiac index less than 2.2 L/min/m^2
Pulmonary artery occlusion pressure (also known as pulmonary artery wedge pressure) greater than 18 mm Hg
Systemic vascular resistance greater than 1,400 dynes/s/cm^{-5}

Noninvasive Findings
Thready, rapid pulse
Narrow pulse pressure
Distended neck veins
Dysrhythmias
Chest pain
Cool, pale, moist skin
Oliguria
Decreased mentation

Pulmonary Findings
Dyspnea
Increased respiratory rate
Inspiratory crackles, possible wheezing
Arterial blood gas measures show a decrease in PaO_2
Respiratory alkalosis

Radiographic Findings
Enlarged heart
Pulmonary congestion

cardiogenic shock. Laboratory studies suggesting myocardial tissue death reveal a continuous release of myocardial bands of creatine phosphokinase (MB-CPK) and cardiac troponin I into the circulation. These cardiac markers are released into the bloodstream by dying cardiac cells into the bloodstream. Each marker has a time course for its peak level indicative of myocardial injury. Brain natriuretic peptide (BNP), another cardiac marker that may be assessed by laboratory analysis, is produced and released by the ventricle when it is stretched due to pressure overload. The BNP can be used to help determine both the presence and severity of heart failure.[13]

Management

Management is aimed at increasing myocardial oxygen delivery, maximizing CO, and decreasing left ventricular workload. The first goals of treatment are to correct reversible problems, protect the ischemic myocardium, and improve tissue perfusion. Early treatment is imperative to preserve myocardial muscle. Reversing the hypoxemia and acidosis can improve the response to other therapies. Fluids should be managed to provide adequate filling pressure without overdistention of the ventricle. Left ventricular filling pressures are often elevated; therefore, diuresis or nitrate infusion may be indicated to achieve optimal preload. Electrolytes, specifically potassium, calcium, and magnesium, may need to be replaced to provide optimal conditions for the damaged myocardial muscle.

Nursing management for the patient with cardiogenic shock centers on conserving myocardial energy and decreasing the workload of the heart. Use of opioid analgesics and sedatives to minimize the sympathetic nervous system response can increase venous capacitance and decrease resistance to ejection. Opioids also relieve ischemic pain. Increasing the oxygen concentration of inspired air is a simple but important step and may require initiation of mechanical ventilation. Nurses need to provide physical care and periods of rest to minimize myocardial energy expenditure.

Dysrhythmias often occur with acute myocardial infarction, ischemia, or acid–base imbalances and can further decrease CO. Correcting these problems with antiarrhythmic agents, cardioversion, or pacing can help restore a stable heart rhythm and enhance CO. The critical care nurse must obtain, follow, and carefully interpret the patient's hemodynamic parameters to achieve the goal of optimal CO. Optimal filling pressures assist in restoring CO but must be attained cautiously. As mentioned, left ventricular filling pressures may be elevated, and diuresis should be used to reduce these pressures. If the left ventricular filling pressure is too low, fluids may be used, but they must be stopped when filling pressures increase without a subsequent increase in CO. In general, a preload (left ventricular end-diastolic pressure [LVEDP]) of 14 to 18 mm Hg should be maintained. Achieving an "optimal filling pressure" by administering fluids and diuretics is not always an easy task. Slow fluid administration or diuresis requires diligent assessment of the effectiveness of the interventions. Invasive measures of fluid status may be used in patients with pulmonary artery catheters, central venous catheters, or specialized sensors on arterial catheters. Although these technologies provide a wealth of information, at this time there is little evidence to show that their use improves patient outcomes. For this reason, excellent clinical examination skills are essential and noninvasive measures such as blood pressure, mental status, and urine output are very important.[7,14,15]

Pharmacological agents can be used to augment CO, but they too must be used cautiously. Many agents can increase myocardial oxygen consumption ($M\dot{v}O_2$) without having an appreciable effect on CO. Decisions to use some pharmacological agents are based on overall risk–benefit considerations. The sympathomimetic drugs norepinephrine and epinephrine hydrochloride may enhance CO by increasing contractility, heart rate, or SVR but simultaneously increase cardiac work. In addition, stimulation of beta-2 receptors by epinephrine may produce dilation in peripheral vascular beds that robs vital organs of blood. Agents with positive inotropic effects that have less activity on vascular tone, such as low-dose dopamine hydrochloride, dobutamine hydrochloride, amrinone, and milrinone, are frequently used with success.[7,16] Table 54-4 lists pharmacological agents used in treating patients in shock states.

Decreasing the workload of the left ventricle can be accomplished through pharmacological afterload reduction or mechanical support devices. It is recommended that vasodilators, such as sodium nitroprusside, nitroglycerin, or angiotensin-converting enzyme (ACE) inhibitors, be administered to reduce SVR and LVEDP in an effort to increase CO and improve left ventricular function.[16] Mechanical support for the failing ventricle includes the intra-aortic balloon pump and left ventricular assist device. Both devices reduce the workload of the left ventricle by supplementing pumping ability (see Chapter 18).

Distributive Shock States

Distributive shock states can be caused by anaphylaxis (anaphylactic shock), loss of sympathetic tone (neurogenic shock), or sepsis (septic shock). The underlying mechanism is decreased venous return resulting from displacement of blood volume away from the heart because of enlargement of the vascular compartment and loss of blood vessel tone (Fig. 54-6). Loss of blood vessel tone occurs as a consequence of a loss of sympathetic innervation to blood vessels (neurogenic shock) or because of vasodilating substances in the blood (anaphylactic and septic shock). Distributive shock states can occur in a variety of settings, although the most common is sepsis.[17]

Anaphylactic Shock

Anaphylaxis results from an allergic reaction to a specific allergen that evokes a life-threatening hypersensitivity response. While foods are the most common cause of anaphylaxis in children, medication and insect-sting allergies are more common causes in adults.[18] If left untreated, vascular collapse can occur, resulting in greatly decreased tissue perfusion and death. Prompt intervention is critical.

ETIOLOGY. Antigens, the substances that elicit the response, can be introduced through injection or ingestion, or through the skin or respiratory tract. Substances capable of evoking anaphylaxis in humans include a multitude of factors (Box 54-5).

Table 54-4 Pharmacological Drugs Used in the Treatment of Shock*

Drug	Heart Rate	Effects on Contractility	Systemic Venous Resistance	Nursing Considerations
Dopamine (Intropin)	↑	↑↑	↑	Hemodynamic effects are dose dependent May increase $M\dot{V}O_2$ demands
Epinephrine (Adrenaline)	↑↑	↑↑	↑	May induce ventricular dysrhythmias May increase $M\dot{V}O_2$ demands β_2 Activity may dilate peripheral beds
Norepinephrine (also known as levarterenol [Levophed])	↑	↑	↑↑↑	Monitor peripheral circulation closely; may increase $M\dot{V}O_2$
Phenylephrine (Neo-Synephrine)			↑↑	May induce dysrhythmias
Vasopressin (Pitressin)		↑	↑↑	Monitor peripheral circulation closely; may increase $M\dot{V}O_2$
Sodium nitroprusside (Nipride)	↑		↓↓	Hemodynamic effects are dose dependent; adjust dosage slowly
Nitroglycerine (Tridil)	↑		↓	Hemodynamic effects are dose dependent; adjust dosage slowly; tolerance may develop
Angiotensin-converting enzyme (ACE) inhibitors	↑		↓	
Amrinone (Inocor)	↑	↑	↓	May increase $M\dot{V}O_2$ demands
Milrinone (Primacor)	↑	↑↑	↓	May increase $M\dot{V}O_2$ demands Monitor for tachyarrhythmias
Dobutamine (Dobutrex)	↑	↑↑	↓	May increase $M\dot{V}O_2$ demands Monitor for tachyarrhythmias

$M\dot{V}O_2$, myocardial oxygen consumption.

*All agents should be administered through a central venous catheter and using a volumetric pump.

Anaphylaxis may be either immunoglobulin E (IgE) or non-IgE mediated. Non-IgE responses occur without the presence of IgE antibodies and are called anaphylactoid reactions. It is thought that direct activation of mediators causes this response. Anaphylactoid reactions are commonly associated with nonsteroidal anti-inflammatory drugs (NSAIDs), including aspirin. If there has been an anaphylactoid reaction to one agent, restrictions should include all NSAIDs because any of them could elicit a second reaction.

IgE-mediated anaphylaxis occurs as a result of the immune response to a specific antigen. The first time the immune system is exposed to the antigen, a very specific IgE antibody is formed and circulates in the blood. When a second exposure to this antigen occurs, the antigen binds to this circulating IgE, which then activates mast cells and basophils, triggering release of histamine, prostaglandins, leukotrienes, and other biochemical mediators that initiate anaphylaxis.

Pathophysiology

The antibody–antigen reaction causes antibody-specific mast cells and basophils to secrete substances such as histamine, leukotrienes, eosinophil chemotactic substance, heparin, prostaglandins, neutrophil chemotactic substance, and platelet-activating factor 2 (Fig. 54-7). These substances, particularly histamine, prostaglandins, and leukotrienes, cause systemic vasodilation, increased capillary permeability, bronchoconstriction, coronary vasoconstriction, and urticaria (hives). Some of the other substances precipitate a continued downward spiral by causing myocardial depression, inflammation, excessive secretion of mucus, and peripheral vasodilation.[19] The diffuse arterial vasodilation creates a maldistribution of blood volume to tissues, and venous dilation decreases preload, decreasing CO. Increased capillary permeability leads to loss of vascular volume, further decreasing CO and subsequently impairing tissue perfusion. Initial symptoms include itching, urticaria, and some difficulty breathing due to bronchoconstriction. Death from circulatory collapse or extreme bronchoconstriction may occur within minutes or hours.

Assessment

Anaphylactic shock may have no predisposing factors. Therefore, avoiding known allergens is usually the best way to prevent anaphylactic shock.

HISTORY. It is necessary to obtain a thorough history of allergies and responses to drugs, foods, blood products, or anesthetic agents. Moreover, it is important to recognize the various clinical presentations.

PHYSICAL FINDINGS. The earlier the symptoms of anaphylaxis appear after exposure to the antigen, the more severe the response. Initially, generalized erythema,

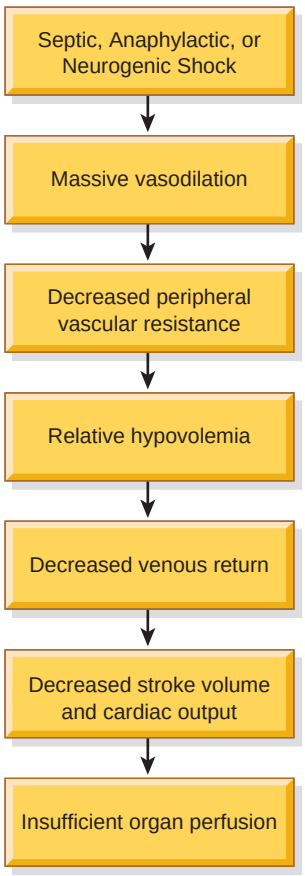


FIGURE 54-6 ▲ Distributive shock states are caused by decreased venous return as a result of displacement of blood volume away from the heart due to enlargement of the vascular compartment and loss of blood vessel tone.

urticaria, and pruritus may occur in response to the antigen. Other symptoms may include anxiety and restlessness, dyspnea, wheezing, chest tightness, a warm feeling, nausea and vomiting, angioedema, and abdominal pain. As the episode progresses, severe respiratory manifestations, such as laryngeal edema or severe bronchoconstriction with stridor, may develop. Hypotension from vasodilation soon occurs and leads to circulatory collapse. As circulatory collapse or hypoxia related to severe bronchoconstriction progresses, the level of consciousness deteriorates to unresponsiveness.

Management

Early recognition and treatment of anaphylaxis is essential. Therapeutic goals include removal of the offending antigen, reversal of effects of the biochemical mediators, and restoration of adequate tissue perfusion. Regardless of the cause of the anaphylactic reaction, treatment depends on clinical symptoms. If the symptoms are mild, immediate therapy includes oxygen and subcutaneous or IV administration of an antihistamine, such as diphenhydramine, to block the effects of histamine. Any patient with life-threatening changes in airway, breathing, or circulation should immediately receive epinephrine to reverse the vasodilation and bronchoconstriction (Box 54-6). If the patient is severely hypotensive or does not respond promptly to epinephrine, rapid infusion (over 1 to 3 minutes) of crystalloid fluids is essential. Other pharmacotherapy includes corticosteroids, bronchodilators, and, if absolutely necessary, vasoconstrictors and positive inotropic agents to combat circulatory collapse.[20,21]

Nursing care involves maintaining an adequate airway and monitoring patient response to the antigen. The nurse also monitors respirations, heart rate, blood pressure, and level of anxiety, and institutes comfort measures related to the dermatological manifestations. If the agent causing the anaphylaxis is unknown, evaluation for allergies and future risk for anaphylaxis should be completed. Patient education regarding prevention and treatment is critical for any person who experiences a significant anaphylactic or anaphylactoid reaction.

Neurogenic Shock

ETIOLOGY. Neurogenic shock results from loss or disruption of sympathetic tone, which causes peripheral vasodilation and subsequent decreased tissue perfusion. The disturbance of sympathetic tone may be caused by any event

BOX 54-5 PATIENT SAFETY

Drugs Commonly Implicated in Anaphylactic and Anaphylactoid Reactions

Antibiotics	Penicillin and its synthetics, cephalosporins, erythromycin, streptomycin, tetracyclines
Anti-inflammatory agents	Salicylates, aminopyrine, ibuprofen, naproxen, and others
Narcotic analgesics	Morphine, codeine
Anesthetics	Procaine, lidocaine, cocaine, thiopental
Anesthetic adjuncts	Succinylcholine, tubocurarine
Other medications	Protamine, chlorpropamide, parenteral iron, iodides, thiazide diuretics
Blood products	Red blood cell, WBC, and platelet transfusions; gamma globulin
Immune sera	Rabies, tetanus, diphtheria antitoxin, snake and spider antivenom
Diagnostic agents	Iodinated radiocontrast agents
Venoms	Bees, wasps, hornets, spiders, snakes, jellyfish
Hormones	Insulin, corticotropin, pituitary extract
Enzymes and other biologicals	Acetylcysteine, pancreatic enzyme supplements
Extracts used in desensitization	Food, pollen, venoms
Foods	Eggs, fish, shellfish, milk, nuts, legumes
Textiles	Latex

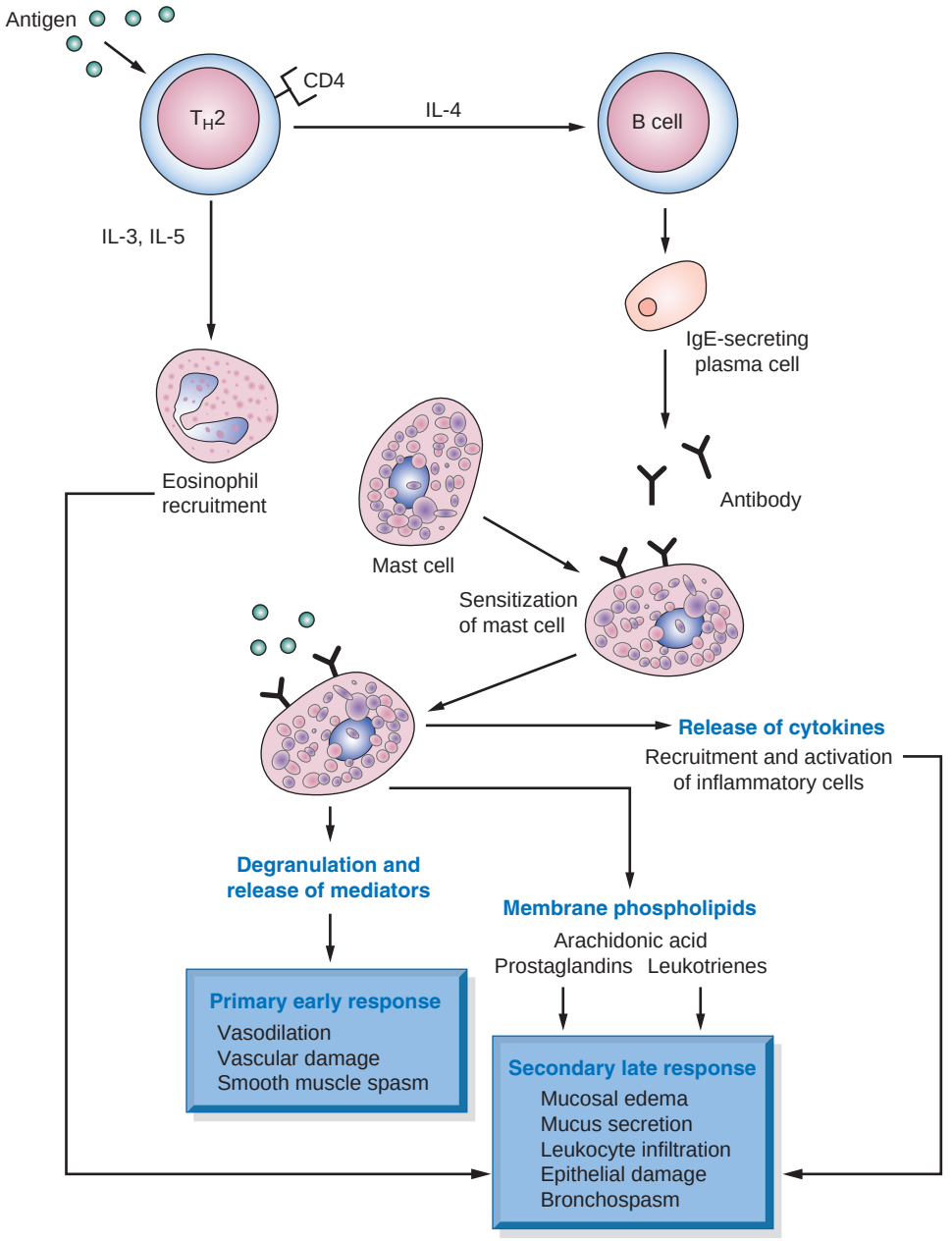


FIGURE 54-7 ▲ IgE-mediated hypersensitivity reaction. IL-3, interleukin 3; IL-4, interleukin 4; IL-5, interleukin 5. (From Porth CM: Concepts of Altered Health States, 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009, p 412.)

that disrupts the sympathetic nervous system. The most common cause of neurogenic shock is a spinal cord injury above the level of T6 because sympathetic innervation occurs above this level. Other causes include spinal analgesia, emotional stress, pain, drugs, or other central nervous system problems.

BOX 54-6 Epinephrine Dosage in Anaphylaxis (Adults)

Epinephrine 1:1,000 dilution (1 mg/mL), 0.2 to 0.5 mL intramuscularly or subcutaneously every 5 minutes, as necessary, should be used to control symptoms and increase blood pressure in patients experiencing anaphylaxis.

From Joint Task Force on Practice Parameters, American Academy of Allergy, Asthma and Immunology, American College of Allergy, Asthma and Immunology, Joint Council of Allergy, Asthma and Immunology: The diagnosis and management of anaphylaxis: An updated practice parameter. J Allergy Clin Immunol 115(3 Suppl 2):S483–S523, 2005

PATHOPHYSIOLOGY. Neurogenic shock is characterized by hypotension, bradycardia, and hypothermia. When sympathetic tone is lost, unopposed parasympathetic tone results in uncontrolled arterial vasodilation and a decrease in SVR. Simultaneous venous vasodilation results in blood pooling and decreases preload. Unopposed parasympathetic stimulation leads to bradycardia, even in the presence of falling blood pressure, which would normally lead to increased heart rate through stimulation of baroreceptors in the aortic arch and carotid sinus. The decrease in both stroke volume (from decreased preload) and heart rate leads to decreased CO, resulting in inadequate tissue perfusion. Hypothermia results from uncontrolled heat loss resulting from excessive vasodilation.[22,23]

ASSESSMENT. Physical findings in the patient with neurogenic shock are largely related to excessive vasodilation and the impaired response to this process. Patients demonstrate decreased central venous pressure (CVP), CO, and SVR combined with bradycardia. Unlike many shock states in which the patient may feel cold and clammy, often the skin is warm due to the uncontrolled vasodilation. In the trauma patient, it is important to exclude hypovolemia before attributing hypotension to neurogenic shock.

MANAGEMENT. Prevention and treatment of hypotension through careful fluid resuscitation is a high priority. The patient's effective circulating volume may be dramatically decreased because of venous pooling. In general, the systolic blood pressure (SBP) should be kept over 90 mm Hg. If fluid administration alone is not adequate to restore the blood pressure, vasopressors may be added. The use of agents with alpha-adrenergic activity promotes vasoconstriction, while beta-adrenergic agonists increase heart rate and contractility.[22] (Refer to Chapter 37 for detailed information on spinal cord injury and associated complications such as neurogenic shock.)

Septic Shock

Septic shock is a complex and generalized process that involves all organ systems. Sepsis, severe sepsis, and septic shock represent progressive stages of the same illness. In 1991, the Society of Critical Care Medicine and the American College of Chest Physicians established universal definitions for the term sepsis and other associated clinical conditions[4] to promote earlier detection of and intervention for these states, improve outcomes, and standardize the terminology used in research protocols. In 2001, a second consensus conference was held to modify the existing definitions for accuracy, reliability, and clinical utility of the diagnosis of sepsis[24] (Box 54-1, p. 1212).

ETIOLOGY. In the United States, it has been estimated that there are as many as 750,000 cases of severe sepsis annually, and that incidence is increasing.[25] The incidence is rising for several reasons: an aging population, increasing number of infections associated with antibiotic-resistant organisms, an increasing number of immunocompromised patients who present with critical illness, more patients undergoing high-risk surgery, and possibly improved identification. The mortality rate for sepsis is approximately 20% and can be as high as 70% in severe sepsis and septic shock. Survivors are often left with a profoundly different quality of life.[25] Risk factors for the development of septic shock include host factors and treatment-related factors (Box 54-7). Approximately one in four patients who present to the emergency department in sepsis will progress to septic shock within 72 hours.[26] Risk factors for progression from sepsis to severe sepsis are listed in Box 54-8.

Septic shock is initiated by an infection. Infections may be due to invading Gram-negative or Gram-positive bacteria, fungi, and viruses. In some patients, multiple causative organisms are identified, but in many patients the causative organism is never identified. Bacteria may be introduced through the pulmonary system, urinary tract, or gastrointestinal system; through wounds; or through invasive devices.

BOX 54-7 PATIENT SAFETY

Risk Factors for the Development of Septic Shock

Host Factors
- Extremes of age
- Malnutrition
- General debilitation
- Chronic debilitation
- Chronic illness
- Drug or alcohol abuse
- Neutropenia
- Splenectomy
- Multiple organ failure

Treatment-Related Factors
- Use of invasive catheters
- Surgical procedures
- Traumatic or thermal wounds
- Invasive diagnostic procedures
- Drugs (antibiotics, cytotoxic agents, steroids)

PATHOPHYSIOLOGY. Septic shock results from complex interactions among invading microorganisms and immune, inflammatory, and coagulation systems, which results in a proinflammatory and procoagulation state (Fig. 54-8). Both Gram-negative and Gram-positive organisms may directly stimulate the inflammatory response and other aspects of the immune system that activate cytokines, complement, and coagulation systems. In response to the presence of microorganisms, macrophages and helper (CD4+) type 1 helper T (Th1) cells secrete the proinflammatory cytokines, such as TNF-α and IL-1β. As previously discussed, these cytokines induce endothelial dysfunction and result in increased capillary permeability. In addition to proinflammatory cytokines, anti-inflammatory cytokines are also released. Type 2 helper T cells (Th2) secrete the anti-inflammatory cytokines IL-4 and IL-10, which balance the proinflammatory response. But in some patients, these proinflammatory cytokines fail to shut down or control the proinflammatory cytokines, and the "out of control" proinflammatory response activates the coagulation cascade.[27]

Another important aspect of sepsis is the imbalance between procoagulant and anticoagulant factors. Endotoxins

BOX 54-8 PATIENT SAFETY

Risk Factors for Progression From Sepsis to Severe Sepsis
- SBP less than 110 mm Hg
- Temperature more than 38.2°C
- Sodium more than 145 mmol/L
- Platelets less than 150 × 10^9/L
- Bilirubin more than 30 mcmol/L
- Mechanical ventilation
- Presence of infection
  - Primary bacteremia
  - Aerobic Gram-negative bacilli
  - Gram-positive cocci
  - Peritonitis
  - Pneumonia

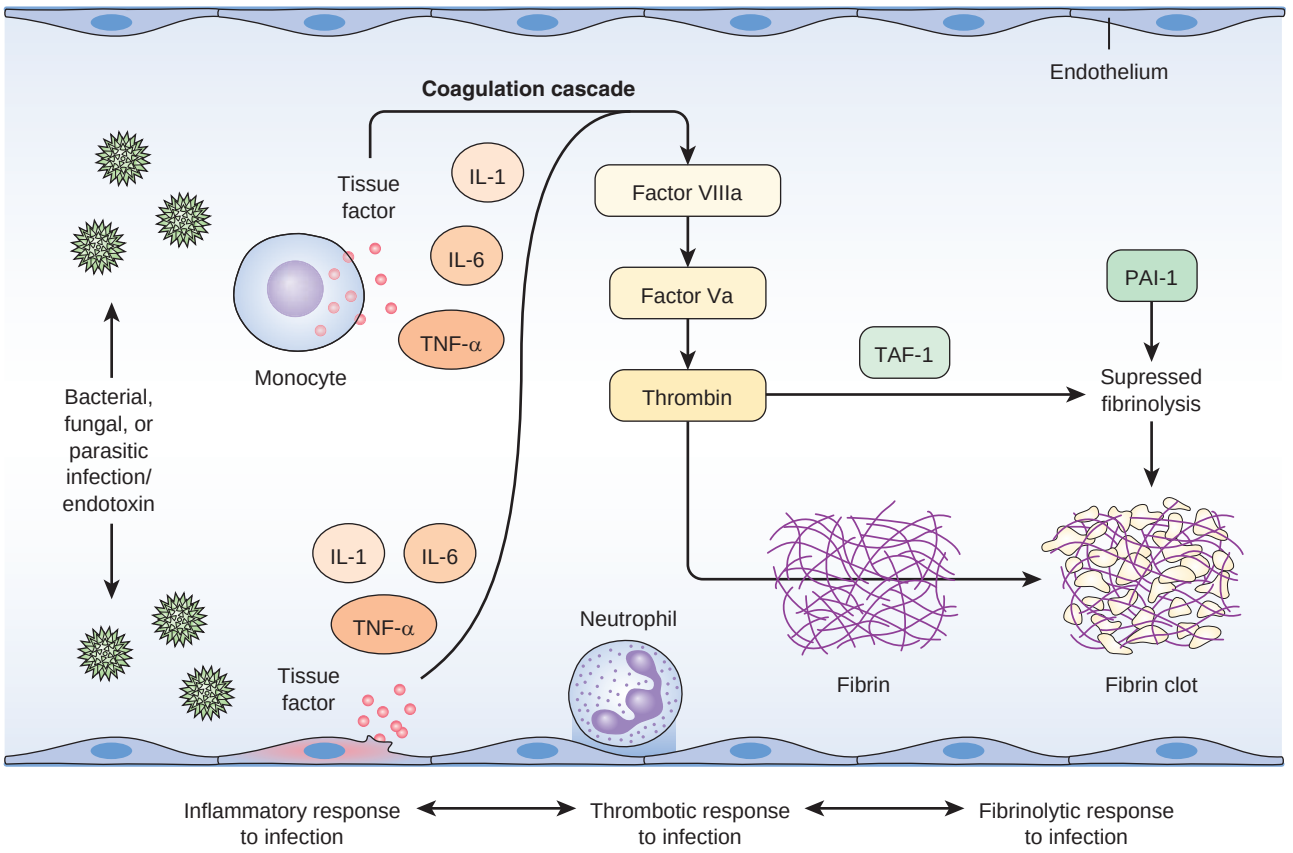


FIGURE 54-8 ▲ Inflammatory/immune response in septic shock. IL-1, interleukin 1; IL-6, interleukin 6; TNF-α, tumor necrosis factor-α; TAFI, thrombin-activated fibrinolysis inhibitor; PAI-1, plasminogen-activator inhibitor-1. (Copyright © 2001, Eli Lilly and Company. All rights reserved.)

stimulate endothelial cells to release tissue factor, which activates the coagulation cascade causing the conversion of fibrinogen to fibrin. Fibrin binds to platelet plugs that have adhered to damaged endothelial cells, forming a stable fibrin clot. These clots form throughout the microvasculature and cause additional injury and ischemia to distal tissues. Normally, anticoagulant factors (protein C, protein S, antithrombin III, tissue factor pathway inhibitor) modulate coagulation, preventing widespread microthrombi formation. Thrombin binds with thrombomodulin on endothelial cells, "activating" protein C. Activated protein C then inactivates factors V and VIII and inhibits the synthesis of plasminogen-activator inhibitor, which then allows plasmin to break down the fibrin–platelet clots.[28] Unfortunately, sepsis lowers the levels of these anticoagulant factors, and the net result is a procoagulant state that contributes to further inflammation.[27] Recognition that the proinflammatory and procoagulant responses result in a loss of homeostasis of almost every organ system is key to understanding sepsis.

Cardiovascular Alterations. In general, septic shock is associated with three major pathophysiological effects on the cardiovascular system: vasodilation, maldistribution of blood flow, and myocardial depression.

Proinflammatory cytokines stimulate the release of NO from endothelial cells. NO is a potent vasodilator and causes widespread vasodilation. Because of this vasodilation, there is decreased venous return to the heart, decreased CO, and decreased SVR. Other inflammatory mediators, including endothelin, are released from endothelial cells and cause vasoconstriction in other vascular beds. This situation of mixed vasodilation and vasoconstriction produces maldistribution of blood flow, particularly in the microcirculation.[29]

In septic shock, myocardial depression is evident in a decreased ventricular ejection fraction, dilation of the ventricles, and a flattening of the Frank-Starling curve after fluid resuscitation. Myocardial depressant factors, such as the cytokines TNF-α, IL-1β, and IL-6, are released as part of the inflammatory cascade. NO also contributes to dysfunction by impairing the ability of cells to utilize available oxygen for ATP production. The heart demonstrates functional impairment of contractility and ventricular performance.[30]

Early in septic shock, activation of the sympathetic nervous system and release of vasodilatory substances such as NO promote development of a hyperdynamic state with high CO and low SVR. Later, as circulating cardiac depressants increase, the heart becomes hypodynamic, with low CO and increased SVR. Hemodynamic parameters, including $ScvO_2$/SvO_2 and measures of metabolic acidosis, should be followed over time to recognize early and progressive cardiac failure.

Pulmonary Alterations. Events initiated by activation of the inflammatory response and its mediators affect the lungs both directly and indirectly. Activation of the sympathetic nervous system and release of epinephrine from the adrenal medulla produce bronchodilation. However, this may be overridden by activity of cytokines, and the net result is bronchoconstriction. More importantly, inflammatory mediators and activated neutrophils cause capillary leak into the pulmonary interstitium, resulting in interstitial edema, areas of

poor pulmonary perfusion (shunting), pulmonary hypertension, and increased respiratory work. As fluid collects in the interstitium, pulmonary compliance is reduced, gas exchange is impaired, and hypoxemia occurs.

The pulmonary alterations described previously may culminate in acute respiratory distress syndrome (ARDS), which is frequently associated with septic shock.[31] Continued fluid accumulation in the pulmonary interstitium may finally spill over into the alveoli, producing alveolar infiltrates that provide fertile areas for bacterial growth. Mechanical ventilation, which is common in patients with ARDS, may provide an avenue of entry for lung infections. Therefore, a secondary pneumonia may develop, possibly caused by a different organism than that which produced the sepsis. See Chapter 27 for more information about ARDS.[31]

Hematological Alterations. Platelet abnormalities also occur in septic shock because endotoxin indirectly causes platelet aggregation and subsequent release of more vasoactive substances (serotonin and thromboxane A_2). Circulating platelet aggregates have been identified in the microvasculature of septic patients. These obstruct blood flow and compromise cellular metabolism. Overactivation of the coagulation cascade without the counterbalance of adequate fibrinolysis compromises tissue perfusion both regionally and globally. Over time, clotting factors are depleted, and a coagulopathy results, with the potential of progressing to disseminated intravascular coagulation (DIC).[28]

Metabolic Alterations. Septic shock induces a hypermetabolic state characterized by an increase in resting energy consumption, extensive protein and fat catabolism, negative nitrogen balance, hyperglycemia, and hepatic gluconeogenesis. Excessive catecholamine release stimulates gluconeogenesis and insulin resistance, which both result in hyperglycemia in critically ill patients who do not have diabetes. Cells are progressively unable to use glucose, protein, and fat as energy sources. Hyperglycemia that is resistant to insulin therapy is a frequent finding in early shock. Eventually, all glycogen energy stores are depleted, cells lack ATP, and cellular pumps fail, progressing to tissue and organ death.

In response to lack of effect of insulin, proteins break down, leading to high blood urea nitrogen and urinary nitrogen excretion. Muscle protein is broken down to amino acids, some of which are used as energy sources for the Krebs cycle or as substrates for gluconeogenesis. In later stages of shock, the liver is unable to use the amino acids because of its own metabolic dysfunction. Amino acids then accumulate in the bloodstream.

As shock progresses, adipose tissue is broken down (lipolysis) to furnish the liver with lipids for energy production. Hepatic triglyceride metabolism produces ketones, which circulate to peripheral cells that can use them in the Krebs cycle for ATP production. As liver function decreases, triglycerides are not broken down; they collect in the mitochondria, inhibit the Krebs cycle, and result in increased anaerobic metabolism and lactate production. The ability of cells to extract and use oxygen is impaired as a result of mitochondrial dysfunction. Oxidants are normally produced as a by-product of oxidative phosphorylation. However, in critical illness, an accumulation of oxidants occurs that results in oxidative stress. Oxidative stress causes lipid peroxidation, protein oxidation, and mutations in mitochondrial DNA, thereby contributing to cell death.

The net effect of these metabolic derangements is that cells become energy starved. This energy deficit is implicated in the emergence of multiple organ failure that frequently develops regardless of interventions designed to support the circulatory and organ systems.[32,33]

ASSESSMENT. A thorough understanding of the mediator responses that occur during sepsis aids in assessing and evaluating the response to treatment.

Physical Findings. Some of the earliest signs of septic shock include changes in mental status (confusion or agitation), increased respiratory rate as compensation for the metabolic acidosis, and either fever or hypothermia. Because of the exaggerated inflammatory response with release of vasoactive mediators, the clinical presentation of the patient is complex. The patient is edematous yet intravascularly depleted, and areas of microthrombi and vasoconstriction obstruct perfusion. As fluid replacement occurs, the leaking capillary beds shift the fluid interstitially, requiring more fluid resuscitation, which may further exacerbate interstitial edema. Because of the inappropriate systemic activation of the coagulation system, clotting factors are depleted, and spontaneous bleeding may occur. Perfusion imbalances cause ischemia in some vascular beds, such as the splanchnic circulation, skin, and extremities, which may lead to necrosis. Consistent with the hyperdynamic state, CO may initially be high; however, it is insufficient to maintain adequate perfusion due to the inappropriately low SVR.

Laboratory Studies. Laboratory and diagnostic studies that may help to identify sepsis are summarized in Box 54-9. Despite the use of such testing, the early diagnosis of sepsis and septic shock is usually made on the basis of patient risk factors and clinical findings (see Box 54-7).

