By Kristen J. Overbaugh, MSN, RN, APRN-BC

ACUTE CORONARY SYNDROME

Even nurses outside the ED should recognize its signs and symptoms.

Overview: Acute coronary syndrome (ACS) is the umbrella term for the clinical signs and symptoms of myocardial ischemia: unstable angina, non–ST-segment elevation myocardial infarction, and ST-segment elevation myocardial infarction. This article further defines ACS and the conditions it includes; reviews its risk factors; describes its pathophysiology and associated signs and symptoms; discusses variations in its diagnostic findings, such as cardiac biomarkers and electrocardiographic changes; and outlines treatment approaches, including drug and reperfusion therapies.

Coronary artery disease, in which atherosclerotic plaque builds up inside the coronary arteries and restricts the flow of blood (and therefore the delivery of oxygen) to the heart, continues to be the number-one killer of Americans. One woman or man experiences a coronary artery disease event about every 25 seconds, despite the time and resources spent educating clinicians and the public on its risk factors, symptoms, and treatment. Coronary artery disease can lead to *acute coronary syndrome* (ACS), which describes any condition characterized by signs and symptoms of sudden myocardial ischemia—a sudden reduction in blood flow to the heart. The term *ACS* was adopted because it was believed to more clearly reflect the disease progression associated with myocardial ischemia. Unstable angina and myocardial infarction (MI) both come under the ACS umbrella.

The signs and symptoms of ACS constitute a continuum of intensity from unstable angina to non–ST-segment elevation MI (NSTEMI) to ST-segment elevation MI (STEMI). Unstable angina and NSTEMI normally result from a partially or intermittently occluded coronary artery, whereas STEMI results from a fully occluded coronary artery. (For more, see Table 1.)
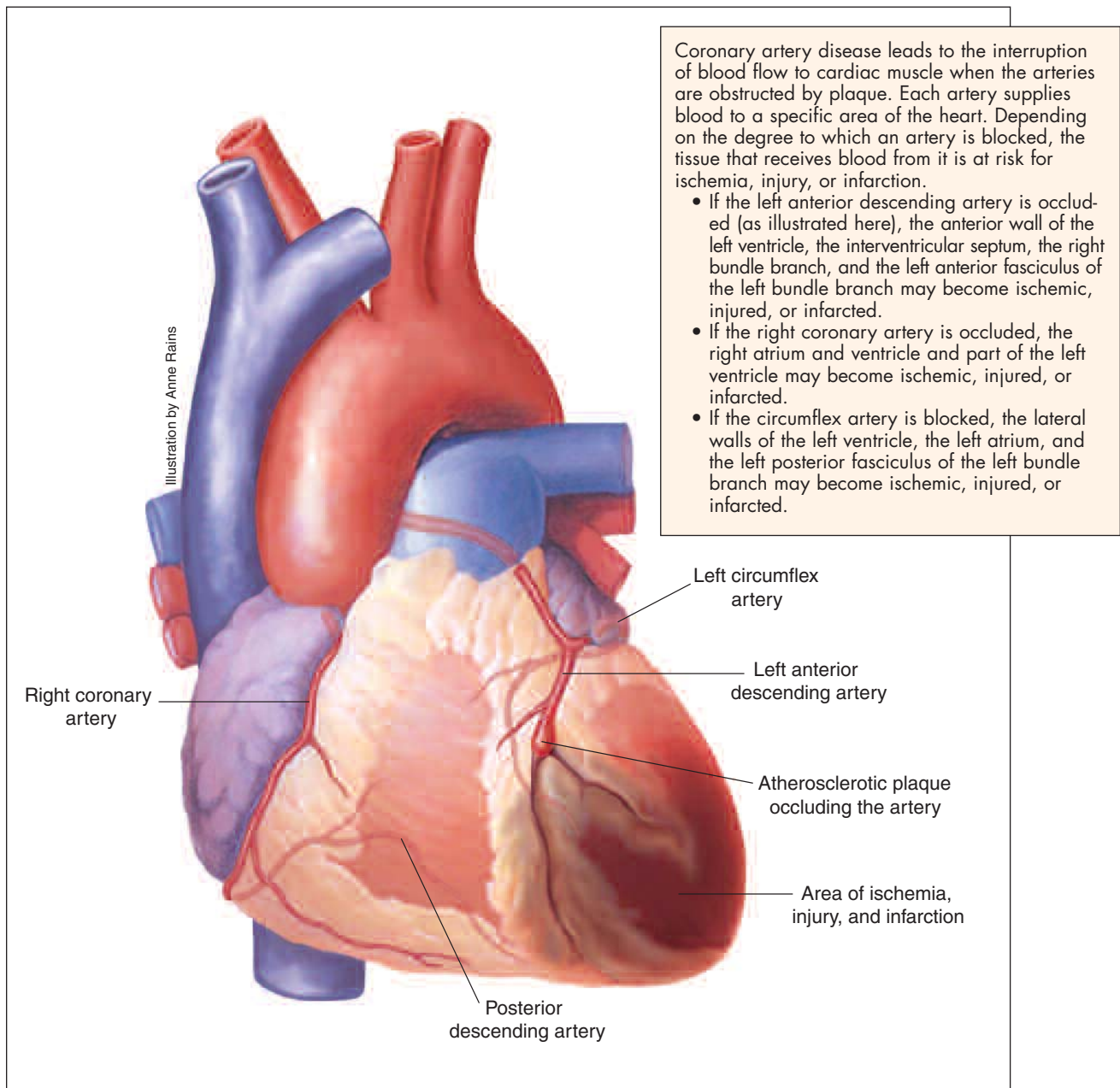
According to the American Heart Association (AHA), 785,000 Americans will have an MI this year, and nearly 500,000 of them will experience another.[1] In 2006 nearly 1.4 million patients were discharged with a primary or secondary diagnosis of ACS, including 537,000 with unstable angina and 810,000 with either NSTEMI or STEMI (some had both unstable angina and MI).[1]

