Shock, Sepsis, and Multiple Organ Dysfunction Syndrome

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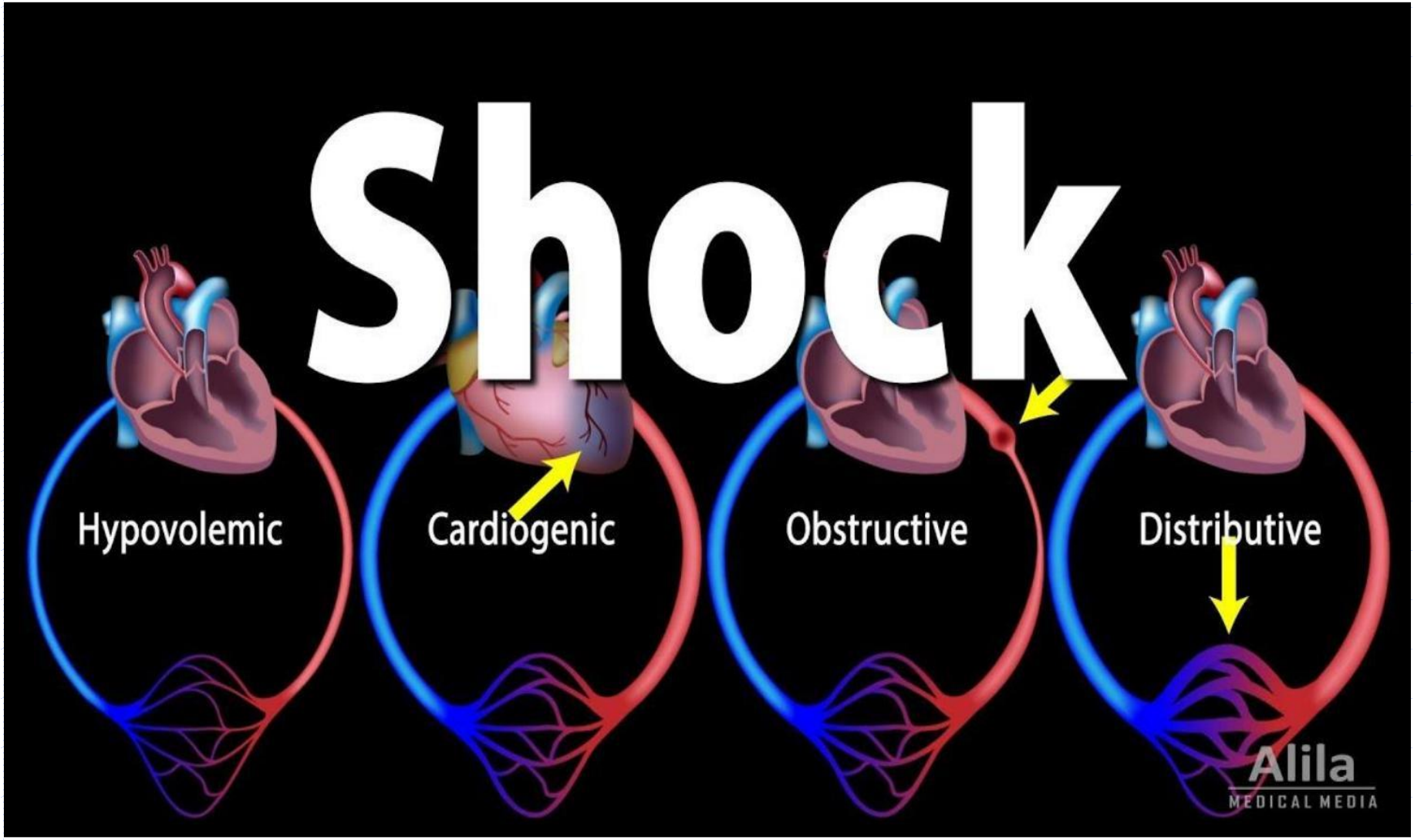
- Shock is a clinical syndrome characterized by inadequate tissue perfusion that results in cellular, metabolic and hemodynamic disturbances.