MANAGEMENT. Septic shock requires a prompt, aggressive, multidisciplinary team approach with monitoring and treatment facilities found in an intensive care unit. The primary treatment goals are to maximize oxygen delivery above cellular oxygen consumption requirements and to halt the exaggerated inflammatory response. The treatment of sepsis involves early, goal-directed therapy. See the Evidence-Based Practice Highlight 54-1. Initiating therapy promptly (within the first 6 hours), with specific hemodynamic goals for resuscitation, has been shown to slow the decompensation of patients in a septic state and decrease the risk for cardiovascular collapse.[34] The Surviving Sepsis Campaign Guidelines,[35] developed in 2003 and revised in 2007 by an international team, outline the evidence-based care of patients with sepsis (Table 54-5, p. 1228). The use of a sepsis protocol or sepsis bundle improves interdisciplinary management of septic shock and is associated with improved survival.[36,37]

Prevention. Because morbidity and mortality from septic shock are so high, it is imperative that preventive infection control measures be in place. In critically ill patients, defense mechanisms are often impaired, and protection from hospital-acquired (nosocomial) infections is essential. Nosocomial infections increase length of hospital stay and are associated with substantial costs, ranging from $5,800 to $12,700 for a case of sepsis and $11,100 to $22,300 for a case of pneumonia.[38] Therefore, a critical aspect of nursing care involves meticulous adherence to aseptic technique, thorough hand washing, and a continuing awareness of potential sites and causes of infection. Sources of equipment-related infections are listed in Box 54-10.

Identification and Treatment of Infection. Immediately on presentation, blood cultures should be drawn and then empiric

BOX 54-9 Physiological Data Helpful in Diagnosing Sepsis

- Cultures: blood, sputum, urine, surgical or nonsurgical wounds, sinuses, and invasive lines; positive results are not necessary for diagnosis.
- CBC: WBCs usually will be elevated and may decrease with progression of shock.
- Chemistry panel: hyperglycemia may be evident, followed by hypoglycemia in later stages.
- Arterial blood gases: metabolic acidosis with mild hypoxemia (PaO_2 < 80 mm Hg) and possibly compensatory respiratory alkalosis ($PaCO_2$ < 35 mm Hg) is present in sepsis.
- CT scan may be needed to identify sites of potential abscesses.
- Chest and abdominal radiographs may reveal infectious processes.
- SvO_2 or $ScvO_2$ can assist in the assessment of adequacy of oxygen delivery and consumption.
- Lactate level: decreasing levels of lactate in the serum indicate aerobic metabolism is able to meet cellular energy requirements. Elevated levels indicate inadequate perfusion and anaerobic metabolism to meet cellular energy requirements.
- Base deficit: elevated levels indicate inadequate perfusion and anaerobic metabolism.
- $EtCO_2$: may detect early indications of inadequate regional and global tissue perfusion

broad-spectrum antibiotic therapy with coverage against Gram-negative and Gram-positive bacteria and anaerobes must be initiated. Once the infectious organism has been isolated, antibiotic therapy should be narrowed to antibiotics effective against that specific organism to try to minimize development of antibiotic resistance. Source identification and control is of paramount importance. If a source is identified, definitive measures to alleviate the cause of sepsis might include resection or drainage of purulent tissues or secretions.[35]

However, antimicrobial treatment of sepsis is not sufficient to treat the generalized inflammatory reactions seen with septic shock. Supportive measures establish and maintain adequate tissue perfusion, and other therapies aim to block or interfere with the action of the various mediators implicated in shock. Aspects of supportive care include the following:

- Restoring intravascular volume
- Maintaining an adequate CO
- Ensuring adequate ventilation and oxygenation
- Restoring balance between coagulation and anticoagulation
- Providing an appropriate metabolic environment

Restoration of Intravascular Volume. Adequate volume replacement is important for reversing hypotension. Patients may require several liters or more of fluid because

EVIDENCE-BASED PRACTICE HIGHLIGHT 54-1

Severe Sepsis: Initial Recognition and Resuscitation

△ Expected Practice

- Assess all patients and immediately notify physician when a patient presents with clinical findings suggestive of sepsis.
- Clinical findings for severe sepsis include:
  - Documented or suspected infection

AND

  - Two or more of the following systemic inflammatory response syndrome (SIRS) criteria:
    - Heart rate greater than 90 beats/min
    - Temperature less than 36°C (96.8°F) or greater than 38.3°C (101°F)
    - Respiratory rate greater than 20 breaths/min or $PaCO_2$ less than 32 mm Hg
    - White blood cell count greater than or equal to 12,000/mm^3 or less than or equal to 4,000 mm^3 or a left shift in the immaturation of granulocytes (bands) greater than 10%

AND

  - At least one of the following indicators of tissue hypoperfusion or sepsis-related acute organ dysfunction:
    - Acute altered mental status
    - Systolic blood pressure less than 90 mm Hg or mean arterial pressure less than 70 mm Hg or a SBP decrease of 40 mm Hg
    - Blood glucose greater than 140 mg/dL in patients without diabetes.
    - Arterial hypoxemia (PaO_2/FiO_2 < 300)
    - Acute oliguria (<0.5 mL/kg/h for at least 2 hours)
    - Creatinine increase greater than 0.5 mg/dL above baseline
    - Coagulation abnormalities (INR > 1.5 or a PTT > 60 seconds)
    - Ileus
    - Thrombocytopenia (platelet count < 100,000/mL)
    - Hyperbilirubinemia (plasma total bilirubin > 2 mg/dL) Lactate greater than 2 mmol/L
- Obtain serum lactate measurements. Hyperlactatemia is defined as lactic acid level greater than 4 mmol/L. (Level D)
- Obtain blood cultures as well as cultures from all potential sites of infection prior to initiating broad spectrum antibiotics. Blood cultures should be drawn prior to initiation of antibiotic therapy and within 1 hour of sepsis diagnosis. (Level D)
- Evaluate for and remove potential sources of infection (eg, obviously infected invasive devices). (Level D)
- Maintain the following therapeutic endpoints during resuscitation: mean arterial pressure at greater than 65 mm Hg, central venous pressure (CVP) 8 to 12 mm Hg, and central venous or mixed venous oxygen saturation greater than 70%. (Level D)
  - Administer fluids to attain a CVP of 8 to 12 mm Hg or greater than or equal to 12 mm Hg if on a ventilator. (Level D)
  - Administer vasopressors if necessary to achieve a mean arterial pressure of 65 mm Hg if fluid replacement is not successful. (Level D)
  - If venous oxygen saturation goal not attained, consider additional fluids, blood transfusion, and/or dobutamine administration. (Level D)
- Maintain blood glucose levels at less than 150 mg/dL. (Level D)
- Consider administration of human recombinant activated protein C (drotrecogin alfa activated) for patients at risk for dying and presenting with septic shock, sepsis with multiple organ failure, and sepsis-induced acute respiratory distress syndrome. (Level D)

Note: Administration of human recombinant activated protein C (drotrecogin alfa activated) is no longer recommended. The FDA sent out notification on October 25, 2011 that Eli Lilly has withdrawn this drug from the market. In a recently completed clinical trial (PROWESS-SHOCK trial), the drug failed to show a survival benefit for patients with severe sepsis and septic shock.

(continued on page 1227)

EVIDENCE-BASED PRACTICE HIGHLIGHT 54-1 (continued)

Severe Sepsis: Initial Recognition and Resuscitation

△ Supporting Evidence

- More than 750,000 cases of severe sepsis occurred annually (year 2000) and mortality ranges from 28% to 50% with an overall hospital mortality of about 30%.[1] Sepsis (infection and two of the SIRS criteria) can rapidly progress to severe sepsis (infection + SIRS criteria + organ dysfunction) to septic shock (persistent hypotension and tissue hypoperfusion despite sufficient fluid resuscitation) within 24 hours.[1–4]
- Treatment should be initiated regardless of where the patient is located within the hospital. A prospective randomized study of 263 emergency department patients diagnosed with severe sepsis or septic shock showed that patients treated aggressively with a goal direction toward tissue oxygenation within the first 6 hours of presentation had a 16% improvement in mortality. Another small retrospective study showed a decrease in mortality in patients identified with signs of severe sepsis and treated within the first 6 hours.[3,5–8]
- Serum lactate levels can be elevated in the setting of a normal or increased cardiac output. The measurement of serum lactate can reflect occult decreases in global tissue perfusion, and as such, may be an indicator of organ malperfusion. The presence and the clearance rate of lactate are associated with increases in patient morbidity and mortality.[3,9]
- Early administration of appropriate antibiotics decreases mortality in patients with Gram-positive and Gram-negative bacteremias. Empiric broad spectrum antibiotics should be initiated when possible after obtaining appropriate cultures but prior to identification of the infecting organism and reassessed after 48 to 72 hours based on culture results and clinical data.[10]
- According to the Surviving Sepsis Campaign guidelines, during the first 6 hours of treatment, the goal is to achieve and maintain a CVP of 8 to 12 mm Hg or 12 to 15 mm Hg for patients receiving mechanical ventilation and a MAP of at least 65 mm Hg with fluid resuscitation.[9] Dobutamine is identified as the medication of choice to increase cardiac output to normal levels or to improve lactate clearance when cardiac output is not being measured. Two large clinical trials did not show a benefit from increasing cardiac output above physiologic normal levels in order to increase oxygen delivery to the tissues.[11–13] Available data do not support the use of low-dose dopamine for renal protection.[14]
- Colloids have not been shown to be of more benefit than crystalloid for fluid resuscitation. One large randomized controlled trial comparing 4% albumin with normal saline in the treatment of patients requiring volume resuscitation found no significant difference in mortality between the groups. Several literature reviews have concluded that choice of fluids does not appear to change outcomes.[15,16]
- In the setting of hypotension, fluid replacement should be optimized before vasopressors are started. No high-level evidence exists to identify the most appropriate vasopressor to use for the treatment of septic shock and selection is based on multiple clinical parameters. However, in the Surviving Sepsis Campaign Guidelines for the Management of Severe Sepsis and Septic Shock, norepinephrine or dopamine are identified as the initial vasopressors of choice to increase vascular tone and blood pressure.[9]
- Two meta analyses concluded that administration of high-dose corticosteroids are of no benefit or may be detrimental to patients with septic shock.[17,18] In vasopressor-dependent shock, the addition of low-dose exogenous cortisol has been shown to improve the uptake of the patient's own and the exogenously administered sympathetic stimulants when serum cortisol levels are low.[19]
- Maintaining glucose levels within normal range (80 to 110 mg/dL) but at least less than 150 mg/dL has been shown to decrease morbidity and mortality in a surgical population but did not focus on septic patients. Maintaining glucose levels less than 150 mg/dL showed reduced morbidity but not mortality in critically ill medical patients with sepsis.[20,21]
- In a large double-blind study, human recombinant activated protein C (drotrecogin alfa activated) decreased mortality by 6% in patients with severe sepsis and decreased mortality by 13% for patients at high risk for death (ie, patients having an APACHE II score of 25 or greater).[22,23]

Note: Administration of human recombinant activated protein C (drotrecogin alfa activated) is no longer recommended. The FDA sent out notification on October 25, 2011 that Eli Lilly has withdrawn this drug from the market. In a recently completed clinical trial (PROWESS-SHOCK trial), the drug failed to show a survival benefit for patients with severe sepsis and septic shock.

AACN Evidence Leveling System

Level A Meta-analysis of quantitative studies or metasynthesis of qualitative studies with results that consistently support a specific action, intervention, or treatment.
Level B Well-designed, controlled studies with results that consistently support a specific action, intervention, or treatment.
Level C Qualitative studies, descriptive or correlational studies, integrative review, systematic reviews, or randomized controlled trials with inconsistent results.
Level D Peer-reviewed professional organizational standards with clinical studies to support recommendations.
Level E Multiple case reports, theory-based evidence from expert opinions, or peer-reviewed professional organizational standards without clinical studies to support recommendations.
Level M Manufacturer's recommendations only.

Excerpted from American Association of Critical-Care Nurses Practice Alert. Available online at http://aacn.org. All references cited in this alert are available with the associated resources related to this chapter. Visit: http://thepoint.lww.com

of mediator-induced vasodilation and capillary leak. Fluid replacement should be guided by hemodynamic parameters, urine output, and indicators of metabolic acidosis (end-tidal carbon dioxide, base deficit, lactic acid levels). The Surviving Sepsis Campaign Guidelines recommend fluid resuscitation with crystalloids or colloids to a target CVP of at least 8 mm Hg (12 mm Hg or higher if the patient is receiving mechanical ventilation).[35] Invasive monitoring devices, such as arterial catheters, some of which can provide CO information, and central venous catheters that can monitor CVP and venous oxygen saturation ($ScvO_2$ or SvO_2), are helpful in guiding fluid resuscitation.[39] A downward trend in the markers of metabolic acidosis is a good indicator of improvement in tissue perfusion. Blood products may be administered even in the absence of bleeding to enhance the delivery of oxygen to cells. Administering the fluid and closely monitoring the response to fluid therapy are important nursing responsibilities (see Table 54-5).

Table 54-5 Surviving Sepsis Campaign Guidelines

Collaborative Care Focus	Surviving Sepsis Guidelines	Interventions and Patient Care Considerations
Oxygenation, ventilation	**Mechanical Ventilation** • For patients requiring mechanical ventilation, a tidal volume of 6 mL/kg should be used, with an upper limit plateau pressure of 30 cm H_2O or less. • Permissive hypercapnia may be tolerated in patients with elevated plateau pressures and tidal volumes. • A minimum amount of positive end-expiratory pressure should be used to prevent lung collapse at end expiration. • The head of bed should be raised to at least 30 degrees unless contraindicated to prevent ventilator-associated pneumonia. • Prone position may be considered in patients with acute respiratory distress syndrome requiring high levels of FiO_2 or plateau pressure. • A weaning protocol for severe and refractory hypoxemia with spontaneous breathing trial should be in place to promote ventilator weaning in patients who are arousable, hemodynamically stable, who have no new life-threatening conditions, and who are not requiring high levels of FiO_2 or ventilatory support.	• Maintain a patent airway. • Auscultate breath sounds every 2–4 h and PRN. • Suction endotracheal airway when appropriate (see Chapter 25). • Hyperoxygenate and hyperventilate before and after each suction pass. • Monitor pulse oximetry and end-tidal CO_2. • Monitor arterial blood gases as indicated by changes in noninvasive parameters. • Monitor intrapulmonary shunt (Qs/Qt and PaO_2/FiO_2). • Monitor airway pressures every 1–2 h. • Consider kinetic therapy. • Consider a daily chest x-ray (see Chapter 27).

Circulation, perfusion	**Initial Resuscitation**
	• Resuscitation should begin as soon as sepsis is identified.
	• Fluid resuscitation should initially begin with boluses of crystalloids.
	• Goals of resuscitation in the initial 6-h period after identifying sepsis should include:
	  • Central venous pressure: 8–12 mm Hg (12 mm Hg or higher if mechanically ventilated)
	  • Mean arterial pressure (MAP) of at least 65 mm Hg
	  • Urine output 0.5 mL/kg/h or more
	  • $ScvO_2$ 70% or higher or SvO_2 65% or higher
	• If venous oxygen saturation target not achieved:
	• Consider additional fluid
	  • Transfuse packed red blood cells to achieve a hematocrit of 30% or higher and/or
	  • Start dobutamine infusion (to a maximum of 20 mcg/kg/min)
		• Administer intravascular fluids and vasopressors per protocol.
		• Lactate level may be used to confirm hypoperfusion in patients who are not hypotensive. Monitor serum lactate level on admission and then at least once daily.
		• Assess vital signs hourly.
		• Assess hemodynamic pressures hourly if patient has a pulmonary artery catheter in place.
		• Monitor SvO_2 via a specialty pulmonary artery catheter or $ScvO_2$ via central venous catheter.
		• Administer red blood cells or inotropic agents as ordered to increase oxygen delivery.

	Ongoing Hemodynamic Management
	• Continue to use fluid challenge techniques as long as associated with clinical improvement.
	• Vasopressors should be considered for patients unresponsive to fluid challenges (inadequate blood pressure and organ perfusion).
	  • Norepinephrine or vasopressin should be used.
	  • Low-dose dopamine should not be used for renal protection as a part of the treatment for severe sepsis.
	  • Epinephrine is recommended as the first alternative agent in septic shock that responds poorly to norepinephrine or vasopressin.
		• Monitor for response to fluid challenge with increases in blood pressure or urine output.
		• Monitor for evidence of intravascular volume overload.
		• Vasopressors should be administered through central venous access whenever possible.
		• For patients on vasopressors, an arterial catheter should be placed as soon as possible for accurate monitoring of blood pressures.

	• Inotropic therapy may be initiated for patients with low cardiac output (CO) despite adequate fluid resuscitation.
	  • Dobutamine may be used to increase CO/index to normal levels.
	  • Patients with hypotension should also receive a vasopressor to maintain MAP.
		• Monitor CO and cardiac index per hospital protocol.
		• Monitor hemoglobin and hematocrit.

	• Blood products: After the initial resuscitation is complete, administer red blood cells only when the hemoglobin is <7 g/dL.
	  • The target hemoglobin is 7–9 g/dL for patients without significant coronary artery disease, acute hemorrhage, or lactic acidosis.
	  • Erythropoietin is not recommended for anemia related to severe sepsis but may be used for anemia of other etiologies (such as renal failure).
	  • Fresh frozen plasma is not routinely used to correct altered coagulation unless there is active bleeding or invasive procedures are planned.
		• During transfusion, observe for signs of transfusion reaction.
		• Monitor coagulation parameters.

(continued on page 1229)

Table 54-5 Surviving Sepsis Campaign Guidelines (continued)

Collaborative Care Focus	Surviving Sepsis Guidelines	Interventions and Patient Care Considerations
Sedation, analgesia, and neuromuscular blockade	• A sedation protocol should be used in conjunction with a standardized sedation scale for patient evaluation. • Intermittent bolus sedation or continuous sedation is recommended. • Avoid neuromuscular blocking agents whenever possible.	• Monitor sedation level per sedation scale. • Continuous infusion of sedative agents should be interrupted daily for assessment of patient status while awake, with subsequent retitration as indicated by sedation protocol and assessment.
Steroid therapy	• Consider IV corticosteroids (hydrocortisone) in patients with septic shock who respond poorly to adequate fluid resuscitation and vasopressors.	• Monitor for elevated glucose levels, gastric ulcers, and other complications of steroid administration.
Fluids, electrolytes, and glycemic control	• Blood glucose: After initial stabilization, blood glucose level should be maintained at <150 mg/dL.	• Monitor intake and output every 1 h. • Monitor electrolytes daily and PRN. • Replace electrolytes as ordered. • Monitor blood urea nitrogen (BUN), creatinine, serum osmolality, and serum electrolyte values daily.
	• Renal replacement therapy with intermittent hemodialysis and continuous renal replacement therapy (CRRT) are considered equivalent. CRRT may be preferable in the hemodynamically unstable patient.	• Monitor fluid balance and hemodynamic stability of patients receiving renal replacement therapy.

Identifying and treating the cause of sepsis	• Cultures should be obtained before antimicrobial therapy is initiated. At least two blood cultures should be obtained:
	  • At least one culture specimen drawn percutaneously
	  • At least one culture specimen from each vascular access device inserted >48 h prior
	  • Culture other sites as clinically indicated (urine, cerebrospinal fluid, wounds, respiratory secretions, or other body fluids).
		• Obtain urine, sputum, and blood cultures as ordered.

	• The patient should be formally evaluated for a focus of infection. Any known or suspected source of infection (ie, necrotic tissue, abscesses, or infected vascular access devices) should be removed or treated. • IV antibiotics should be started as early as possible and always within the first hour of recognizing severe sepsis or septic shock. • Initial therapy should include medications with activity against the likely pathogen, with consideration of patterns of resistance in the hospital and community.	• Obtain wound and central vascular line tip culture specimens as ordered. • Administer antibiotics as ordered. • Monitor serum antibiotic levels as ordered. • Consider infectious disease consult. • Monitor systemic inflammatory response syndrome criteria: increased WBCs, increased temperature, tachypnea, and tachycardia.
Preventing new infection	• The antimicrobial regimen should be reassessed daily to optimize activity and prevent development of resistance.	• Adjust antibiotics based on culture results. • Use strict aseptic technique during procedure, and monitor technique of others. • Maintain sterility of invasive catheters and tubes.

Deep venous thrombosis (DVT) prophylaxis	• Patients with sepsis should receive prophylaxis against DVT.
	  • Low-dose unfractionated heparin or low-molecular-weight heparin is preferred.
	  • For patients with a contraindication to pharmacological prophylaxis, mechanical therapy should be used (graduated compression device or intermittent compression device).
	  • For patients at high risk, both pharmacological and mechanical prophylaxis should be considered.
		• Monitor for signs and symptoms of deep venous thrombosis (redness, swelling, tenderness, or pain in calf).

Stress ulcer prophylaxis	• All patients with sepsis should receive stress ulcer prophylaxis. • The preferred agents are H_2 blockers or proton pump inhibitors.	• Monitor for signs and symptoms of peptic ulcer disease (abdominal pain, gastrointestinal bleeding).
Consideration for limitation of support	• Communicate likely outcomes and realistic goals of treatment to patients and family. • Consider less aggressive support or withdrawal of support if in the best interest of the patient.	• Consult social services, clergy, and palliative care team as appropriate. • Provide for adequate rest and sleep.

Adapted from Dellinger RP, Levy MM, Carlet JM, et al: Surviving Sepsis Campaign: International Guidelines for management of severe sepsis and septic shock. Crit Care Med 36:296–327, 2008

BOX 54-10 *PATIENT SAFETY*

Equipment-Related Sources of Infections

- Intravascular catheters (arterial and venous)
- Endotracheal/tracheostomy tubes
- Indwelling urinary catheters
- Surgical wound drains
- Intracranial monitoring devices and catheters
- Orthopedic hardware
- Nasogastric tubes
- Gastrointestinal tubes

Maintenance of Adequate Cardiac Output. In the early phase of septic shock, CO may be normal or elevated. However, the CO is not adequate to maintain tissue oxygenation and perfusion because of decreased SVR and peripheral vasodilation. As septic shock progresses, CO begins to decrease because of cardiac dysfunction. Because oxygen delivery is dependent on CO, maintenance of CO is a primary therapeutic goal.

If adequate volume replacement does not improve tissue perfusion, vasoactive drugs are administered to support circulation. The Surviving Sepsis Campaign Guidelines recommend the catecholamines norepinephrine and dopamine as first-line vasopressors for patients in septic shock. Vasopressin or epinephrine may be used as second-line agents.[17,35] For patients with a low CO or SvO_2 despite adequate fluid resuscitation, dobutamine is the recommended first-line inotropic agent[35] (see Table 54-5).

Maintenance of Adequate Ventilation and Oxygenation. Maintaining a patent airway, augmenting ventilation, and ensuring adequate oxygenation in the patient with septic shock usually requires endotracheal intubation and mechanical ventilation. Because of the ARDS-like picture, low tidal volume (known as lung-protective) ventilation strategies are frequently necessary (for nursing management of patients on mechanical ventilation, see Chapter 25). Assessment of circulatory support, ventilation, and oxygenation is essential. The patient's DaO_2 and VO_2 needs are evaluated frequently. The goal is to maximize DaO_2 to ensure that VO_2 remains independent of DaO_2. Aerobic metabolism is maintained, and tissue energy needs are satisfied through the delivery of adequate oxygen to the cells.

Restoration of Balance Between Coagulation and Anticoagulation. There has been intense investigation of drugs aimed directly at the bacterial toxins and mediators implicated in the inflammatory response seen in sepsis and SIRS. Drotrecogin alfa (activated) (Xigris), a form of human recombinant activated protein C, is not recommended for use in patients with sepsis based on a recently completed clinical trial (PROWESS-SHOCK trial). The drug failed to show a survival benefit for patients with severe sepsis and septic shock.[40] Currently research does not show improved outcomes in patients who receive these medications.

Maintenance of Metabolic Environment. The many and varied metabolic derangements associated with septic shock necessitate frequent monitoring of hematological, renal, and hepatic function. Nutritional stores are depleted, and the patient requires supplemental nutrition to prevent malnutrition and to optimize cellular function. Enteral nutrition is the preferred route of nutritional support because it maintains the integrity of the gastrointestinal tract, decreases infection, and decreases mortality in patients with a septic or hypotensive event.[41]

Intolerance of enteral feeding may necessitate the use of total parenteral nutrition, but ideally, a small amount of enteral nutrition can still be delivered. Recent research suggests that specific nutrients, such as glutamine, omega-3 fatty acids, antioxidants, nucleic acid, and arginine, may help support the immune system during states of stress.[41,42] (See Chapter 40 for a discussion of nutritional support.)

▲ Multiple Organ Dysfunction Syndrome

MODS is defined as a progressive physiological failure of several organ systems in acutely ill patients following an acute threat to systemic homeostasis such that homeostasis cannot be maintained without intervention.[4] MODS can result from SIRS or any type of shock as a consequence of the inability to maintain end-organ perfusion and oxygenation.

Etiology

The exact etiology of MODS is unknown. Release of systemic inflammatory mediators found in SIRS (see Table 54-1, p. 1214) may play a role in the etiology of MODS.[2] In addition, a loss of integrity of mucosal barrier function may liberate bacterial toxins from the gut. These toxins circulate systemically, damaging multiple organs. Finally, tissue hypoxia caused by microvascular thromboses probably also contributes to MODS.[6]

Pathophysiology

Several mechanisms may contribute to the pathophysiology of MODS; it appears to result from a cascade of bacterial factors, endothelial injury, inflammatory mediators, disturbed hemostasis, and microcirculatory failure (see Fig. 54-3, p. 1213). Mitochondrial dysfunction and reduced ATP production is implicated in organ failure.[43] It is suggested that MODS may even be an adaptive state allowing organs time to recover from injury and insult.[44]

Damage to organs may be primary or secondary and cause organ failure. A primary insult refers to a direct injury to an organ that results in organ dysfunction. For example, severe blunt chest trauma injures the lungs and may cause ARDS. Secondary insult is due to mechanisms operable in shock states. For example, a wound infection may cause sepsis, but the resultant SIRS and septic shock may cause ARDS (ARDS is discussed in Chapter 27).

In MODS, there is "crosstalk" between tissue and organ systems; dysfunction and the inflammatory response in one organ may trigger dysfunction in another.[45,46] Therefore, failure of a particular organ makes the failure of a second or third organ more likely. MODS has been described as a "continuum of physiologic derangements"[47] (p 369).

Usually, the first organs to manifest signs of dysfunction are the lungs, heart, and kidneys. Liver failure tends to occur later because the liver has a considerable compensatory capacity. If hypoperfusion persists, all vital organs may fail. It is paramount that interventions increase end-organ perfusion and oxygenation and lessen the inflammatory response during the clinical management of shock states to prevent or limit MODS.

The lungs are typically the first organ system to fail.[45] They are particularly vulnerable to failure because the capillary beds act as a filter that is exposed to cytokines, mediators, and activated neutrophils. Capillary leakage causes interstitial edema, which impairs pulmonary gas exchange. The epithelial cells lining the alveoli are affected by inflammatory mediators. The disruption of the epithelium allows fluid, mediators, and coagulation factors to flood the alveoli, further impairing pulmonary gas exchange.[31] Respiratory failure associated with MODS is not different than ARDS and is discussed in detail in Chapter 27.

The cardiovascular system dysfunction includes reduced CO secondary to dysrhythmias and myocardial depression, as well as abnormalities in the peripheral vascular system, including vasodilation and hypotension that is unresponsive to fluid administration, increased capillary permeability, and maldistribution of blood flow. The most common hematological dysfunction is thrombocytopenia, which is due to increased consumption of platelets due to microthrombi formation and sequestration of platelets in the spleen, as well as impaired thrombopoiesis as a result of bone marrow suppression. This increases the risk for DIC in MODS. (See Chapter 49 for a discussion of DIC.)

Neurological dysfunction can be manifested by altered levels of consciousness, confusion, and delirium. The dysfunction may be secondary to poor cerebral perfusion or an increase in metabolic substances that are neurotoxic (ammonia), or they can be due to electrolyte imbalances. Renal dysfunction can occur secondary to poor renal perfusion and prolonged ischemia to renal tubular cells or intrarenal causes such as nephrotoxic drugs. Renal failure may also be a direct result of mechanical ventilation from altered cardiovascular function or to ventilator-induced lung injury and resultant cytokine release.[46] Progressive liver dysfunction results in hepatic failure. Hepatic failure affects multiple body systems because the liver has so many functions, including synthesis of albumin, clotting factors, and drug metabolism. And, as previously discussed, hepatic failure can lead to impaired mitochondrial function and the ability of cells to use oxygen.

Assessment

Early recognition and management of MODS is essential to improve the likelihood of survival.[48]

Assessment of vital signs for signs of SIRS including hypotension, tachycardia, tachypnea, hypothermia, and hyperthermia is crucial in all hospitalized patients, particularly those at risk for developing shock and MODS. Close surveillance of laboratory values for changes in coagulation parameters, platelet count, WBCs, lactate, renal function, and other studies discussed in this chapter provides early indicators that a patient may be developing organ dysfunction. Several scoring systems exist to determine the extent of MODS, but to date there has not been uniform acceptance of one tool over another.[49] Nursing diagnoses for shock states, SIRS, and MODS are shown in Box 54-11.

BOX 54-11 EXAMPLES OF NURSING DIAGNOSES

For the Patient With Shock States, Systemic Inflammatory Response Syndrome, and Multiple Organ Dysfunction Syndrome

- Risk for Decreased Cardiac Tissue Perfusion
- Risk for Ineffective Cerebral Tissue Perfusion
- Decreased Cardiac Output
- Deficient Fluid Volume
- Ineffective Breathing Pattern
- Impaired Gas Exchange
- Impaired Spontaneous Ventilation
- Impaired Physical Mobility
- Imbalanced Nutrition: Less Than Body Requirements
- Acute Pain
- Fear
- Anxiety

Management

Nurses have a key role in preventing, recognizing, and managing patients with MODS. Prevention strategies include enforcement of measures to prevent nosocomial infections, such as proper positioning (head of bed elevated during mechanical ventilation), oral care, turning and skin care, invasive catheter care, and wound care. Unfortunately, no specific medical treatment for MODS, other than supportive care, is available. Management focuses on treating hemodynamic and metabolic derangements as described in the above management of sepsis (see Table 54-5). Treatment directed at specific organ systems, other than supportive measures such as continuous renal replacement therapy and low tidal volume ventilation, has not been shown to result in improved survival in patients with MODS. This may reflect the interdependence of organ systems and the systemic character of MODS. However, evidence suggests that early identification of patients with a high likelihood of developing MODS and early normalization of $ScvO_2$, arterial lactate concentration, base deficit, and pH lead to a more benign hospital course with decreased inpatient mortality.[39,47]

▲ Clinical Applicability Challenges

CASE STUDY

Ms. H., an 81-year-old female, is brought to the emergency department one afternoon in an ambulance from her skilled nursing facility. Her care givers report that Ms. H., who is normally alert, oriented, and communicative, has been becoming increasingly drowsy and lethargic over the past 24 hours. She has had no fever. She has a history of type 2 diabetes, hypertension, coronary artery disease, and an ischemic stroke 5 years ago that left her with some residual right-sided weakness. Her medications include metoprolol, lisinopril, atorvastatin, aspirin, metformin, and a multivitamin.

(*continued on page 1232*)

CASE STUDY *(Continued)*

On physical examination, Ms. H. is pale, diaphoretic, and lethargic. She arouses to voice but is able only to state her first name. Her vital signs on arrival in the emergency department are as follows: blood pressure, 84/52 mm Hg; heart rate, 130; respiratory rate, 26; temperature, 36.9°C; and oxygen saturation by pulse oximetry is 89% on room air. She is placed on 2 L/min of oxygen via nasal cannula, with improvement in saturation to 95%. Two large-bore intravenous (IV) catheters are started, and specimens are sent immediately for laboratory analysis, including two sets of blood cultures. She is given a liter bolus of normal saline solution. Meanwhile, a Foley catheter is inserted, yielding 40 mL of concentrated urine that is sent for immediate analysis and culture if indicated. Her laboratory results are as follows: sodium, 143 mmol/L; potassium, 3.9 mmol/L; blood urea nitrogen, 32 mg/dL; creatinine, 0.9 mg/dL; glucose, 104 mg/dL; lactate, 2.1 mmol/L; hemoglobin, 11.1 g/dL; hematocrit, 33.5%; and white blood cell (WBC) count, 14.2 1,000/mcl. Her urinalysis reveals 58 WBCs and is positive for nitrites and leukocyte esterase. Chest x-ray findings are normal.

After the first liter of fluid, her blood pressure is 88/54 mm Hg, and a second liter bolus of normal saline solution is given. She is also given ceftriaxone 1 g IV and ampicillin 2 g IV and is admitted to the medical intensive care unit. A central line is inserted for fluid and medication administration and monitoring of central venous pressure (CVP) and superior vena cava oxygen saturation ($ScvO_2$). An arterial line is inserted for continuous blood pressure monitoring. She receives an additional liter of normal saline solution to achieve CVP between 8 and 12 mm Hg, but her mean arterial pressure (MAP) hovers around 55 to 60 mm Hg and urine output remains about 20 mL/h. Norepinephrine is started at 1 mcg/min and titrated to 8 mcg/min to achieve a MAP of 65 mm Hg or greater.

Six hours into her intensive care unit stay, Ms. H develops a fever at 38.7°C. At this time, her $ScvO_2$ is 64% and the lactate level has increased to 3.4 mmol/L. Dobutamine is started at 5 mcg/kg/min, her urine output begins to improve, and her $ScvO_2$ rises to 71%. Her heart rate decreases to 105 beats/min. She is started on subcutaneous heparin for venous thromboembolism prophylaxis as well as famotidine for stress ulcer prophylaxis, and antibiotic therapy is continued. Her glucose level has risen to 160 mg/dL, so an insulin drip is started to maintain glucose level between 110 and 140 mg/dL. Overnight, she remains on norepinephrine and dobutamine, receives an additional liter of normal saline solution to maintain CVP at 8 to 12 mm Hg, and makes 30 to 60 mL/h of urine.

The next morning, Ms. H.'s lactate level has decreased to 1.8 mmol/L, and $ScvO_2$ remains 70% to 75%. Other laboratory results include hemoglobin 10.1 g/dL, hematocrit 30.2%, and WBC count 13.6 1,000/mcl. The nurse is able to decrease the norepinephrine incrementally without a drop in MAP. The laboratory reports growth of Gram-negative rods in the urine sent for culture, and antibiotic therapy is narrowed to a fluoroquinolone. On examination, Ms. H. is more alert and is oriented to place and person. Her heart rate is 85 to 90; respiratory rate 16, and temperature 36.5°C. In the afternoon, her lactate level is 1.4 mmol/L and $ScvO_2$ 72%. The dobutamine is discontinued. Close monitoring of her MAP, CVP, and $ScvO_2$ is continued. She remains stable overnight and is transferred to the step-down unit for continuing care the following day.

1. Discuss some of the challenges encountered in trying to deliver timely antibiotic therapy to patients presenting in the emergency department with severe sepsis.
2. Discuss why steroid therapy was not indicated for Ms. H. based on the Surviving Sepsis Campaign Guidelines.
3. Discuss the serum lactate level as a measure of tissue perfusion and prognostic indicator in shock states.