The AHA and the American College of Cardiology (ACC) recently updated practice guidelines and performance measures to help clinicians adhere to a standard of care for all patients who present with symptoms of any of the three stages of ACS.[2-5] Nurses not specializing in the care of patients with cardiovascular disease may not be familiar with current practice guidelines and nomenclature, but they nevertheless play significant roles in detecting patients at risk for ACS, facilitating their diagnosis and treatment, and providing education that can improve outcomes. Many patients admitted with a diagnosis of NSTEMI or unstable angina are cared for by physicians other than cardiologists and are therefore less likely to receive evidence-based care. Nurses caring for these patients can be instrumental in promoting adherence to practice guidelines.

WHO'S AT RISK FOR CORONARY ARTERY DISEASE?

Nonmodifiable factors that influence risk for coronary artery disease include age, sex, family history, and ethnicity or race. Men have a higher risk than

Figure 1. The Coronary Arteries and Ischemia

Illustration by Anne Rains

Coronary artery disease leads to the interruption of blood flow to cardiac muscle when the arteries are obstructed by plaque. Each artery supplies blood to a specific area of the heart. Depending on the degree to which an artery is blocked, the tissue that receives blood from it is at risk for ischemia, injury, or infarction.
- If the left anterior descending artery is occluded (as illustrated here), the anterior wall of the left ventricle, the interventricular septum, the right bundle branch, and the left anterior fasciculus of the left bundle branch may become ischemic, injured, or infarcted.
- If the right coronary artery is occluded, the right atrium and ventricle and part of the left ventricle may become ischemic, injured, or infarcted.
- If the circumflex artery is blocked, the lateral walls of the left ventricle, the left atrium, and the left posterior fasciculus of the left bundle branch may become ischemic, injured, or infarcted.

Left circumflex artery

Left anterior descending artery

Right coronary artery

Atherosclerotic plaque occluding the artery

Area of ischemia, injury, and infarction

Posterior descending artery

women. Men older than age 45, women older than age 55, and anyone with a first-degree male or female relative who developed coronary artery disease before age 55 or 65, respectively, are also at increased risk. Modifiable risk factors include elevated levels of serum cholesterol, low-density lipoprotein cholesterol, and triglycerides; lower levels of high-density lipoprotein cholesterol; and the presence of type 2 diabetes, cigarette smoking, obesity, a sedentary lifestyle, hypertension, and stress.

PATHOPHYSIOLOGY OF ACS
ACS begins when a disrupted atherosclerotic plaque in a coronary artery stimulates platelet aggregation and thrombus formation. It's the *thrombus* occluding the vessel that prevents myocardial perfusion (see figure 1). In the past, researchers supposed that the narrowing of the coronary artery in response to thickening plaque was primarily responsible for the decreased blood flow that leads to ischemia, but more recent data suggest that it's the rupture of an

unstable, vulnerable plaque with its associated inflammatory changes—or as Hansson puts it in a review article in the *New England Journal of Medicine,* "most cases of infarction are due to the formation of an occluding thrombus on the surface of the plaque."[6]

Myocardial cells require oxygen and adenosine 5β-triphosphate (ATP) to maintain the contractility and electrical stability needed for normal conduction. As myocardial cells are deprived of oxygen and anaerobic metabolism of glycogen takes

Angina continues to be recognized as the classic symptom of ACS. Chest pain associated with NSTEMI is normally longer induration and more severe than chest pain associated with unstable angina.

over, less ATP is produced, leading to failure of the sodium–potassium and calcium pumps and an accumulation of hydrogen ions and lactate, resulting in acidosis. At this point, infarction—cell death—will occur unless interventions are begun that limit or reverse the ischemia and injury. During the *ischemic phase,* cells exhibit both aerobic and anaerobic metabolism. If myocardial perfusion continues to decrease, aerobic metabolism ceases and eventually anaerobic metabolism will be significantly reduced. This period is known as the *injury phase.* If perfusion is not restored within about 20 minutes, myocardial necrosis results and the damage is irreversible. Impaired myocardial contractility, the result of scar tissue replacing healthy tissue in the damaged area, decreases cardiac output, limiting perfusion to vital organs and peripheral tissue and ultimately contributing to signs and symptoms of shock. Clinical manifestations include changes in level of consciousness; cyanosis; cool, clammy skin; hypotension; tachycardia; and decreased urine output.[7] Patients who have experienced an MI are therefore at risk for developing cardiogenic shock.