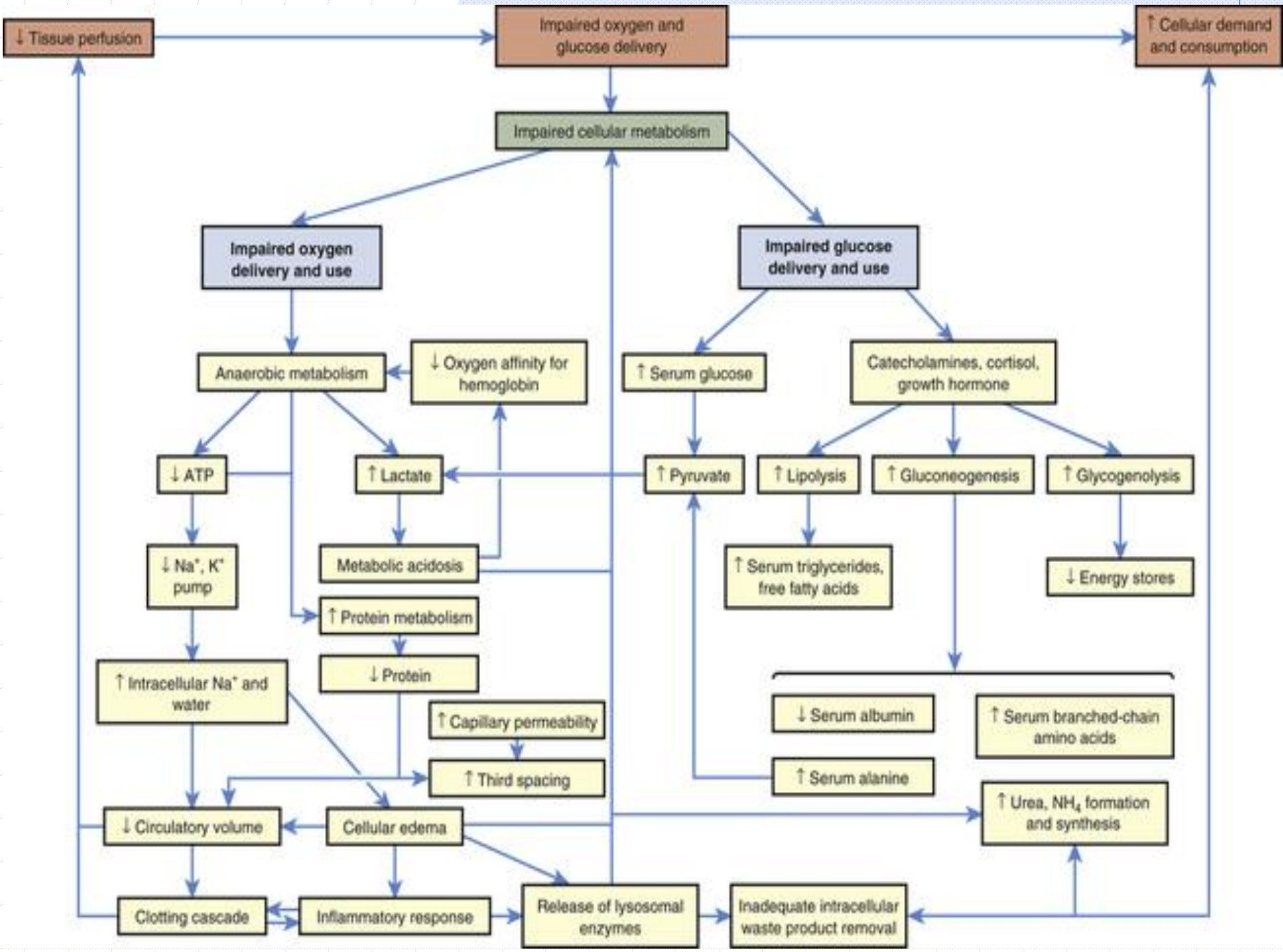
- Impaired tissue perfusion occurs when there is an imbalance between cellular oxygen supply and cellular oxygen demand.