References

1. Nichols D, Nielsen D: Oxygen Delivery and Consumption: A Macrocirculatory Perspective. Crit Care Clin 26:239–253, 2010
2. Von Rueden KT, Bolton PA, Vary T: Traumatic shock and multisystem organ dysfunction. In McQuillan K, Makic MB, Whalen E (eds): Trauma Nursing: Resuscitation Through Rehabilitation, 4th ed. Philadelphia, PA: Saunders, 2009, pp 200–227
3. Nanas S, Gerovasili V, Renieris P, et al: Non-invasive assessment of the microcirculation in critically ill patients. Anaesth Int Care 37(5):733–739, 2009
4. American College of Chest Physicians/Society of Critical Care Medicine: Consensus conference: Definitions for sepsis and multiple organ failure and guidelines for use of innovative therapies in sepsis. Crit Care Med 20:864–874, 1992
5. Vincent JL. Definition of Sepsis and Non-infectious SIRS. In Cavaillon JM, Adrie C (eds): Sepsis and Non-infectious Systemic Inflammation: From Biology to Critical Care. New York: Wiley Blackwell, 2009, pp 3–10
6. Gando S: Microthrombosis and multiple organ dysfunction syndrome. Crit Care Med 38(2 Suppl):S35–S42, 2010.
7. Topalian S, Ginsberg F, Parrillo J: Cardiogenic shock. Crit Care Med 36:S66–S74, 2008
8. Arnold RC, Shapiro NI, Jones AE, et al: Multicenter study of early lactate clearance as a determinant of survival in patients with presumed sepsis. Shock 32:35–39, 2009
9. Jansen TC, van Bommel J, Bakker J: Blood lactate monitoring in critically ill patients: A systematic health technology assessment. Crit Care Med 37:2827–2839, 2009
10. Finfer S, Bellomo R, Boyce N, et al; SAFE Study Investigators: A comparison of albumin and saline for fluid resuscitation in the intensive care unit. N Engl J Med 350(22):2247–2256, 2004
11. Perel P, Roberts I, Pearson M: Colloids versus crystalloids for fluid resuscitation in critically ill patients. Cochrane Database Syst Rev (4): CD000567, 2009
12. Van den Elsen MJ, Leenen LP, Kesecioglu J: Hemodynamic support of the trauma patient. Curr Opin Anesth 23:269–275, 2010
13. Green SM, Green JA, Januzzi JL: Natriuretic peptide testing for heart failure therapy guidance in the inpatient and outpatient setting. Am J Ther 16:171–177, 2009

14. Beningon S, Ferris P, Nirmalan M: Emerging trends in minimally invasive haemodynamic monitoring and optimization of fluid therapy. Eur J Anaesthesiol 26:893–905, 2009
15. Sevransky J: Clinical assessment of hemodynamically unstable patients. Curr Opin Crit Care 15:234–238, 2009
16. Antman EM, Anbe DT, Armstrong PW, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction; A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1999 Guidelines for the Management of patients with acute myocardial infarction). J Am Coll Cardiol 44:E1–E211, 2004.
17. Holmes CL, Walley KR: Vasoactive drugs for vasodilatory shock in ICU. Curr Opin Crit Care 15:398–402, 2009
18. Tang ML, Obsorne N, Allen K: Epidemiology of anaphylaxis. Curr Opin Allergy Clin Immunol 9:351–6, 2009
19. Peavy RD, Metcalfe DD: Understanding the mechanisms of anaphylaxis. Curr Opin Allergy Clin Immunol 8:310–315, 2008
20. El-Shanawany T, Williams PE, Jolles S: Clinical immunology review series: an approach to the patient with anaphylaxis. Clin Exp Immunol 153:1–9, 2008
21. American College of Allergy, Asthma and Immunology, Joint Council of Allergy, Asthma and Immunology: The diagnosis and management of anaphylaxis: an updated practice parameter. Joint Task Force on Practice Parameters, American Academy of Allergy, Asthma and Immunology. J Allergy Clin Immunol 115(3 Suppl 2):S483–S523, 2005
22. Furlan JC, Fehlings MG: Cardiovascular complications after acute spinal cord injury: Pathophysiology, diagnosis, and management. Neurosurg Focus 25:E13, 2008
23. Garstang S, Miller-Smith, SA: Autonomic nervous system dysfunction after spinal cord injury. Phys Med Rehabil Clin N Am 18:275–296, 2007
24. Levy MM, Fink MP, Marshall JC: 2001 SCCM/ESICM/AACP/ATS/SIS International sepsis definitions conference. Crit Care Med 31: 1250–1256, 2003
25. Martin GS, Mannino DM, Eaton S, et al: The epidemiology of sepsis in the United States from 1979 through 2000. N Engl J Med 348:1546–1554, 2003
26. Glickman S, Cairns C, Otero R, et al: Disease progression in hemodynamically stable patients presenting to the emergency department with sepsis. Acad Emerg Med 17:383–390, 2010
27. Cinel I, Opal S: Molecular biology of inflammation and sepsis: A primer. Crit Care Med 37:291–304, 2009
28. Toussaint S, Gerlach H: Activated protein C for sepsis. N Engl J Med 361:2646–2652, 2009
29. Spanos A, Jhanji S, Vivian-Smith A, et al: Early microvascular changes in sepsis and severe sepsis. Shock 33:387–391, 2010
30. Zanotti-Cavazzoni SL, Hollenberg SM: Cardiac dysfunction in severe sepsis and septic shock. Curr Opin Crit Care 15:392–397, 2009
31. Cehovic GA, Hatton KW, Fahy BG: Adult respiratory distress syndrome. Int Anesth Clin 47:83–95, 2009
32. Abraham E, Singer M: Mechanisms of sepsis-induced organ dysfunction. Crit Care Med 35:2408–2416, 2007
33. Dunser MW, Hasibeder WR: Sympathetic overstimulation during critical illness: Adverse effects of adrenergic stress. J Int Care Med 24: 293–316, 2009
34. Rivers EP, Coba V, Whitmill M: Early goal-directed therapy in severe sepsis and septic shock: A contemporary review of the literature. Curr Opin Anesth 21:128–140, 2008
35. Dellinger RP, Levy MM, Carlet JM, et al: Surviving Sepsis Campaign: International Guidelines for management of severe sepsis and septic shock. Crit Care Med 36:296–327, 2008
36. Barochia A, Cui X, Vitberg D, et al: Bundled care for septic shock: An analysis of clinical trials. Crit Care Med 38(2):668–678, 2010
37. Levy MM, Dellinger P, Townsend S, et al: The surviving sepsis campaign: Results of an international guideline-based performance improvement program targeting severe sepsis. Crit Care Med 38(2):367–374, 2010
38. Eber MR, Laxminarayan R, Perencevich EN, et al: Clinical and economic outcomes attributable to health care-associated sepsis and pneumonia. Arch Intern Med 170:347–353, 2010
39. Maddirala S, Khan A: Optimizing hemodynamic support in septic shock using central and mixed venous oxygen saturation. Crit Care Clin 26:323–333, 2010
40. U.S. Food and Drug Administration (FDA): FDA Drug Safety Communication: Voluntary market withdrawal of Xigris [drotrecogin alfa (activated)] due to failure to show a survival benefit. October 25, 2011. http://www.fda.gov/Drugs/DrugSafety/ucm277114.htm
41. Martindale RG, McClave SA, Vanek VW, et al: Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient: Society of Critical Care Medicine and American Society for Parenteral and Enteral Nutrition: Executive Summary. Crit Care Med 37:1757–1761, 2009
42. Beale RJ, Sherry T, Lei K, et al: Early enteral supplementation with key pharmaconutrients improves Sequential Organ Failure Assessment score in critically ill patients with sepsis: Outcome of a randomized, controlled, double-blind trial. Crit Care Med 36:131–144, 2008
43. Harrois A, Huet O, Duranteau J: Alterations of mitochondrial function in sepsis and critical illness. Curr Opin Anaesthesiol 22(2):143–149, 2009
44. Mongardon N, Dyson A, Singer M: MOF an outcome parameter or a transient, adaptive state of critical illness. Curr Opin Crit Care 15: 431–436, 2009
45. Wang H, Ma S: The cytokine storm and factors determining the sequence and severity of organ dysfunction in multiple organ dysfunction syndrome. Am J Emerg Med 26:711–715, 2008
46. Ricci Z, Ronco C: Pulmonary/renal interaction. Curr Opin Crit Care 16:13–18, 2010
47. Philip S, Barie P, Hydo L, et al: Multiple organ dysfunction syndrome in critical surgical illness. Surg Infect 10(5):369–377, 2009
48. Strehlow MC: Early identification of shock in critically ill patients. Emerg Med Clin North Am 28(1):57–66, 2010
49. Sauaia A, Moore EE, Johnson JL, et al: Validation of postinjury multiple organ failure scores. Shock 31(5):438–447, 2009

55 Trauma

Carla A. Aresco

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Compare and contrast mechanisms of trauma injury.
2. Describe phases of initial assessment and related care of the trauma patient.
3. Discuss the assessment of patients with thoracic, abdominal, musculoskeletal, and maxillofacial trauma.
4. Explain the management and nursing care of patients with thoracic, abdominal, musculoskeletal, and maxillofacial trauma.
5. Describe early and late complications of trauma and the impact of these complications on mortality.

Trauma is defined by the *American Heritage Dictionary* as "a wound, especially one produced by sudden physical injury."[1] Injury is defined by the National Committee for Injury Prevention and Control as "unintentional or intentional damage to the body resulting from acute exposure to thermal, mechanical, electrical, or chemical energy or from the absence of such essentials as heat or oxygen."[2] Unintentional injuries include motor vehicle crashes (MVCs), poisonings, falls, drownings, fires, and burns. Intentional injuries (eg, suicide attempts, assaults, and homicides) include injuries from poisoning, hanging, drowning, firearms, cutting, and jumping. This chapter specifically discusses mechanical injury.

Trauma is one of the leading causes of critical illness and death in the United States. In 2007, unintentional injury was the number one cause of death in age groups 1 through 44 years and the fifth leading cause of death in all age groups.[3] At present, MVCs, with the increased number of cars and drivers, have made civilian trauma an international epidemic; globally, the war on terror has increased the importance and incidence on military trauma.[4]

▲ Mechanism of Injury

Knowing the mechanism of injury is important because this information can help explain the type of injury, predict the eventual outcome, and identify common injury combinations. In addition, an injury may exist in a trauma patient without the classic signs. The mechanism of injury may indicate the need for additional diagnostic workup and reassessment.

The mechanism of injury is related to the type of injuring force and the subsequent tissue response. Injury occurs when the force deforms tissues beyond their failure limits. Wounds vary depending on the injuring agent.[5] The effect of injury also depends on personal and environmental factors, such as the person's age and sex, the presence or absence of underlying disease process, and the geographic region.

Force may or may not be penetrating. The injury delivered from force depends on the energy delivered and the area of contact. In penetrating injury, the concentration of force is to a small area. In blunt or nonpenetrating injury, the energy is distributed over a large area. The predominant feature affecting the impact is speed, or acceleration:

$$\text{Force} = \text{mass} \times \text{acceleration}$$

Blunt Injury

Mechanisms of blunt injury include MVCs, falls, assaults, and contact sports. Multiple injuries are common with blunt trauma, and these injuries are often more life threatening than penetrating injuries because the extent of the injury is less obvious and the diagnosis can be more difficult.

Blunt injury is caused by a combination of forces. These forces include acceleration, deceleration, shearing, crushing, and compressive resistance:

- Acceleration is an increase in the velocity (or speed) of a moving object.
- Deceleration, on the other hand, is a decrease in the velocity of a moving object.
- Shearing occurs across a plane when structures slip relative to each other.
- Crushing occurs when continuous pressure is applied to a body part.
- Compressive resistance is the ability of an object or structure to resist squeezing forces or inward pressure.

In blunt trauma, it is the direct impact that causes the greatest injury. Injury occurs when there is direct contact between the body surface and the injuring agent. Indirect forces are transmitted internally with dissipation of energy to the internal structure. The extent of injury from an indirect force depends on transference of energy from an object to the body. Injury occurs as a result of energy released and the tendency for the tissues to be displaced on impact. Acceleration–deceleration injuries are the most common causes of blunt trauma.

In an MVC, the vehicle size and design change injury patterns. Small cars are involved in more crashes per mile and cause more deaths than larger vehicles. Before the crash, the occupant and the car are traveling at the same speed. During the crash, both the occupant and the car decelerate to zero, but not necessarily at the same rate. There are actually three collisions involved in one crash. The first is the car with another object; the second is the occupant's body with the interior of the car; and the third is the internal tissues with the rigid body surface structure. For example, rapid deceleration in an MVC can cause direct injury to tissue. Subsequently, injury occurs as internal organs impinge on bony internal structures and cause major vessels to undergo stretching and bowing.

Wearing shoulder and lap restraints reduces the incidence and severity of injury by reducing the force with which a person strikes a surface, thereby preventing the occupant from striking multiple surfaces and being ejected from the vehicle[4] (Fig. 55-1). The occupant's position in the vehicle also makes a difference in the blunt injury received. When a vehicle strikes a pedestrian, it is important to visualize the size of the vehicle and the size of the pedestrian. The area of impact can vary depending on these factors (Fig. 55-2).

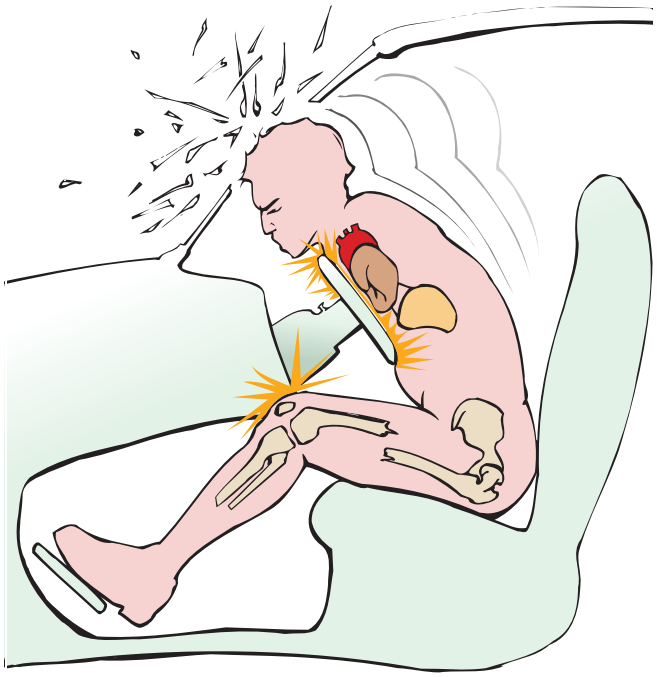


FIGURE 55-1 ▲ In a motor vehicle crash, if the driver is not wearing a seat belt, damage may occur in various sections of the body. Common injuries occur to the skull, scalp, face, sternum, ribs, heart, liver, or spleen. Bones of the pelvis and lower extremities also may be damaged.

Penetrating Injury

Penetrating trauma refers to an injury produced by foreign objects penetrating the tissue. The severity of the injury is related to the structures damaged. The mechanism of injury is caused by the energy created and dissipated by the penetrating object into the surrounding areas. The amount of tissue damaged by a bullet is determined by the amount of energy that transfers into the tissue along with the amount of time it takes for the transfer to occur. It is important to note that the external appearance of the wound does not reflect the extent of internal injury.

Velocity determines the extent of cavitation and tissue damage (Fig. 55-3). Low-velocity missiles localize the injury to a small radius from the center of the tract and have little disruptive effect. They cause little cavitation and blast effect, essentially only pushing the tissue aside. High-velocity missiles can cause more serious injury because of the amount of energy and cavitation produced.[6] The damage depends on three factors: the density and compressibility of the tissue injured, the missile's velocity, and the fragmentation of the primary missile. High-velocity bullets compress and accelerate tissue away from the bullet, causing a cavity to form around the bullet and its entire tract.[6]

Shotguns are short-range, low-velocity weapons that use multiple lead pellets encased in a larger shell for ammunition. Each pellet is a missile (Fig. 55-4). It is important to obtain a brief description of the mechanism of gunshot injuries, including the weapon, the ammunition, and ballistics. This essential information is used to guide the assessment of patients who sustain injuries from these weapons. All trauma patients must be undressed and inspected for entrance and exit wounds during the assessment process.

A stab wound or impalement is a low-velocity injury. The main injury determinants are length, width, and trajectory of the penetrating object and the presence of vital organs in the area of the wound. Although the injuries tend to be localized, deep organs and multiple body cavities can be penetrated.

▲ Initial Assessment and Management

When a trauma patient is brought to the emergency department or the trauma resuscitation unit, it is imperative to obtain a thorough history of the preceding events. This initial evaluation aids in assessment and treatment and can decrease morbidity and mortality. During this initial assessment, it is important to obtain as much detail as possible about the circumstances surrounding the injury, including the mechanism of injury. To facilitate initial assessment, intervention, and triage of the trauma victim, the American College of Surgeons (ACS) Committee on Trauma has developed guidelines. These guidelines provide an organized, standardized approach to the initial assessment of trauma patients, increasing the speed of the primary assessment and minimizing the risk that injuries will be overlooked.

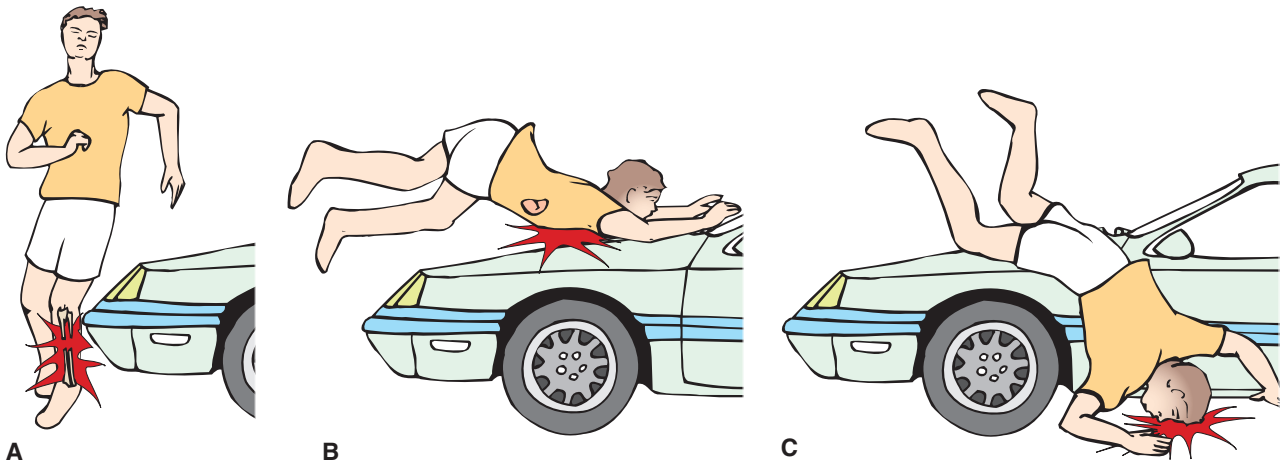


FIGURE 55-2 ▲ Pedestrians may be hurt critically when hit by a moving vehicle. **A:** A common injury is the fracture of the tibia and fibula at the time of impact. **B:** Impact when the pedestrian strikes the hood of the car may cause fractured ribs and a ruptured spleen. **C:** Injuries to the head and additional fractures of the extremities may occur as the pedestrian rolls off the braking car or is thrown by the impact.

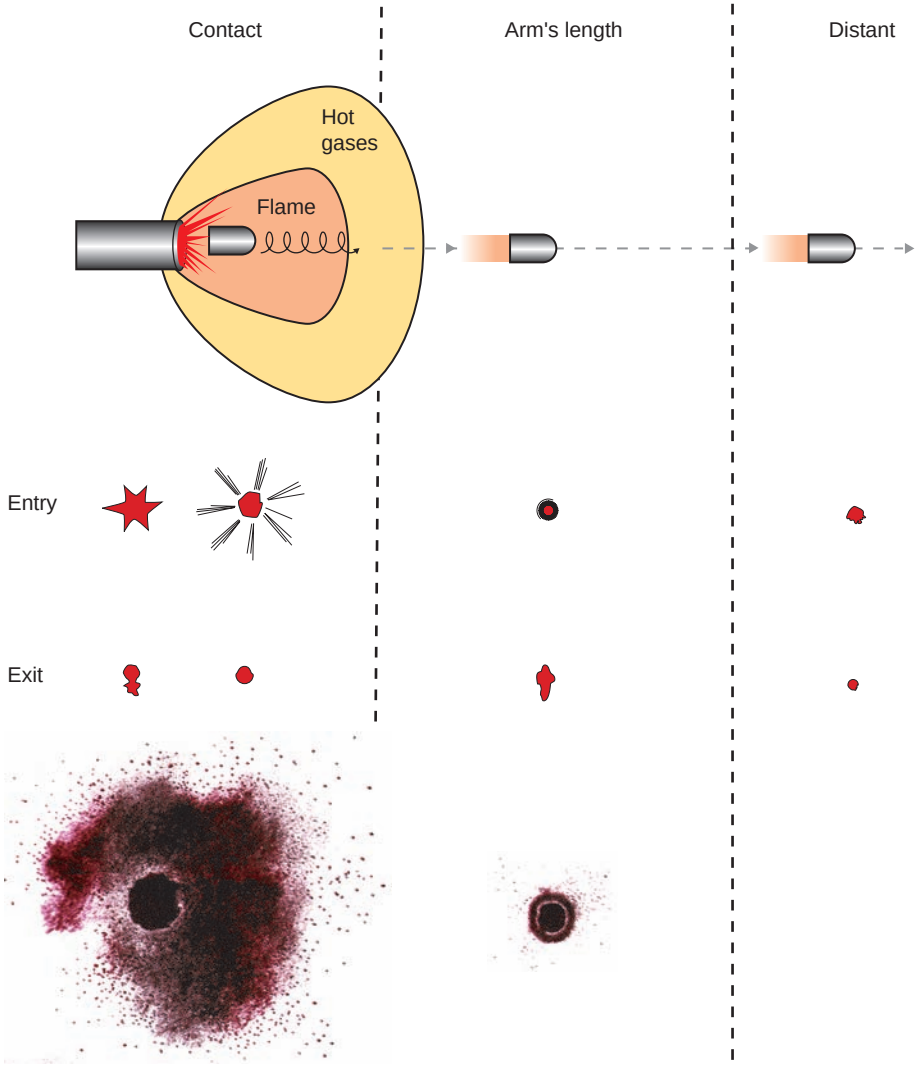


FIGURE 55-3 ▲ Diagrammatic views of the effects of gunshot wounds on the body surface. Kinetic energy is dependent on the distance from which the weapon is fired and the tissue involved. Entry and exit wounds are shown when in direct contact, at arm's reach, and at a distance. The bottom illustrations show entry wounds of .22 rifle at 5-cm range (**left**) and at 20-cm range (**right**). The drilled-in entry wound and faint powder markings are indicated.

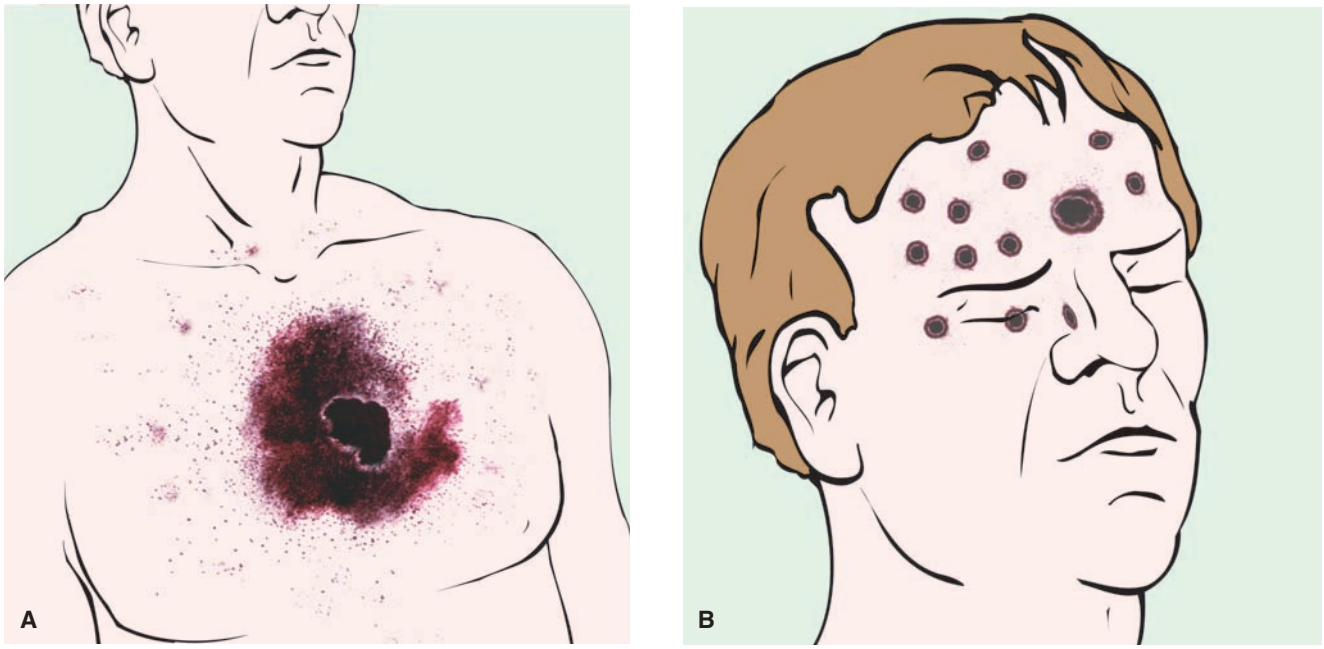


FIGURE 55-4 ▲ Damage is caused by shotguns at two different distances. **A:** At close range, opening is extensive and is surrounded by blood splatters and powder burns. **B:** At medium range (8 to 10 ft), the larger entry wound is surrounded by individual pellet wounds.

Prehospital Management

The trauma patient has a greater chance of a positive outcome if admission to a trauma center and definitive care are initiated within 1 hour of injury. Care begins in the prehospital arena and is continued throughout the hospital stay. Many current studies discuss the prehospital management of trauma patients and seem to conclude that the principal factor influencing prehospital care is the transport time to the trauma center. The swift transport of trauma patients to the most appropriate facility and the appropriate identification and treatment of injuries are essential components of modern trauma systems.[7]

The advanced trauma life support guidelines state that the emphasis for assessment and management in the prehospital phase should be placed on maintaining the airway, ensuring adequate ventilation, controlling external bleeding and preventing shock, maintaining spine immobilization, and transporting the patient immediately to the closest appropriate facility.[8] The prehospital priority of maintaining adequate airway, breathing, and circulation (ABC) may be difficult because of the mechanism of injury. It is imperative that cervical spine immobilization be maintained at all times during airway management and transport to definitive care. After assessing and managing the ABCs, the trauma patient's neurological status is assessed, including level of consciousness and pupil size and reaction. Once this primary assessment is complete, a secondary assessment is performed to determine any other injuries.

The prehospital providers must consider the facility that will receive the patient (Table 55-1). Transporting the patient to a level I facility allows definitive care to be initiated earlier in the process, thereby reducing patient mortality. Trauma systems have been designated to get the "right patient to the right resources in the right time frame." Creation of trauma centers and trauma programs has a positive effect on outcomes in severely injured patients.[9] Transport of the patient to a lesser facility for "stabilization," followed by transport to the definitive care setting later on, is associated with higher patient mortality. For select severe injuries, level I trauma centers have significantly better survival and functional outcomes than level II, III, or IV centers.

Research has shown that injury-related mortality is significantly reduced with organized systems of trauma care, including prehospital care, acute care, and rehabilitation.[10] Inclusive trauma systems have been designed to care for all injured patients and involve all acute care facilities to the extent that their resources allow.[10]

In-Hospital Management

In-hospital patient management entails a rapid primary evaluation and resuscitation of vital functions, a more detailed secondary survey, a tertiary survey to identify specific injuries, and initiation of definitive care. Table 55-2 shows the process, the initial assessment and management of the patient with trauma, commonly referred to as the "ABCDEs" of trauma care. According to the ACS, adhering to this sequence allows for the efficient identification of life-threatening conditions.[7] Optimal care of the trauma patient includes a preplanned emergency phase involving a predetermined response team with defined roles and expectations. This is necessary so that multiple procedures can be performed simultaneously. The team leader is a physician. It is the leader's responsibility to assess the patient, order and interpret the diagnostic studies, and prioritize the diagnostics and therapeutic concerns.

Table 55-1 Trauma Center Designation

	Level I	Level II	Level III	Level IV
Admission requirements	1,200 patients per year; 20% with an Injury Severity Score (ISS) 15 or more or 35 patients per surgeon with an ISS of 15 or more	Varies depending on geographical area, population, resources available, and system maturity	No requirement	No requirement
Surgeon availability	24-h in-house attending surgeon	Rapidly available	Promptly available	24-h emergency coverage
Research center	Required	Not required	Not required	Not required
Education, prevention, and outreach	Required	Required	Required	Required

Source: Scalea TM, Boswell SA: Initial management of traumatic shock. In McQuillan KA, Von Rueden KT, Hartsock RL, et al (eds): Trauma Nursing, 3rd ed. Philadelphia, PA: WB Saunders, 2002.

Primary Survey

During the primary survey, each priority of care is dealt with in order. The patient's assessment does not continue to the next phase until each preceding priority is effectively managed. For example, if a patient does not have a patent airway, breathing and ventilation cannot be established. Therefore, it is during this initial phase that life-threatening injuries are identified and managed. So, if the patient does not have a patent airway, endotracheal intubation, chest tube insertion, and central vascular line access may be initiated and intravenous (IV) fluid and blood products may be administered to maintain life-sustaining vital signs before moving on to the next phase of the evaluation. Initial radiographs and procedures are dependent on the findings in the primary survey. However, chest, abdomen, and pelvis imaging is generally completed at this time.

Assessing the patient for evidence of hypovolemia is essential. Blood loss can result from an external injury, associated with obvious bleeding, or from an internal injury, where bleeding may not be obvious. Any of these injuries can lead to inadequate tissue perfusion, which equals traumatic shock. It is necessary to first stop the bleeding with compression or surgery and then replace the lost intravascular volume. Some signs of hypovolemia include pallor, poor skin integrity, diaphoresis, tachycardia, and hypotension. Usually, trauma patients arrive at the trauma center with a large-bore IV line already in place, with IV fluid running in rapidly.

During the resuscitation period, electrocardiography (ECG) is performed. The patient is placed on a monitor with pulse oximetry and end-tidal carbon dioxide monitoring. A Foley catheter and a nasogastric or orogastric tube are

Table 55-2 Initial Assessment and Management of the Patient With Trauma

Parameter	Assessment	Interventions
Airway	Air exchange Airway patency	Jaw thrust, chin lift Removal of foreign bodies Suctioning Oropharyngeal or nasopharyngeal airway Endotracheal intubation (orally or nasally) Cricothyrotomy
Breathing	Respirations (rate, depth, effort) Color Breath sounds Chest wall movement and integrity Position of trachea	Supplemental oxygen Ventilation with bag–valve device Treatment of life-threatening conditions (eg, tension pneumothorax)
Circulation	Pulse, blood pressure Capillary refill Obvious external bleeding Electrocardiogram	Hemorrhage control: direct pressure, elevate extremity, pneumatic antishock garment Intravenous therapy: crystalloids, blood transfusion Treatment of life-threatening conditions (eg, cardiac tamponade) Cardiopulmonary resuscitation
Disability	Level of consciousness Pupils	—
Exposure	Inspection of body for injuries	—

placed, and blood specimens are sent to the laboratory for evaluation. Blood work includes evaluation of electrolytes, hemoglobin and hematocrit, blood type and crossmatch, and arterial blood gases (ABGs), if the patient is believed to have a high level of injury.

The nurse also assesses the patient for hypothermia. The trauma patient is often subjected to environmental factors, which, along with his or her altered physiological state and possibly wet clothing, predispose the patient to hypothermia. Some measures, such as the infusion of room-temperature IV fluids or exposure of the patient's body to inspect for injuries, can exacerbate hypothermia. Warm fluids and blankets are used whenever possible to increase body temperature or maintain normothermia.

Secondary Survey

Once the primary survey is completed, a more detailed secondary survey is initiated. This survey begins at the head and works down to the patient's feet. Non-life-threatening injuries are revealed during this survey. During this time, a plan is developed and the appropriate diagnostic tests (eg, radiographs, ultrasound studies, computed tomography [CT] scans, angiographic studies) are ordered. This is also the time when a more detailed patient history can be obtained, as well as important information regarding the mechanism of injury. The nurse asks the field providers for information regarding the incident because the patient may not be able to speak or may not remember the event. Family and friends might be helpful in providing additional information about the patient.

Questions the nurse asks before or during the trauma patient's arrival to the hospital include the following:

- Was the person involved in an MVC? Was the person wearing a restraining device? If the person was hit by a vehicle, was the person on foot or on a bike? What kind of vehicle was involved? Where was the person at the time of impact? What was the speed, point of impact, type of impact? Was there a fatality at the scene?
- Is blunt or penetrating trauma the main concern?
- Did the patient fall? How far? Was the fall off a ladder or down a flight of stairs?

Based on the information obtained from field providers or family members of the patient, other injuries may be suspected, and further investigation may be warranted. This is especially true in intubated, comatose, or paralyzed patients who are unable to verbalize their complaints. The nurse continuously reassesses the trauma patient because injuries often go undetected.

Tertiary Survey

The tertiary survey is completed on all trauma patients admitted to the intensive care unit (ICU). It is necessary to identify all of the patient's injuries completely. To do this, another head-to-toe examination is completed, an assessment of the patient's response to resuscitation is made, films are reviewed with the radiologist, laboratory values are reviewed, and every effort is made to obtain or complete a preinjury medical history. Delays in injury identification are common. However, if the injury is found within 24 hours of admission, it is not considered a missed injury.

Fluid Resuscitation

Most trauma patients have a fluid volume deficit that must be corrected. The goal of fluid resuscitation is to maintain perfusion to the vital organs, especially the heart and the brain, by restoring circulating volume.[11,12] It is essential to have adequate intravascular volume and oxygen-carrying capacity to transport needed nutrients to the tissues.[8] Fluid administration is one of the most basic concepts in resuscitation and is also a part of the daily routine of medically managed patients in the hospital.[11,12] To guide fluid resuscitation, the nurse uses physical assessment and hemodynamic parameters. Two factors affect the choice of fluid: how the volume loss occurred and which solutes need to be replaced.[13] It is important to address the underlying problem causing the loss of fluids, electrolytes, or both. With aggressive fluid resuscitation, many patients have total-body edema and ascites. The two main complications of aggressive fluid resuscitation are hypothermia and coagulopathy.[13]

Crystalloids

Typically, crystalloids are used for the trauma patient. Crystalloids contain water and other electrolytes that are premixed into the fluid. These electrolytes include sodium, potassium, and chloride. Crystalloids closely mimic the body's extracellular fluid and can be used to expand both intravascular and extravascular fluid volume. There tends to be a larger requirement for crystalloid replacement compared with the amount of blood lost. Crystalloids can be further broken down by their tonicity. The tonicity is based on the amount of sodium in the solution. Crystalloids can be classified as isotonic, hypotonic, and hypertonic (Table 55-3).

Hypertonic saline has been shown to enable a more rapid restoration of cardiac function with a smaller volume of fluid. It is supplied either in a 3%, 7.5%, or 23.4% sodium chloride (NaCl) solution. As little as 4 mL/kg, if given rapidly, may have the same hemodynamic effect as several liters of isotonic crystalloid. Hypertonic saline solution has the effect of shifting water into the plasma. This water comes from the red blood cells, interstitial space, and tissue.[14] The result is a rapid increase in blood volume, which supports and improves hemodynamics. Hypertonic saline solution increases the mean arterial pressure and cardiac output, which then leads to peripheral vasodilation. Some studies have indicated that

Table 55-3 Intravenous Fluid

Isotonic	• Example: 0.9 normal saline solution • Equivalent to the tonicity of the human body • Causes minimal shifts between intracellular and extracellular fluid
Hypotonic	• Example: 5% dextrose in water (D_5W) • Tonicity is less than that of human body • May cause swelling, pulls into extracellular space
Hypertonic	• Example: 3% saline solution • Tonicity is more than that of human body • Pulls fluid into the intravascular space

Modified from American Association of Critical-Care Nurses: Clinical Reference for Critical-Care Nursing, 4th ed. Aliso Viejo, CA: American Association of Critical-Care Nurses, 1998.

no change in survival rates were demonstrated in patients given hypertonic saline solution versus lactated Ringer's solution.[13]

The initial management of trauma patients often requires the infusion of 2 L of isotonic crystalloid as rapidly as possible, while trying to achieve a normal heart rate and blood pressure.[12] However, research has shown that the infusion of crystalloids in patients with hypotension can cause more harm by displacing a hemostatic clot, only to cause more bleeding.[11–13] The infusion of crystalloid also further dilutes the patient's hemoglobin and can increase intraperitoneal blood loss. Makley et al suggest that "conventional fluid resuscitation may prime the inflammatory response for the development of organ injury and late complications including ARDS." This is thought to be related to hemorrhagic shock with tissue ischemia, and subsequent reperfusion injury promotes a systemic inflammatory response, which contributes to the development of late complications.[15]

Colloids

Colloids can also be given to resuscitate a trauma patient. Colloids, such as albumin, dextran, and hetastarch, create oncotic pressure, which encourages fluid retention and movement of fluid into the intravascular space. Colloids have a longer duration of action because they are larger molecules and stay in the intravascular compartment longer.[14] They are also more efficient in expanding plasma volume, use a smaller volume, and increase colloid osmotic pressure.[14]

Proponents of colloid use have argued that less volume of fluid is necessary to achieve hemodynamic stability and that the fluid is retained in the intravascular space longer. Despite possible advantages, there is no clear evidence that colloids are superior to crystalloids for resuscitation of the trauma patient. Potential complications, such as anaphylaxis and coagulopathy, have been reported with certain colloids. These potential adverse affects, together with higher costs, make colloids less desirable than crystalloids for use in resuscitation of trauma patients.