In an attempt to support vital functions, the sympathetic nervous system responds to ischemic changes in the myocardium. Initially, both cardiac output and blood pressure decrease, stimulating the release of the hormones epinephrine and norepinephrine, which in the body's attempt to compensate increase the heart rate, blood pressure, and

afterload, ultimately increasing myocardial demand for oxygen. As oxygen demand increases at the same time that its supply to the heart muscle decreases, ischemic tissue can become necrotic. Low cardiac output also leads to decreased renal perfusion, which in turn stimulates the release of renin and angiotensin, resulting in further vasoconstriction. Additionally, the release of aldosterone and antidiuretic hormone promotes sodium and water reabsorption, increasing preload and ultimately the workload of the myocardium.[8]
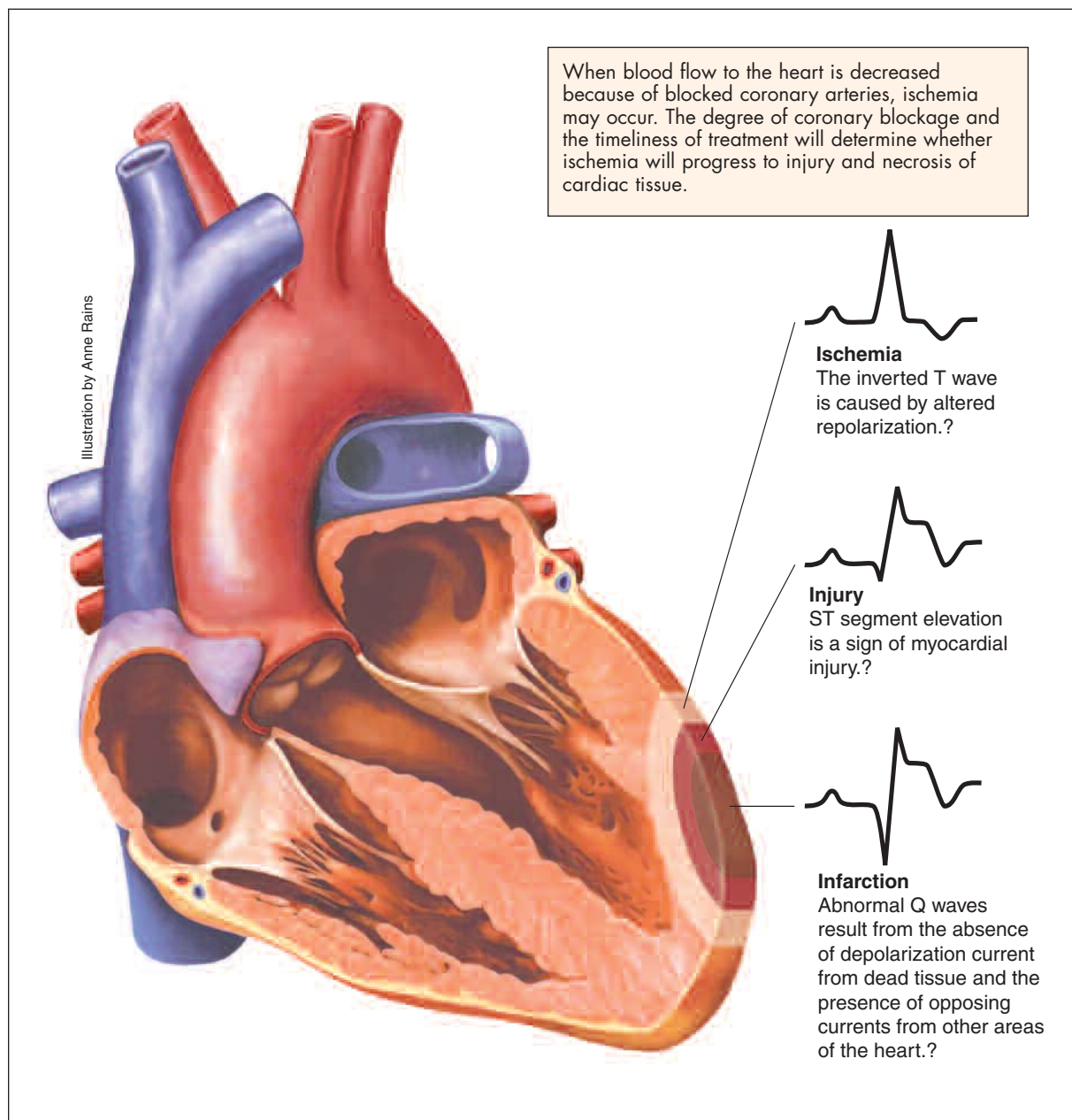
Mastering the concepts of preload and afterload will guide the nurse in understanding the pharmacologic management of ACS. *Preload,* the blood volume or pressure in the ventricle at the end of diastole, increases the amount of blood that's pumped from the left ventricle (the stroke volume). Ischemia decreases the ability of the myocardium to contract efficiently; therefore, in a patient with ACS an increase in preload hastens the strain on an already oxygen-deprived myocardium, further decreasing cardiac output and predisposing the patient to heart failure. As I'll describe in further detail below, medications such as nitroglycerin, morphine, and β-blockers act to decrease preload. These medications, along with angiotensin-converting enzyme (ACE) inhibitors, also decrease *afterload,* which is the force the left ventricle has to work against to eject blood.[9] In myocardial ischemia, the weakened myocardium cannot keep up with the additional pressure exerted by an increase in afterload.