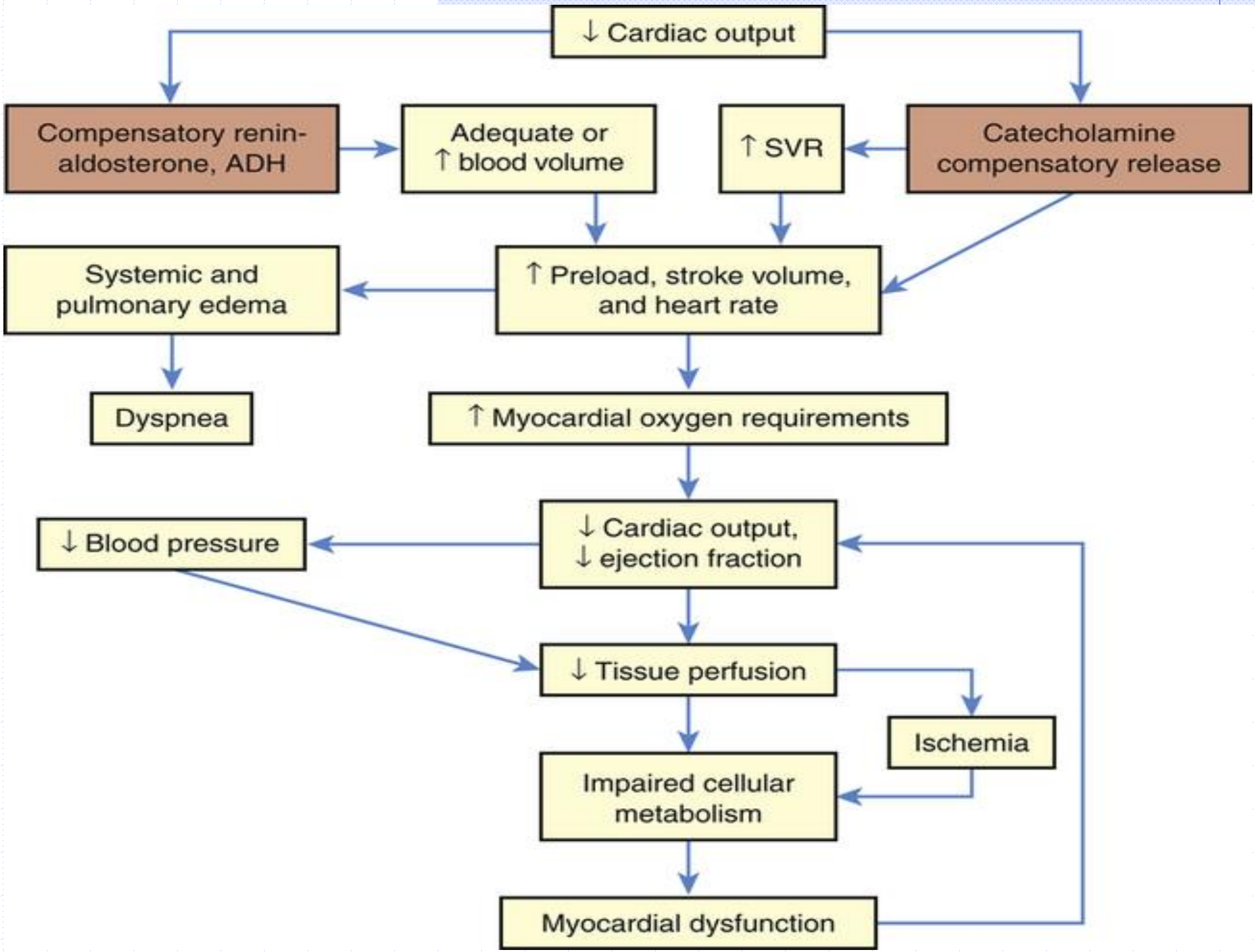
- Shock can result from ineffective cardiac function, inadequate blood volume, or inadequate vascular tone.
- Shock can progress to organ failure and death unless compensatory mechanisms reverse the process, or clinical interventions are successfully implemented.
- Shock frequently results in systemic inflammatory response syndrome (SIRS) and multiple organ dysfunction syndrome (MODS)

Pathology and Pathophysiology of Shock



Marc Imhotep Cray M.D.


↓ Tissue perfusion
Impaired oxygen and glucose delivery
↑ Cellular demand and consumption
Impaired cellular metabolism
Impaired oxygen delivery and use
Impaired glucose delivery and use
Anaerobic metabolism
↓ Oxygen affinity for hemoglobin
↑ Serum glucose
Catecholamines, cortisol, growth hormone
↓ ATP
↑ Lactate
↑ Pyruvate
↑ Lipolysis
↑ Gluconeogenesis
↑ Glycogenolysis
↓ Na⁺, K⁺ pump
Metabolic acidosis
↑ Serum triglycerides, free fatty acids
↓ Energy stores
↑ Protein metabolism
↓ Protein
↑ Intracellular Na⁺ and water
↑ Capillary permeability
↓ Serum albumin
↑ Serum branched-chain amino acids
↑ Third spacing
↑ Serum alanine
↓ Circulatory volume
Cellular edema
↑ Urea, NH4 formation and synthesis
Clotting cascade
Inflammatory response
Release of lysosomal enzymes
Inadequate intracellular waste product removal


↓ Cardiac output
Compensatory renin-aldosterone, ADH
Adequate or ↑ blood volume
↑ SVR
Catecholamine compensatory release
Systemic and pulmonary edema
↑ Preload, stroke volume, and heart rate
Dyspnea
↑ Myocardial oxygen requirements
↓ Blood pressure
↓ Cardiac output, ↓ ejection fraction
↓ Tissue perfusion
Ischemia
Impaired cellular metabolism
Myocardial dysfunction