Blood Products

Blood products are considered an excellent resuscitation fluid. Red blood cells increase oxygen-carrying capacity and allow for volume expansion.[12] It is well known that the maintenance of adequate oxygen delivery is critical in the bleeding trauma patient; therefore, packed red blood cells are the mainstay of treatment. Blood also stays in the intravascular space for longer periods of time compared with the other resuscitation fluids.[12] Although there is some concern about blood-borne pathogens and transfusion reactions, it is essential to understand the advantages offered by blood transfusion.

Blood should be transfused when patients are hemodynamically unstable or are showing signs of tissue hypoxia despite crystalloid infusion. Crossmatched blood is preferred but may not be available if emergent transfusion prohibits type and crossmatching of the patient's blood. O-negative blood is the preferred type of uncrossmatched blood, especially in women of childbearing age. O-positive blood may be used in male and postmenopausal female patients. If the patient requires large amounts of blood, transfusion of fresh frozen plasma and platelets is initiated. It is important to replace coagulation factors and platelets not contained in blood. In the event of massive blood transfusions, the risk for acute respiratory distress syndrome (ARDS) and disseminated intravascular coagulation (DIC) is heightened. An extended period of hypotension increases the possibility of renal failure.

Autotransfusion is another common modality used in the hemorrhaging trauma patient. Obviously, the nature of trauma prevents patients from donating their own blood, as they may in an elective surgery. However, sometimes blood is salvaged. Most often, blood is saved from a chest tube underwater seal device. A cell saver is connected into the system, and the blood from the wound collects there. Once full, the cell saver is disconnected from the underwater seal device, and this blood is then transfused into the patient using a macroaggregate filter.

Blood Substitutes

Blood substitutes have been developed but have not been approved for use in all countries. These agents do not require crossmatching and do not carry the risk for blood-borne pathogen transmission. Blood substitutes have a long shelf life and are not immunosuppressive. Blood substitutes have oxygen-carrying capacity and oxygen dissociation capability of natural hemoglobin, and in addition, they have the ability to maintain hemodynamic stability and intravascular perseverance.[13] Some examples of blood substitutes are perfluorocarbons and hemoglobin solutions.[13]

Damage Control

Damage-control surgery avoids widespread procedures on unstable patients, stabilizes potentially fatal problems at initial operation, and utilizes staged surgery after initial resuscitation.[16] It is designed to avoid or correct the lethal triad of hypothermia, acidosis, and coagulopathy before definitive management can be performed. Uncontrolled bleeding and iatrogenic interventions ultimately result in hypothermia, coagulopathy, and acidosis. Each of these exacerbates the other, causing a spiraling cycle with death as the ultimate result.[16–18]

Damage control involves using a staged approach to patients with multiple injuries. The stages are as follows:

- Stage 1: Stop hemorrhage, control contamination, and perform closure methods to close wounds temporarily.
- Stage 2: Correct physiological abnormalities in the ICU by warming and ensuring adequate resuscitation as well as correcting coagulopathy.
- Stage 3 (final stage): Definitive operative management

The philosophy of damage control is to abbreviate surgical interventions before the development of irreversible physiological end points.[16–18] Traditionally, damage control was used for abdominal trauma, but it is now used for all types of trauma that require immediate surgery.

Definitive Care

Increasingly, trauma care consists of nonoperative management of stable patients. Traditionally, solid organ injuries, both blunt and penetrating, were treated with surgery. Today, many trauma surgeons are choosing nonoperative management for their patients whenever possible. Ever more sophisticated techniques for visualization of internal structures,

BOX 55-1 EXAMPLES OF NURSING DIAGNOSES

For the Person With Trauma

- Deficient Fluid Volume related to hemorrhage, third spacing
- Impaired Gas Exchange related to pulmonary trauma, respiratory complications (eg, acute respiratory distress syndrome [ARDS], pain)
- Impaired Tissue Integrity related to trauma, surgery, invasive procedures, immobility
- Anxiety related to critical illness, fear of death or disfigurement, role changes within social setting, or permanent disability
- Risk for Decreased Cardiac Tissue Perfusion, Risk for Ineffective Cerebral Tissue Perfusion: Multiple organs related to decreased cardiac output, decreased oxygenation, decreased gas exchange
- Risk for Infection related to trauma, invasive procedures
- Acute Pain related to trauma
- Dysfunctional Gastrointestinal Motility related to intra-abdominal trauma, ileus, or both
- Ineffective Coping, Compromised Family Coping related to trauma

such as CT, ultrasonography, and angiography, have reduced the need for immediate surgical exploration in many cases. Without question, CT offers improved accuracy and high sensitivity and specificity.[19] It provides more information to aid in diagnosing injuries that may have been missed without it. Using CT to discharge a patient earlier, or clear him or her for an earlier surgery with another service, makes sense from a patient care and economic perspective. In addition, many of these techniques can be used for management, as well as diagnosis. For example, angiographic interventions may be used to embolize a hemorrhaging internal vessel, obviating the need for invasive surgical intervention. Patients who are treated nonoperatively require frequent assessment and are admitted to the ICU to facilitate this.

To observe the patient effectively, the nurse must be aware of potential injuries and associated signs and symptoms. Examples of nursing diagnoses are given in Box 55-1. Attention is also given to the management of preexisting medical conditions and the identification of injuries missed during treatment of life-threatening problems. Once again, knowledge regarding the mechanism of injury is necessary. Finally, the patient is monitored for the development of complications. The critical care nurse must be aware of potential complications and related risk factors associated with various injuries. Certain situations, such as prolonged extrication, prolonged hypothermia, respiratory or cardiac arrest, massive fluid resuscitation, or massive blood transfusions, suggest an increased likelihood of severe injuries and a greater chance of complications and death after trauma. A collaborative care guide for the patient with multisystem trauma is given in Box 55-2.

▲ Assessment and Management of Specific Injuries

Although this section discusses traumatic injuries related to specific areas of the body, the nurse must keep in mind that head-to-toe assessment is required for each trauma patient. Physical assessment of each organ system is indicated, as described in previous chapters throughout this text.

Thoracic Trauma

Thoracic injuries range from simple abrasions and contusions to life-threatening insults to the thoracic viscera. Although these injuries are associated with a high mortality rate, most can be managed with simple chest tube insertion, mechanical ventilation, aggressive pain control, and other supportive care. Great vessel injuries or disruption to the heart usually result in immediate death. Early deaths (30 minutes to 3 hours after injury) are related to cardiac tamponade, tension pneumothorax, aspiration, or airway obstruction.[20]

Immediate life-threatening injuries require evaluation and treatment during the primary survey. Examples of these include airway obstruction, tension pneumothorax, cardiac tamponade, open pneumothorax, massive hemothorax, and flail chest (Fig. 55-5). Potentially life-threatening injuries, such as thoracic aortic disruption, tracheobronchial disruption, myocardial contusion, traumatic diaphragm tear, esophageal disruption, and pulmonary contusion, should be addressed during the secondary survey.[20]

In thoracic injury, the first priority is always airway management. This includes immediate airway control as well as adequate oxygenation and protection from aspiration. Airway obstruction may be the result of another injury or the primary problem. The most common causes of airway obstruction are the tongue, avulsed teeth, dentures, secretions, and blood. Other causes of airway obstruction include injuries to the trachea, thyroid cartilage, or cricoid process.

Tracheobronchial Trauma

Injuries to the trachea or bronchi can be caused by blunt or penetrating trauma and frequently are accompanied by esophageal and vascular damage. Ruptured bronchi often are present in association with upper rib fractures and pneumothorax. Severe tracheobronchial injury is associated with a high mortality rate; however, with continued improvements in prehospital care and transport, more of these patients are surviving.

Airway injuries often are subtle. Presenting signs include dyspnea (occasionally the only sign), hemoptysis, cough, subcutaneous emphysema, anxiety, hoarseness, stridor, air hunger, hypoventilation, use of accessory muscles, sternal and subscapular retractions, diaphragm breathing, apnea, and cyanosis. Cyanosis may be a late sign. Often, trauma patients are anemic and do not have enough hemoglobin to develop cyanosis. A chest radiograph can alert the physician to a possible injury; however, diagnosis usually is made with bronchoscopy or during surgery. Tracheobronchial injury is considered whenever a persistent air leak accompanies a pneumothorax.

Small lung lacerations or pleural tears can be managed conservatively with mechanical ventilation delivered through an endotracheal tube or tracheostomy. Larger injuries may require surgical repair. Simultaneous independent lung ventilation, in which each lung is ventilated

BOX 55-2 *COLLABORATIVE CARE GUIDE for the Patient With Multisystem Trauma*

Outcomes	Interventions
Oxygenation/Ventilation	
The patient will maintain a patent airway.	• Auscultate breath sounds. • Perform frequent assessments. • Intubate if needed. • Provide supplemental oxygen PRN.
The patient will maintain an SaO_2 of 95% or more and have adequate ABG values.	• Provide pulmonary toilet (chest physiotherapy and incentive spirometry). • Intubate. • Monitor ABGs.
The patient will be able to take deep breaths and will be free of anxiety.	• Use mechanical ventilation if necessary to support adequate ventilation. • Provide adequate pain medication to promote deep breathing (patient-controlled analgesia [PCA], epidural, around-the-clock medications) • Medicate before pain increases. • Use antianxiety drugs as necessary.
Circulation/Perfusion	
The patient will maintain an adequate blood pressure, heart rate, and respiratory rate.	• Monitor respiratory rate and depth. • Use ECG monitor. • Administer intravenous (IV) fluids and packed red blood cells to ensure adequate intravascular volume and oxygen-carrying capacity. • Administer medications, such as vasoactive and inotropic agents, after intravascular volume is restored. • Install pulmonary artery catheter/A-line. • Assess skin color and capillary refill time.
The patient will not experience deep venous thrombosis.	• Use prophylactic anticoagulants unless contraindicated. • Apply antiembolic stockings. • Use pneumatic compression devices.
Fluids/Electrolytes	
The patient will maintain an adequate intake and output.	• Monitor blood pressure, heart rate, central venous pressure, pulmonary capillary wedge pressure, IV fluid. • Use Foley catheter to monitor urine output. • Consider insensible fluid loss in output. • Monitor laboratory values.
The patient will maintain electrolyte balance.	• Replace electrolytes PRN. • Monitor ECG.
Mobility/Safety	
The patient's range of motion will be maintained.	• Consult physical/occupational therapist. • Use splints PRN. • Do range-of-motion exercises every 8 h. • Out of bed as tolerated.
Skin Integrity	
The patient will not experience skin breakdown.	• Monitor skin every 4 h. • Turn patient every 2 h and PRN. • Use pressure-relieving devices. • Remove splints to monitor skin. • Provide prescribed wound care. • Monitor wound for evidence of infection.
Nutrition	
The patient will maintain an adequate calorie intake to meet metabolic needs.	• Arrange dietary/nutrition consult. • Use total parenteral nutrition/lipids if enteral nutrition contraindicated. • Tube feeds: Encourage enteral nutrition when possible. • Check prealbumin and electrolytes. • Monitor for weight loss.

(*continued on page 1243*)

BOX 55-2 COLLABORATIVE CARE GUIDE for the Patient With Multisystem Trauma (continued)

Outcomes	Interventions
Comfort/Pain Control	
The patient will maintain a pain score of <5.	• Administer adequate pain medication. • Use PCA/epidural PRN. • Arrange pain consult if needed. • Use sedation as needed. • Monitor vital signs.
Psychosocial	
The patient will maintain as much control as possible.	• Inform patient of procedures. • Establish a schedule with the patient if possible. • Provide an alternate means of communication if necessary, such as lip reading, writing, and a communication board.
The patient and family will cope effectively with the traumatic event.	• Provide repeated information. • Encourage use of appropriate coping. • Encourage use of support systems. • Arrange social work consult.
Teaching/Discharge Planning	
The patient will be involved in discharge planning.	• Discuss discharge with patient. • Allow patient to make decisions if possible.
The patient will understand injuries and complications of injuries.	• Provide discharge instructions accordingly with injury. • Provide patient with list of injuries.

separately (each with a dedicated ventilator), may also be used.

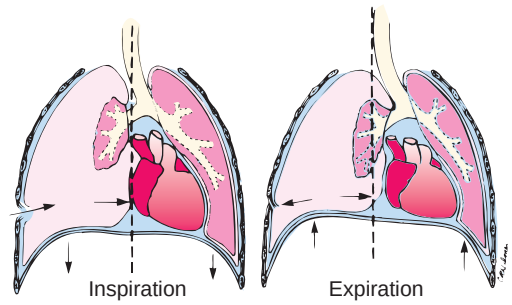
Nursing care involves the assessment of oxygenation and gas exchange, along with appropriate pulmonary care. During the first few days, the physician may perform a bronchoscopy to visualize the repair site and provide more effective secretion removal. Pneumonia is a potential short-term complication, whereas tracheal stenosis may occur later.

Bony Thorax Fractures

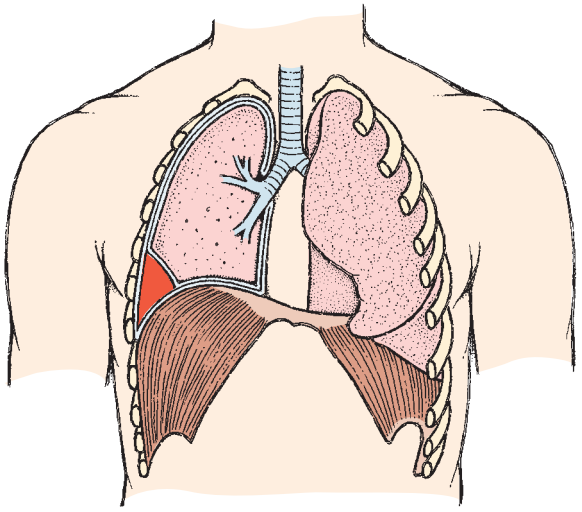
Rib fractures, sternal fractures, and flail chest are thoracic fractures commonly seen in trauma patients. They occur when the applied force exceeds the strength of the thoracic cage. Rib fractures are common injuries. They are clinically significant as (1) markers of serious intrathoracic and abdominal injuries, (2) sources of significant pain, and (3) predictors of pulmonary deterioration. With rib fractures, the most common associated thoracic injuries are pneumothorax, hemothorax, and pulmonary contusion, and the most frequently injured abdominal organs are the liver and the spleen.[21] The greatest concerns for nurses caring for patients with such injuries are pain, ineffective ventilation, and secretion control. Ribs 1 and 2 are usually protected by the clavicle, scapula, humerus, and surrounding muscles. If these ribs are fractured, it often signifies that high-impact trauma and other injuries, for example, to the aorta, the thorax, and the spine, are very likely and should be investigated. Ribs 4 through 10 are most commonly fractured in blunt trauma. If these ribs are fractured, there are often associated underlying lung injuries. Fractures of the lower ribs (8 through 12) can also be associated with injury to the liver or other abdominal structures.[21] Sternal fractures are associated with blunt trauma.

Flail chest is an injury that involves multiple rib fractures. These fractures can be anterior, posterior, or lateral, and usually a sternal fracture is present as well. The stability of the thorax is disrupted, and the rib cage no longer moves in unison. The diagnosis of flail chest is made on the basis of a fracture of two or more ribs, in two or more separate locations, causing an unstable segment. This creates a free-floating segment of rib or sternum.[21] The injured area does not respond to the action of the respiratory muscles; rather, it moves in accordance with the changes in intrapleural pressure. The flail segment movement is paradoxical, hence the term paradoxical breathing. The flail segment causes a decrease in the normal negative pressure of the chest, thereby decreasing ventilation and causing some degree of hypoxia. The flail segment follows pleural pressure instead of respiratory muscle activity. As the patient's pulmonary status worsens, the paradoxical movement of the flail segment increases. Initially, muscular splinting may mask the injury until the patient becomes fatigued. This patient may require intubation and mechanical ventilation.[21]

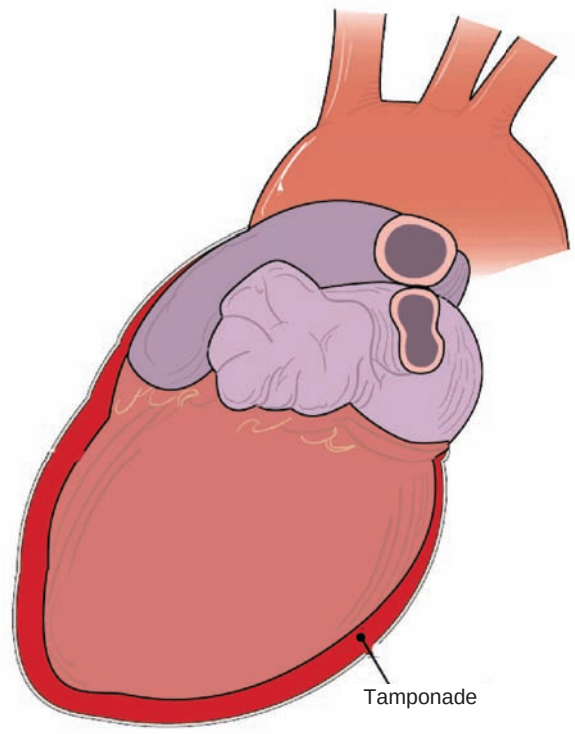
Initial management of patients with bony thorax fractures includes airway management, pain management, and oxygen therapy to maintain adequate saturation. The nurse must consider the underlying structures and the possible injury to them. Treatment of flail chest includes turning the patient with the injured side down to improve oxygenation. This is often difficult because of the need to maintain cervical spine immobilization. Other treatment modalities include internal splinting, accomplished by placing the intubated patient on positive-pressure ventilation. Sometimes, surgical repair is



A. Tension pneumothorax



B. Hemothorax



C. Cardiac tamponade

FIGURE 55-5 ▲ **A:** Tension pneumothorax. **B:** Hemothorax. **C:** Cardiac tamponade. (**B**, Courtesy of Neil O. Hardy, Westpoint, Conn. **C**, Courtesy of LifeART image © 2007 Lippincott Williams & Wilkins. All rights reserved.)

performed, especially if a thoracotomy is necessary for other reasons. Surgical repair may help decrease the need for prolonged mechanical ventilation.

Pleural Space Injuries

For the purpose of this chapter, the term pleural space injuries is used in reference to pneumothorax (intrapleural air collection), hemothorax (intrapleural blood collection), and hemopneumothorax (interpleural air and blood collections). Pleural space injuries are caused by disruption of an intrathoracic structure that allows air or blood to build up in the pleural layers, thereby leading to a decrease in negative intrathoracic pressure. Sometimes, air and blood continue to build up in the pleural cavity, causing increased tension, which leads to a tension pneumothorax or a tension hemothorax. Either blunt or penetrating trauma may result in a pleural space injury.

The mechanism of injury may lead the nurse to suspect a pleural space injury. For example, an unrestrained driver whose chest hits the steering wheel has a great potential for this injury. When assessing the patient, respiratory distress may be evident along with altered ventilation, which leads to impaired gas exchange. Impaired gas exchange may be evidenced by restlessness, anxiety, tachypnea, decreased oxygenation, poor color, and diaphoresis. The nurse continuously reassesses the patient because even if the original injury is small, it can expand, causing a life-threatening emergency.

Chest radiography is usually used to diagnose pleural space injuries. Sometimes, if the pneumothorax is less than 20% of the chest cavity, it may not be seen initially on chest film. A chest CT scan often shows the smaller pleural space injuries.

Treatment of pleural space injuries includes appropriate management of the patient's airway, ventilation, and oxygenation. A large-bore chest tube, such as a 40-French tube, is often inserted to reexpand the lung and drain the air or blood. This tube is inserted in the fourth or fifth intercostal space at the midaxillary line. For trauma patients with a simple pneumothorax, a chest tube may be placed in the second intercostal space at the midclavicular line. Once the tube is inserted, it is attached to an underwater seal system and then attached to suction. The effects of treatment are assessed by chest radiograph, physical examination, and noting of improved oxygenation. Often, there is an air leak in the underwater seal system of the chest tube drainage device that ceases within a few days.

The nurse monitors the amount of blood that drains into the chest tube drainage device. Drainage of more than 250 mL/h for 2 consecutive hours may indicate a missed injury or the need for further exploration and should be reported.[5]

A massive hemothorax is defined as 1.5 to 4 L of intrathoracic blood loss and truly constitutes a life-threatening injury. A massive hemothorax is often caused by severe thoracic injuries, and the source of bleeding is a large systemic blood vessel or mediastinal structure. Patients with massive hemothorax often arrive at the emergency department or trauma resuscitation unit in cardiopulmonary arrest. These patients require immediate thoracotomy to control bleeding. The patients who are not in cardiopulmonary arrest present

with signs of hypovolemic shock (see Chapter 54), dyspnea, tachypnea, and cyanosis. Initial management of these patients includes treatment of the shock state. Two large-bore IV lines should be established and resuscitation fluid administered. The amount of fluid administered depends on the patient's response.[5,21]

A left massive hemothorax is more common than a right one and is often associated with aortic rupture. The chest cavity is large enough to hold most of the patient's circulating blood volume. Because of this, the bleeding stops only when the pressure in the pleural cavity is equal to or greater than the pressure in the damaged vessel. Placement of a chest tube in a patient with massive hemothorax could lead to exsanguination by eliminating the tamponade caused by a closed chest injury. If a chest tube is inadvertently placed, it should be clamped until exploratory thoracotomy can be performed.

Tension pneumothorax is a life-threatening condition that requires immediate recognition. It may be the result of a primary injury to the thorax or a delayed complication related to tracheobronchial injury or mechanical ventilation. Tension pneumothorax is caused by air entering the pleural space and becoming trapped without an exit. A one-way valve closed system is formed. This causes a compression of one or more of the intrathoracic structures (trachea, heart, lungs, and great vessels) and prohibits them from functioning adequately. The outcome is ventilation failure, compromised venous return, and insufficient cardiac output.[21]

Tension pneumothorax is often difficult to diagnose in the trauma patient because of other injuries the patient may have, as well as the presence of a shock state. It may not be diagnosed until the patient has decompensated. The nurse may notice that it is difficult to ventilate the patient, despite an open airway. There is often a drop in the patient's oxygenation. Other signs of tension pneumothorax include chest asymmetry, tracheal shift, neck vein distention (unless the patient is hypovolemic), decreased breath sounds on the affected side, and evidence of decreased cardiac output (eg, decreased blood pressure and poor tissue perfusion).

Treatment of tension pneumothorax requires immediate decompression of the trapped air. This is done initially by placing a 14- or 16-gauge needle into the pleural space, usually between the second to fourth anterior intercostal space. An immediate rush of air should escape, and the patient's ventilation should improve. Supplemental oxygen is provided to the patient before decompression. After emergent decompression, the needles are changed to chest tubes. This is done to allow the lungs to expand as well as to prevent a reoccurrence. Lastly, additional assessment is necessary to determine the cause of tension pneumothorax. The nurse must continue to assess and reassess the patient.

Pulmonary Contusion

A pulmonary contusion is a bruising of the lung parenchyma, often caused by blunt trauma. It is the most common lung injury.[20,21] It is often apparent on the chest radiograph and CT as ill-defined, patchy, ground-glass density regions of opacification in mild contusion to widespread areas of consolidation in more severe injury. CT is more sensitive in diagnosing pulmonary contusions because it may take up to 24 hours for the pulmonary contusion to show up on chest radiograph.[21] However, the presence of a scapular fracture, rib fractures, or a flail chest should lead to the suspicion of a possible underlying pulmonary contusion. In fact, pulmonary contusion should be anticipated in any patient who sustains significant high-energy blunt chest trauma. The most common mechanism for pulmonary contusion is MVCs.

Pulmonary contusion occurs when rapid deceleration ruptures capillary cell walls, causing hemorrhage and extravasation of plasma and protein into alveolar and interstitial spaces. This results in atelectasis and consolidation, leading to intrapulmonary shunting and hypoxemia. Presenting signs and symptoms include dyspnea, rales, hemoptysis, and tachypnea. Severe contusions also result in increasing peak airway pressures, hypoxemia, and respiratory acidosis. Pulmonary contusion may mimic ARDS; both are poorly responsive to high fractions of inspired oxygen (FiO_2). ARDS is discussed in detail in Chapter 27. The greater the degree of pulmonary contusion, the greater the degree of ventilatory impairment.[21]

Treatment of pulmonary contusion is supportive. Patients with a mild contusion require close observation with frequent ABG measurements or pulse oximetry monitoring. Additional nursing interventions include frequent respiratory assessment, pulmonary care, and pain control. Chest physiotherapy and continuous epidural analgesia also may be beneficial. An oximetric pulmonary artery catheter and arterial line usually are placed to facilitate monitoring of ABGs, hemodynamics, and respiratory parameters (oxygen delivery, oxygen consumption, intrapulmonary shunt).

Severe pulmonary contusion may require ventilatory support with positive end-expiratory pressure (PEEP). Although alveolar ventilation improves as PEEP is added, blood flow to alveoli may diminish, leading to an increased intrapulmonary shunt. To optimize tissue perfusion and oxygenation, each change in PEEP requires assessment of the status of the shunt, oxygen delivery, and other indicators of tissue perfusion (cardiac output, blood pressure, and urine output). Adequate pain control is necessary and may require epidural or intrapleural infusions of analgesics or an intracostal nerve block. In severe cases of respiratory compromise, increased sedation or paralysis may be indicated to decrease energy expenditure and oxygen requirements. A rotation bed also may be considered to promote pulmonary toilet and respiratory gas exchange. Positioning the patient with the injured side up is beneficial in the case of a severe unilateral contusion. In rare instances, when the patient does not respond to traditional mechanical ventilation, prone positioning and high-frequency jet ventilation may be used. Another mode of ventilation commonly used is airway pressure-release ventilation.

Fluid management also is important. Intake and output, daily weights, central venous pressure, and pulmonary artery and capillary wedge pressures are monitored to guide fluid administration. Medications may need to be more concentrated to compensate for excess fluid intake, and diuretics may be required periodically. Severe fluid restriction is not indicated. Instead, fluid balance should be maintained at a normal level (euvolemia) to support optimal cardiac output and oxygen delivery. The contused lung should show radiographic signs of improvement within 72 hours. The

presence of persistent infiltrates may indicate complications, such as pneumonia or superimposed ARDS. Long-term sequelae include prolonged reduced functional residual capacity, dyspnea, and fibrosis.

Blunt Cardiac Injuries

Blunt cardiac injuries encompass a wide spectrum of clinical manifestations ranging from asymptomatic myocardial bruise to cardiac rupture and death.[21] These injuries include cardiac wall rupture, valvular disruption, coronary artery dissection, and cardiac contusions. Table 55-4 describes the blunt cardiac injury (BCI) classification according to the sequelae of the injury. There are few clinical signs and symptoms of BCI. Chest pain is usually the most common, and others secondary to thoracic injuries often occur (eg, dyspnea, chest wall ecchymosis, flail chest).[21]

Cardiac contusions, the most common form of blunt cardiac injuries, are usually caused by blunt trauma as the heart hits the sternum during rapid deceleration. A contusion can also develop if the heart is compressed between the sternum and the spine. Symptoms vary from none (common) to severe congestive heart failure and cardiogenic shock.[22] Complaints of chest pain must be evaluated carefully after trauma. Nonspecific ECG changes, which can include any type of dysrhythmia, are frequently seen. A dysrhythmia always indicates a cardiac contusion until proven otherwise. Atrial dysrhythmias and conduction disturbances may be seen with right-sided cardiac injuries; ventricular disturbances are more likely after left-sided cardiac injuries.

A cardiac contusion is suspected when there is a history of severe anterior blunt trauma and the patient has chest wall bruising and fractures of the ribs or sternum. A 12-lead ECG is performed to detect any electrical abnormalities. Most patients with cardiac contusions have ECG abnormalities on admission.[22] However, there is no correlation between the complexity of the dysrhythmia and the degree of the cardiac contusion. These patients are placed on continuous cardiac monitoring, and blood is drawn for cardiac isoenzyme and troponin studies. Although cardiac enzymes lack specificity in terms of diagnosis, they are used to guide therapy.

Controversy exists over the standard of care for patients with cardiac contusion. Because there is no standard in diagnosing this injury, there is also no standard in treatment.[22]

Table 55-4 Blunt Cardiac Injury (BCI) Classification According to the Sequelae of the Injury

Classification	Description
1	BCI with cardiac free wall rupture
2	BCI with septal rupture
3	BCI with coronary artery injury
4	BCI with cardiac failure
5	BCI with complex dysrhythmias
6	BCI with minor electrocardiographic or cardiac enzymes abnormalities

From Schultz JM, Trunkey DD: Blunt cardiac injury. Crit Care Clin 20:57–70, 2004.

Continuous monitoring must be done to evaluate for symptomatic dysrhythmias, especially ventricular irritability and conduction defects. Echocardiography or multigated angiography may be helpful in determining any muscle defect or damage. In general, patients are treated to relieve their symptoms.

Penetrating Cardiac Injury

In most cases, a penetrating injury to the heart results in prehospital death. Those who survive do so because of cardiac tamponade. Cardiac tamponade and hypovolemic shock are the common presenting signs.[20] The right ventricle is injured most often because of its anterior location.[5] Occasionally, small stab wounds to the ventricles seal themselves because of the thick ventricular musculature. Treatment of hemodynamically stable patients remains controversial. In some instances, monitoring with serial CT scanning or with pericardial and pleural ultrasonography is acceptable. In other cases, surgery to create a thoracoscopic pericardial window may be necessary to aid in the diagnosis of ongoing hemorrhage and to drain pericardial fluid collections. In the presence of ongoing hemorrhage and shock, lost blood volume is replaced, and the patient is immediately transported to the operating room for a median sternotomy and exploration. In severe cases, a thoracotomy in the emergency department may be required as a lifesaving measure.

After surgical repair, appropriate central lines and arterial line are placed to facilitate careful hemodynamic monitoring. Vasopressors or inotropic agents may be necessary to maintain adequate blood pressure and cardiac output. Fluid and electrolyte balance, along with cardiac rhythm, must be monitored closely. Heart sounds are assessed to detect murmurs, indicating valvular or septal defects, and for signs of congestive heart failure. Chest and mediastinal tube drainage are recorded frequently. Fresh frozen plasma and platelets are administered, as indicated, to correct coagulopathies. Complications include continued hemorrhage and postcardiotomy syndrome.

Cardiac Tamponade

Cardiac tamponade, known as both a symptom and injury, can result from both penetrating and blunt trauma. It is a life-threatening injury that needs to be immediately assessed and treated. Cardiac tamponade is caused by blood filling the pericardium and compressing the heart, causing decreased cardiac filling, which leads to reduced cardiac output and eventually shock.[23] Bleeding into the pericardial sac (hemopericardium) or a small pericardial rupture may or may not cause cardiac tamponade, depending on the amount of pressure in the pericardium.[23]

The pericardial sac normally holds about 25 mL of fluid, which serves to cushion and protect the heart. Only a small amount of pericardial blood (50 to 100 mL) is necessary to increase intrapericardial pressure. Continued bleeding increases the pressure rapidly, and the patient presents with signs and symptoms of cardiac tamponade.

Classic symptoms include decreased blood pressure, muffled heart sounds, and increased central venous pressure manifested by distended neck veins (Beck's triad).[22] Another key sign to cardiac tamponade is pulsus paradoxus, "an inspiratory

systolic fall in arterial pressure of 10 mm Hg or more during normal breathing."[23] This is caused by a fall in cardiac output. Because these signs may be obscured in the hypovolemic trauma victim, patients with a history of precordial trauma must be treated with a high index of suspicion. Diagnosis of cardiac tamponade is not easy. An echocardiogram is most useful in the diagnosis and is readily available, but the cardiac tamponade is a clinical diagnosis.[23] Treatment of cardiac tamponade includes airway control, oxygenation, hemodynamic support, and rapid transport to a definitive care center. The rapid transfusion of IV fluids increases venous pressure and ultimately improves cardiac output as well as provides the needed time to prepare for interventions. Ultimately, the treatment of cardiac tamponade is drainage of blood from the pericardial sac (pericardiocentesis).[22] This is the only life-sustaining intervention.[23] Nursing management of a patient with cardiac tamponade includes airway protection and ventilatory support, hemodynamic support, and assistance with interventions provided to the patient.

Aortic Injuries

Death from aortic disruption occurs immediately in 75% to 90% of cases.[24] Disrupting the blood flow in the aorta inhibits perfusion to vital organs and extremities. The location and size of the disruption determine its significance.[24] Blunt aortic injury is usually associated with sudden deceleration or compression forces. MVCs are the primary causes of this injury.[20] Of the patients who make it to the hospital alive, 75% are hemodynamically unstable and 50% die before repair. Many of these patients have other significant injuries. Aortography is the gold standard for diagnosis of aortic injuries, although transesophageal echocardiography and chest CT play an increasingly important role.[20]

There are three common locations of vessel rupture. Because the thoracic aorta is very mobile, the tears occur at points of fixation. The most common is at the aortic isthmus, just distal to the left subclavian artery, where the vessel is attached to the chest wall by the ligamentum arteriosum. The two other sites of rupture are in the ascending aorta, where the aorta leaves the pericardial sac, and at the entry to the diaphragm. The inner layers of the vessel tear on impact from deceleration. The outer layers remain intact and balloon out into a pseudoaneurysm. A partial circumferential hematoma may also be tamponaded by surrounding tissues. Both of these mechanisms may prolong survival, but only for a limited time.

An understanding of the injury history can raise the suspicion of aortic injury. Penetrating mediastinal injuries or thoracic injuries caused by blunt trauma should raise suspicion. Other injuries that may raise suspicion include first or second rib fractures, high sternal fractures, clavicular fractures at the sternal margin, and massive left-sided hemothorax.

Loss of effective blood transport because of major vessel rupture is the main physiological problem associated with aortic rupture. The goal of assessment is to identify evidence of poor perfusion beyond the aortic lesion. Many patients are asymptomatic on presentation. Findings associated with aortic injuries are given in Box 55-3.

A supine chest radiograph is obtained to aid in diagnosis of an aortic injury. After spinal injury has been ruled out, an upright chest radiograph may be obtained as well. If a widened mediastinum is detected on radiograph, additional evaluation is necessary for definitive management. Although an aortic tear is sometimes seen on CT scan, aortography is the study used for definitive diagnosis.[20,24]

BOX 55-3 PATIENT SAFETY

Signs and Symptoms of Aortic Injuries

- Pulse deficit in any area, particularly lower extremities or left arm
- Hypotension unexplained by other injuries
- Upper extremity hypertension relative to lower extremities
- Interscapular pain or sternal pain
- Precordial or interscapular systolic murmur caused by turbulence across the disrupted area
- Hoarseness caused by hematoma pressure around the aortic arch
- Respiratory distress or dyspnea
- Lower extremity neuromuscular or sensory deficit

Adapted from Frawley PM: Thoracic trauma. In McQuillan KA, Flynn Makic MB, Whalen E, et al (eds): Trauma Nursing, 4th ed. Philadelphia, PA: WB Saunders, 2009, pp 614–677.