SIGNS AND SYMPTOMS

The degree to which a coronary artery is occluded typically correlates with presenting symptoms and with variations in cardiac markers and electrocardiographic findings. Angina, or chest pain, continues to be recognized as the classic symptom of ACS. In unstable angina, chest pain normally occurs either at rest or with exertion and results in limited activity. Chest pain associated with NSTEMI is normally longer in duration and more severe than chest pain associated with unstable angina. In both conditions, the frequency and intensity of pain can increase if not resolved with rest, nitroglycerin, or both and may last longer than 15 minutes. Pain may occur with or without radiation to the arm, neck, back, or epigastric area. In addition to angina, patients with ACS also present with shortness of breath, diaphoresis, nausea, and lightheadedness. Changes in vital signs, such as tachycardia, tachypnea, hypertension, or hypotension, and decreased oxygen saturation (SaO_2) or cardiac rhythm abnormalities may also be present.[2]

Atypical ACS symptoms. Many women present with atypical symptoms, resulting in delayed diagnosis and treatment.[10] Women frequently experience shortness of breath, fatigue, lethargy, indigestion,

Figure 2. Acute Coronary Syndrome: From Ischemia to Necrosis

When blood flow to the heart is decreased because of blocked coronary arteries, ischemia may occur. The degree of coronary blockage and the timeliness of treatment will determine whether ischemia will progress to injury and necrosis of cardiac tissue.

Ischemia
The inverted T wave is caused by altered repolarization.?

Injury
ST segment elevation is a sign of myocardial injury.?

Infarction
Abnormal Q waves result from the absence of depolarization current from dead tissue and the presence of opposing currents from other areas of the heart.?

Illustration by Anne Rains

and anxiety prior to an acute MI and may not attribute those symptoms to heart disease.[11] It's also important for clinicians to realize that women tend to experience pain in the back rather than substernally or in the left side of the chest and do not characterize it as pain, but may instead report a numb, tingling, burning, or stabbing sensation[12]; in fact, a recent study found that, when compared with men, women diagnosed with ACS more often reported indigestion, palpitations, nausea, numbness in the hands, and atypical fatigue than chest pain.[13]

Silent ischemia. Ischemia can also occur without any obvious signs or symptoms. The classic Framingham Heart Study was initiated in 1948 to explore contributing factors for cardiovascular disease and has provided the scientific community with much of what is known today about heart disease (for more information, visit www.framinghamheartstudy.org). Findings from this longitudinal study of 5,209 participants found that 50% of patients diagnosed with an MI experienced silent ischemia and did not exhibit any of the classic symptoms of ACS.[3] Populations more likely to experience a silent MI include people with diabetes, women, older adults, and those with a history of heart failure.[3] As the prevalence of diabetes rises, silent ischemia may also become more common.

Table 1. Unstable Angina, NSTEMI, and STEMI: How They Differ

Unstable angina, non–ST-segment myocardial infarction (NSTEMI), and ST-segment myocardial infarction (STEMI) differ with regard to duration, severity, and treatments, yet those differences can be difficult to remember. Here they are presented side by side. Look for the highlighted areas to see where they differ from one another.

Unstable Angina

Cause
- Thrombus partially or intermittently occludes the coronary artery

Signs and Symptoms
- Pain with or without radiation to arm, neck, back, or epigastric region
- Shortness of breath, diaphoresis, nausea, lightheadedness, tachycardia, tachypnea, hypotension or hypertension, decreased arterial oxygen saturation (SaO_2) and rhythm abnormalities
- Occurs at rest or with exertion; limits activity

Diagnostic Findings
- ST-segment depression or T-wave inversion on electrocardiography
- Cardiac biomarkers not elevated

Treatment
- Oxygen to maintain oxygen saturation level at > 90%
- Nitroglycerin or morphine to control pain
- β-blockers, angiotensin-converting enzyme inhibitors, statins (started on admission and continued long term), clopidogrel (Plavix), unfractionated heparin or low-molecular-weight heparin, and glycoprotein IIb/IIIa inhibitors

Non–ST-Segment Elevation Myocardial Infarction (NSTEMI)

Cause
- Thrombus partially or intermittently occludes the coronary artery

Signs and Symptoms
- Pain with or without radiation to arm, neck, back, or epigastric region
- Shortness of breath, diaphoresis, nausea, lightheadedness, tachycardia, tachypnea, hypotension or hypertension, decreased arterial oxygen saturation (SaO_2) and rhythm abnormalities
- Occurs at rest or with exertion; limits activity
- Longer in duration and more severe than in unstable angina

Diagnostic Findings
- ST-segment depression or T-wave inversion on electrocardiography
- Cardiac biomarkers are elevated

Treatment
- Oxygen to maintain SaO_2 level at > 90%
- Nitroglycerin or morphine to control pain
- β-blockers, angiotensin-converting enzyme inhibitors, statins (started on admission and continued long term), clopidogrel (Plavix), unfractionated heparin or low-molecular-weight heparin, and glycoprotein IIb/IIIa inhibitors
- Cardiac catheterization and possible percutaneous coronary intervention for patients with ongoing chest pain, hemodynamic instability, or increased risk of worsening clinical condition

Anderson JL, et al. *Circulation* 2007;116(7):e148-e304; Hazinski MF, et al., editors. *Handbook of emergency cardiovascular care for healthcare providers.* Dallas: American Heart Association; 2008.

DIAGNOSING ACS

The patient's clinical history, presenting symptoms, biomarker levels, and electrocardiographic results are all evaluated.