CLASSIFICATION OF SHOCK

TYPE OF SHOCK	PHYSIOLOGICAL ALTERATION
Hypovolemic	Inadequate intravascular volume
Cardiogenic	Inadequate myocardial contractility
Obstructive	Obstruction of blood flow
Distributive Anaphylactic Neurogenic Septic	Inadequate vascular tone

STAGES OF SHOCK

- I: Initiation
- I: Compensatory
- II: Progressive
- V: Refractory

Initiation

Decrease Tissue oxygenation caused by:

- Decrease Intravascular volume (hypovolemic)
- Myocardial contractility (cardiogenic)
- Obstruction of blood flow (obstructive)
- Decrease Vascular tone (distributive)
- Septic (mediator release)
- Anaphylactic (histamine release)
- Neurogenic (suppression of SNS)

Initiation

CLINICAL PRESENTATION

No observable clinical indications

Decrease CO may be noted with invasive hemodynamic monitoring

Compensatory

Neural compensation by SNS

- Heart rate and contractility
- Vasoconstriction
- Redistribution of blood flow from nonessential to essential organs
- Bronchodilation

Endocrine compensation(RAAS, ADH, glucocorticoids release)

- Renal reabsorption of sodium, chloride, and water
- Vasoconstriction
- Glycogenolysis and gluconeogenesis

Compensatory

CLINICAL PRESENTATION

- Increase Heart rate (except neurogenic)
- Narrowed pulse pressure
- Rapid, deep respirations causing respiratory alkalosis
- Thirst ,Cool, moist skin, Oliguria ,Diminished bowel sounds, Restlessness progressing to confusion and Hyperglycemia
- increase Urine specific gravity and decrease creatinine clearance

Progressive

- Progressive tissue hypoperfusion
- Anaerobic metabolism with lactic acidosis
- Failure of sodium-potassium pump
- Cellular edema

Progressive

CLINICAL PRESENTATION

- Dysrhythmias
- Decrease BP with narrowed pulse pressure
- Tachypnea
- Cold, clammy skin and Anuria
- Absent bowel sounds
- Lethargy progressing to coma
- Hyperglycemia
- Increase BUN, creatinine, and potassium
- Respiratory and metabolic acidosis

Refractory

- Severe tissue hypoxia with ischemia and necrosis
- Worsening acidosis
- SIRS
- MODS

Refractory

CLINICAL PRESENTATION

- Life-threatening dysrhythmias
- Severe hypotension despite vasopressors
- Respiratory and metabolic acidosis
- Acute respiratory failure
- Acute respiratory distress syndrome
- Disseminated intravascular coagulation
- Hepatic dysfunction/failure
- Acute kidney injury
- Myocardial ischemia/infarction/failure
- Cerebral ischemia/infarction

Hypovolemic Shock

- Occurs when the circulating blood volume is inadequate to fill the vascular network.
- Intravascular volume deficits may be caused by external or internal losses of either blood or fluid
  - External plasma losses may be seen in patients with burn injuries who have significant fluid shifts from the intravascular space to the interstitial space

CAUSES

- External loss of blood: GI hemorrhage, surgery ,trauma
- External loss of fluid: Diarrhea, Diuresis Burns
- Internal sequestration of blood fluid Hemothorax ,Dissecting aortic aneurysm ,Femur or pelvic fracture ,Ascites ,Pleural effusion

CLINICAL PRESENTATION

- Increase HR, Decrease BP,Tachypnea
- Decrease CO,CI,RAP,PAP,PAOP,SvO2
- Increase SVR, Hematocrit (dehydration)
- Decrease Hematocrit (blood loss)
- Tachypnea, cool ,pale skin
- Decreased mental status
- Flat neck veins

Clinical Correlates of Hemorrhage

	Class I	ClassII	Class III	Class IV
Blood loss (mL)	> 750	750 - 1500	1500 - 2000	> 2000
Blood loss (% total)	> 15%	15 - 30%	30 - 40%	> 40%
Pulse rate	< 100	> 100	> 120	> 140
Blood pressure	Normal	Normal	↓	↓
Pulse pressure	Normal or ↑	↓	↓	↓
Orthostasis	Absent	Minimal	Marked	Marked
Capillary refill	Normal	Delayed	Delayed	Delayed
Resp rate	14 - 20	20 - 30	30 - 40	> 34
UO (mL/hr)	> 30	20 - 30	5 - 15	< 5
CNS mental status	Slight anxiety	Mild anxiety	Anxious/confused	Confused/lethargic
CI (L/min)	↓ 0-10%	↓ 20-50%	↓ 50-75%	↓ >75%

American College of Surgeons, 1989

MANAGEMENT

- Eliminate and treat the cause
- Replace lost volume with appropriate fluid
- The patient requires a minimum of two IV catheters, one in a peripheral vein and one in a central vein. Peripheral access via a large-gauge catheter (14 or 16 gauge) in a large vein
- Blood, blood products, crystalloids, and colloids are used alone, or in combination, to restore intravascular volume.