A positive aortogram indicates the need for surgical repair. The torn aorta may require end-to-end anastomosis or, more commonly, the placement of a synthetic graft. Cardiopulmonary bypass may be necessary for repair of the ascending aorta or the aortic arch. However, repair of the descending thoracic aorta is usually accomplished during aortic cross-clamping. Because this maneuver occludes distal blood flow, it is imperative that the cross-clamp time be as brief as possible (preferably <30 minutes). To prevent leakage from the repair site, vasodilators may be administered after surgery to reduce afterload. After replacement of intravascular volume, a vasopressor may be added to support adequate blood pressure. Nursing care focuses on hemodynamic monitoring with a pulmonary artery catheter and titrating medications to maintain optimal blood pressure. Autotransfusion may also be necessary.

Complications are related to the level of the tear and the extent of altered perfusion. Hypoperfusion and resulting damage to organs below the level of the laceration can result from the injury itself or from prolonged cross-clamping during repair. Serious complications resulting from prolonged cross-clamp time include renal failure, bowel ischemia, lower extremity weakness, or permanent paralysis of the lower extremities. Other sequelae, such as ARDS or DIC, can be a consequence of hemorrhagic shock and multiple blood transfusions.

Abdominal Trauma

Abdominal trauma can be caused by both blunt and penetrating injuries. Abdominal injuries can rapidly lead to death secondary to hemorrhage, shock, and sepsis. Missed abdominal injuries are a frequent cause of trauma deaths. Compared with penetrating trauma, blunt abdominal trauma is associated with more fatalities because many of the injuries are "hidden," and often, more obvious but less severe injuries lead to a delay in diagnosis. Deaths that occur more than 48 hours after abdominal injury are due to sepsis and

its complications. In intra-abdominal trauma, rarely does single-organ or single-system injury occur.

The abdomen contains both solid and hollow organs. The solid organs include the liver, spleen, pancreas, and kidneys. The hollow organs include the intestines, stomach, gallbladder, and urinary bladder. Clinicians divide the abdomen into three main regions to facilitate description of the location of the injury. The three areas are the following:

- The peritoneal area, which includes the diaphragm, liver, spleen, stomach, transverse colon, and the portion covered by the bony thorax
- The retroperitoneal area, which includes the aorta, vena cava, pancreas, kidney, ureters, and parts of the duodenum and colon
- The pelvis, which includes the rectum, bladder, uterus, and the iliac vessels

MVCs are the most common cause of blunt abdominal trauma, although assaults, falls, pedestrian–motor vehicle collisions, and industrial accidents also contribute to blunt abdominal injuries. These injuries occur as the result of compressive, crushing, shearing, and deceleration forces. Diagnosis of blunt abdominal trauma can be difficult, especially if there are multisystem injuries. If the patient has abdominal tenderness or guarding, hemodynamic instability, lumbar spine injury, pelvic fracture, retroperitoneal or intraperitoneal air, or unilateral loss of the psoas shadow on radiograph, visceral damage should be suspected.

Blunt trauma is likely to cause serious damage to solid organs, and penetrating trauma most often damages the hollow organs. The compression and deceleration of blunt trauma leads to fractures of solid organ capsules and parenchyma, whereas hollow organs can collapse and absorb the force. However, the bowel, which occupies most of the abdominal cavity, is prone to injury by penetrating trauma. In general, solid organs respond to trauma with bleeding. Hollow organs rupture and release their contents into the peritoneal cavity, causing inflammation and infection.

Stab wounds, impalements, and gunshot wounds can cause penetrating trauma. Injury patterns differ depending on the mechanism. If the mechanism of penetrating trauma is a stab wound, knowledge of the size, shape, and length of the instrument used is helpful in determining the extent of intra-abdominal damage. However, it is estimated that only half of stab wounds enter the abdomen, which means that compared with gunshot wounds, stab wounds are less destructive and have a decreased morbidity and mortality.[25] Impalement is considered a "dirty" wound. "Dirty" wounds can result in high mortality secondary to the infection that is caused by bacterial contamination and subsequent multisystem organ failure. Gunshot wounds (missile injuries) are difficult to evaluate. The amount of major vessel disruption and multiple organ involvement are predictors of mortality. The velocity and amount of energy dispersed by the bullet often determine the extent of injury. A bullet can rebound off organs or bones, changing its trajectory and causing massive internal damage to organs and vessels. The blast effect from bullets can also cause significant intra-abdominal injury.

Abdominal trauma requires continual assessment. Frequently, unrecognized abdominal trauma is a cause of preventable death. The nurse must be organized and methodical in the approach to patient assessment. The nurse needs to understand the mechanism of injury as well as the patient's complaints to perform an adequate assessment and identify potentially life-threatening abdominal injuries. It is important to remember that in blunt trauma, the validity of the physical examination alone is questionable. It is often unreliable if alcohol, illicit drugs, analgesics, or narcotics were involved or if the patient has a reduced level of consciousness. In penetrating trauma, the physical examination tends to be more reliable.

Usually, a primary survey is completed, and the patient is resuscitated before the abdomen is assessed. During the secondary survey, the abdomen is assessed and reassessed, and laboratory and diagnostic tests are performed. An orogastric or nasogastric tube and a Foley catheter are placed during the secondary survey phase.

Often, diagnosis of penetrating trauma requires local wound exploration. However, it is important to note the site of the injury because wound exploration depends on mechanism and location. If the injury is in the anterior abdominal region (anterior costal margins to the inguinal creases between the anterior axillary lines), the likelihood that the peritoneum has been penetrated is low. If the injury is in the thoracoabdominal region (fourth intercostal space anteriorly and seventh intercostal space posteriorly to inferior costal margins), exploration is not recommended because there is an increased risk for tension pneumothorax. The patient requires a laparoscopy, thoracoscopy, or exploratory laparotomy. Exploration tends to be difficult in flank or back wounds. The patient usually requires a triple-contrast CT.

Diagnostic testing may include focused abdominal sonography for trauma (FAST), diagnostic peritoneal lavage (DPL), a chest radiograph (to determine gross abnormalities as well as any organ displacement), and an abdominal CT scan. Many trauma centers are performing FAST on all trauma patients. This is an ideal diagnostic study because it is portable, fast, and reproducible.[26] It is performed by placing an ultrasound probe over various areas on the abdomen to determine whether free fluid is located in those areas. The areas evaluated are Morison's pouch in the right upper quadrant, the pericardial sac, the splenorenal region in the left upper quadrant, and the pelvis (suprapubic region).[27] If the results of FAST are positive and the patient is hemodynamically unstable, an exploratory laparotomy is performed. The FAST allows the practitioner to forego other diagnostic tests and proceed to immediate operation.[27]

A DPL is a quick diagnostic procedure that is used during the resuscitation phase of care in hemodynamically unstable trauma patients to diagnose intra-abdominal bleeding (Box 55-4). DPL is not performed as regularly now because the FAST has proved to be very effective and efficient in diagnosing the immediate need for surgical intervention. Other indications for use may include the following:

1. Unexplained hypotension, decreased hematocrit, or shock
2. Equivocal results of abdominal examination
3. Altered mental status caused by brain injury or alcohol or drug intoxication
4. Spinal cord injury
5. Distracting injuries, such as major orthopedic fractures or chest trauma.

BOX 55-4 Diagnostic Peritoneal Lavage

Indications
- Blunt abdominal injury with:
  - Altered mental status
  - Unexplained hypotension, decreased hematocrit, shock
  - Equivocal results of abdominal examination
  - Spinal cord injury
  - Distracting injuries (eg, orthopedic fractures, chest trauma)
- Penetrating abdominal trauma (if exploration is not indicated)

Possible Contraindications
- History of multiple abdominal operations
- Third-trimester pregnancy
- Advanced cirrhosis of the liver
- Morbid obesity
- Known history of coagulopathy

Technique
1. Insert lavage catheter into peritoneal cavity through 1- to 2-cm incision.
2. Attempt to aspirate peritoneal fluid.
3. Infuse normal saline or Ringer's lactate by gravity.
4. Turn patient from side to side (unless contraindicated).
5. Allow fluid to run back into bag by gravity.
6. Send specimens to laboratory.

Positive Results
- 10 to 20 mL gross blood on initial aspirate
- Greater than 100,000 red blood cells/mm^3
- Greater than 500 white blood cells/mm^3
- Elevated amylase level
- Presence of bile, bacteria, or fecal matter

If the results of DPL are positive and the patient is hemodynamically unstable, an exploratory laparotomy is performed.

There are several contraindications to performing a DPL. These include morbid obesity, third-trimester pregnancy, advanced cirrhosis, a history of coagulopathy, and a history of multiple abdominal surgeries.[26] There is an increased risk for omental laceration and visceral or vascular perforation if DPL is performed in patients with these findings.

When performing DPL, it is important to first ensure that the patient has a Foley catheter and an orogastric or nasogastric tube in place to decompress the stomach and the bladder. Decompression of the stomach and bladder guards against accidental perforation when the lavage catheter is placed. Once the Foley catheter and an orogastric or nasogastric tube are placed, the lavage catheter is inserted into the peritoneal space. If fewer than 10 mL of frank blood are returned, a 1-liter bag of warm crystalloid (lactated Ringer's solution or 0.9% normal saline solution) is infused into the peritoneum. After the infusion is complete, the IV bag is placed in a dependent position to allow the fluid to exit the abdomen by gravity. A sample of the fluid is then sent to the laboratory for evaluation.

CT scans are now being used more often in trauma patients. In blunt trauma, the CT scan has become the mainstay of diagnosis for abdominal injury, with a 92% to 97% sensitivity and 98% specificity. Often, the CT scan is performed with both IV and oral contrast agents to visualize the organs and note any disruption. The CT scan allows visualization of the peritoneal, retroperitoneal, and pelvic areas and permits estimation of the amount of fluid in these areas. CT scans are also used to grade solid organ injuries. Limitations to the use of CT include penetrating trauma, the amount of time required to perform the study, the need to transport the patient out of the resuscitation area, and the requirement that the patient must be hemodynamically stable and have limited movement during the study.

Trauma to the Esophagus and Diaphragm

Esophageal injury is rare and very difficult to diagnose because of the lack of clinical signs initially and the severity of other injuries. Esophageal injuries often go unrecognized until sepsis develops. Esophageal injuries have a high death rate.[5] Penetrating trauma is the most likely cause of esophageal injury, and usually the cervical esophagus is where the injury occurs. The clinical symptoms are subtle. Presenting symptoms that should lead to a suspicion of esophageal injury include a hemothorax or pneumothorax without rib fractures.

Diagnosis includes CT scan of the chest, abdomen, and pelvis with and without contrast. Esophagoscopy, flexible endoscopy, and swallow studies are also performed. Treatment for esophageal injury is surgical repair. The patient is kept without anything by mouth (NPO) with a nasogastric tube to continuous suction, and antibiotic therapy is initiated. Nursing considerations include paying close attention to the airway of the patient, ventilation, oxygenation, and hemodynamic support.

Diaphragm rupture is more common in blunt injury than in penetrating injury. Such rupture occurs more frequently on the left side because there the diaphragm is not protected by the liver. Injury is often secondary to the rising and falling associated with respiration. If a diaphragm rupture is suspected, it is necessary to look for both thoracic and abdominal injury. It is not uncommon to see abdominal contents in the thorax, which subsequently causes bowel strangulation in approximately 30% of patients. Respiratory compromise may also be seen because of impairment of lung capacity and displacement of normal lung tissue.

The clinical picture of diaphragm rupture depends on the size and site of injury. This injury is often difficult to diagnose because there is minimal bleeding and the patient is often asymptomatic. Clinical findings may include marked respiratory distress, dyspnea, decreased breath sounds on the affected side, positive bowel sounds in the thorax, palpation of abdominal contents when inserting a chest tube, and paradoxical movement of the abdomen when breathing.

Chest radiography is the initial modality used to diagnose diaphragm rupture. However, findings are often normal or nonspecific. The presence of abdominal contents in the chest denotes an injury. If an injury is suspected, ultrasound and a CT scan should be performed. DPL may be falsely negative. The only definitive treatment for diaphragm rupture is surgical repair.

Trauma to the Stomach and Small Bowel

Significant gastric injury is rare. Small bowel injuries are much more common. Although frequently damaged by penetrating trauma, the small bowel can also burst when subjected to blunt trauma. The multiple convolutions occasionally form a closed loop, which can rupture when subjected to increased pressure caused by impact with a steering wheel or

BOX 55-5 PATIENT SAFETY

Complications Related to Stomach and Small Bowel Trauma

- Intolerance to tube feedings
- Peritonitis
- Postoperative bleeding
- Hypovolemia caused by third spacing
- Development of a fistula or obstruction

seat belt. The bowel's mobility around fixed points (such as the ligament of Treitz) predisposes it to shearing injuries with deceleration.

Blunt small bowel or gastric injury can present with blood in the nasogastric aspirate or hematemesis. Physical signs often are absent, and CT findings may be subtle and nonspecific. Close observation is required; often, the diagnosis is not made until peritonitis develops. Penetrating injuries usually cause positive results on DPL. Although a mild bowel contusion can be managed conservatively (gastric decompression and withholding oral intake), surgery usually is necessary to repair penetrating wounds or bowel rupture.

Postoperative decompression with a gastric tube is maintained until bowel function returns. In most cases, a feeding jejunostomy tube is placed distal to the repair site, and tube feedings can be initiated early in the postoperative course. As the concentration and rate of feedings are advanced slowly, frequent assessment for signs of intolerance (distention, vomiting) is essential.

Because the stomach and small bowel contain an insignificant amount of bacteria, the risk for sepsis is small after rupture of these organs. If the injury goes unrecognized, there is a risk for sepsis. On the other hand, the acidic gastric juice is irritating to the peritoneum and may cause peritonitis. Potential complications related to stomach and small bowel trauma are listed in Box 55-5. Some of these conditions may necessitate additional surgical procedures.

Trauma to the Duodenum and Pancreas

The pancreas and duodenum are discussed together because these retroperitoneal organs are closely related anatomically and physiologically. A great deal of force is necessary to injure these organs because they are well protected deep in the abdomen. Most injuries are related to penetrating trauma. Injuries to adjacent organs almost always are present. The retroperitoneal location makes these injuries difficult to diagnose with DPL. An abdominal CT scan is very useful in this instance. Signs and symptoms may include an acute abdomen, increased serum amylase levels, epigastric pain radiating to the back, nausea, and vomiting.

Small lacerations or contusions may require only the placement of drains, whereas larger wounds need surgical repair. Most pancreatic injuries require postoperative closed-suction drainage to prevent fistula formation. Distal pancreatectomy and Roux-en-Y anastomosis are two procedures commonly performed for injuries to the body and tail of the pancreas. Occasionally, the spleen also must be removed because of its multiple vascular attachments to the pancreas. Damage to the head of the pancreas is associated with duodenal injury and severe hemorrhage because of the close proximity of vascular structures. Surgical procedures used in these cases include pancreaticoduodenectomy, Roux-en-Y anastomosis, and, on rare occasions, total pancreatectomy.

Postoperative nursing assessment and care are similar for the various procedures. Patency of drains must be maintained, and the patient must be monitored for the development of fistulas, the most common complication. Skin protection is important if a cutaneous fistula does develop because of the high enzyme content of pancreatic fluid. Assessment of fluid and electrolyte balance is also important because a pancreatic fistula results in fluid loss, along with loss of potassium and bicarbonate. Pancreatic stimulation can be decreased by administering parenteral hyperalimentation or jejunal feedings instead of an oral diet. The onset of diabetes mellitus is rare unless a total pancreatectomy is performed.

Primary repair or resection with reanastomosis is sufficient to manage most penetrating duodenal injuries. A duodenostomy tube may be placed for decompression and a jejunostomy tube for feeding. Blunt trauma to the duodenum can cause an intramural hematoma, which may lead to duodenal obstruction. The diagnosis is made with a diatrizoate (Gastrografin) upper gastrointestinal study. A complete obstruction usually requires surgical drainage of the hematoma.

Trauma to the Colon

Usually, injury to the colon results from penetrating trauma. The nature of the injury most often dictates surgical exploration (exploratory laparotomy). Primary repair may be considered if the patient is hemodynamically stable and the injury is small and without fecal contamination. In some situations, such as injury to the left colon or when there is massive blood loss, an exteriorized repair or colostomy is required. A cecostomy tube may be placed for colon decompression. Subcutaneous tissue and skin of the incision site are often left open to decrease the chance of wound infection. The colon has a high bacteria count; spillage of the contents predisposes the patient to intra-abdominal sepsis and abscess formation.

Postoperative nursing care focuses on prevention of infection. Dressing changes are necessary for open incisions, and prophylactic antibiotics may be used. In the case of an exteriorized colon repair, resection and end-to-end anastomosis is performed, and the repair site is exteriorized to facilitate identification of a leak. The exteriorized colon must be kept moist and covered with a nonadherent dressing or bag to protect the integrity of the sutures. Because sepsis is a major complication of colon injuries, a series of radiographic and surgical procedures may be required to locate and drain abscesses.

A teaching guide for patients who have undergone a laparotomy can be found in Box 55-6.

Trauma to the Liver

Along with the spleen, the liver is the most commonly injured abdominal organ.[28] Both blunt and penetrating trauma can cause hepatic injuries. Fractures of the right lower ribs increase suspicion for a liver injury. Presenting signs and symptoms may include right upper quadrant pain, rebound tenderness, hypoactive or absent bowel sounds, or signs of hypovolemic shock. Box 55-7 presents the liver injury scale.

Hemodynamically stable patients with liver injuries are now managed nonoperatively. Observation is now considered

BOX 55-6 TEACHING GUIDE After a Laparotomy

Patient Activity

- No tub baths or showers while the staples/stitches are in place.
- If you are tired, rest.
- Only lift what you can easily lift with one hand.
- You may eat your normal diet.
- Take your temperature once a day at the same time and write it down.
- Maintain a normal schedule with your bowel movements.
- If you become constipated, drink more fruit juices.
- Do not drive until you have your doctor's permission.

Wound Care

- It is important to keep the staple/stitch line clean.
- Monitor your wound closely.
- Cleanse the area once a day. To do this, you will need 4" × 4" gauze pads and a solution of half peroxide/half saline solution.
- Wash your hands.
- Open the gauze pad and leave the pad on the paper.
- Pour a small amount of the peroxide/saline solution on the center of the pad while it is lying on the paper.
- Pick up the pad, pulling all four corners together without touching the center.
- Wipe over the stitches/staples from top to bottom, covering them well with the solution. It is normal to see bubbles when cleaning with this solution. Wipe the area only once with a single gauze pad.
- Repeat.
- Allow the area to air dry.
- Tape a gauze pad over the stitch or staple line to prevent rubbing or irritation caused by a belt or waistband.

Signs of Infection

- Swelling around the site
- Increased redness
- Increased tenderness
- Warmth around the site
- Wound edges separating
- Increased drainage
- Foul-smelling drainage
- Change in color of drainage
- Temperature of 101°F or higher
- Vomiting, diarrhea, or constipation

the standard treatment for liver injuries unless the patient is hemodynamically unstable or has frank peritonitis.[29] The liver is manifested to heal itself. It is thought that venous injuries occur in low-pressure systems and have little need for an operation. Also noted have been cases of worsening bleeding after manipulation in the operating room.[29] In this case, serial CT scans are performed to verify bleeding cessation. However, in some cases, the patient's unstable clinical condition dictates the need for surgery. Hepatic trauma can cause a large blood loss into the peritoneum, but bleeding may stop spontaneously. In some instances, bleeding vessels may be ligated or embolized. Small lacerations are repaired, whereas larger injuries may require segmental resection or débridement. In the case of uncontrollable hemorrhage, the liver is packed. After packing, the abdomen may be closed or simply covered and left open. An additional surgical procedure is required within the next few days to remove the packing and repair the laceration. Large liver injuries also need postoperative drainage of bile and blood with closed-suction drains.

After surgery, coagulopathies may be present. Incomplete hemostasis also is a possibility and must be differentiated from coagulopathy-induced bleeding. Severe bleeding resulting from incomplete hemostasis requires clot removal, packing, and additional repair. With a coagulopathy, bleeding arises from numerous sites, whereas with incomplete hemostasis, the bleeding is mainly from the surgical site.

Nursing care of patients with liver injuries includes the replacement of blood products while monitoring the hematocrit and coagulation studies. Assessment of the character and amount of tube drainage, along with fluid balance, also is essential. Potential complications of liver injury include hepatic or perihepatic abscess, biliary obstruction or leak, sepsis, ARDS, and DIC. In 6 to 8 weeks, physical examination findings should be improved. To prevent rebleeding, patients should not participate in contact sports until a repeated CT scan shows healing of the injury.

BOX 55-7 Liver Injury Scale

Hematomas

- **Grade I hematomas:** subcapsular and nonexpanding; involving less than 10% of surface area
- **Grade II hematomas:** subcapsular, less than 1-cm intraparenchymal hematoma; involving 10% to 50% of surface area
- **Grade III hematomas:** expanded and subcapsular, involving more than 50% of surface area, or ruptured and actively bleeding; intraparenchymal hematoma 2 cm or more or expanding
- **Grade IV hematomas:** ruptured parenchyma with active bleeding

Lacerations

- **Grade I lacerations:** capsular tear less than 1 cm deep and nonbleeding
- **Grade II lacerations:** actively bleeding capsular tear 1 to 3 cm deep without trabecular vessel involvement
- **Grade III lacerations:** more than 3 cm deep
- **Grade IV lacerations:** involving 25% to 50% hepatic lobe parenchymal disruption
- **Grade V lacerations:** involving more than 50% hepatic lobe parenchymal disruption; vascular injury includes retrohepatic vena cava and juxtahepatic venous injuries
- **Grade VI lacerations:** vascular hepatic avulsion

From Eckert KL: Penetrating and blunt abdominal trauma. Crit Care Nurse Q 28(1):41–59, 2005.

Trauma to the Spleen

Along with the liver, the spleen is the most commonly injured abdominal organ, usually as a result of blunt trauma.[28] In 60% of patients sustaining blunt trauma, the spleen is the only organ injured.[28] Because of its vascularity, the spleen has a tendency to lose blood rapidly.[30] Presence of left lower rib

fractures increases suspicion for a splenic injury. Presenting signs and symptoms include left upper quadrant pain radiating to the left shoulder (Kehr's sign), hypovolemic shock, and the nonspecific finding of an increased white blood cell count. FAST, DPL or abdominal CT is usually necessary for diagnosis. Box 55-8 provides the splenic injury scale.

Management for splenic injuries includes observation, embolization, or surgery, depending on the hemodynamic stability of the patient, their preexisting conditions, and the grade of splenic injury. Hemodynamically unstable patients with a positive FAST or DPL require immediate surgery to determine the source of bleeding. Hemodynamically stable patients with low-grade injury (grades I to III) without any evidence of bleeding on CT scan or FAST can be observed closely.[28] Patients with contrast extravasation on CT or with abdominal blush on CT may have a higher rate of failure and may require embolization. Also, patients with neurological compromise, who are unreliable to observe, may require surgery.[28]

Approximately 50% to 70% of hemodynamically stable patients are treated nonoperatively with observation and embolization. These patients tend to be adults with lower-grade injuries and most children. An observation period of 5 days is the standard. These patients are admitted to a monitored bed and watched carefully for hypotension and signs of bleeding. They have hemoglobin and hematocrit (H&H) analyses every 6 hours until stable. They are initially placed on bed rest, although there is no clinical significance to support this. Patients are kept NPO until their H&H is stable and then they begin a diet. Patients with a higher-grade injury require longer observation.[28]

Early complications include recurrent bleeding, subphrenic abscess, and pancreatitis resulting from surgical trauma. Rupture of an expanding subscapular hematoma or pseudoaneurysm may present days or weeks after an initial normal examination. Late complications consist of thrombocytosis and overwhelming postsplenectomy sepsis (OPSS). Because the spleen plays an important role in the body's response to infection, a splenectomy predisposes the patient to an increased risk for infection. This risk is especially high among children and highest in those younger than 2 years old. Pneumococcus, an encapsulated microorganism resistant to phagocytosis, is the organism that most often infects patients after splenectomy. OPSS frequently begins with the onset of pneumococcal pneumonia, which progresses to a fulminant sepsis. Postsplenectomy patients increase their immunity toward pneumococcal infections by receiving a polyvalent pneumococcal vaccine (Pneumovax). Complications of OPSS include adrenal insufficiency and DIC. OPSS has a high incidence and mortality rate, especially within 1 year of surgery. Patient and family teaching should focus on information about signs and symptoms of infection.

BOX 55-8 Splenic Injury Scale

Hematomas

- **Grade I hematomas:** involving less than 10% of surface area; subcapsular and nonexpanding
- **Grade II hematomas:** subcapsular; involving 10% to 50% of surface area or less than 1-cm intraparenchymal hematoma
- **Grade III hematomas:** expanding, subcapsular, and ruptured, with active bleeding, involving more than 50% of surface area; subcapsular hematoma 2 cm or more or expanding intraparenchymal hematoma
- **Grade IV hematomas:** involving ruptured parenchyma with active bleeding

Lacerations

- **Grade I lacerations:** nonbleeding capsular tear less than 1 cm deep
- **Grade II lacerations:** actively bleeding capsular tear 1 to 3 cm deep without trabecular vessel involvement
- **Grade III lacerations:** more than 3 cm deep or involving trabecular vessels
- **Grade IV lacerations:** including hilar vessel with more than 25% devascularization
- **Grade V lacerations:** involving completely shattered spleen; hilar vascular injury with total devascularization of the spleen

From Eckert KL: Penetrating and blunt abdominal trauma. Crit Care Nurse Q 28(1):41–59, 2005.

Trauma to the Kidneys

Injury to the kidney may lead to a "free" hemorrhage, contained hematoma, or the development of an intravascular thrombus. Sudden deceleration injury can cause the kidney to move, avulsing smaller renal vessels or tearing the renal artery intima, which also may lead to vessel thrombosis. Blunt and penetrating trauma can also cause a laceration or contusion of the renal parenchyma or rupture of the collecting system. Lower rib or lumbar vertebral fractures, along with liver and spleen injuries, should raise suspicion of an associated renal injury. Signs and symptoms, when present, consist of hematuria, pain, a flank hematoma, or ecchymosis over the flank. Because the bleeding is retroperitoneal, it can be difficult to detect. A helical CT scan, ultrasound, or an IV pyelogram (less commonly used) usually provides the diagnosis.

Renal injuries are graded based on their severity. Increased grade correlates with decreased function.[31] Many renal injuries can be managed conservatively with observation and bed rest until gross hematuria resolves. However, in some instances (mainly for vascular injury), surgical repair or nephrectomy is necessary.

Postoperative assessment and support of renal function are imperative. Optimal fluid balance must be maintained. Low-dose dopamine may be ordered to promote renal perfusion. Major complications consist of arterial or venous thrombosis and acute renal failure. Other complications include bleeding, perinephric abscess, the development of a urinary fistula, and late onset of hypertension.

Trauma to the Bladder

The bladder can be lacerated, ruptured, or contused, most often as the consequence of blunt trauma (usually because of a full bladder at the time of injury). Bladder injuries frequently are associated with pelvic fractures. Gross hematuria is typically noted with bladder rupture.[32] Presence of blood at the urethral meatus, a scrotal hematoma, or a displaced prostate gland requires examination for urethral injuries with a CT scan or conventional cystography before the insertion of a urinary catheter.[32]

A bladder injury can cause intraperitoneal or extraperitoneal urine extravasation. Extraperitoneal extravasation, usually associated with pelvic fractures, can often be managed with urinary catheter drainage. However, intraperitoneal extravasation (associated with a high-force injury) requires surgery. This injury has a high mortality rate because of associated injuries that occur secondary to the force involved. A suprapubic cystostomy tube may be placed. Complications are infrequent, but infection resulting from the urinary catheter or sepsis from extravasation of infected urine can occur. Patients may complain of an inability to void or of shoulder pain (caused by extravasation of urine into the peritoneal space).

Musculoskeletal Injuries

Although musculoskeletal injuries take a long time to heal and can often result in lifelong disability, they are usually not considered life threatening unless there is a traumatic amputation or pelvic fracture. Routinely, the musculoskeletal assessment is done in the secondary survey after hemodynamic stabilization. These injuries do require prompt recognition and stabilization to promote optimal recovery and function.

Although there are a variety of causes of trauma-related musculoskeletal injuries, the major ones include MVCs, falls, assaults, and industrial, farming, and home accidents. Musculoskeletal injuries are often associated with other injuries to the body.

It is important to understand the circumstances surrounding, and the mechanism involved in, musculoskeletal trauma. Force might be applied to one area, but the transferred energy and distribution of force may cause injury somewhere else. For example, in a person who falls off a two-story building and lands on his or her feet, one would expect to find calcaneus or ankle fractures, but the transference of energy may also cause a pelvic or lumbar spine fracture. Obviously, if the patient is conscious, he or she can verbalize his or her pain. However, many times, fractures and sprains go unrecognized because the patient is not able to verbalize and communicate the location of his or her pain.

As in all trauma patients, initial assessment begins with the primary survey. Once this is complete and the patient is hemodynamically stable, the secondary assessment is conducted. When trauma patients are admitted to the resuscitation unit, cervical spine, chest, and pelvis films are obtained first. Sometimes, thoracic and lumbar spine films are obtained, depending on the mechanism of injury. The initial pelvic film tells the nurse whether the patient has a life-threatening pelvic fracture. If this is the case, immobilization of the pelvis should be maintained to prevent exsanguination. Immobilization of the pelvis is achieved with a C-clamp, external fixator, pelvic binder, or sheets wrapped tightly around the patient to attempt to stop the bleeding.

During the secondary survey, if limb swelling, ecchymosis, or deformity is noted, that extremity should be immobilized. Proper imaging is ordered to determine the extent of the injury. The nurse tests the extremities for capillary refill (<2 seconds is normal), pulses, crepitus, muscle spasm, movement, sensation, and pain.

The most common studies used to diagnose musculoskeletal injuries are plain radiographs, CT, and magnetic resonance imaging (MRI). When obtaining radiographs, it is important to get two views of the affected area. It is also important to assess the joint above and below the injured area. If the affected area is in a place that is difficult to visualize on plain films, a CT scan usually gives a better picture. An MRI also gives more specific detail about the area surrounding the injury and about the injury itself.

There are many types of musculoskeletal injuries, including fractures, fracture dislocations, amputations, and trauma to the soft tissue (ie, skin, muscle, tendons, ligaments, and cartilage). Fracture classification is based on type, cause, and anatomical location. Several fracture types are shown in Figure 55-6. If the skin is broken at the fracture site, the injury is considered to be an "open" fracture. If the skin is intact, the injury is said to be a "closed" fracture. An open fracture is further classified as grade I, II, or III, depending on the tissue damage involved.

Dislocation occurs when the articulating surfaces of a joint are no longer in contact because of joint disruption. Joint mobility may be restricted. There may also be associated vascular or nerve injury with dislocations. Ligamentous injury usually accompanies dislocations because ligaments stretch or tear at the time of dislocation.

Amputations are classified according to the amount of tissue, nerve, and vascular damage. A cut or guillotine amputation has clean lines and well-defined edges, whereas a crush amputation has more soft tissue damage and the edges are not as well defined. An avulsion amputation occurs when a force stretches and tears away tissues, causing nerves and vessels to be torn in different areas than the bone.

As with any injury, musculoskeletal trauma requires continuous assessment. It is not uncommon for vascular or neurological compromise, or both, to develop in patients with musculoskeletal injury. Any musculoskeletal injury involving bone or soft tissue can cause neurological or vascular compromise because nerves and blood vessels are located in such close proximity to the bones and muscles. The nerves and muscles are very sensitive to impaired circulation and compression.

It is also important to continually assess the patient for hypovolemia. As stated earlier, traumatic amputation and major pelvic ring fractures are known for their extensive blood loss. Other orthopedic injuries can also cause substantial blood loss. Very rarely do patients sustain severe musculoskeletal injuries without other systemic injuries; therefore, other sources of blood loss should also be investigated.

Pelvic fractures may occur in patients with isolated simple fractures as well as in critically injured patients with multisystem injuries. The primary causes of pelvic fractures are MVCs, MCCs, and motor vehicle–pedestrian collisions.[33,34] Although pelvic fractures are thought to contribute to traumatic death, they usually are not the main cause.[33] Mortality rates have ranged from 18% to 40%, and death within the first 24 hours was most often a result of acute blood loss.[35] The risk for death is increased in patients who have open pelvic fractures or who have been hit by a motor vehicle. Pelvic ring fractures are associated with high-energy mechanisms, and patients often have soft tissue

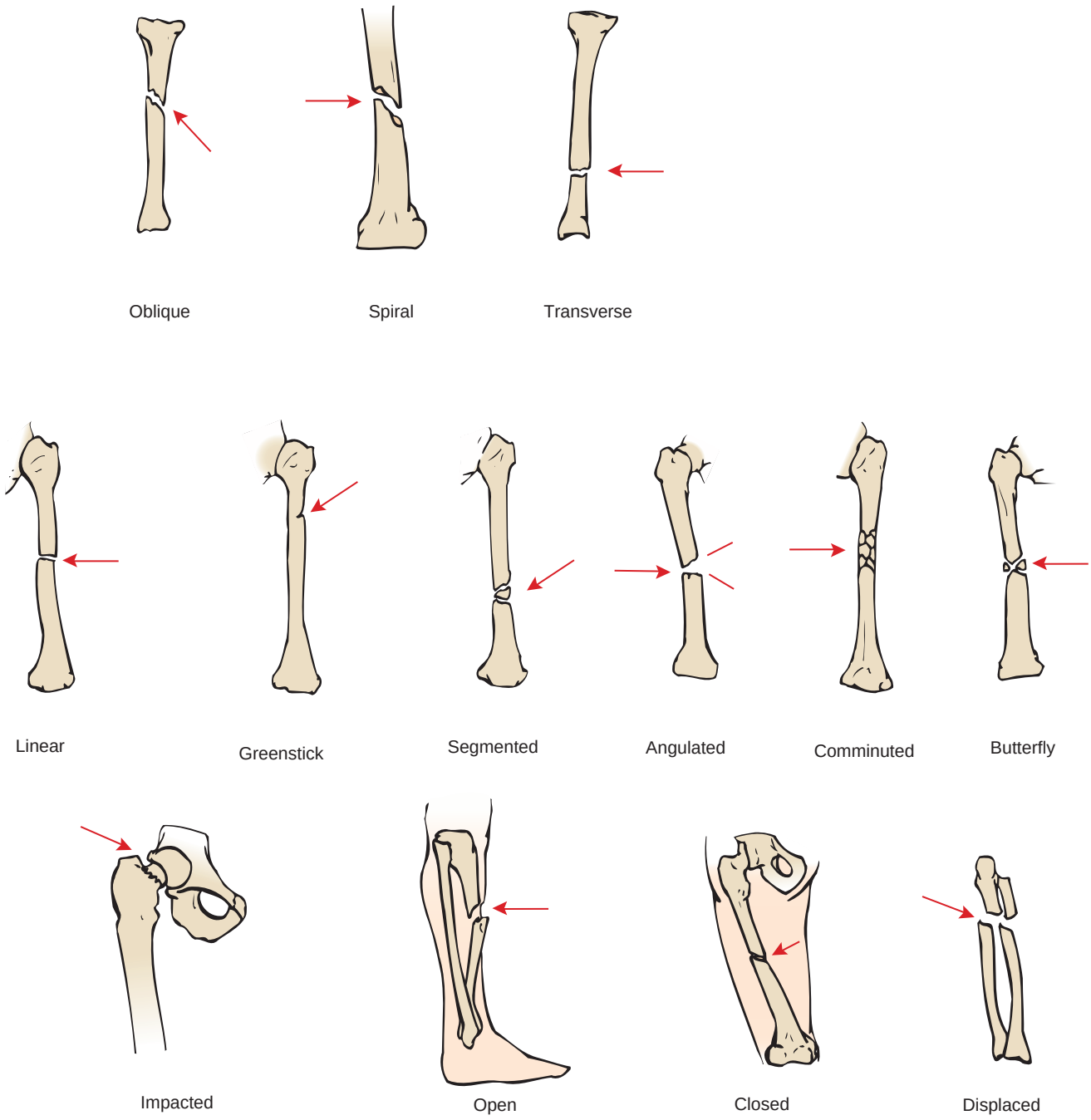


FIGURE 55-6 ▲ There are many different types of fractures.

injury.[36] In hemodynamically unstable patients, it is imperative to find the site of bleeding and control it. Bleeding can occur from three major sources: arterial, venous, and/or cancellous bone.[35]

Physical examination for pelvic fractures begins with inspection for abrasions, lacerations, contusions, and symmetry of the lower extremities. Palpation to assess for rotational and vertical instability is then necessary. Rectal and vaginal examinations should be performed to assess for a urethral tear in males and an open fracture in females.[34]

Radiographic evaluation of pelvic injuries includes an anteroposterior view. This film can detect up to 90% of pelvic fractures. Other radiographs include pelvic inlet and outlet views and lateral sacral views. CT scan is also used to evaluate the sacroiliac joints as well as the extent of the injury.[34] See Box 55-9 for classification of pelvic fractures.