Cardiac biomarkers. Injured myocardial cells release proteins and enzymes known as cardiac biomarkers into the blood. These markers help practitioners determine whether the patient is having or has recently had an acute MI (either an NSTEMI or a STEMI). The utility of various biomarkers is determined by the timing and duration of their elevation as well as by the extent of their cardiac speci-

ST-Segment Elevation Myocardial Infarction (STEMI)

Cause

- Thrombus fully occludes the coronary artery

Signs and Symptoms

- Pain with or without radiation to arm, neck, back, or epigastric region
- Shortness of breath, diaphoresis, nausea, lightheadedness, tachycardia, tachypnea, hypotension or hypertension, decreased arterial oxygen saturation (SaO_2), and rhythm abnormalities
- Occurs at rest or with exertion; limits activity
- Longer in duration and more severe than in unstable angina (irreversible tissue damage [infarction] occurs if perfusion is not restored)

Diagnostic Findings

- ST-segment elevation or new left bundle branch block on electrocardiography
- Cardiac biomarkers are elevated

Treatment

- Oxygen to maintain SaO_2 level at > 90%
- Nitroglycerin or morphine to control pain

- β-blockers, angiotensin-converting enzyme inhibitors, statins (started on admission and continued long term), clopidogrel (Plavix), unfractionated heparin or low-molecular-weight heparin
- Percutaneous coronary intervention within 90 minutes of medical evaluation
- Fibrinolytic therapy within 30 minutes of medical evaluation

Levels of troponins I and T increase within four to six hours of myocardial injury; troponin I levels remain elevated for four to seven days, and troponin T levels remain elevated for 10 to 14 days. Normal reference ranges for cardiac biomarkers vary among laboratories; in order to diagnose myocardial necrosis a single troponin elevation greater than the 99th percentile of an agreed-upon reference control group is required.[14]

Cardiac troponins are the preferred biomarkers for diagnosing acute MI because elevated levels correlate with a more accurate diagnosis, predict a high risk of future cardiac events even when levels of the myocardium-specific biomarker creatine kinase-MB (CK-MB) are normal or only mildly ele-

Nurses can use the mnemonic 'MONA' to recall initial treatment strategies

vated, and elicit fewer false positives when concurrent skeletal muscle injury is present (after trauma or surgery, for example). But if a laboratory is unable to process troponins, CK-MB is considered a reasonable alternative. CK-MB is a cardiac-specific enzyme that's released within four to six hours of injury and remains elevated for 48 to 72 hours after injury. Two consecutive levels of CK-MB greater than the 99th percentile of a reference control group contribute to the diagnosis of acute MI.[14]

Myoglobin, a heme protein, is not cardiac specific, yet it's still considered a valuable biomarker because it's the first to rise after myocardial damage. If a patient presents with ACS symptoms that started less than three hours earlier, CK-MB and troponin levels may not yet be elevated. In such a case, myoglobin can rule out or lead to an early diagnosis of acute MI and prompt decisive therapy.[14]

Electrocardiographic findings. The AHA and the ACC recommend that a 12-lead electrocardiogram (ECG) be performed in patients with symptoms consistent with ACS and interpreted by an experienced physician within 10 minutes of ED arrival.[2] Findings on a 12-lead ECG help the practitioner to differentiate between myocardial ischemia, injury, and infarction; locate the affected area; and assess related conduction abnormalities. Electrocardiographic findings reflective of unstable angina or NSTEMI include ST-segment depression and inverted T waves. ST depression will normally resolve when the ischemia or pain has resolved, although T-wave inversion may persist. Providers should review electrocardiographic findings as well as levels of cardiac biomarkers to dis-

ficity. The cardiac troponins, troponin T and troponin I, are the most cardiac-specific biomarkers. These structural proteins are not normally found in serum; therefore elevated serum levels may predict the degree of thrombus formation and microvascular embolization associated with coronary lesions.

tinguish between unstable angina and NSTEMI.[2] On the other hand, ST elevation on a 12-lead ECG in two contiguous leads is diagnostic of STEMI. With STEMI, T-wave inversion may also be present. These changes normally subside within hours of an MI. Abnormal Q waves appear on an ECG in the presence of an MI as a result of alterations in electrical conductivity of the infarcted myocardial cells. Once an abnormal Q wave has developed it usually remains permanently on the ECG. Therefore, an abnormal Q wave on an ECG does not necessarily signal a current acute MI, but could indicate an old MI.[15] (See Figure 2.)

DRUG THERAPY

Initial drug therapy for patients presenting with angina includes aspirin, oxygen, nitroglycerin, and morphine sulfate (see Tables 2 and 3). Nurses can use the mnemonic "MONA" to recall these initial treatment strategies (although MONA doesn't specify the correct order).

Patients should be given 162 to 325 mg of *aspirin* by mouth (crushed or chewed) as soon as possible after symptom onset, unless contraindicated. Aspirin inhibits platelet aggregation and vasoconstriction by inhibiting the production of thromboxane A2.[16] Aspirin is contraindicated in patients with active peptic ulcer disease, bleeding disorders, and an allergy to aspirin.

Oxygen should be administered at 2 to 4 L/min by nasal cannula to maintain an SaO_2 level greater than 90%.[16] Nurses should be alert for signs of hypoxemia, such as confusion, agitation, restlessness, pallor, and changes in skin temperature. By increasing the amount of oxygen delivered to the myocardium, supplemental oxygen will decrease the pain associated with myocardial ischemia.