Fluid Resuscitation For Hypovolemic Shock

Initial fluid resuscitation consists of isotonic

Crystalloid	Colloid	Whole Blood
0.9 % NaCl	5 % plasmanate or Albumin	
Lactated Ringer's Solution	6 % hetastarch	

Choice of solution is based on

1. *O_2-carrying capacity (e.g., hemoglobin, hematocrit)*
2. *Cause of hypovolemic shock*
3. *Accompanying disease states*
4. *Degree of fluid loss, and*
5. *Required speed of fluid delivery.*

Rate of Fluid Administration

- Patients in shock typically require and tolerate infusion at the maximum rate.
- Adults are given 1 L of crystalloid (20 mL/kg in children) or, in hemorrhagic shock, 5 to 10 mL/kg of colloid or red blood cells, and the patient is reassessed.
- With an infusion pump allow infusion of 1 L of crystalloid in 10 to 15 minutes and 1 unit of red blood cells in 20 minutes
- a pressure infusion device can infuse 1 unit of red blood cells in < 5 minutes.

- The 3-for-1 rule is used which recommends the replacement of 300 mL of isotonic solution for every 100 mL of blood lost.
- Hemodynamic monitoring provides objective data to guide fluid replacement

Cardiogenic shock

- Occur when the heart fails to act as an effective pump. A decrease in myocardial contractility results in decreased cardiac output and impaired tissue perfusion.
- Cardiogenic shock is one of the most difficult types of shock to treat and carries a hospital mortality of 67%

POSSIBLE CAUSES

- Myocardial infarction
- Myocardial contusion
- Cardiomyopathy
- Myocarditis
- Severe heart failure
- Dysrhythmias
- Valvular dysfunction
- Ventricular septal rupture

CLINICAL PRESENTATION

- Increase HR
- Dysrhythmias
- Decrease BP
- Chest pain, left or right ventricular failure
- Tachypnea
- Oliguria
- Cool, pale skin
- Decrease Mentation
- Decrease CO,CI,SvO2
- Increase PAP,RAP,PAOP,SVR

MANAGEMENT

- Improve contractility with inotropic agents
- Mechanical support
- Emergency revascularization
- Reduce preload
- Reduce afterload
- Prevent or treat dysrhythmias

- Prevention and treatment of cardiogenic shock is aimed at promoting myocardial contractility, decreasing the myocardial oxygen demand, and increasing the oxygen supply to the damaged tissue. percutaneous coronary interventions, intracoronary stent placement, or both, fibrinolytic agents
- When primary percutaneous coronary intervention is not available, glycoprotein IIb/IIIa inhibitors, and beta-blockers given

- Diuretics (e.g., furosemide) and venous vasodilators (e.g., morphine, nitroglycerin, nitroprusside) reduce preload and venous return to the heart. Nitroglycerin at low doses less (1 mcg/kg/min) causes venous vasodilation to decrease preload. At higher doses more (1 mcg/kg/min) arterial vasodilation decreases afterload

- Positive inotropic agents (e.g., dobutamine) are given to increase the contractile force of the heart. As contractility increases, ventricular emptying improves, filling pressures decrease (RAP, PAOP), and stroke volume improves.

- Afterload reduction may be achieved by the cautious administration of arterial vasodilators (e.g., nitroprusside) to decrease SVR, increase stroke volume, and increase cardiac index.
- Blood pressure must be carefully monitored to keep the mean arterial pressure above 65 mm Hg to ensure organ perfusion

- IABP improves coronary artery perfusion, reduces afterload, and improves perfusion to vital organs
- VADs are used to treat cardiogenic shock by allowing the ventricle to recover or to support the patient awaiting cardiac transplant as a bridge to transplant

Obstructive shock

- Obstructive shock (also known as extracardiac obstructive shock)
- occurs when there is a physical impairment to adequate circulatory blood flow.