Treatment goals of pelvic fractures are to control bleeding and to prevent loss of function and infection (sepsis) caused by open fractures.[34,36] Application of a pelvic binder or external fixator is used for temporary stabilization and to control bleeding. Embolization is also indicated for hemorrhage control.[34,36] Permanent orthopedic repair of pelvic fractures is usually performed within 24 to 72 hours after injury when the patient is adequately resuscitated and hemodynamically

BOX 55-9 Classification Schemes for Pelvic Fractures

Tile's Classification

Type A, Stable
A1, without involvement of pelvic ring
A2, with involvement of pelvic ring

Type B, Rotationally Unstable
B1, open book
B2, ipsilateral lateral compression
B3, contralateral lateral compression

Type C, Rotationally and Vertically Unstable
C1, rotationally and vertically unstable
C2, bilateral
C3, with associated acetabular fracture

Young and Burgess Classification

Lateral Compression (LC)
I, Sacral compression on side of impact
II, Iliac wing fracture on side of impact
III, LCI or LCII injury on side of impact with contralateral open-book injury

Anterior Posterior Compression
I, slight widening of pubis symphysis or anterior part of sacroiliac joint with intact anterior and posterior sacroiliac ligaments
II, widened anterior part of sacroiliac joint with disrupted anterior and intact posterior sacroiliac ligaments
III, complete disruption of the sacroiliac joint

Vertical Shear
Vertical displacement anteriorly and posteriorly
Combined mechanism
Combination of other injury patterns

From Frakes MA, Evans T: Major pelvic fractures. Crit Care Nurse 24(2):18–32, 2004.

stable. This can be accomplished with either internal or external fixation.

Infection is common in open injuries. Ideally, patients with musculoskeletal trauma are brought to the operating room within 6 hours of injury for a washout of the affected area. Sometimes, antibiotic prophylaxis is started; however, this practice is controversial. A tetanus booster is given if indicated to all patients with open injuries. Other serious complications of musculoskeletal injuries include compartment syndrome, deep venous thrombosis (DVT), pulmonary embolus, and fat embolus syndrome.

Compartment Syndrome

Compartment syndrome occurs when the pressure within the fascia-enclosed muscle compartment is increased, causing blood flow to the muscles and nerves in the compartment to become compromised. The final result is cellular anoxia.[37] It is suspected based on mechanism of injury. This ischemia then leads to tissue damage, which compromises nerve and muscle function. A prolonged elevation of compartmental pressure leads to death of the muscles and nerves involved.[37] Normal compartment pressure is between 0 and 8 mm Hg. Capillary blood flow is believed to become compromised at 20 mm Hg, pain develops at a pressure between 20 and 30 mm Hg, and ischemia occurs at pressures greater than 30 mm Hg.[37] Patients with higher diastolic pressures are able to tolerate higher tissue pressures without ischemic damage. Fasciotomy is recommended when the compartment pressure approaches 20 mm Hg below the diastolic pressure. Hypotensive trauma patients may experience significant muscle ischemia at lower compartment pressures.

Patients with compartment syndrome complain of increased pain in the affected area. Compartment syndrome occurs most often with long bone fractures in the lower leg or forearm.[37] The pain is described as being "out of proportion" to the injury. The most reliable early sign of compartment syndrome is decreased sensation. The compartment involved is firm, and the patient eventually has paresthesia. Pallor and pulselessness are late signs of compartment syndrome. When the compartment syndrome has progressed to the point that the patient is showing late signs, loss of the affected extremity is threatened. The nurse must constantly monitor the affected extremity and compare it with the nonaffected extremity. If any of the signs or symptoms of compartment syndrome are present, the orthopedic or general surgeon should be notified immediately so that compartment pressures can be measured. If it is deemed that the compartment pressures are high, a fasciotomy is performed to release the pressure and save the extremity. The extremity should never be elevated when compartment syndrome is suspected because this will decrease arterial inflow and exacerbate ischemia.[37]

Deep Venous Thrombosis

DVT is a significant risk for all trauma patients, especially those with musculoskeletal injuries. It is known as a common, life-threatening complication of major trauma. The danger of DVT is that it may progress to pulmonary embolus. The administration of low-dose heparin or low–molecular-weight heparin and the use of intermittent pneumatic compression devices are recommended to prevent DVT.[38]

The pathophysiology of DVT, and later pulmonary embolus, is related to Virchow's triad:

1. Venous stasis from decreased blood flow, decreased muscular activity, and external pressure on the deep veins
2. Vascular damage or concomitant pathological state
3. Hypercoagulability

The nurse assesses for signs and symptoms of DVT on a regular basis. These include the presence of Homans' sign (calf pain on dorsiflexion of the foot), swelling of the affected area, tachycardia, fever, and distal skin color and temperature changes.[39] If these signs or symptoms are found, they should be reported immediately. Sometimes, an acute pulmonary embolus is the first indication of DVT.

Pulmonary Embolus

A pulmonary embolus occurs when a blood clot dislodges from the vein, travels through the heart, and lodges in the pulmonary artery, obstructing blood flow. Sudden onset of dyspnea is the classic sign of a pulmonary embolus, but signs and symptoms vary, depending on the size of the clot and the number of vessels occluded. Signs and symptoms may include a decline in oxygenation, substernal chest pain,

hypovolemic relative shock, tachypnea, shortness of breath, anxiety, a feeling of impending doom, a low-grade fever, an altered level of consciousness, and a pale, dusky, or cyanotic skin color. Often, a patient with a pulmonary embolus also has a DVT.[40]

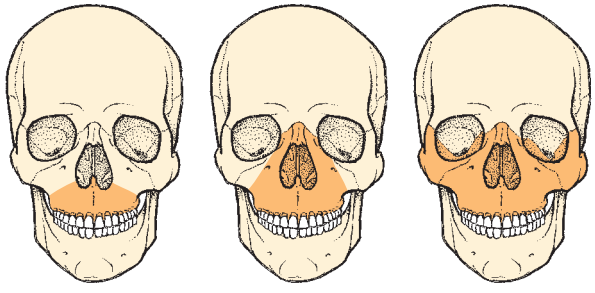
Fat Embolism Syndrome

Fat emboli are fat globules in the lung tissue and peripheral circulation after a long bone fracture or major trauma. Fat emboli may or may not cause systemic symptoms. Fat embolism syndrome is a serious (but rare) manifestation of fat emboli that involves a classic triad of symptoms: hypoxemia, neurological decline, and petechial rash.[41] It usually occurs within 24 to 72 hours of injury. Clinical indications of this syndrome include tachypnea, dyspnea, and hypoxemia.[41] Many patients develop neurological changes after respiratory distress. These changes are usually completely reversible. Petechial rash often occurs on the head, neck, anterior thorax, axilla, and subconjunctiva. It typically lasts 5 to 7 days.[41] Nurses should be aware of the potential for fat embolism syndrome and monitor the patient for hypoxemia with pulse oximetry.

Maxillofacial Trauma

Despite laws that mandate lower speed limits and the use of air bags and seat belts, the incidence of maxillofacial trauma remains high because the face is unprotected during rapid deceleration. The degree of maxillofacial injury is directly related to the force at impact when the face makes contact with a stationary object. As the force increases, the amount of energy that is dispersed increases, causing an increase in injury. Penetrating injury is less common than blunt injury in patients with maxillofacial trauma.

As with any trauma patient, initial management priorities remain ABC. The trauma team cannot be distracted from these priorities by obvious deformities that may be associated with maxillofacial injuries.[42] Maxillofacial trauma can cause airway obstruction and death if airway and breathing are not adequately and urgently established. When the primary survey is completed, an adequate assessment of the maxillofacial injuries is performed. Figure 55-7 shows diagnosis of maxillae fractures according to Le Fort's classification.



A. Le Fort I **B**. Le Fort II **C**. Le Fort III

FIGURE 55-7 ▲ Le Fort fractures. **A:** Le Fort I: Transverse disarticulation of the maxillary dentoalveolar process from the remaining basal bone of maxilla and midface. **B:** Le Fort II: Pyramidal fracture involving entire maxilla and nasal complex. **C:** Le Fort III: Complete craniofacial–midface disassociation. (Courtesy of Neil O. Hardy, Westpoint, CT.)

When assessing for maxillofacial injuries, the nurse assesses soft tissues as well as bony structures. The nurse inspects the face for symmetry and then palpates systematically to observe for any movement of bony structures. Cranial nerves are assessed. Often, maxillofacial injuries coincide with head injuries, reinforcing the importance of a thorough neurological examination (see Chapter 33). Any midface fractures that communicate through the orbit require a thorough ocular assessment and frequent reassessment.

Most maxillofacial injuries involve the soft tissue. In any soft tissue injury, there is potential for contamination. Therefore, each patient's immunization to tetanus is assessed. If needed, a tetanus booster is given. All wounds are assessed for dirt, grease, particles, and other contaminants. Many wounds require an operation for washout to débride the tissue and clean the area.[42] These injuries are usually not life threatening and are treated in the appropriate order. However, even a small abrasion to a person's face can lead to a lifetime of disfigurement; therefore, all injuries must be attended to appropriately.

As stated earlier, in any type of trauma, but especially in maxillofacial trauma when the patient's airway may be compromised, it is imperative to assess and maintain an adequate airway for the patient continuously. Loss of an artificial airway (eg, inadvertent removal of the endotracheal tube) can be life threatening because of soft tissue swelling. It is also essential to assess for and treat hypovolemia secondary to hemorrhage from facial arteries. Epistaxis may also occur with any fracture that communicates with the nose. The nurse continuously assesses the patient's neurological status and reports any abnormalities. The patient's pain and anxiety must be assessed and treated. Many patients with maxillofacial injuries are robbed of their senses. They may be unable to see, smell, taste, or speak secondary to their injury. This is an anxiety-provoking situation; patients require continuous reassurance and medication as necessary. Many maxillofacial injuries require multiple surgeries before the patient is definitively treated.

▲ Complications of Multiple Trauma

Complications associated with multiple trauma are numerous (Box 55-10). Because most trauma patients are in the ICU when these complications develop, the nurse plays an essential role in detecting, preventing, and treating them.

The unexpected nature of trauma tends to amplify fear and anxiety. Therefore, nursing care must also provide psychosocial support for the seriously injured patient and his or her family. A multidisciplinary approach that recognizes concerns and offers frequent explanations is recommended. Special considerations for older trauma patients can be found in Box 55-11.

Death after multiple traumatic injuries, when it occurs, may occur immediately, or it may occur as a result of early or late complications. Immediate deaths occur at the scene and within minutes of the injury. Most common causes of immediate deaths are brainstem or high spinal cord injury, cardiac rupture, transection of the great vessels, and airway obstruction.

BOX 55-10 PATIENT SAFETY

Delayed Complications of Multiple Trauma

Hematologic
- Hemorrhage, coagulopathy, disseminated intravascular coagulation

Cardiac
- Dysrhythmia, heart failure, ventricular aneurysm

Pulmonary
- Atelectasis, pneumonia, emboli (fat or thrombotic), ARDS

Gastrointestinal
- Peritonitis, adynamic ileus, mechanical bowel obstruction, acalculous cholecystitis, anastomotic leak, fistula, bleeding

Hepatic
- Liver abscess, liver failure

Renal
- Hypertension, myoglobinuria, renal failure

Orthopedic
- Compartment syndrome

Skin
- Wound infection, dehiscence, skin breakdown

Systemic
- Sepsis

BOX 55-11 CONSIDERATIONS FOR THE OLDER PATIENTS

Trauma
- Trauma is the seventh most frequent cause of death among the elderly.
- The older person is injured less frequently than the younger person; however, when an older person does sustain injuries, the injuries are more likely to be life threatening.
- The injuries occurring in the elderly population tend to be less severe but are associated with a greater risk for death.
- Falls are the most prominent cause of trauma in the older person.
- Constant monitoring is essential with the older trauma patient.
- Providers should have a decreased threshold for invasive monitoring with an elderly patient, secondary to predisposing conditions and past medical history.
- Management considerations are as follows:
  - Consider cervical osteoarthritis when intubation is necessary.
  - Pain management should be more local if possible (eg, epidural catheter, nerve block).
  - Fluid management should be done cautiously. Older adults require adequate rapid fluid replacement without excess. Consider a pulmonary artery or central venous pressure line for guidance in fluid replacement.
  - Older adults tend to become hypothermic more quickly than younger people. Use warm fluid and warming devices as indicated.

Adapted from Plummer E: Trauma in the elderly. In McQuillan KA, In McQuillan KA, Flynn Makic MB, Whalen E, et al (eds): Trauma Nursing, 4th ed. Philadelphia, PA: WB Saunders, 2009, pp 835–849.

Early Complications

Severe head injuries and hemorrhage are the early complications of multiple trauma most often responsible for causing death within hours of the injury, usually in the emergency department or operating room. Often, death at this stage can be prevented with quick assessment, resuscitation, and management of injuries.

Management of head injuries is discussed in Chapter 36. To prevent exsanguination, hemorrhage must be controlled and volume resuscitation begun with the infusion of crystalloids and blood. Patients may require emergent surgical ligation or packing, or embolization by angiography. Massive hemorrhage complicated by hypothermia, metabolic acidosis, and coagulopathy is highly lethal.

Late Complications

Late complications of multiple trauma include hypovolemic shock, infection and septic shock, ARDS, and multiple organ dysfunction syndrome (MODS).

Hypovolemic Shock

Massive hemorrhage or continued bleeding because of incomplete hemostasis or an undiagnosed injury can lead to hypovolemic shock and eventually decreased organ perfusion. The various organs respond differently to the decrease in perfusion caused by hypovolemia. Multiple blood transfusions are often necessary, further increasing the likelihood of ARDS and MODS.

Infection and Septic Shock

Another frequent and potentially serious complication of multiple trauma is infection. The risk for infection is increased after close-range shotgun blasts, high-velocity penetrating injuries, penetrating wounds to the colon, prolonged surgery, multiple blood transfusions, and injury to multiple organs. Other risk factors include advanced age, underlying immunosuppression, and a history of diabetes mellitus.

Infections can range from a minor wound infection to fulminant sepsis syndrome and septic shock. In septic shock, the release of toxins causes dilation of vessels, leading to venous pooling that results in a decreased venous return. Initially, cardiac output increases to compensate for decreased systemic vascular resistance. Eventually, the compensatory mechanisms fail, and cardiac output falls along with blood pressure and organ perfusion (ie, septic shock).

The source of infection must be found and eradicated to treat sepsis effectively. The nurse must watch for the sometimes subtle indicators of sepsis. Hyperthermia or hypothermia and altered mental status are often present early in the septic process, as well as tachycardia, tachypnea, and an increase in the white blood cell count. These findings should prompt further assessment to detect a possible infectious source.

When sepsis is suspected, cultures are obtained, antibiotics are prescribed, radiological studies are done, and exploratory surgery frequently is performed. Intra-abdominal abscess is a frequent cause of sepsis. Some abscesses can be drained percutaneously, whereas others require surgery. After the surgical drainage of an abdominal abscess, the incision is left open with drains in place to allow healing and prevent recurrence. Other sources of infection are invasive lines, the urinary tract, and the lungs. Pneumonia is a common cause of sepsis in trauma patients. Risk factors for pneumonia include advanced age, aspiration, underlying pulmonary disease, thoracic or abdominal surgery, and prolonged intubation.

Hemodynamics are altered and metabolic demands are increased during sepsis. The typical patient exhibits elevated cardiac output, decreased systemic vascular resistance, and increased oxygen consumption. Hemodynamics must be supported and a balance between oxygen delivery and oxygen consumption maintained. Research suggests that early nutritional support decreases the development of sepsis and MODS. Enteral feeding should be used whenever possible because it is associated with a lower incidence of sepsis than total parenteral nutrition.

Acute Respiratory Distress Syndrome

ARDS refers to a "syndrome of lung injury characterized by dyspnea, severe hypoxemia, decreased lung compliance and diffuse bilateral pulmonary infiltrates."[43] ARDS can be caused by direct (pneumonia, aspiration of gastric contents, inhalation injury, fat emboli, etc.) or indirect (insult anywhere in the body: sepsis, multiple blood transfusions, shock, burns, etc.) injuries.[43]

Sepsis may predispose the patient to ARDS (see Chapter 27). In addition to sepsis, specific injuries (eg, head trauma, pulmonary contusion, multiple major fractures), massive blood transfusions, aspiration, and pneumonia can also increase the likelihood of ARDS. With a mortality rate between 50% and 80%, ARDS is characterized by hypoxemia with shunting, decreased lung compliance, tachypnea, dyspnea, and the appearance of diffuse bilateral pulmonary infiltrates.

Treatment of ARDS is multifaceted. Initially, therapy is aimed at treatment of the primary cause. Fluid and hemodynamic management, management of infection, adequate nutrition, mechanical ventilation, and supportive oxygen delivery are incorporated in the therapeutic regimen. The main goal of ARDS treatment is to increase oxygen in the body.[43]

Systemic Inflammatory Response Syndrome

Systemic inflammatory response syndrome (SIRS) describes a pathophysiological response to a cascade of events precipitated by shock, which usually occurs after trauma. A controlled inflammatory response takes place, designed to heal wounds and ward off infection. Continuous stimulation or severe infection may result in sustained inflammation—SIRS. The result is an imbalance of cellular oxygen supply and demand, causing an oxygen extraction deficit.[8]

Multiple Organ Dysfunction Syndrome

Sixty percent of trauma patients have clinical signs of sepsis without an apparent bacterial source. Many factors have been associated with the development of MODS, including hemorrhage, massive blood transfusion, hypovolemic shock, and sepsis. Characterized by the failure of two or more organs, MODS accounts for many late deaths in trauma patients. Usually, the lungs are the first organs to fail (heralded by the onset of ARDS), followed by the liver, gastrointestinal tract, and kidneys.

Liver failure can result from initial damage, vascular compromise, shock, or sepsis. Jaundice is a common indicator of deteriorating liver function, although other causes, such as posttraumatic biliary obstruction, must be ruled out. Liver function tests are diagnostic. Liver failure can lead to a decreased level of consciousness, abnormal clotting study results, and hypoglycemia (see Chapter 41).

Gastrointestinal failure manifests with hemorrhage from stress ulcers requiring blood transfusion. Prophylactic neutralization of gastric acid can minimize the risk for bleeding (see Chapter 41).

Renal failure can be precipitated by a renal injury, ischemia, radiographic contrast material, rhabdomyolysis, hypovolemia (due to hemorrhage, third spacing), or sepsis. Initial signs include increasing blood urea nitrogen and serum creatinine levels. Renal failure may be polyuric or oliguric. Dialysis may be necessary (see Chapter 30).

Cardiovascular failure, DIC, metabolic changes (eg, hyperglycemia, metabolic acidosis), and central nervous system changes, ranging from confusion to obtundation, also may be evident in MODS (see Chapter 49 for a discussion of DIC).

Psychosocial Considerations

As medical advances extend life expectancy, the elderly population (65 and older) are expected to be greater than 20% of the population by the year 2020.[42] Elderly trauma patients are the fastest growing segment of patients admitted to trauma centers and have a six-time risk of dying when compared with similarly injured younger patients.[42,43] This age group is more active and mobile than ever; however, this leads to an increased risk of injury. This also leads to multiple discussions on end-of-life care, of losing independence, and health care cost.

Another important issue in trauma care is patients' families. It is very difficult to care for a trauma patient because you are caring not only for the patient but also for the family. Trauma is an unexpected event that occurs and disrupts the life of many. Both patients and families go through grief and denial. Many family members are unable to accept that their loved one was injured because they just saw them minutes ago and they were "fine." Many ICUs and resuscitation areas are offering 24-hour visiting and presence during resuscitation to support the patient and the family.

▲ Clinical Applicability Challenges

CASE STUDY

Mr. S. is a 42-year-old male patient who was struck and run over by a trailer. At the time of admission he was awake, very tachypneic, and complaining of difficulty breathing and abdominal pain. His vital signs were as follows: blood pressure was 80/50 mm Hg, heart rate 127 beats/min, and respiratory rate 33 per minute. He is pale and diaphoretic and now complaining of being thirsty.

During Mr. S.'s primary survey, he is intubated and placed on the ventilator. Although his respiratory rate is improved with intubation, he continues to be tachycardic and hypotensive. There is no evidence of outward bleeding.

Once radiographs are obtained, Mr. S. is found to have multiple right-sided rib fractures as well as a pulmonary contusion. He is also noted to have a crushed pelvis on x-ray. His FAST examination is positive for blood in his abdomen. Because he continues to be unstable, he is given multiple units of blood products. Although he is becoming increasingly more difficult to ventilate, he is taken to the operating room emergently for a damage-control laparotomy and then transferred to the ICU with a pelvic binder in place.

1. After reviewing this case study, is there any other information that you would like to know in order to adequately care for Mr. S.?

2. Mr. S. has a pelvic binder in place; what nursing considerations do you need to be aware of when caring for this patient? If Mr. S. continues to show signs of instability despite blood products and an operation, what would you expect the next procedure to be?

3. What expectations do you have for Mr. S.'s lungs after the operation, especially knowing that he was becoming more difficult to ventilate and has rib fractures and a pulmonary contusion?

References

1. The American Heritage Dictionary, 2nd ed. Boston, MA: Houghton Mifflin, 1991
2. National Safety Council: Accident Facts, 2000. Chicago, IL: National Safety Council, 2000
3. National Center for Injury Prevention and Control. Available at: http://webappa.cdc.gov/cgi-bin/brocker.exe
4. Dutton RP: Trauma. Curr Opin Crit Care 15:525–526, 2009
5. Haider AH, Chang DC, Hout ER, et al: Mechanism of injury predicts patient mortality and impairment after blunt trauma. J Surg Res 153:138–142, 2009
6. Shahani R, Galla JD: Penetrating chest trauma. Available at: http://emedicine.medscape.com/article/425698-overview, 2008
7. Onzuka J, Worster A, McCreadie B: Is computerized tomography of trauma patients associated with a transfer delay to a regional trauma centre? CJEM 10(3):205–208, 2008
8. American College of Surgeons Committee on Trauma: Advanced Trauma Life Support: Program for Physicians. Chicago, IL: American College of Surgeons, 1997
9. Simon R, Stone M, Cucuzzo J: The impact of a new trauma center on an existing nearby trauma center. J Trauma 67(3):645–650, 2009
10. MacKenzie E, Weir S, Rivara F, et al: The value of trauma center care. J Trauma 69(1):1–10, 2010
11. Sambasivan CN, Schreiber MA: Emerging therapies in traumatic hemorrhage control. Curr Opin Crit Care 15:560–568, 2009
12. Takasu A, Minagawa Y, Ando S, et al: Improved survival time with combined early blood transfusion and fluid administration in uncontrolled hemorrhagic shock in rats. J Trauma 68(2):312–316, 2010
13. Diez C, Varon A: Airway management and initial resuscitation of the trauma patient. Curr Opin Crit Care 15:542–547, 2009
14. Moore F, Davis J, Moore E, et al: Western trauma association (WTA) critical decisions in trauma management of adult splenic trauma. J Trauma 65(5):1007–1011, 2008
15. Makley AT, Goodman MA, Fiend LAW, et al: Resuscitation with fresh whole blood ameliorates the inflammatory response after hemorrhagic shock. J Trauma 68(2):305–311, 2010
16. Cirocchi R, Abraha I, Montedori A, et al: Damage control surgery for abdominal trauma (review). The Cochrane Collaboration. John Wiley & Sons, Ltd., 2010
17. Kushimoto S, Miyauchi M, Yokota H, et al: Damage control surgery and open abdominal management: recent advances and our approach. J Nippon Med Sch 76(6):280–290, 2009
18. Matsumoto H, Mashiko K, Sakamoto Y, et al: A new look at criteria for damage control surgery. J Nippon Med Sch 77:13–20, 2010
19. Barrios C, Malinoski D, Dolich M, et al: Utility of thoracic computed tomography after blunt trauma: When is chest radiograph enough? Am Surg 10(75):966–969, 2009
20. Legome E: General approach to blunt thoracic trauma in adults. Available at: www.uptodate.com, 2010
21. Yarlagadda C: Cardiac tamponade. Available at: http://emedicine.medscape.com/article/152083-overview, 2010
22. Cook C, Gleason T: Great vessel and cardiac trauma. Surg Clin North Am 89:797–820, 2009
23. Lawson R, Goosen J: Abdominal stab wound exploration. Available at: http://emedicine.medscape.com/article/82869-overview, 2009
24. Jana T: Bedside ultrasonography, trauma evaluation. Available at: http://emedicine.medscape.com/article/104363-overview, 2010
25. Alameda County Medical Center/Highland General Hospital: Trauma Service. Focused abdominal sonography for trauma
26. Maung AA, Kaplan LJ: Diagnosis and management of splenic injury in the adult trauma patient. Available at: www.uptodate.com, 2010
27. Lyuboslavsky Y, Pattillo M: Stable patients with blunt liver injury: Observe do not operate!! Crit Care Nurs Q 32(1):14–18, 2009
28. Klepac S: Spleen trauma. Available at: http://emedicine.medscape.com/article/373694-overview, 2009
29. Smith K, Schauberger JS, Kenney P, et al: Kidney trauma. Available: http://emedicine.medscape.com/article/379085-overview, 2009

30. Rackley R, Vasuvada SP, Battino BS: Bladder trauma. Available at: http://emedicine.medscape.com/article/441124-overview, 2009
31. Papakostidis C, Giannovides PV: Pelvic ring injuries with haemodynamic instability: Efficacy of pelvic packing, a systemic review. Injury 40(Suppl 4):s53–s61, 2009
32. Fiechtl J: Adult pelvic trauma. Available at: www.uptodate.com, 2010
33. Davis J, Moore F, McIntyre R, et al: Western trauma association critical decisions in trauma: Management of pelvic fracture with hemodynamic instability. J Trauma 65(5):1012–1015, 2008
34. Frevert S, Dahl B, Lonn L: Update on the roles of angiography and embolisation in pelvic fractures. Injury 39:1290–1294, 2008
35. Stacciolini A: Acute compartment syndrome. Available at: www.uptodate.com, 2010
36. Lip G, Hull RD: Treatment of deep vein thrombosis. Available at: www.uptodate.com, 2010
37. Landaw SA, Bauer K: Approach to the diagnosis and therapy of deep vein thrombosis. Available at: www.uptodate.com, 2010
38. Thompson BT, Hales CA: Overview of acute pulmonary embolism. Available at: www.uptodate.com, 2010
39. Weinhouse GL: Fat embolism syndrome. Available at: www.uptodate.com, 2010
40. McKay MP, Mayersak RJ: Facial trauma in adults. Available at: www.uptodate.com, 2010
41. Laux L, McGonigal M, Thieret T, et al: Use of prone positioning in a patient with acute respiratory distress syndrome: A case review. Crit Care Nurs Q 31(2):178–183, 2008
42. Zarzaur BL, Magnotti LJ, Croce MA, et al: Long-term survival and return on investment after nonneurologic injury: Implications for the elderly trauma patient. J Trauma 69(1):93–98, 2010
43. Newell MA, Rotondo MF, Toschlog EA, et al: The elderly trauma patient: An investment in the future? J Trauma 67(2):337–340, 2009
44. Mancini M: Blunt chest trauma. http://emedicine.medscape.com/article/428723-overview
45. Legome E: Blunt cardiac injury (BCI) in adult trauma. www.uptodate.com, 2010

WANT TO KNOW MORE?

A wide variety of resources to enhance your learning and understanding of this chapter are available on thePoint.

You will find:

- Chapter outlines
- Additional selected readings
- NCLEX-style review questions
- Internet resources
- And more!

56 Drug Overdose and Poisoning

Eric Schuetz and Julie Schuetz

LEARNING OBJECTIVES

Based on the content in this chapter, the reader should be able to:

1. Explain the initial assessment and management of acutely poisoned or overdosed patients.
2. Describe the groups of symptoms that may help identify the drugs or toxins to which a patient may have been exposed.
3. Compare and contrast methods used to prevent absorption and enhance elimination of a drug or toxin.
4. Formulate a plan of care for the poisoned patient.

In 2008, more than 2.49 million exposures to various drugs and toxins were reported to the American Association of Poison Control Centers. Of these exposures, 1,756 resulted in death.[1] The types of toxic exposure reported to poison control centers are diverse: herbal remedies purchased at health food stores, snake and arthropod envenomations, alcohol or drugs, fumes emitted by faulty furnaces, poisonous plants, and industrial hazardous material spills or releases.

Because of clinical experience and new research information, therapy for toxic exposure changes rapidly. Health care professionals may find it challenging to keep abreast of the most advanced therapy. Fortunately, phone consultation with poison control centers offers rapid access to this information. A local poison control center can be reached nationwide by calling 1-800-222-1222. The services of a local poison control center are a useful resource for both health care professionals and the public. Nurses, pharmacists, and physicians with specialized training in clinical toxicology staff such centers.

This chapter presents general guidelines for the assessment and management of the acutely poisoned or overdosed patient. It lists commonly observed poisonings and contains a collaborative care guide for the patient with cocaine toxicity. The chapter ends with a section discussing prevention through patient teaching.

▲ The Poisoned or Overdosed Patient

Poisonings and drug overdoses can cause quick physical and mental changes in a person. Bystanders usually are the ones who must initiate care and call a poison control center or emergency number.

Poisoning

The most common routes of exposure in poisoning are inhalation, ingestion, and injection. Toxic chemical reactions can compromise cardiovascular, respiratory, central nervous, hepatic, gastrointestinal (GI), and renal systems.

Most exposures to toxic fumes occur in the home. Poisoning may result from the improper mixing of household cleaning products or malfunctioning household appliances that release carbon monoxide. Burning wood, gas, oil, coal, or kerosene also produces carbon monoxide. Carbon monoxide gas is colorless, odorless, tasteless, and nonirritating, which makes it especially dangerous.

The ingestion of poisons and toxins occurs in various settings and in different age groups. Poisoning in the home usually occurs when children ingest household cleaners or medicines. Improper storage of these items contributes to such accidents. Plants, pesticides, and paint products are also potential household poisons. Because of mental or visual impairment, illiteracy, or a language barrier, older adults may ingest incorrect amounts of medications. In addition, poisoning may occur in the health care environment when medications are administered improperly.

Similarly, poisoning can also occur in the health care environment when a medication normally given only by the subcutaneous or intramuscular route is given intravenously (IV) or when the incorrect medication is injected. Poisoning by injection can also occur in the setting of substance abuse, as when an addict inadvertently injects bleach or too much heroin.

Substance Abuse and Overdose

Admission of most poisoned patients to a critical care unit is for an intentional or suspected suicidal overdose. As part of their histories, these patients frequently have mental illness, substance abuse problems, or both. Often, withdrawal symptoms complicate the assessment of potential toxidromes. A toxidrome is a group of signs and symptoms (syndrome) associated with overdose or exposure to a particular category of drugs and toxins.

Commonly abused substances are nicotine, alcohol, heroin, marijuana, narcotic analgesics, amphetamines, benzodiazepines, and cocaine. Some children and adolescents turn to common household substances because they are readily available. People who attempt to manage stress through substance abuse require a comprehensive treatment program to address their coping and adaptation problems.

▲ Assessment

A health care facility's systematic approach to the assessment of the poisoned or overdosed patient includes performing triage, obtaining the patient's history, performing a physical examination, and conducting laboratory studies.

Triage

Although some type of triage usually is performed at the scene or by an emergency response team, triage is always the first step performed in the emergency department. Two essential questions to be considered in the triage evaluation are the following: (1) Is the patient's life in immediate danger? (2) Is the patient's life in potential danger? If the patient's life is in immediate danger, the goals of immediate treatment are patient stabilization and evaluation and management of airway, breathing, and circulation (ABC).

History

A history of the patient's exposure provides a framework for managing the poisoning or overdose. Key points include identifying the drugs or toxins, the time and duration of the exposure, first aid treatment given before arrival at the hospital, allergies, and any underlying disease processes or related injuries. This information may be obtained from the patient, family members, friends, rescuers, or bystanders.

Physical Examination

A quick but thorough physical examination is essential. Preliminary examination results lead to the in-depth evaluation and serial assessment of affected systems (actual or anticipated). As noted previously, a toxidrome is a group of signs and symptoms associated with overdose or exposure to a particular category of drugs and toxins. Recognizing the presence of a toxidrome may help identify the toxins or drugs to which the patent was exposed, and the crucial body systems that may be involved. Table 56-1 lists four common toxidromes with their signs and symptoms and common causes.

Laboratory Studies

Relevant clinical laboratory data are vital to the assessment of the poisoned or overdosed patient. Tests that provide clues to the agents taken by the patient include electrolytes, hepatic function, urinalysis, electrocardiography, and serum osmolality tests. A serum level measurement of acetaminophen is obtained in all patients who have overdosed because acetaminophen is a component of many prescription and over-the-counter preparations. In the event of an acetaminophen overdose, the result of the level is plotted against the time since ingestion on the Rumack-Matthew nomogram (Fig. 56-1). Serum level measurements are also available for carbamazepine, iron, ethanol, lithium, aspirin, and valproic acid and may be obtained if these agents are suspected in an overdose.