Nitroglycerin tablets (0.3 to 0.4 mg) should be administered sublingually every five minutes, up to three doses. If there's no relief after the first dose and the patient is experiencing chest pain and is not in an acute care facility, 911 should be called.[2]

Nitroglycerin causes venous and arterial dilation, which reduces both preload and afterload and ultimately decreases myocardial oxygen demand. It's available in sublingual tablets or spray or can be given intravenously. Because nitroglycerin can cause hypotension, patients should be helped to a bed or into a sitting position before taking it. Nurses must assess for a drop in blood pressure or changes in pain level every five to 10 minutes after administering nitroglycerin. The drug may cause a tingling sensation when administered sublingually. If there is no relief after three oral doses and the physician decides to start an infusion, IV nitroglycerin is started at 10 to 20 micrograms per minute and slowly titrated by 10 micrograms every three to five minutes until the pain is resolved or the patient

becomes hypotensive. The maximum dosage is 200 micrograms per minute.[16] Nitroglycerin is contraindicated in patients who have taken sildenafil (Viagra) in the last 24 hours.

If the patient's pain hasn't improved after administration of nitroglycerin, *morphine sulfate* may be given at an initial dose of a 2-to-4-mg IV push that can be repeated every five to 15 minutes until the pain is controlled.[16] Morphine causes venous and arteriolar vasodilation, reducing both preload and afterload, and the drug's analgesic properties decrease the pain and anxiety associated with ACS. However, morphine can cause hypotension and respiratory depression, so nurses should closely monitor the patient's blood pressure level, respiratory rate, and SaO_2 level for changes.

Adjunctive drug therapy can also be used to improve outcomes in ACS patients. The early use of β-blockers during or after MI is now considered controversial. According to 2008 performance measures jointly written by the ACC and the AHA,

Nurses must assess for a drop in blood pressure or changes in pain level every five to 10 minutes after administering nitroglycerin.

β-blockers decrease rates of reinfarction and death from arrhythmias in NSTEMI and STEMI patients but don't necessarily improve overall mortality rates, especially in patients with heart failure or hemodynamic instability.[5] If no contraindications exist and β-blocker therapy is deemed appropriate, it should be initiated within 24 hours and continued after discharge.[5] Patients started on β-blocker therapy need to be monitored for hypotension, bradycardia, signs of heart failure, hypoglycemia, and bronchospasm.

ACE inhibitors decrease the risks of left-ventricular dysfunction and death in ACS patients and should be administered within 24 hours and continued upon discharge unless contraindicated.[16] ACE inhibitors are also especially beneficial in ACS patients with diabetes. Nurses need to assess for hypotension, decreased urine output, cough, hyperkalemia, and renal insufficiency in patients receiving ACE inhibitors.[17] In patients with an intolerance to ACE inhibitors, angiotensin-receptor blockers can be considered as alternative therapy.[2]

Statins should be prescribed in patients with unsta-

Table 2. Initial Drug Therapy for Acute Coronary Syndrome (ACS)

Drug Therapy	Dosing*	Nursing Considerations
Aspirin	162–325 mg orally, crushed or chewed; then 81–325 mg daily	Contraindicated in active peptic ulcer disease, hepatic disease, bleeding disorders, and aspirin allergy
Oxygen	2–4 L by nasal cannula	Maintain oxygen saturation at > 90%
Nitroglycerin	0.3–0.4 mg sublingual tablets every 5 min (up to 3 doses) or 1–2 sublingual sprays every 5 min (up to 3 times) or 10 µg/min by IV (titrate 10 µg every 3–5 min based on pain and blood pressure assessments)	Assess for pain relief Monitor blood pressure, cease medication if systolic blood pressure < 90 or 100 mmHg
Morphine sulfate	2–4 mg IV push (may repeat every 5–15 min until pain controlled)	Indicated when pain not improved with nitroglycerin Assess for pain relief Monitor blood pressure and respiratory status

* Dosages may vary depending on selected drug.

Anderson JL, et al. *Circulation* 2007;116(7):e148-e304; Gluckman TJ, et al. *JAMA* 2005;293(3):349-57; Hazinski MF, et al., editors. *Handbook of emergency cardiovascular care for healthcare providers.* Dallas: American Heart Association; 2008; Stringer KA, Lopez LM. Myocardial infarction. In: Wells BG, et al., editors. *Pharmacotherapy handbook.* 5th ed. New York: McGraw-Hill; 2003. p. 112-22.

ble angina, NSTEMI, or STEMI whose low-density lipoprotein cholesterol level is above 100 mg/dL.[5] In patients with a diagnosis of NSTEMI or STEMI, a lipid panel should be ordered during hospitalization.

Clopidogrel (Plavix) inhibits platelet aggregation and can be administered to unstable angina and NSTEMI patients with a known allergy to aspirin. Clopidogrel may also be added to aspirin therapy in ACS patients scheduled for diagnostic angiography or in those receiving conservative treatment. Contraindications are similar to those for aspirin therapy, and clopidogrel should not be administered if coronary artery bypass surgery is planned within the next five to seven days because it increases a patient's risk of bleeding.[2]

Glycoprotein IIb/IIIa inhibitors are the antiplatelet agents used in unstable angina and NSTEMI patients who are scheduled for an invasive diagnostic procedure. These drugs bind to the platelet surface integrin glycoprotein IIb/IIIa receptor sites and inhibit the binding of fibrinogen and subsequent platelet aggregation. If a percutaneous coronary intervention (PCI) is planned and can be performed without delay, the glycoprotein IIb/IIIa inhibitor of choice is abciximab (ReoPro).[2] If the PCI is not planned or is delayed, the glycoprotein IIb/IIIa inhibitors eptifibatide (Integrilin) or tirofiban (Aggrastat) are preferred. These agents may also be considered in patients opting for conservative treatment. Glycoprotein IIb/IIIa inhibitors confer the greatest benefits in patients scheduled for PCI who have elevated cardiac troponin levels.[2]

Options for *anticoagulant therapy* in patients with unstable angina or NSTEMI include enoxaparin (Lovenox), unfractionated heparin, bivalirudin (Angiomax), and fondaparinux (Arixtra).[2] These agents are recommended in patients scheduled for diagnostic testing. Enoxaparin or unfractionated heparin is strongly recommended in patients who choose conservative treatment, but fondaparinux is preferred in those at higher risk for bleeding.