POSSIBLE CAUSES

Impaired diastolic filling:

- Cardiac tamponade
- Tension pneumothorax
- Constrictive pericarditis
- Compression of great veins

Increased right ventricular afterload:

- Pulmonary embolism (PE)
- Severe pulmonary hypertension
- Increased intrathoracic pressure

Increased left ventricular afterload:

- Aortic dissection
- Systemic embolization
- Aortic stenosis
- Abdominal hypertension

CLINICAL PRESENTATION

- Increase HR, Dysrhythmias ,decrease BP
- Chest pain ,Dyspnea ,Oliguria ,Cool, pale skin ,Decreased mental status
- Jugular venous distention
- Cardiac tamponade: muffled heart sounds, pulsus paradoxus
- Tension pneumothorax: diminished breath sounds on affected side, tracheal shift away from affected side
- Pulmonary embolism: right ventricular failure

- Aortic dissection: ripping chest pain, pulse differences between left and right side, widened mediastinum
- Decrease CO, CI , SvO2 , SVR
- Increase or normal RAP, PAP, PAOP
- Increase PVR

MANAGEMENT

- Eliminate source of obstruction or compression
- Pericardiocentesis for cardiac tamponade
- Fibrinolytics, anticoagulants for PE
- Emergency decompression for tension pneumothorax

Distributive Shock

- also known as vasogenic shock, describes several different types of shock that present with widespread vasodilation and decreased SVR.
- Neurogenic, anaphylactic, and septic shock are forms of distributive shock
- All forms of distributive shock, stroke volume, cardiac output, and blood pressure decrease, resulting in decreased tissue perfusion and impaired cellular metabolism

Anaphylactic shock

- A severe allergic reaction can precipitate a second form of distributive shock known as anaphylactic shock.
- Antigens, which are foreign substances to which someone is sensitive, initiate an antigen-antibody response.

POSSIBLE CAUSES

- Foods: fish, shellfish, eggs, milk, wheat, strawberries, peanuts,
- Drugs: antibiotics, ACE inhibitors, aspirin, local anesthetics, narcotics, barbiturates, contrast media
- Blood and blood products
- Bites and stings
- Chemicals: latex, lotions, soap, perfumes

CLINICAL PRESENTATION

- Increase HR ,dysrhythmias ,Decrease BP ,Chest pain ,Tachypnea
- Flushed, warm to hot skin ,Oliguria
- Restlessness, change in LOC, seizures
- Nausea, vomiting, abdominal cramping, diarrhea
- Decrease CO,CI,RAP,PAP,PAOP,SvO2,SVR
- Increase IgE
- Dyspnea, cough, stridor, wheezing, dysphagia
- Urticaria, angioedema

MANAGEMENT

- Remove offending agent or slow absorption: remove stinger; apply ice to sting or bite; discontinue drug, dye, blood; lavage stomach if antigen ingested; flush skin with water
- Maintain airway, oxygenation, and ventilation; intubation may be necessary
- Modify or block the effects of mediators: epinephrine, antihistamines, steroids
- Maintain MAP

Neurogenic shock

- There is an interruption of impulse transmission or a blockage of sympathetic outflow resulting in vasodilation, inhibition of baroreceptor response, and impaired thermoregulation.
- Consequently, these reactions create vasodilation with decreased SVR, venous return, preload, and cardiac output and a relative hypovolemia

POSSIBLE CAUSES

- General or spinal anesthesia
- Epidural block
- Cervical spinal cord injury
- Drugs: barbiturates, phenothiazines, sympathetic blocking agents

CLINICAL PRESENTATION

- Decrease HR, BP
  CO,CI,RAP,PAP,PAOP,SvO2,SVR
- Hypothermia
- Warm, dry, flushed skin
- Oliguria
- Neurological deficit

MANAGEMENT

- Eliminate and treat the cause
- Maintain MAP
- Maintain adequate heart rate
- VTE prophylaxis

Septic Shock

- Septic shock is one component of a continuum of progressive clinical insults including SIRS, sepsis, and MODS

CLINICAL CONDITION AND DEFINITION	DIAGNOSTIC CRITERIA
Infection: Inflammatory response to microorganisms	Fever
SIRS: Systemic inflammatory response to a clinical insult including infection, pancreatitis, ischemia, trauma, or hemorrhagic shock	Tachycardia (HR+= 90 beats/min) Respiratory rate +20 breaths/min or PaCO2 less32 mm Hg Temperature +38° C (hyperthermia) or _36° C (hypothermia)
Sepsis: Systemic response to infection manifested by two or more of the symptoms noted with SIRS	Leukocytosis (WBC count +12,000 cells/microliter) or leukopenia (WBC count _4000 cells/microliter
Septic shock: Sepsis with hypotension despite adequate fluid resuscitation, along with perfusion abnormalities	Hypotension Lactic acidosis, oliguria, acute change in mental status Patients receiving inotropic agents or vasopressors may not exhibit hypotension
MODS: Altered organ function in acutely ill patients	Acute respiratory distress syndrome ,Acute tubular necrosis, Hepatic dysfunction/failure, Disseminated intravascular coagulation