▲ Management

Management of the poisoned or overdosed patient seeks to prevent absorption of and further exposure to the agent. After triage to determine the status of the patient's ABC, the patient must be stabilized. Treatment begins with first aid at the scene and continues in the emergency department and often in the critical care unit. Advanced general management involves further steps to prevent absorption and enhance elimination of the agent. Antidotes, antivenins, or antitoxins, when available, may be administered. The

Table 56-1 Toxidromes

Toxidrome	Signs/Symptoms	Common Causes
Anticholinergic agents	Delirium; dry, flushed skin; dilated pupils; elevated temperature; decreased bowel sounds; urinary retention; tachycardia	Antihistamines, atropine, jimson weed
Cholinergic agents	Excessive salivation, lacrimation, urination, diarrhea, and emesis; diaphoresis, bronchorrhea, bradycardia, fasciculations, central nervous system (CNS) depression, constricted pupils	Organophosphate insecticides (eg, malathion, diazinon); carbamate insecticide (eg, carbaryl, propoxur)
Opioid agents	CNS depression, respiratory depression, constricted pupils, hypotension, hypothermia	Opiates (eg, codeine, morphine, propoxyphene, heroin), diphenoxylate (eg, diphenoxylate/atropine sulfate [Lomotil])
Sympathomimetic agents	Agitation, tachycardia, hypertension, seizures, metabolic acidosis	Amphetamines, cocaine, theophylline, caffeine

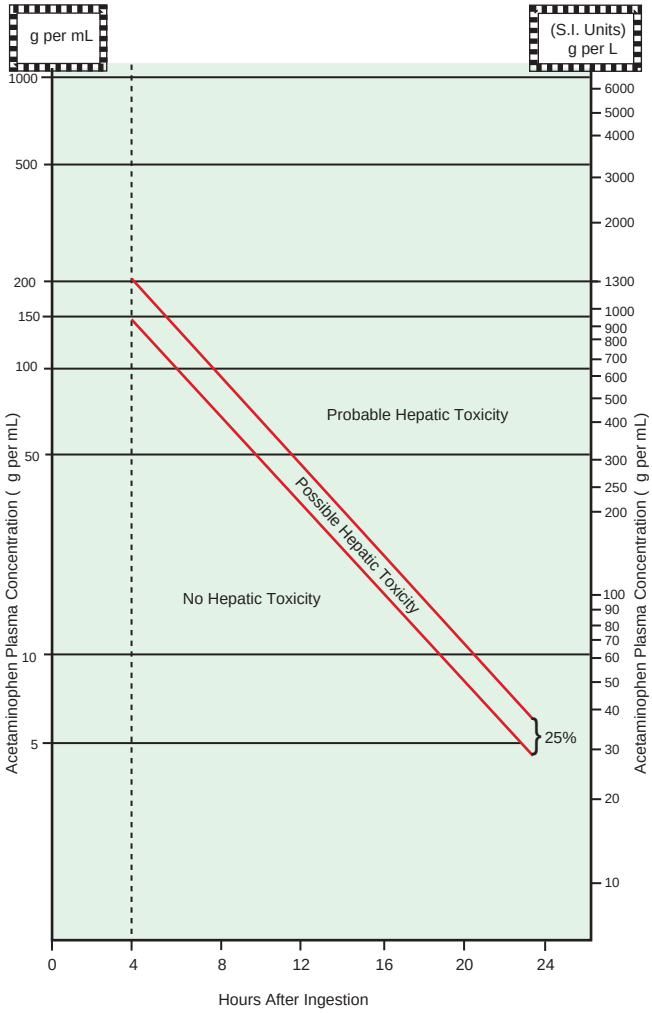


FIGURE 56-1 ▲ Semilogarithmic plot of plasma acetaminophen levels versus time. (From Rumack BH, Matthew HJ: Acetaminophen poisoning and toxicity. Pediatrics 55:871–876, 1975.)

health care team must further support vital functions and monitor and treat multisystem effects. Patient and family teaching to prevent future exposures is another part of the nurse's management strategy. Examples of nursing diagnoses for the poisoned or overdosed patient are listed in Box 56-1.

Stabilization

Stabilization of patients includes performing the steps summarized in Box 56-2, which are also discussed in the following list:

- *Airway patency*. Nasotracheal or endotracheal intubation may be necessary to adequately maintain and protect the patient's airway.

BOX 56-1 EXAMPLES OF NURSING DIAGNOSES

For the Poisoned or Overdosed Patient

- Ineffective Breathing Pattern related to overdose of medication
- Decreased Cardiac Output related to ingestion of poisonous substance
- Impaired Verbal Communication related to overdose of medication
- Acute Confusion related to overdose of medication.
- Impaired Gas Exchange related to carbon monoxide poisoning
- Impaired Swallowing related to poisoning

- *Breathing*. Mechanical ventilation may be necessary to support the patient. Many drugs and toxins depress the respiratory drive. Patients therefore may require ventilator assistance until the drugs or toxins are eliminated from the body.
- *Circulation*. Complications range from shock caused by fluid loss to fluid overload. These are often related to the patient's hydration status and the ability of the cardiovascular system to adjust to drug- or toxin-induced changes. For example, rattlesnake envenomations often cause third spacing of fluid into the area of the bite, leading to intravascular hypovolemia. As a consequence, the patient develops hypotension, which usually responds to aggressive IV fluid therapy. Some toxic drug ingestions impair myocardial contractility, and fluid overload may result because of the heart's inability to pump effectively. In these cases, fluid balance needs to be carefully controlled. Invasive monitoring (eg, central venous pressure, pulmonary artery catheter, Foley catheter with urometer) and drug therapy may be necessary to prevent or minimize complications such as pulmonary edema.
- *Cardiac function*. Many drugs and toxins cause cardiac conduction delays and dysrhythmias. The history of the drugs or toxins involved may not be reliable or even known, especially when patients are found unconscious or have attempted suicide. In these cases, continuous cardiac monitoring and 12-lead electrocardiograms (ECGs) help detect cardiotoxic effects.
- *Acid–base balance and electrolyte homeostasis*. Electrolyte abnormalities and metabolic acidosis frequently occur, which may require serial measurements of electrolytes and arterial blood gases (ABGs), as well as other specific

BOX 56-2 NURSING INTERVENTIONS

For the Stabilization of the Poisoned or Overdosed Patient

- Assess, establish, and maintain the airway.
- Evaluate respiratory effort.
- Maintain adequate circulation.
- Monitor cardiac function.
- Maintain or correct acid–base balance and electrolyte homeostasis.
- Assess mentation.
- Identify injuries and disease processes that increase risk.
- Measure vital signs and temperature frequently to track changes.

laboratory tests. For example, serial measurements of electrolytes, ABGs, and salicylate levels are the means of evaluating aspirin toxicity.

- *Mentation*. Many factors can affect the patient's mental status. Hypoglycemia and hypoxemia are two that can be life threatening but easily addressed by administering oxygen and IV dextrose until laboratory results are available. Naloxone (Narcan) is a narcotic antagonist that reverses narcotic-induced central nervous system and respiratory depression. It is often initially given to comatose patients. However, it must be given cautiously because it can precipitate withdrawal in narcotic-dependent individuals, which may present as violent, agitated behavior, thus placing nurses and other health care providers in danger. In the critical care unit, it may be necessary to continue to administer boluses of naloxone to a patient because of its short duration of action compared with the duration of action of most opioids. In such circumstances, it may be necessary to give naloxone by continuous infusion.[2]
- *Injuries associated with toxic exposure and underlying disease processes*. Any injuries associated with toxic exposure and other underlying disease processes identified during the initial physical examination are treated or monitored, or both. For example, the street drug phencyclidine (PCP) may provoke violent, agitated, bizarre behavior, leading to trauma during the acute toxic phase. Also, for instance, the patient with preexisting ischemic heart disease may not be able to tolerate the hypoxemia associated with carbon monoxide poisoning as well as a young, healthy patient.
- *Vital signs and temperature*. The critical or potentially critical patient's vital signs and temperature are measured frequently to track changes indicating additional problems.

Initial Decontamination

First aid may be given by a bystander, health care provider, or emergency response team or in the emergency department. The physicochemical properties of the agent and the amount, route, and exposure time help determine the type and extent of management required. Decontamination methods for ocular, dermal, inhalation, and ingestion exposures follow.

Ocular Exposure

Many substances can accidentally splash into the eyes. When this happens, the eyes must be flushed to remove the agent. Immediate irrigation with lukewarm water or normal saline solution is recommended. Continuous flooding of the eyes with a large glass of water or low-pressure shower should be done for 15 minutes. The patient should blink the eyes open and closed during the irrigation. An ophthalmological examination is needed if ocular irritation or visual disturbance persists after irrigation.

Dermal Exposure

When dermal exposure occurs, the patient should flood the skin with lukewarm water for 15 to 30 minutes. Most companies that produce or use chemical agents have showers for this purpose. The patient should remove any clothing that may have been contaminated. After standing under running water for the allotted time, the patient should then wash the area gently with soap and water and rinse thoroughly.

Some toxins may require further decontamination. For example, three separate soap-and-water washings or showers are recommended to decontaminate organophosphate pesticides (eg, malathion or diazinon). Protective clothing should be worn to reduce the risk for toxicity while handling contaminated clothing or assisting with skin decontamination.

Although it may seem logical to apply an acid to neutralize a base exposure and a base to neutralize an acid exposure, this can be quite dangerous. Neutralization is the reaction between an acid and a base in which the H^+ of the acid and the OH^- of the base react to produce H_2O (water) and heat. The heat produced by this reaction is significant enough to cause burns. Therefore, neutralizing the skin after a dermal exposure is not recommended.

Inhalation Exposure

A person who has experienced an inhalation exposure should be moved to fresh air as quickly as possible. The responder must also protect himself or herself from the airborne toxin. Further evaluation is needed if the patient experiences respiratory irritation or shortness of breath. Large-scale exposures or those that occur at the workplace may require consultation with a Hazmat team, a group of individuals specially trained to manage exposures to hazardous materials.

Ingestion Exposure

Milk or water dilutes ingested irritants, such as bleach, or caustics, such as drain cleaner. After such an ingestion, adults should drink 8 oz of milk or water, and children should drink 2 to 8 oz (based on their size). Further evaluation is necessary after dilution if there is mucosal irritation or burns. Because of the risk for aspiration, ingestions should not be diluted when they are accompanied by seizures, depressed mental status, or loss of the gag reflex. Again, neutralization is not used because of the risk for thermal burn.

Gastrointestinal Decontamination

Gastric lavage, adsorbents, cathartics, and whole-bowel irrigation are used to prevent absorption of, and forestall toxicity from, almost all drugs and a variety of toxins. The American Academy of Pediatrics no longer recommends the use of emetics (such as of ipecac syrup) for GI decontamination.

Gastric Lavage

Gastric lavage is a method of GI decontamination. Fluid (usually normal saline solution) is introduced into the stomach through a large-bore orogastric tube and then drained in an attempt to reclaim part of the ingested agent before it is absorbed. A small-bore nasogastric tube is ineffective for lavage because particulate matter, such as tablets or capsules, is too large to pass through the tube. If airway protection is necessary, the patient should be intubated before lavage begins.

As noted, a large-bore orogastric tube (36- to 40-French in adults and 16- to 28-French in children) is used to evacuate particulate matter, including whole tablets and capsules. For the lavage, the patient is positioned in the left lateral decubitus position, with the head lower than the feet. Before beginning, the tube should be coated with a jelly lubricant

such as hydroxyethylcellulose. The position of the tube must be confirmed after passing, either by aspirating and checking the pH of the aspirate or by insufflation of air, while listening over the stomach. The lavage is accomplished by attaching a funnel or syringe to the end of the tube and instilling aliquots of 150 to 200 mL (50 to 100 mL in children) of 100°F (38°C) saline solution into the stomach. Placing the funnel and tube below the patient allows the fluid to return by gravity. This procedure is repeated until clear fluid returns or 2 L of fluid has been used. The contents of the stomach can then be collected for drug or toxin identification.

Complications of gastric lavage include esophageal perforation, pulmonary aspiration, electrolyte imbalance, tension pneumothorax, and hypothermia (when cold lavage solutions are used). Lavage is contraindicated in cases of ingestion of caustics or hydrocarbons with a high aspiration potential. Because of the associated risks and the lack of clear evidence supporting its use, gastric lavage should be used only if the patient has ingested a life-threatening amount of a substance and the procedure is undertaken within an hour of the ingestion.[3]

Adsorbents

An adsorbent is a solid substance that has the ability to attract and hold another substance to its surface ("to adsorb"). Activated charcoal is an effective nonspecific adsorbent of many drugs and toxins. Activated charcoal adsorbs, or traps, the drug or toxin to its large surface area and prevents absorption from the GI tract. Box 56-3 identifies both drugs and toxins that are adsorbed effectively by activated charcoal and those not adsorbed effectively.

Activated charcoal is a fine, black powder that is given as a slurry with water, either orally or by nasogastric or orogastric tube, as soon as possible after the ingestion. Commercially available activated charcoal products may be mixed with 70% sorbitol to decrease grittiness, increase palatability, and serve as a cathartic. The usual dose that is given is one 50-g bottle. Administration of more than one dose is controversial and usually is limited to overdoses of large quantities of aspirin, valproic acid, and theophylline. Activated charcoal is used cautiously in patients with diminished bowel sounds and is contraindicated in patients with bowel obstruction.[3]

BOX 56-3 Adsorption of Drugs and Toxins by Activated Charcoal

Drugs and Toxins Well Adsorbed by Activated Charcoal
- Acetaminophen
- Amphetamines
- Antihistamines
- Aspirin
- Barbiturates
- Benzodiazepines
- β-Blockers
- Calcium channel blockers
- Cocaine
- Opioids
- Phenytoin
- Theophylline
- Valproic acid

Drugs and Toxins Not Well Adsorbed by Activated Charcoal
- Acids
- Alkalis
- Alcohols
- Iron
- Lithium
- Metals

Cathartics

A cathartic is a substance that causes or promotes bowel movements. The use of cathartics alone in the management of poisoning is not an acceptable means of GI decontamination. In theory, cathartics decrease the absorption of drugs and toxins by speeding their passage through the GI tract, thereby limiting their contact with mucosal surfaces. Magnesium citrate or 70% sorbitol often is used. Currently, however, there is no clinical evidence that shows that a cathartic can reduce the bioavailability of drugs or improve the outcome of poisoned patients. Data regarding the effectiveness of mixing cathartics with activated charcoal are not yet available. Clearly, more research needs to be done in this area of clinical practice.[3]

Whole-Bowel Irrigation

The goal of whole-bowel irrigation is to give large volumes of a polyethylene glycol with electrolytes solution rapidly (1 to 2 L/h) to flush the patient's bowel mechanically without creating electrolyte disturbances. Used as a bowel preparation for colonoscopy, it is also used as a GI decontamination procedure for patients who have ingested bags or vials of narcotics to avoid arrest, for drug smugglers who pack their GI tracts with narcotics (either orally or rectally), and for patients who have overdosed on modified-release pharmaceuticals.

Commercial products used in whole-bowel irrigation include GoLYTELY and Colyte. Both products are dispensed as powders and are given after adding water. Whole-bowel irrigation is contraindicated in the patient with bowel obstruction or perforation.[3]

Enhanced Elimination of the Drug or Toxin

The pharmacological and kinetic characteristics of a drug or toxin greatly influence the severity and length of the clinical course in the acutely poisoned or overdosed patient. The absorption rate, body distribution, metabolism, and elimination must be considered when choosing methods to eliminate the drug or toxin from the body

Multiple-Dose Activated Charcoal

Administering multiple doses of activated charcoal can result in greater adsorption of certain drugs such as aspirin, valproic acid, and theophylline. Multiple-dose activated charcoal is given orally, by nasogastric tube, or by orogastric tube every 2 to 6 hours. Complications of multiple-dose activated charcoal include aspiration and bowel obstruction.[3]

Alteration of Urine pH

Alkalinizing the patient's urine enhances excretion of drugs that are weak acids by increasing the amount of ionized

drug in the urine. This form of enhanced elimination is also termed ion trapping. The urine is alkalinized by administering a continuous IV infusion of 1 to 3 ampules of sodium bicarbonate per 1 liter of fluid. Urine alkalinization is frequently used in patients experiencing a salicylate overdose. Complications of alkalinization include cerebral or pulmonary edema and electrolyte imbalances. Urine acidification is no longer recommended because of low drug clearance and the risk for complications such as rhabdomyolysis.

Hemodialysis

Hemodialysis is the process of altering the solute composition of blood by removing it from an artery, diffusing it across a semipermeable membrane (between the blood and a salt solution), then returning it into a vein. It is used in moderate to severe intoxications to remove a drug or toxin rapidly when more conservative methods (eg, gastric lavage, activated charcoal, antidotes) have failed or in patients with decreased renal function. Hemodialysis requires consultation with a nephrologist and specially trained nurses to perform the procedure and monitor the patient. Low molecular weight, low protein binding, and water solubility are factors that make a drug or toxin suitable for hemodialysis. Drugs and toxins that may be removed by hemodialysis include ethylene glycol (commonly found in antifreeze), methanol, lithium, salicylates, and theophylline.[4]

Hemoperfusion

Hemoperfusion removes drugs and toxins from the patient's blood by pumping the blood through a cartridge of adsorbent material, such as activated charcoal. An advantage of hemoperfusion over hemodialysis is that the total surface area of the dialyzing membrane is much greater with the hemoperfusion cartridges. As in hemodialysis, drugs that have high tissue-binding characteristics and a large volume distributed outside the circulation are not good candidates for hemoperfusion because little drug is found in the blood. Although rarely used in the poisoned and overdosed population, hemoperfusion has been used successfully in patients experiencing a theophylline overdose.[4]

Chelation

Chelation involves the use of binding agents to remove toxic levels of metals from the body, such as mercury, lead, iron, and arsenic. Examples of chelating agents are dimercaprol (BAL in oil), calcium disodium edetate (EDTA), succimer (DMSA), and deferoxamine. Concerns about the toxicity of the chelators; their tissue distribution characteristics; and the stability, distribution, and elimination of the chelator–metal complex make chelation a complicated procedure.

Hyperbaric Oxygenation Therapy

In hyperbaric oxygenation therapy (HBO) therapy, oxygen is administered to a patient in an enclosed chamber at a pressure greater than the pressure at sea level (eg, 1 atm). This therapy is sometimes used in carbon monoxide poisoning. Elimination of carbon monoxide is as follows: in room air, the half-life of carbon monoxide is 5 to 6 hours; in 100% oxygen, it is 90 minutes; and in an HBO chamber, it is 20 minutes. Complications of HBO therapy include pressure-related otalgia, sinus pain, tooth pain, and tympanic membrane rupture. Confinement anxiety, convulsions, and tension pneumothorax also have been observed in patients receiving HBO therapy.[5]

Antagonists, Antitoxins, and Antivenins

In pharmacology, an antagonist is a substance that counteracts the action of another drug. Although the general public often believes there is an antidote for every drug or toxin, the opposite is closer to the truth. There are, in fact, very few antidotes. Antidotes for specific intoxications are listed in Table 56-2.

Antitoxins neutralize a toxin. For instance, botulism antitoxin trivalent (equine) is available through the Centers for Disease Control and Prevention (CDC) to counteract the effects of botulism.

Antivenins are antitoxins that neutralize the venom of the offending snake or spider. There are several antitoxins; each is active against a specific venom. Recently approved

Table 56-2 Antidotes for Specific Drugs and Toxins

Drug/Toxin	Antidote
Acetaminophen	*N*-acetylcysteine (NAC) (Mucomyst [PO], Acetadote [IV])
Anticholinergics	Physostigmine (Antilirium)
Benzodiazepines	Flumazenil (Romazicon)
β-Blocking agents	Glucagon
Calcium channel blockers	Glucagon, calcium chloride, hyperinsulinemia-euglycemia
Carbon monoxide	Oxygen
Cyanide	Lilly Cyanide Antidote Kit: amyl nitrite, sodium nitrite, and sodium thiosulfate, Cyanokit (hydroxocobalamin)
Digoxin	Digoxin-specific fab fragments (Digibind or DigiFAB)
Ethylene glycol	Fomepizole (Antizol)[6], ethanol
Methanol	Fomepizole (Antizol)[7], ethanol
Nitrites	Methylene blue
Opioids	Naloxone (Narcan)
Organophosphate insecticides	Atropine, pralidoxime

by the U.S. Food and Drug Administration (FDA) is crotalidae polyvalent immune Fab (CroFab), a product that is produced using a purification process that removes the Fc fragment and leaves only the Fab fragments of the immunoglobulins. Typically, this process results in a product that causes fewer reactions in humans. Antivenin (*Latrodectus mactans;* equine) is available for black widow spider bites as well as for envenomations by the eastern and Texas coral snake (*Micrurus fulvius;* equine). However, there are many venomous snakes and spiders for which no antivenin exists. Envenomation from one of these species is treated with symptomatic and supportive care.[6,7]

Continuous Patient Monitoring

Seriously poisoned or overdosed patients may require continued monitoring for hours or days after exposure. Physical examination, the use of diagnostic tools, and careful assessment of clinical signs and symptoms provide information about the patient's progress and direct medical and nursing management. Diagnostic tools include the following:

- *Electrocardiography*. Electrocardiography can provide evidence of drugs causing dysrhythmias or conduction delays (eg, tricyclic antidepressants).
- *Radiology*. Many substances are radiopaque or can be visualized using a contrast-enhanced computed tomography scan (eg, heavy metals, button batteries, some modified-release tablets or capsules, aspirin concretions, cocaine or heroin containers). Chest radiographs provide evidence of aspiration and pulmonary edema.
- *Electrolytes, ABGs, and other laboratory tests*. Acute poisoning can cause an imbalance in a patient's electrolyte levels, including sodium, potassium, chloride, carbon dioxide content, magnesium, and calcium. Signs of inadequate ventilation or oxygenation include cyanosis, tachycardia, hypoventilation, intercostal muscle retractions, and altered mental status. Such signs should be evaluated by pulse oximetry and ABG measurements. Seriously poisoned patients require routine screening of electrolytes, ABGs, creatinine, and glucose; complete blood count; and urinalysis.
- *Anion gap*. The anion gap is a simple, cost-effective tool that uses common serum measurements, such as sodium, chloride, and bicarbonate, to help evaluate the poisoned patient for certain drugs or toxins. The anion gap represents the difference between unmeasured anions and cations in the blood. Using measured anions and a cation, the anion gap is calculated using the following formula:

$$[Na] - ([Cl] + [HCO_3]) = \text{anion gap}$$

The normal value for the anion gap is approximately 8 to 16 mEq/L. An anion gap that exceeds the upper normal value can indicate metabolic acidosis caused by an accumulation of acids in the blood. Drugs, toxins, or medical conditions that can produce an elevated anion gap include iron, isoniazid (INH), lithium, lactate, carbon monoxide, cyanide, toluene, methanol, metformin, ethanol, ethylene glycol, salicylates, hydrogen sulfide, strychnine, diabetic ketoacidosis, uremia, seizures, and starvation. Although these substances and processes can cause an elevated anion gap, a normal anion gap alone does not preclude a toxic exposure.

- *Osmolal gap*. The osmolal gap is the difference between the measured osmolality (using the freezing point depression method) and the calculated osmolality. The calculated osmolality is derived using laboratory values for the major osmotically active substances in the serum, such as sodium, glucose, and blood urea nitrogen (BUN). Like the anion gap, it is a simple, cost-effective tool for evaluating the poisoned patient for certain drugs or toxins. The calculated osmolality (using serum electrolyte values) is defined as follows:

$$2(Na^+) + \frac{\text{glucose}}{18} + \frac{\text{BUN}}{2.8} = \text{calculated osmolality}$$

The osmolal gap is then calculated as follows:

Measured osmolality − calculated osmolality = osmolal gap

An osmolal gap that exceeds 10 mOsm is abnormal. Toxins that can cause an elevated osmolal gap include ethanol, ethylene glycol, and methanol. If an ethanol level is known, it can be factored into the following equation:

$$2(Na^+) + \frac{\text{glucose}}{18} + \frac{\text{BUN}}{2.8} + \frac{\text{BAL}}{4.6} = \text{calculated osmolality}$$

where BAL is the blood alcohol level measured in milligrams per deciliter.

- *Toxicology screens*. A toxicology screen is a laboratory analysis of a body fluid or tissue to identify drugs or toxins. Although saliva, spinal fluid, and hair may be analyzed, blood or urine samples are used more frequently. The number and type of drugs assessed by toxicology screens vary. Each screen tests for specific drugs or agents. For example, drug abuse screens usually identify several common street or prescription drugs, whereas a coma panel detects common drugs that cause CNS depression. Comprehensive screens include many drugs (ranging from antidepressants to cardiac drugs to alcohols) and are more expensive. A number of factors limit the role of toxicology screens in managing poisonings or overdoses. The test sample must be collected while the drug or toxin is in the body fluid or tissue used for testing. For example, cocaine is a rapidly metabolized drug; however, its metabolite, benzoylecgonine, can be detected in the urine for several hours after cocaine use. Also, a toxicology screen with a negative result does not necessarily mean that no drug or toxin is present, but rather that none of the drugs or toxins for which a patient has been screened is present. For example, γ-hydroxybutyrate is not included in toxicology screens because it is rapidly metabolized to small, unmeasurable molecules.

Patient care in some of the more common poisonings and overdoses is summarized in Table 56-3. Clinical manifestations are included in the table. Management of the patient who is toxic with cocaine is summarized in Box 56-4 on page 1272–1273.[8,9]

Patient Teaching

One of the interventions the nurse can perform in the emergency department or intensive care unit is preventive

Text continued on page 1273

Table 56-3 Common Patient Care in Poisonings and Overdoses

Drug/Substance	Clinical Presentation and Assessment	Intervention
Acetaminophen (APAP)		
Common OTC antipyretic and analgesic Often sold as a component of combination drugs for pain, cough, cold, and sleep Examples: OTC remedies such as Tylenol, Tylenol Extended Relief, Tempra, Liquiprin, Panadol, Excedrin PM (diphenhydramine-APAP) and in controlled-substance combination drugs such as oxycodone-APAP (Percocet), codeine-APAP (Tylenol #3), hydrocodone-APAP (Vicodin) Acetaminophen toxicity: hepatotoxicity and occasionally renal dysfunction, 1–3 d postingestion	• Phase 1 (up to 24 h postingestion): anorexia, nausea, malaise • Phase 2 (24–48 h postingestion): clinical picture improves, increase in AST, ALT, and total bilirubin, prolongation of prothrombin time • Phase 3 (72–96 h postingestion): peak hepatotoxicity usually observed • Coagulopathies • Jaundice • AST and ALT may rise into the 10,000–20,000 IU/L range and return to normal without the patient experiencing long-term sequelae. • Chronic toxicity well described in the medical literature	*Prevention of absorption:* • Activated charcoal *Laboratory:* • Draw acetaminophen level at 4 h (or later if patient presents late to the health care facility), plot level on the Rumack-Matthew nomogram (see Fig. 56-1, p. 1263) to determine whether antidote is indicated. • Monitor daily AST, ALT, total bilirubin, blood urea nitrogen, creatinine, and prothrombin time in patients with a toxic acetaminophen level. *Treatment:* • Antidote: NAC • Oral: NAC, Mucomyst • Loading dose: 140 mg/kg orally • Maintenance doses: 70 mg/kg orally every 4 h for a total of 17 maintenance doses • Dilute NAC (20% solution) 3:1 with a soft drink or juice • Repeat any dose not retained 1 h, may need large doses of antiemetics to control vomiting[10] • IV: Acetadote • Loading dose: 150 mg/kg in 200 mL of D_5W IV over 60 min • First maintenance dose: 50 mg/kg in 500 mL of D_5W IV over 4 h • Second maintenance dose: 100 mg/kg in 1,000 mL of D_5W IV over 16 h • Supportive care
Amphetamines		
Group of drugs used therapeutically for narcolepsy, short-term treatment of obesity, and attention-deficit disorder As drugs of abuse, used for ability to stimulate CNS to combat fatigue or produce a “high” Prescription amphetamines and related agents: methylphenidate (Ritalin), dextroamphetamine (Dexedrine), mixed salts of amphetamine (Adderall) Street names: speed, uppers, crank, E, X, ecstasy, ice, crystal	• Flushing • Diaphoresis • Restlessness • Talkativeness • Irritability • Confusion • Panic • Seizures • Intracranial hemorrhage • Hypertension • Tachycardia • Chest pain • Myocardial infarction • Cardiac dysrhythmias • Palpitations • Peripheral vasoconstriction • Nausea • Vomiting • Chronic amphetamine toxicity may lead to the development of paranoia or hallucinations. • IV abusers may also have complications such as hepatitis, sepsis, abscesses, and HIV infection.	*Prevention of absorption:* • Activated charcoal *Laboratory:* • Monitor electrolytes and acid–base status • Urine drug screen may detect amphetamines *Treatment:* • External cooling measures for hyperthermia • Benzodiazepines to control agitation • Severe hypertension controlled with IV nitroprusside (Nipride), other drugs suggested • Supportive care

(*continued on page 1269*)

Table 56-3 Common Patient Care in Poisonings and Overdoses (continued)

Drug/Substance	Clinical Presentation and Assessment	Intervention
Benzodiazepines		
Antianxiety agents, anticonvulsants, muscle relaxants, and sedatives Examples: alprazolam (Xanax), clonazepam (Klonopin), diazepam (Valium), lorazepam (Ativan), midazolam (Versed) Primarily cause CNS and respiratory depression. Due to their low order of toxicity, fatalities unlikely unless ingested with other CNS depressants	• Respiratory depression • Airway protection/gag reflex • Lethargy • Coma • Confusion • Slurred speech • Ataxia	*Prevention of absorption:* • Activated charcoal *Laboratory:* • Urine drug screen may detect benzodiazepines. *Treatment:* • Flumazenil reverses CNS and respiratory depression; due to risk in unmasking controlled seizures, flumazenil is contraindicated in the face of simultaneous potential seizure causing overdose. • Supportive care
Carbon Monoxide		
Colorless, odorless gas that is a component of automobile exhaust, natural gas or propane furnace emissions, cigarette smoke, wood stove emissions, and pollution Methylene chloride, a component found in some paint strippers, is metabolized in the body to carbon monoxide after inhaled or ingested It displaces oxygen from the hemoglobin, leading to hypoxia It is absorbed rapidly by inhalation and combines readily with hemoglobin due to a greater affinity than oxygen Fetal carboxyhemoglobin levels are possibly 10%–15% greater than the maternal carboxyhemoglobin level	• Flu-like symptoms • Headache • Nausea • Vomiting • Syncope • Fatigue • Weakness • Lack of concentration • Irritability • Chest pain, especially in people with underlying cardiovascular disease • Occasionally, irreversible changes in memory and personality • Fetotoxicity • People usually report feeling better when not in the area of the carbon monoxide; for example, if the exposure is occurring in the home because of a faulty furnace, the person will often report a decrease or resolution of symptoms when away from the home.	*Prevention of absorption:* • Fresh air *Laboratory:* • Carboxyhemoglobin levels *Treatment:* • 100% oxygen until all signs and symptoms resolve • Thorough neurological examination • Hyperbaric oxygenation therapy (HBO) to decrease half-life; however, due to lack of available HBO chambers, use is limited and efficacy not well documented by research • Supportive care
Cocaine		
Common street drug that produces a temporary feeling of well-being for the user Routes of exposure: IV, snorting, smoking Street names: crack, rock, coke, snow, blow Toxic effects related to the rapid onset of CNS and cardiac stimulation	• Tachycardia • Hypertension • Cardiac dysrhythmias • Chest pain • Myocardial infarction • Aortic dissection • Bowel infarction • Hyperthermia • Anxiety • Seizures • Tactile hallucinations (“cocaine bugs”) • Cerebral hemorrhage • Cerebral infarction • Rhabdomyolysis • Rapid onset of toxic effects • In pregnant women, abruptio placentae or abortion possible • Chronic snorting, nasal septal perforation If clinical presentation is inconsistent with cocaine alone, possibly adulterants, substitutes, coingestants, or withdrawal	*Prevention of absorption (for ingested packets):* • Activated charcoal • Whole-bowel irrigation *Laboratory:* • Urine drug screen to detect metabolite of cocaine: benzoylecgonine • Cardiac enzymes as indicated to rule out myocardial infarction *Treatment:* • Benzodiazepines such as diazepam (Valium) usually control hyperactivity, hypertension, tachycardia, anxiety, hyperthermia, and seizures. • Phenobarbital may be necessary if seizures not controlled with benzodiazepines. • Life-threatening hyperthermia may be reduced by external cooling measures. • Cardiac monitoring and serial 12-lead ECG are used to evaluate dysrhythmias and myocardial ischemia.[8] • Monitor for other organ ischemia or infarction. • Provide supportive care.

(Continued on page 1270)

Table 56-3 Common Patient Care in Poisonings and Overdoses (continued)

Drug/Substance	Clinical Presentation and Assessment	Intervention
Halogenated Hydrocarbons		
Agents used as propellants and refrigerants Freon, dichlorodifluoromethane (Freon 12) and trichloromonofluoromethane (Freon 11) included in this category Exposures to leaking household air conditioners are usually minor, causing transient eye, nose, and throat irritation; dizziness; and palpitations More concentrated exposures such as in industrial spills or deliberate abuse ("huffing") associated with possible fatal ventricular dysrhythmias (due to myocardial sensitization to catecholamines) and pulmonary edema	• Eye, nose, and throat irritation • Cough • Dizziness • Disorientation • Palpitations • Bronchial constriction • Pulmonary edema • Ventricular dysrhythmias • Frostbite possible with dermal exposures	*Prevention of absorption:* • Fresh air *Laboratory:* • No specific laboratory tests *Treatment:* • Quiet environment • Cardiac monitoring • Frostbite: complete rewarming • Supportive care
Heroin		
Common street drug that produces a temporary euphoria in the user Routes of exposure: IV, snorting Street names: dope, smack, junk	• Miosis • Decreased respiratory drive • Decreased level of consciousness • "Nodding"	*Prevention of absorption:* • Not applicable *Laboratory:* • As clinically indicated • Serum toxicology screen *Treatment:* • Careful administration of naloxone • Referral to substance abuse counselor
Lysergic Acid Diethylamide (LSD)		
Common name for psychedelic drug LSD Common drug of abuse since its rise in popularity in the 1960s Street drug: available in tablet, capsule, sugar cubes, or as a substance on blotting paper known as "blotter acid" One source of LSD is ingestion of morning glory seeds In addition to the psychedelic experience, may result in physical effects and behavior-related trauma during the acute toxic phase	• Anxiety • Impaired color perception • Impaired judgment • Paranoia or ideas of persecution • Time distortions • Blood pressure normal • Tachycardia • Tachypnea • Slight temperature elevation • Flashbacks (transient recurrences of a psychedelic experience) possible after a period of abstinence, may recur for years • Trauma due to behavioral changes associated with LSD use	*Prevention of absorption:* • Activated charcoal • Cathartic *Laboratory:* • Urine drug screen *Treatment:* • Acute anxiety may be managed with IV or oral diazepam (Valium). • A quiet, nonstimulating environment may be useful while trying to help the patient who is experiencing a bad reaction. • Evaluate for evidence of trauma. • Provide supportive care.
Methanol		
Highly toxic antifreeze and solvent Available forms: most windshield washer fluids, Sterno canned heat, and components of some paints, gasoline additives, and shellacs Toxic effects: life-threatening acidosis and irreversible blindness, caused by the toxic metabolite, not the methanol itself	• Blurred vision • Decreased visual acuity • Subjective description of vision as if walking in a snowstorm • Retinal edema • Hyperemia of the optic disk • Headache • Vertigo • Lethargy • Confusion • Coma • Nausea • Vomiting • Abdominal pain • Metabolic acidosis	*Prevention of absorption:* • Syrup of ipecac • Gastric lavage • Activated charcoal and cathartic are of little value *Laboratory:* • Methanol level drawn 1 h postingestion • Serial electrolytes • If using ethanol therapy, serial glucose and blood ethanol level monitored every hour initially *Treatment:* • Treatment is aimed at preventing the formation of toxic metabolites with either Antizol (4-methylpyrazole: 4-MP) or ethanol.