Table 3. Adjunctive Drug Therapy for Acute Coronary Syndrome (ACS)

Drug Therapy	Dosing*	Nursing Considerations
β-blockers • metoprolol (Lopressor) • atenolol (Tenormin) • propranolol (Inderal)	Administer oral dose within 24 hours of symptom onset and continue upon discharge	Contraindicated when heart rate < 60 beats per minute, systolic blood pressure < 100 mmHg, and in heart blocks, moderate-to-severe left ventricular failure, pulmonary edema, acute asthma, or reactive airway disease Monitor for hypotension, bradycardia, signs of heart failure, hypoglycemia, and bronchospasm
Angiotensin-converting enzyme inhibitors • enalapril (Vasotec) • captopril (Capoten) • lisinopril (Prinivil, Zestril) • ramipril (Altace)	Administer oral dose within 24 hours of symptom onset and continue upon discharge	Assess for hypotension, decreased urine output, cough, hyperkalemia, and renal insufficiency Contraindicated in renal failure, hyperkalemia, angioedema, and pregnancy Monitor vital signs and blood glucose
Statins • atorvastatin (Lipitor) • pravastatin (Pravachol) • simvastatin (Zocor)	Administer oral dose upon discharge when low-density lipoprotein cholesterol >100 mg/dL	Instruct patients to take at bedtime and limit grapefruit consumption Contraindicated in pregnancy Monitor lipids, liver function, and creatine kinase levels, and assess for myopathy
Clopidogrel (Plavix)	Administer loading dose, followed by 75 mg/day; continue on discharge	Contraindicated in active peptic ulcer disease, bleeding disorder, hepatic disease, or if coronary artery bypass graft surgery is planned within 5–7 days Can be used in patients allergic to aspirin
Glycoprotein IIb/IIIa inhibitors • abciximab (ReoPro) • eptifibatide (Integrilin) • tirofiban (Aggrastat)	*Abciximab (ReoPro)* preferred if PCI is planned and can be performed without delay *eptifibatide (Integrilin)* or *tirofiban (Aggrastat)* preferred if PCI is not planned or is delayed	Contraindicated with active bleeding, bleeding disorder, surgery or trauma within last month, or platelets < 150,000/mm^3 Monitor blood tests for anemia and clotting disorders
Anticoagulation agents • unfractionated heparin • low-molecular-weight heparin • enoxaparin (Lovenox) • fondaparinux (Arixtra) • bivalirudin (Angiomax)	Indicated for unstable angina, NSTEMI, and STEMI	Monitor complete blood count, platelets, bleeding times, blood urea nitrogen, and creatinine levels

* Dosages may vary depending on selected drug.

Anderson JL, et al. *Circulation* 2007;116(7):e148-e304; Gluckman TJ, et al. *JAMA* 2005;293(3):349-57; Hazinski MF, et al., editors. *Handbook of emergency cardiovascular care for healthcare providers.* Dallas: American Heart Association; 2008; Stringer KA, Lopez LM. Myocardial infarction. In: Wells BG, et al., editors. *Pharmacotherapy handbook.* 5th ed. New York: McGraw-Hill; 2003. p. 112-22.

Table 4. Common Fibrinolytic Drugs

Drug	Weight Dependent?	Half-Life	Dosing
Alteplase (Activase)	Yes	4–8 min	IV bolus dose, then 90-min continuous infusion
Reteplase (Retavase)	No	13–16 min	Two rapid IV bolus doses of 10 units each 30 min apart
Tenecteplase (TNKase)	Yes	20–24 min	Single IV bolus dose

Peacock WF, et al. *Am J Emerg Med* 2007;25(3):353-66.

REPERFUSION THERAPY

Reperfusion therapy is recommended in patients diagnosed with STEMI. Reperfusion strategies include a variety of PCIs and fibrinolytic drug therapy. The goal of reperfusion therapy is to restore blood flow to ischemic myocardial tissue and prevent further complications. Reperfusion therapy should be initiated within a defined time frame to improve patient outcomes.[18]

PCI refers to invasive procedures in which a catheter is inserted, normally through the femoral artery, into the occluded coronary artery in order to open blockages and restore blood flow. Percutaneous transluminal coronary angioplasty (PTCA) is the insertion of a catheter with a balloon tip that's inflated to open the artery. A metal mesh device known as a coronary stent can also be inserted after angioplasty to keep the artery open. Drug-eluting stents are coated with medications that prevent restenosis by reducing inflammation and the formation of thrombin. Blockages can also be destroyed in a procedure known as an arthrectomy, in which a mechanical device or rotational technology is used to cut or shave the plaque. Once the artery is opened with PTCA or a coronary stent, radiation is delivered to the lesion (through brachytherapy), which helps prevent narrowing or reocclusion.