POSSIBLE CAUSES

Immunosuppression:

- Extremes of age ,Malnutrition ,Alcoholism or drug abuse ,Malignancy ,History of splenectomy ,Chronic health problems
- Immunosuppressive therapies

Significant bacteremia:

- Invasive procedures and devices
- Traumatic wounds or burns
- GI infection or untreated disease ,Peritonitis
- Food poisoning Prolonged hospitalization
- Translocation of GI bacteria (associated with NPO status)

- Gram-negative bacteria such as Escherichia coli, Klebsiella species, or Pseudomonas species are a common cause of infections in adults. Common sites of infection include the pulmonary system, urinary tract, gastrointestinal system
- Gram-positive bacteria such as Staphylococcus aureus can also lead to sepsis and septic shock
- Pneumonia (VAP)is a common trigger for sepsis.
- Urinary tract infection is an often overlooked cause of secondary bloodstream infections

CLINICAL PRESENTATION

Early, hyperdynamic, warm:

- Increase HR
- Normal or decrease BP
- widened Pulse pressure
- Skin warm, flushed
- Confusion
- Oliguria
- Increase CO, CIS , SvO2
- Decrease RAP, PAP, PAOP, SVR

Late, hypodynamic, cold:

- Increase HR
- Decrease BP
- Decrease Pulse pressure
- Skin cool, pale
- Decrease LOC
- Anuria
- Hypothermia
- decrease CO, CI, SvO2
- Variable RAP, PAP, PAOP, SVR
- Positive culture

MANAGEMENT

- Good hand-washing techniques
- Avoid invasive procedures
- Identify source of infection
- Meticulous oral and airway care
- Meticulous catheter and wound care
- Avoid NPO status: initiate and maintain enteral nutrition
- Antibiotics as indicated by culture results
- Control hyperthermia
- Maintain MAP