(*continued on page 1271*)

Table 56-3 Common Patient Care in Poisonings and Overdoses (continued)

Drug/Substance	Clinical Presentation and Assessment	Intervention
Methanol		
		• Hemodialysis usually indicated for methanol levels more than 50 mg/dL, visual changes, renal failure, or refractory acidosis. • Folic acid administration to assist with oxidation of the toxic metabolite formic acid to carbon dioxide • Supportive care
Salicylates		
Group of drugs used primarily for anti-inflammatory, antipyretic, and analgesic properties Common sources: aspirin, some formulations of Alka-Seltzer, Aspergum, PeptoBismol, sunscreens, liniments such as Icy Hot, and oil of wintergreen (methylsalicylate) Life-threatening metabolic acidosis, cerebral edema, and pulmonary edema from salicylism Aspirin ingestions difficult to manage due to the formation of a mass of aspirin in the gastrointestinal (GI) tract called a concretion Concretion formation leads to delayed absorption and therefore delayed toxicity Chronic salicylism more common in older adults and easily missed due to lack of careful history taking Higher salicylate levels tolerated with acute overdose as opposed to chronic toxicity	• Tinnitus • Tachypnea • Pulmonary edema • Confusion • Lethargy • Seizures • Cerebral edema • Respiratory alkalosis coupled with metabolic acidosis (initially) • Hypokalemia • Platelet dysfunction • Hypothrombinemia • GI hemorrhage • Nausea • Vomiting • Hyperthermia • Dehydration	*Prevention of absorption:* • Syrup of ipecac • Gastric lavage • Multiple-dose activated charcoal • Single-dose cathartic *Laboratory:* • Serial salicylate levels • Serial electrolytes • Arterial blood gas (ABG) as indicated • Hematological and coagulation studies *Treatment:* • IV hydration • Urinary excretion is enhanced by urine alkalinization (urine pH = 7.5–8.0); IV fluid is usually D_5W with 20–40 mEq KCl and 2–3 ampules of sodium bicarbonate/L to infuse at a rate of 2–3 mL/kg/h to achieve equal urine output Note: It is difficult to alkalinize the urine without a normal serum potassium level. • Potassium is replaced IV as needed. • Monitor onset of cerebral or pulmonary edema; chest radiograph is taken as needed. • Hemodialysis is indicated for renal failure, cerebral edema, pulmonary edema, refractory acidosis, chronic salicylate level more than 50 mg/dL, or acute salicylate level more than 100 mg/dL postingestion. • Provide supportive care Note: Treatment is based on serial salicylate levels and clinical presentation; each case is individually assessed and managed.
Tricyclic Antidepressants (TCA)		
Class of drugs prescribed for depression and chronic pain Examples: amitriptyline (Elavil), clomipramine (Anafranil), desipramine (Norpramin), doxepin (Adapin, Sinequan), imipramine (Tofranil), nortriptyline (Pamelor, Aventyl), protriptyline (Vivactil), and trimipramine (Surmontil)	• Tachycardia • Ventricular dysrhythmias (including ventricular tachycardia and ventricular fibrillation) • Cardiac conduction delays (eg, QRS >100 ms) • Hypotension • Agitation • Sedation • Seizures • Coma • Dry, flushed skin • Decreased GI motility • Urinary retention • Metabolic acidosis	*Prevention of absorption:* • Syrup of ipecac contraindicated because of the rapid onset of sedation or seizures • Gastric lavage • Activated charcoal • Cathartic *Laboratory:* • Serum TCA levels not clinically useful in managing overdoses • Urine drug screen for TCAs • Serial electrolytes and ABGs as indicated *Treatment:* • Prepare for rapid onset of cardiovascular collapse • Seizures may be treated initially with IV benzodiazepines (diazepam, lorazepam) and, if necessary, phenobarbital.

(Continued on page 1272)

Table 56-3 Common Patient Care in Poisonings and Overdoses (continued)

Drug/Substance	Clinical Presentation and Assessment	Intervention
Tricyclic Antidepressants (TCA)		
		• Ventricular dysrhythmias may initially be controlled with systemic alkalinization (keeping blood pH +7.45–7.55 using IV boluses of sodium bicarbonate or intubation and hyperventilation); ventricular dysrhythmias not controlled with systemic alkalinization may be controlled with lidocaine or bretylium (Bretylol); do not use procainamide (Pronestyl) or quinidine due to effects on cardiac conduction similar to those of TCAs. • Cardiac conduction delays (eg, QRS > 100 ms) also are treated with systemic alkalinization as outlined in previous point; conduction delays not responsive to systemic alkalinization may be treated with phenytoin • Hypotension may be addressed initially with Trendelenburg position and IV fluids; if necessary, follow with dopamine infusion; norepinephrine (Levophed) may be necessary. • Provide supportive care.

ALT, alanine aminotransferase; AST, aspartate aminotransferase; OTC, over the counter.

BOX 56-4 *COLLABORATIVE CARE GUIDE for the Patient With Cocaine Toxicity*

Outcomes	Interventions
Oxygenation/Ventilation	
Arterial blood gases (ABGs) are within normal limits.	• Monitor pulse oximetry and ABGs. • Validate significant changes in pulse oximetry with co-oximetry arterial saturation measurement.
Respiratory rate and depth are within normal limits.	• Monitor every 15 min, then every 1 h. • Prepare for intubation and mechanical ventilation (see Collaborative Care Guide for the Patient on Mechanical Ventilation, p. 538).
Circulation/Perfusion	
Blood pressure and heart rate are within normal limits. Patient is free of dysrhythmias. There is no evidence of myocardial dysfunction, such as altered electrocardiogram (ECG) or cardiac enzymes.	• Monitor vital signs every 15 min then hourly. • Provide continuous ECG monitoring. • Monitor 12-lead ECG daily and PRN. • Monitor cardiac enzymes, magnesium, phosphorus, calcium, and potassium as ordered. • Assess for chest pain. • Monitor ECG for dysrhythmias and changes consistent with evolving myocardial infarction.
Patient is euthermic.	• Assess temperature every 15–30 min, then hourly. • Provide a cool environment, and institute cooling strategies (eg, hypothermia blanket, tepid sponge bath), as indicated.
Fluids/Electrolytes	
Patient's urine output >30 mL/h (or >0.5 mL/kg/h)	• Take intake and output hourly. • Administer fluids and diuretics to maintain intravascular volume and renal function per order.
There is no evidence of electrolyte imbalance or renal dysfunction.	• Monitor electrolytes daily and PRN. • Replace electrolytes as ordered. • Monitor blood urea nitrogen, creatinine, serum osmolality, and urine electrolytes daily.

(Continued on page 1273)

BOX 56-4 COLLABORATIVE CARE GUIDE for the Patient With Cocaine Toxicity (continued)

Outcomes	Interventions
Mobility/Safety	
There is no evidence of seizure activity.	• Monitor for seizure activity. • Administer anticonvulsants. • Assess anticonvulsant levels daily if indicated. • Maintain calm, quiet environment.
Patient does not harm self.	• Institute seizure precautions. • Institute fall precautions. • Assess need for physical or chemical restraint to protect from self-injury. • Monitor agitation and administer sedation when appropriate. • Evaluate risk for suicide and take measures to protect patient.
Skin Integrity	
There is no evidence of skin breakdown.	• Document skin integrity every 8 h. • Turn and reposition every 2 h. • Use Braden Scale to assess risk for skin breakdown.
Nutrition	
Caloric and nutrient intake meets metabolic requirements per calculation (eg, basal energy expenditure).	• Provide parenteral or enteral nutrition if patient is NPO. • Consult dietitian or nutritional support service. • Monitor protein and calorie intake. • Monitor albumin, prealbumin, transferrin, cholesterol, triglycerides, and glucose.
Comfort/Pain Control	
Patient will have minimal discomfort related to withdrawal from cocaine and other substances.	• Obtain toxicology screen to identify other substances used by the patient. • Treat drug withdrawal and overdose symptoms promptly and with appropriate intervention (eg, remove from circulation, administer antidote, administer methadone).
Psychosocial	
Patient and family acknowledge substance abuse.	• Assess patient and family response to overdose. • Support healthy coping behaviors. • Consult substance abuse counselor and social worker. • Encourage patient discussion regarding use of illegal drugs, support system, financial concerns, and readiness for substance abuse treatment.
Teaching/Discharge Planning	
Patient and family have information about treatment and self-help resources. Patient and family each have a plan for follow-up care.	• Assess patient and family knowledge and understanding of substance abuse. • Provide literature and explanations to patient and family regarding substance abuse, treatment, relapse, legal issues, and self-help groups. • Refer family to self-help resources. • If patient agrees, initiate referral for substance abuse rehabilitation. • Coordinate referral with patient, family, and social worker to address other possible issues (eg, housing, financial issues, long-term care planning).

teaching. All patients (and parents of pediatric patients) who have survived a toxic encounter should be taught how to prevent such an incident from recurring. Parents of young children need information on child-proofing their home. Family teaching guidelines for lead poisoning are included in Box 56-5. Finally, a summary of poison prevention for the older patient can be found in Box 56-6.

In addition, carbon monoxide detectors alert families to problems in their homes. Utility companies and local health and fire authorities can help identify and remove sources of fumes.

BOX 56-5 TEACHING GUIDE Lead Poisoning

- Lead is commonly found in old homes, in paint, plumbing, and dinnerware.
- Lead is excreted more slowly than it is absorbed, leading to a buildup of lead in the body.
- Accumulation of lead in high levels is frequently missed through lack of blood lead level screening, and not detected until effects such as learning disabilities are diagnosed.
- Children can be tested for lead by their health care providers.
- The local health department can provide lead poisoning treatment and information about lead abatement programs.

BOX 56-6 CONSIDERATIONS FOR THE OLDER PATIENT

Accidental Poisoning

- Poison control centers receive many calls from or related to older adults regarding accidental poisonings.
- Telephone numbers for the health care provider and the poison control center should be in a readily accessible place.
- The older population uses more medicine than any other age group.
- Older people may be more susceptible to the effects of drugs.
- When questions arise concerning drugs, a responsible adult should not hesitate to call a health care provider.
- The patient should not change the dose or discontinue taking prescription drugs without first consulting the physician or nurse.
- Avoid doubling medication when a pill is forgotten. The patient should seek the advice of his or her physician, nurse, or pharmacist.
- Medications and alcohol should not be mixed without first checking with the pharmacist for possible interactions.
- The pharmacist can provide large-print labels.
- A medication calendar or diary will help the older person keep track of the dosing schedule.
- Pill dispensers are helpful for the patient who has to take a variety of pills or who has trouble remembering the prescribed schedule.
- When a medication is discontinued, the remaining medication should be discarded with regard for the environment; for example, it may be returned to a pharmacy for responsible disposal.

▲ Clinical Applicability Challenges

CASE STUDY

D.L. is a 40-year-old, 143-pound (65-kg) woman who is brought to the emergency department by her husband. He states that D.L. took 100 acetaminophen 500 mg tablets 1 hour ago as a suicide gesture. She appears alert, with the following vital signs: blood pressure, 132/76 mm Hg; pulse, 94 beats/min; respirations, 18 breaths/min; and oxygen saturation, 99% on room air. Her pupils were equal and reactive to light. Her skin was warm and dry. She had no complaints of pain. The nurse called the poison control center who recommended that the patient be given 50 g of activated charcoal, and a stat acetaminophen blood level be measured with a repeat level measured at 4 hours postingestion along with liver function tests. The patient drank the charcoal with no complaints of nausea or vomiting, and the initial acetaminophen level came back at 259 mcg/mL. The 4-hour postingestion acetaminophen level was 269 mcg/mL. Initial aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were 28 and 26, respectively, and D.L.'s international normalized ratio (INR) was 1.0. The poison control center recommended that she be given the oral loading dose of *N*-acetylcysteine (NAC) of 140 mg/kg, as she has not had any vomiting or nausea and would likely tolerate the therapy. The patient successfully received her dose of 9,100 mg of the NAC and was admitted to a regular medical bed for further dosing and observation. The poison center recommended that measures of the acetaminophen level, liver function tests, and INR should be repeated 2 hours prior to the end of the second maintenance infusion of the NAC and that the poison center routinely followed up daily to ensure the continuity of care.

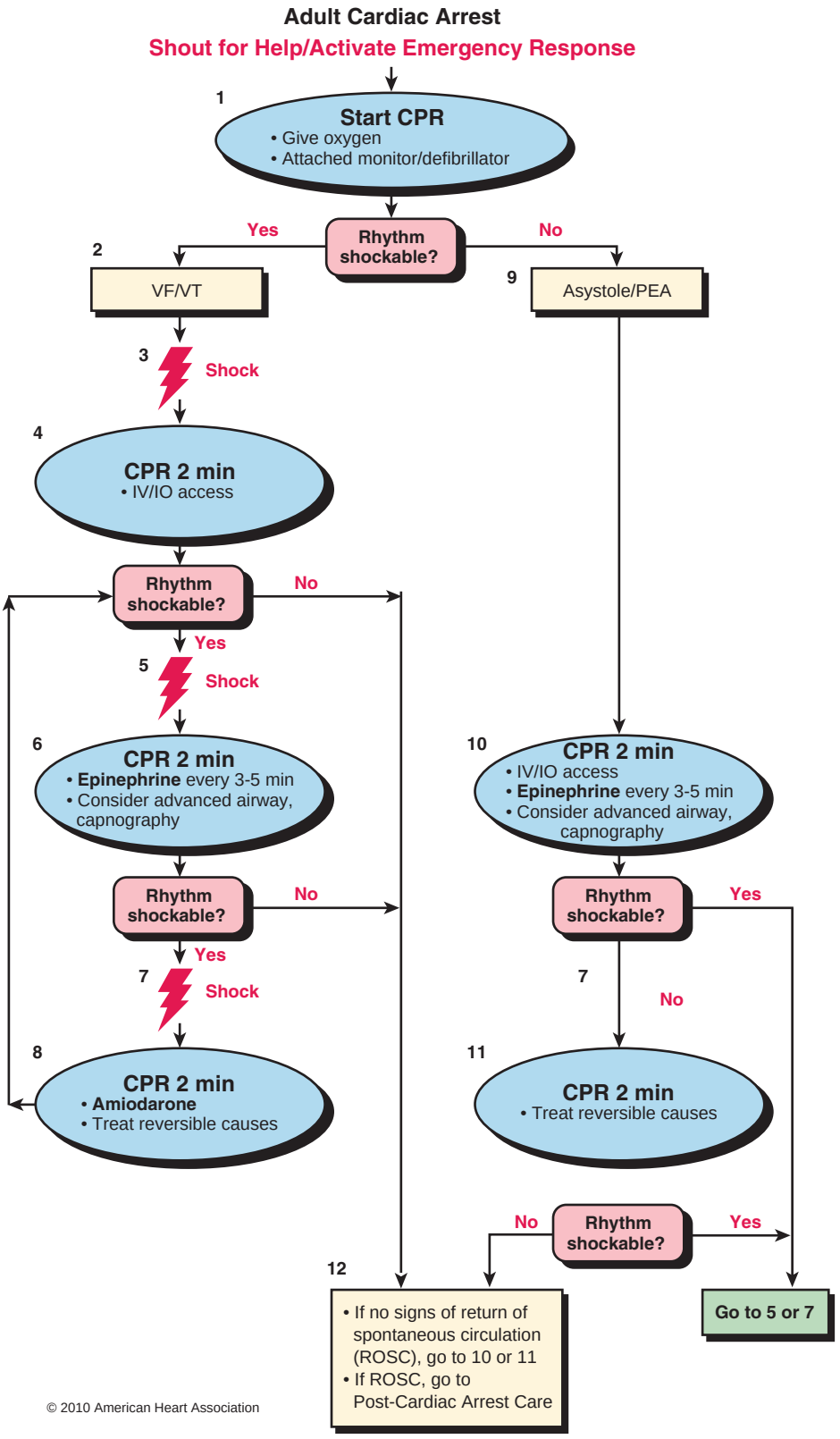
Overnight, the patient continued to receive 70 mg/kg (4,550 mg) doses every 4 hours. Morning laboratory values revealed an acetaminophen concentration of 22 mcg/mL at approximately 16 hours postingestion. The AST/ALT values were 44 and 36, respectively, and the INR was 1.2. The following morning, the patient's acetaminophen level was less than 2 mcg/mL, her AST/ALT values were 40 and 47, her INR was 1.3, and her NAC dosing was discontinued, and she was transferred to the psychiatric floor.

1. What are the primary body systems affected by an acetaminophen overdose? Discuss the effects and the implications for nursing care.
2. Why is the time of ingestion so important in an acetaminophen overdose? Discuss the relationship between the time of ingestion and the measured acetaminophen levels.
3. How would the management change if the patient came in several hours after the overdose?
4. What would the options be if the patient were to have intractable vomiting of the oral doses of NAC? Discuss the differences in risk, benefit, and cost of oral vs. IV NAC.
5. What would be the recommendation if, at the end of her prescribed course of oral NAC, D.L still had a measurable acetaminophen level? Elevated AST/ALT? INR more than 2.0?

References

1. American Association of Poison Control Centers: 2008 Toxic exposure surveillance system poisoning data. Retrieved March 06, 2010, from http://www.aapcc.org
2. Smith SW: Drugs and pharmaceuticals: Management of intoxication and antidotes. EXS 100:397–460, 2010
3. American Academy of Clinical Toxicology, European Association of Poison Centers and Clinical Toxicologists: Position statements. J Toxicol Clin Toxicol 35:699–762, 1997
4. Goldfrank L, Flomenbaum N, Lewin N, et al: Toxicologic Emergencies, 8th ed. New York, NY: McGraw-Hill Medical, 2006, p 617
5. Pepe G, Castelli M, Nazerian P, et al: Delayed neuropsychological sequelae after carbon monoxide poisoning: Predictive risk factors in the Emergency Department. A retrospective study. Scand J Trauma Resusc Emerg Med 19(1):16, 2011
6. Betten DP, Vohra RB, Cook MD, et al: Antidote Use in the Critically Ill Poisoned Patient. J Intensive Care Med 21:255–277, 2006
7. Haddad L, Shannon M, Winchester J: Clinical Management of Poisoning and Drug Overdose, 4th ed. Philadelphia, PA: WB Saunders, 2007, p 438
8. McCord J, Jneid H, Hollander J, et al: Management of cocaine-associated chest pain and myocardial infarction: A scientific statement from the American Heart Association Acute Cardiac Care Committee of the Council on Clinical Cardiology. Circulation 117:1897–1907, 2007
9. Hoffman RS: Treatment of patients with cocaine-induced arrhythmias: bringing the bench to the bedside. Br J Pharmacol 69(5): 448–457, 2010
10. Wright RO, Anderson AC, Lesko SL, et al: Effect of metoclopramide dose on preventing emesis after oral administration of *N*-acetylcysteine for acetaminophen overdose. J Toxicol Clin Toxicol 37:35–42, 1999

ACLS GUIDELINES



CPR Quality
- Push hard (≥2 inches [5 cm]) and fast (≥100/min) and allow complete chest recoil
- Minimize interruptions in compressions
- Avoid excessive ventilation
- Rotate compressor every 2 minutes
- If no advanced airway, 30:2 compression-ventilation ratio
- Quantitative waveform capnography
  – If P_{ETCO_2} <10 mm Hg, attempt to improve CPR quality
- Intra-arterial pressure
  – If relaxation phase (diastolic) pressure <20 mm Hg, attempt to improve CPR quality

Return of Spontaneous Circulation (ROSC)
- Pulse and blood pressure
- Abrupt sustained increase in P_{ETCO_2} (typically ≥40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Shock Energy
- **Biphasic:** Manufacturer recommendation (eg, initial dose of 120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- **Monophasic:** 360 J

Drug Therapy
- **Epinephrine IV/IO Dose:** 1 mg every 3-5 minutes
- **Vasopressin IV/IO Dose:** 40 units can replace first or second dose of epinephrine
- **Amiodarone IV/IO Dose:** First dose: 300 mg bolus. Second dose: 150 mg.

Advanced Airway
- Supraglottic advanced airway or endotracheal intubation
- Wave form capnography to confirm and monitor ET tube placement
- 8-10 breaths per minute with continuous chest compressions

Reversible Causes
– **H**ypovolemia
– **H**ypoxia
– **H**ydrogen ion (acidosis)
– **H**ypo-/hyperkalemia
– **H**ypothermia
– **T**ension pneumothorax
– **T**amponade, cardiac
– **T**oxins
– **T**hrombosis, pulmonary
– **T**hrombosis, coronary

FIGURE A-1 ▲ ACLS cardiac arrest algorithm. (Reprinted with permission. 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, Part 8: Adult Advanced Cardiovascular Life Support. Circulation 122:S729–S767, 2010: © 2010, American Heart Association, Inc.)

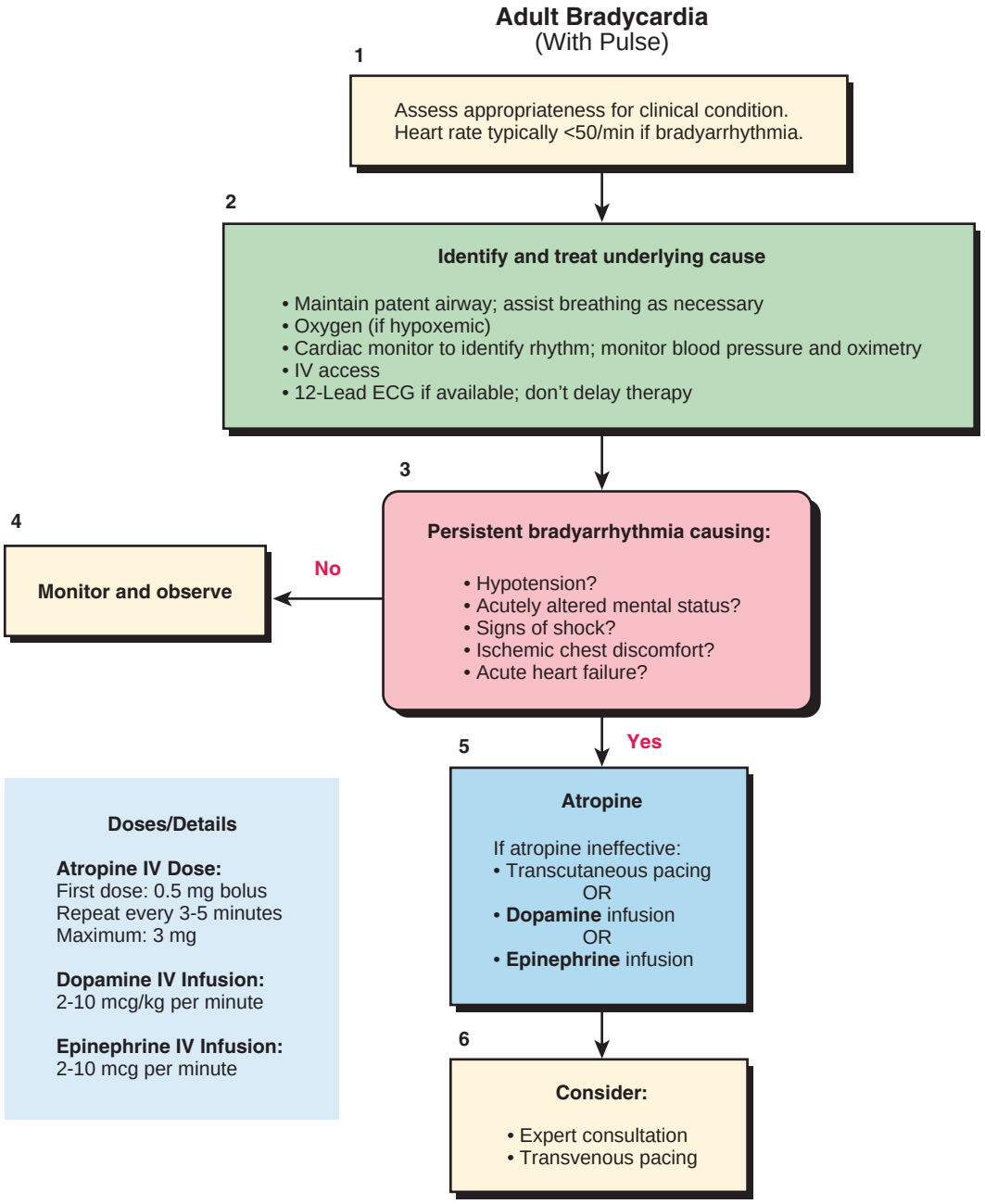


FIGURE A-2 ▲ ACLS bradycardia algorithm. (Reprinted with permission. 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, Part 8: Adult Advanced Cardiovascular Life Support. Circulation 122:S729–S767, 2010: © 2010, American Heart Association, Inc.)

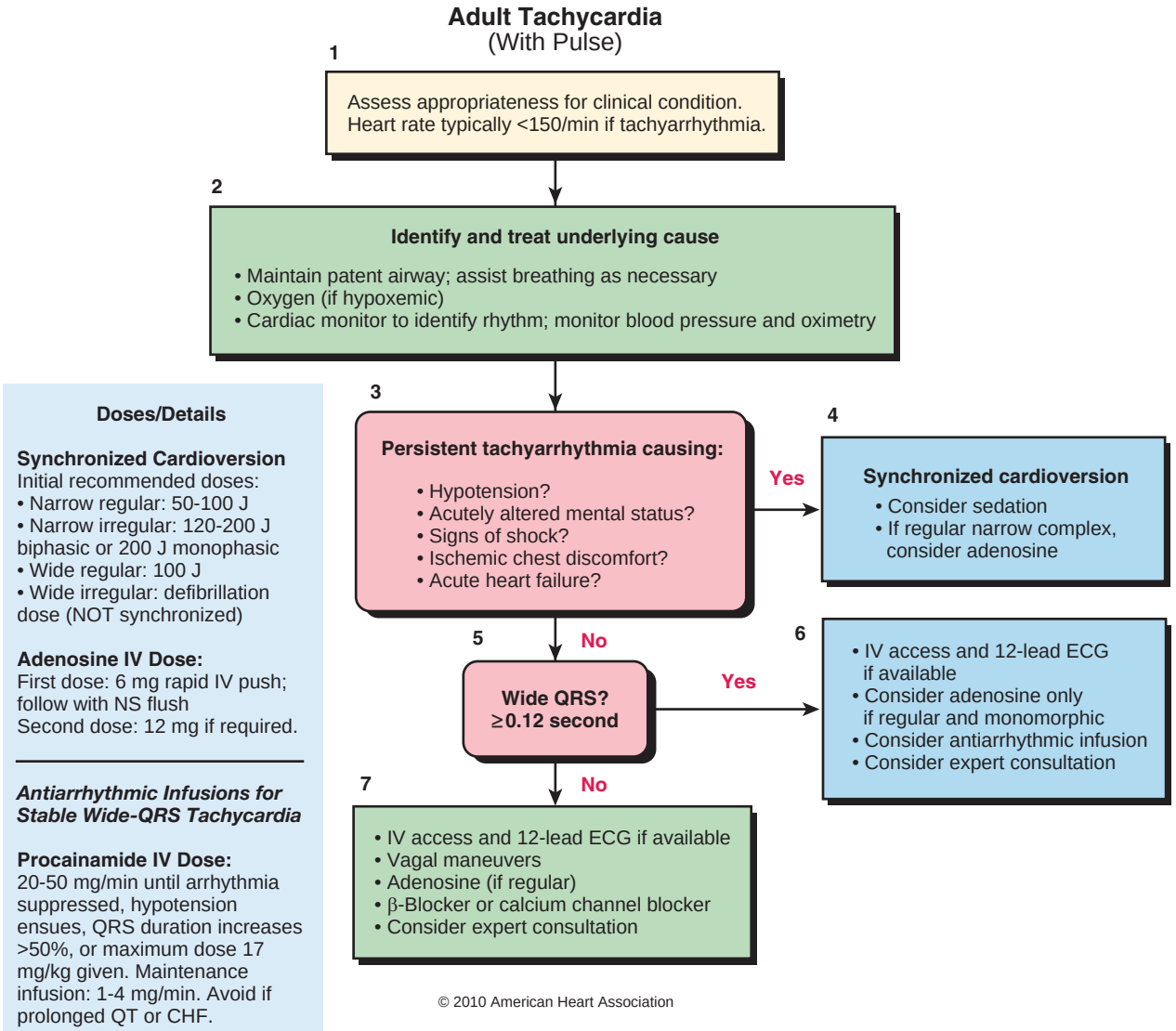


FIGURE A-3 ▲ ACLS tachycardia algorithm. (Reprinted with permission. 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, Part 8: Adult Advanced Cardiovascular Life Support. Circulation 122:S729–S767, 2010: © 2010, American Heart Association, Inc.)

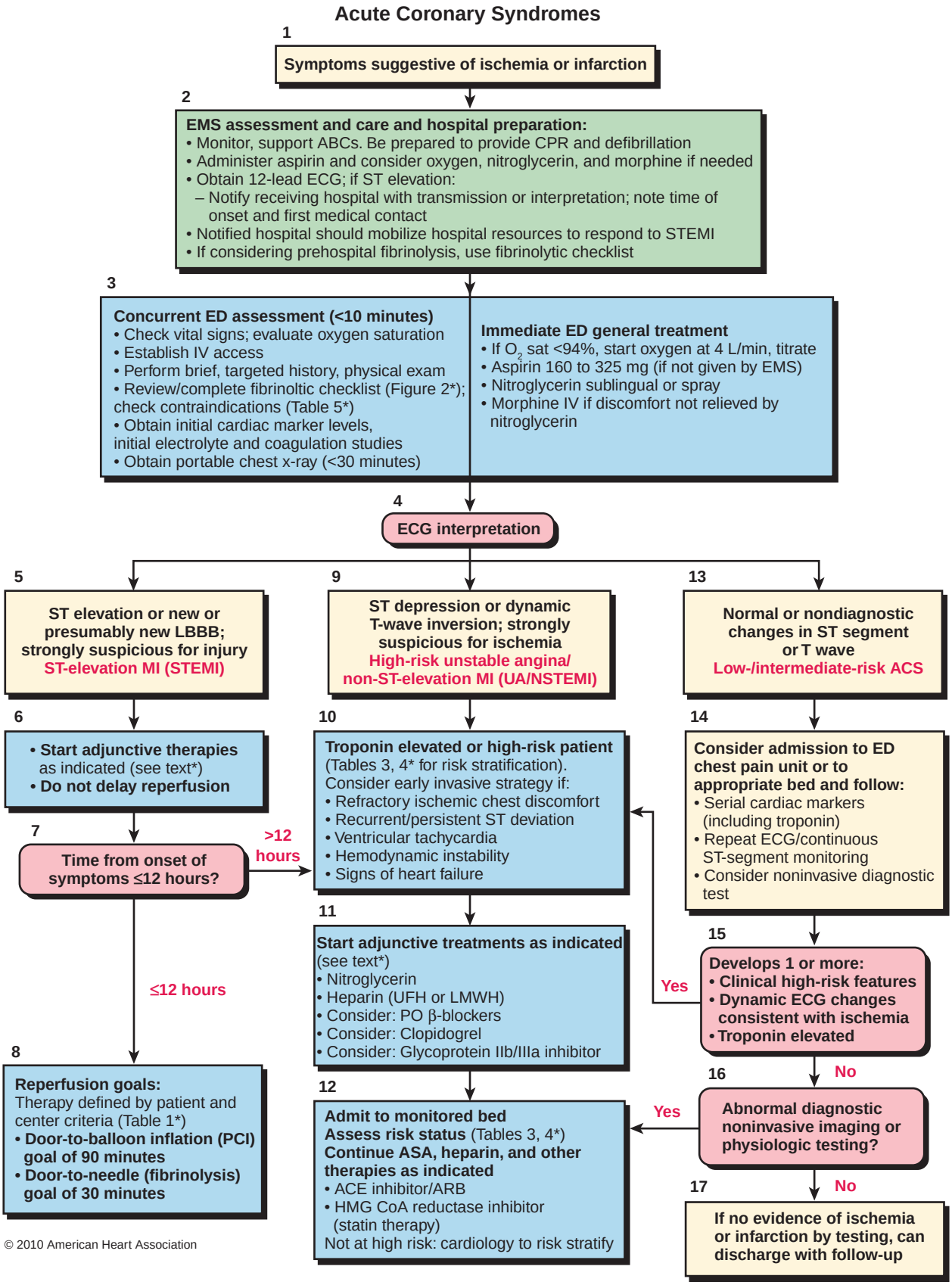


FIGURE A-4 ▲ *Refer to actual guidelines. Acute coronary syndromes algorithm. (Reprinted with permission. 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, Part 10: Acute Coronary Syndromes. Circulation 122:S787–S817, 2010: © 2010, American Heart Association, Inc.)

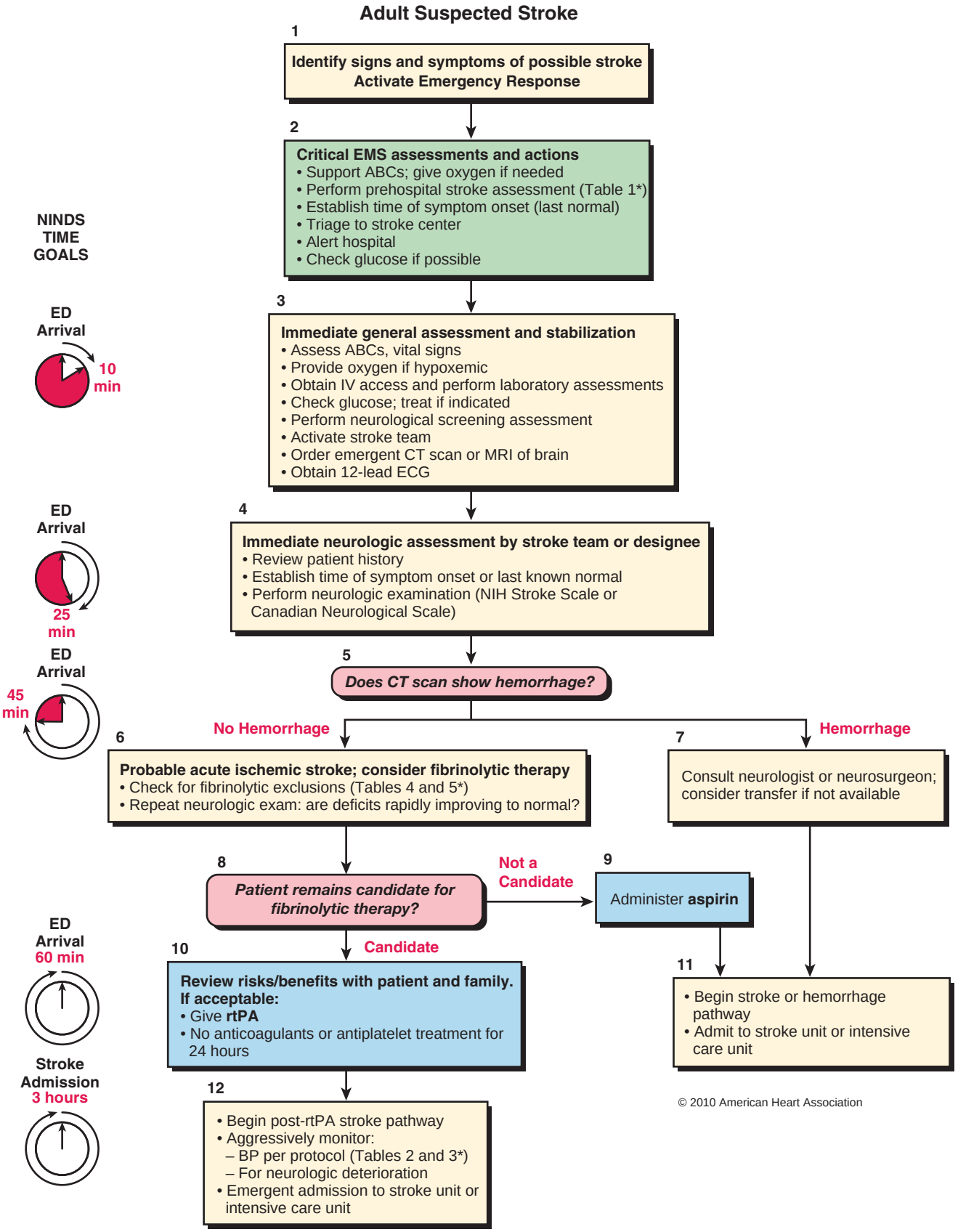


FIGURE A-5 ▲ *Refer to actual guidelines. Goals for management of patients with suspected stroke algorithm. (Reprinted with permission. 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, Part 11: Adult Stroke. Circulation 122:S818–S828, 2010: © 2010, American Heart Association, Inc.)

Index

Note: Page numbers followed by "b" indicate boxed material; those followed by "f" indicate figures; those followed by "t" indicate tables.

B

C

E

F

H

M

N

O

P

S

T

U

V

W

Y

Z