PCI is indicated if the onset of ACS symptoms occurred more than three hours earlier, if fibrinolytic therapy is contraindicated, if the patient is at high risk for developing heart failure, or if the STEMI diagnosis is not absolute. PCI should be performed within 90 minutes of medical evaluation. The degree of coronary occlusion and the structure and viability of the affected vessel may exclude candidates from consideration for PCI.[18]

Possible complications include bleeding or hematoma from the arterial insertion site, decreased peripheral perfusion, retroperitoneal bleeding, cardiac arrhythmias, coronary spasm or MI, acute renal failure, stroke, and cardiac arrest. Postprocedure care should include frequent monitoring of vital signs and cardiac rhythm as well as assessment of peripheral pulses, arterial insertion site, pain, and intake and output.

Fibrinolytic therapy refers to the administration of "clot-busting" drugs, which dissolve existing thrombi by converting plasminogen to plasmin and degrading fibrin clots. The drugs most commonly used are alteplase (recombinant tissue–type plasminogen activator [rt-PA]; Activase), reteplase (Retavase), and tenecteplase (TNKase) (see Table 4).

Fibrinolytic therapy is most effective when given within three hours after symptom onset, although benefits have been seen when these drugs were administered up to 12 hours afterward; giving them after 24 hours, however, can be harmful. Fibrinolytic therapy should be initiated within 30 minutes of medical evaluation.[18] Contraindications include bleeding disorder, recent surgery or other invasive procedure, trauma, active peptic ulcer disease, use of anticoagulants, recent ischemic stroke, cerebrovascular disease, uncontrolled hypertension, and brain tumor. Complications include bleeding and hemorrhage.[16-18] The success of reperfusion therapy depends largely on the timeliness of its initiation; nurses who don't work in EDs or on critical care or cardiovascular specialty units need to remain alert to the possibility of ACS in their patients. ▼

For more than 80 additional continuing nursing education articles related to cardiovascular topics, go to www.nursingcenter.com/ce.

Kristen J. Overbaugh is an instructor at Central New Mexico Community College in Albuquerque. The author of this article has disclosed no ties, financial or otherwise, to any company that might have an interest in the publication of this educational activity. Contact author: koverbaugh@cnm.edu.

REFERENCES

1. Lloyd-Jones D, et al. Heart disease and stroke statistics—2009 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation* 2009;119(3):e21-e181.
2. Anderson JL, 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* 2007;116(7):e148-e304.

3. Antman EM, 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* 2004;110(9):e82-e292.

4. Antman EM, et al. 2007 Focused update of the ACC/AHA 2004 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. *Circulation* 2008;117(2):296-329.

5. Krumholz HM, et al. ACC/AHA 2008 performance measures for adults with ST-elevation and non-ST-elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Performance Measures (Writing Committee to develop performance measures for ST-elevation and non-ST-elevation myocardial infarction). *J Am Coll Cardiol* 2008;52(24):2046-99.

6. Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. *N Engl J Med* 2005;352(16):1685-95.

7. Matfin G, Porth CM. Heart failure and circulatory shock. In: Porth CM, editor. *Essentials of pathophysiology: concepts of altered health states*. 2nd ed. Philadelphia: Lippincott Williams and Wilkins; 2007. p. 419-41.

8. Brashers VL. Alterations in cardiovascular function. In: McCance KL, Huether SE, editors. *Pathophysiology: the biologic basis for disease in adults and children*. 4th ed. St. Louis: Mosby; 2002. p. 980-1047.

9. Stewart SL, Vitello-Cicciu JM. Cardiovascular clinical physiology. In: Kinney MR, et al., editors. *AACN's clinical reference for critical care nursing*. 4th ed. St. Louis: Mosby; 1998. p. 249-76.

10. Pilote L, et al. A comprehensive view of sex-specific issues related to cardiovascular disease. *CMAJ* 2007;176(6):S1-S44.

11. Rosenfeld AG. State of the heart: building science to improve women's cardiovascular health. *Am J Crit Care* 2006;15(6):556-67.

12. Ryan CJ, et al. Typical and atypical symptoms: diagnosing acute coronary syndromes accurately. *Am J Nurs* 2005; 105(2):34-6.

13. DeVon HA, et al. Symptoms across the continuum of acute coronary syndromes: differences between women and men. *Am J Crit Care* 2008;17(1):14-25.

14. Morrow DA, et al. National Academy of Clinical Biochemistry laboratory medicine practice guidelines: clinical characteristics and utilization of biochemical markers in acute coronary syndromes. *Circulation* 2007;115(13):e356-e375.

15. Dressler D. Management of patients with coronary vascular disorders. In: Smeltzer SC, et al., editors. *Brunner and Suddarth's textbook of medical–surgical nursing*. 11th ed. Philadelphia: Lippincott Williams and Wilkins; 2008. p. 858-913.

16. Hazinski MF, et al., editors. *Handbook of emergency cardiovascular care for healthcare providers*. Dallas: American Heart Association; 2008.

17. *Springhouse nurse's drug guide 2007*. 8th ed. Philadelphia: Lippincott Williams and Wilkins; 2006.

18. Peacock WF, et al. Reperfusion strategies in the emergency treatment of ST-segment elevation myocardial infarction. *Am J Emerg Med* 2007;25(3):353-66.