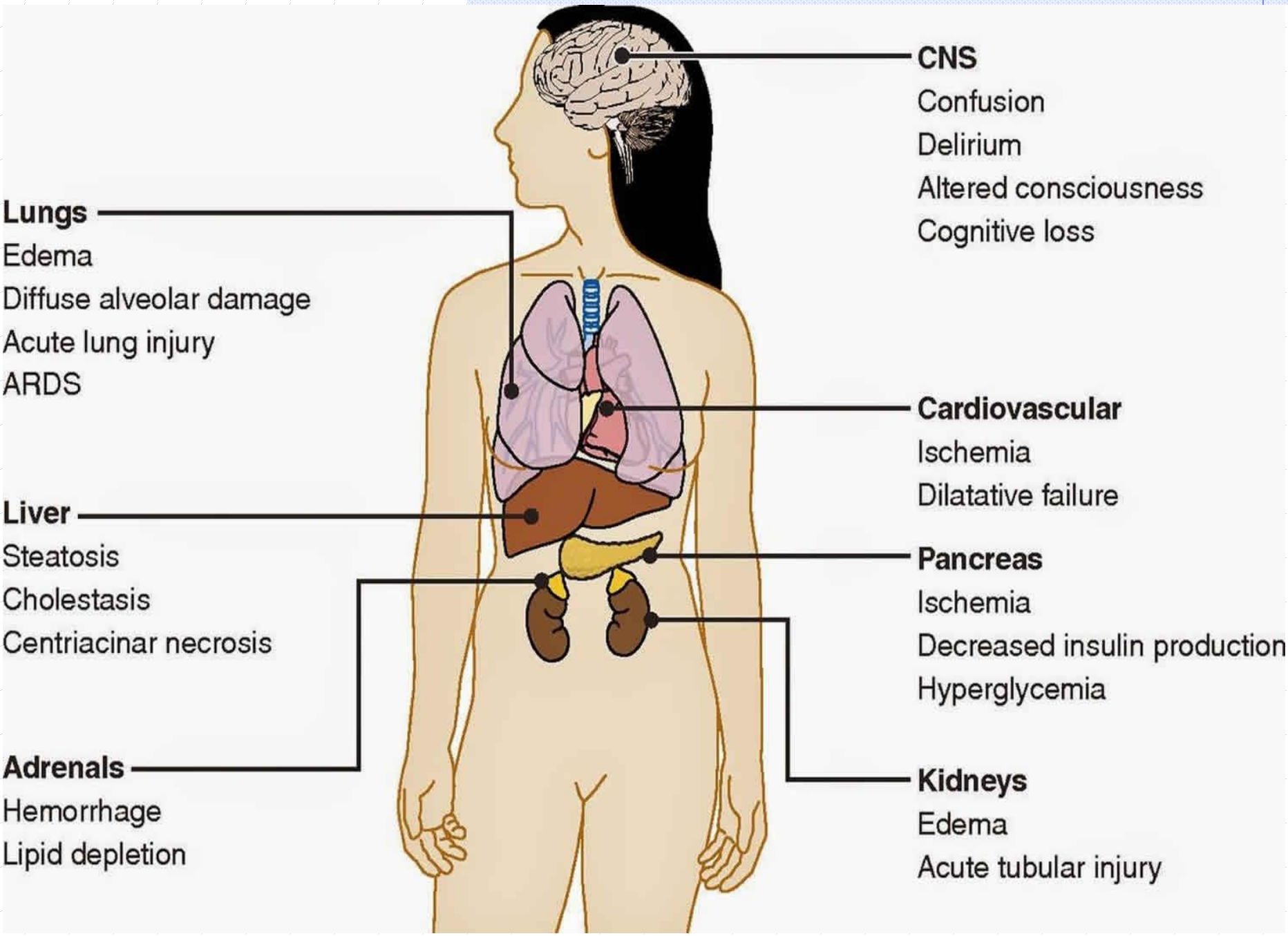
MULTIPLE ORGAN DYSFUNCTION SYNDROME

- MODS is the progressive dysfunction of two or more organ systems as a result of an uncontrolled inflammatory response to severe illness or injury
- Organ dysfunction can progress to organ failure and death
- The most common causes of MODS are sepsis and septic shock; however, MODS can occur after any severe injury or disease process that activates a massive systemic inflammatory response including any classification of shock

- Failure of two or more organs is associated with an estimated 45% to 55% mortality, 80% mortality when three or more organ systems fail, and 100% mortality if three or more organ systems fail for longer than 4 days

- In primary MODS there is direct injury to an organ from shock, trauma, burn injury, or infection with impaired perfusion that results in dysfunction.
- Decreased perfusion may be localized or systemic. As a result of this insult, the stress response and inflammatory response are activated with the release of catecholamines and activation of mediators that affect cellular activity.

- Secondary MODS is a consequence of widespread systemic inflammation that results in dysfunction of organs not involved with the initial insult.
- It occurs in response to altered regulation of the acute immune and inflammatory responses


CNS
Confusion
Delirium
Altered consciousness
Cognitive loss
Lungs
Edema
Diffuse alveolar damage
Acute lung injury
ARDS
Cardiovascular
Ischemia
Dilatative failure
Liver
Steatosis
Cholestasis
Centriacinar necrosis
Pancreas
Ischemia
Decreased insulin production
Hyperglycemia
Adrenals
Hemorrhage
Lipid depletion
Kidneys
Edema
Acute tubular injury

Medications Commonly Used in Shock

- Dopamine At 10-20 mcg/kg/min

stimulates alpha receptors to cause vasoconstriction and increased SVR

- Norepinephrine Stimulates alpha receptors to cause vasoconstriction

Used in vasodilatory states (distributive shock) to restore vascular tone

Stimulation of beta receptors to increase contractility and increase HR

Norepinephrine

Dose : 2-12 mcg/min IV infusion and titrated upward as needed to a maximum of 30 mcg/min

Central venous catheter preferred

Side effects : Tachycardia ,Ventricular dysrhythmias ,Hypertension ,Anxiety

Headache ,Tremor ,Dizziness,

Chest pain ,Metabolic acidosis

- Phenylephrine (Neosynephrine)

Stimulates alpha receptors to cause vasoconstriction

Used in vasodilatory states (distributive shock) to restore vascular tone

Dose :2-10 mcg/kg/min IV infusion

Central venous catheter preferred

- Vasopressin Vasoconstriction via smooth muscle contraction of all parts of capillaries, arterioles, and venules
  Used in vasodilatory states (distributive shock) to restore vascular tone
  Dose :0.01 to 0.03 unit/min IV infusion
  Central venous catheter preferred

- Nitroglycerin Vasodilation by direct smooth muscle relaxation, predominantly Venous
- Used in preload and/or afterload reduction (cardiogenic shock)
- Initial dose 5-10 mcg/min IV infusion; increase by 5-10 mcg/min every 5 minutes until desired results are achieved
- (control of chest pain and decreased preload)

- Nitroprusside (Nipride) Vasodilation by direct smooth muscle relaxation, predominantly arterial

Used in preload and/or afterload reduction (cardiogenic shock)

Dose :0.5-10 mcg/kg/min IV infusion