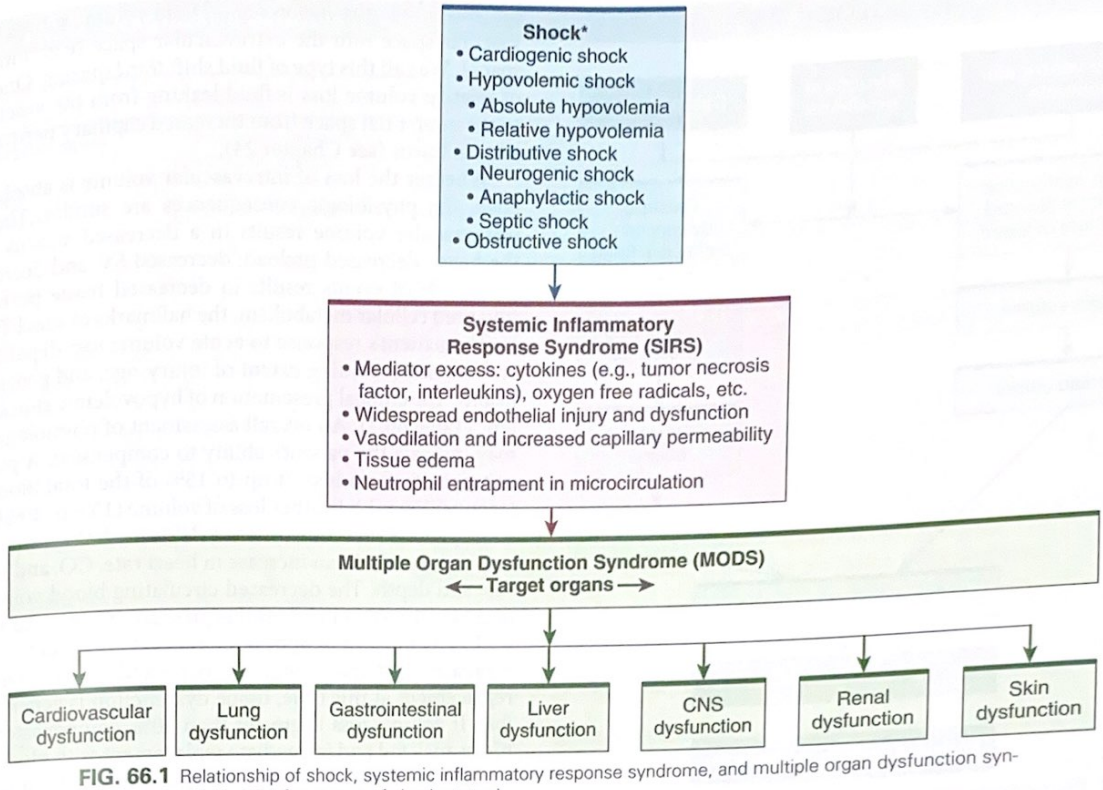


FIG. 66.1 Relationship of shock, systemic inflammatory response syndrome, and multiple organ dysfunction syndrome. (*See Table 66.1 for causes of shock states.)

TABLE 66.1 Classification of Shock States

Types and Causes	Associated Conditions
Cardiogenic Shock	
• Diastolic dysfunction: inability of the heart to fill	Cardiac tamponade, ventricular hypertrophy, cardiomyopathy
• Dysrhythmias	Bradydysrhythmias, tachydysrhythmias
• Structural factors	Valvular stenosis or regurgitation, ventricular septal rupture, tension pneumothorax
• Systolic dysfunction: inability of the heart to pump blood forward	MI, cardiomyopathy, blunt cardiac injury, severe systemic or pulmonary hypertension, myocardial depression from metabolic problems
Hypovolemic Shock	
Absolute Hypovolemia	
• External loss of whole blood	Hemorrhage from trauma, surgery, GI bleeding
• Loss of other body fluids	Vomiting, diarrhea, excessive diuresis, diabetes insipidus, diabetes
Relative Hypovolemia	
• Fluid shifts	Burn injuries, ascites
• Internal bleeding	Fracture of long bones, ruptured spleen, hemothorax, severe pancreatitis
• Massive vasodilation	Sepsis
• Pooling of blood or fluids	Bowel obstruction

Types and Causes	Associated Conditions
Distributive Shock	
Anaphylactic Shock	
• Hypersensitivity (allergic) reaction to a sensitizing substance	Contrast media, blood or blood products, drugs, insect bites, anesthetic agents, food or food additives, vaccines, environmental agents, latex
Neurogenic Shock	
• Hemodynamic consequence of spinal cord injury and/or disease at or above T5	Severe pain, drugs, hypoglycemia, injury
• Spinal anesthesia	
• Vasomotor center depression	
Septic Shock	
• Infection	Pneumonia, peritonitis, urinary tract, invasive procedures, indwelling lines and catheters
• At-risk patients	Older adults, patients with chronic diseases (e.g., diabetes, chronic kidney disease, HF), patients receiving immunosuppressive therapy or who are malnourished or debilitated
Obstructive Shock	
• Physical obstruction impeding the filling or outflow of blood resulting in reduced CO	Cardiac tamponade, tension pneumothorax, superior vena cava syndrome, abdominal compartment syndrome, pulmonary embolism

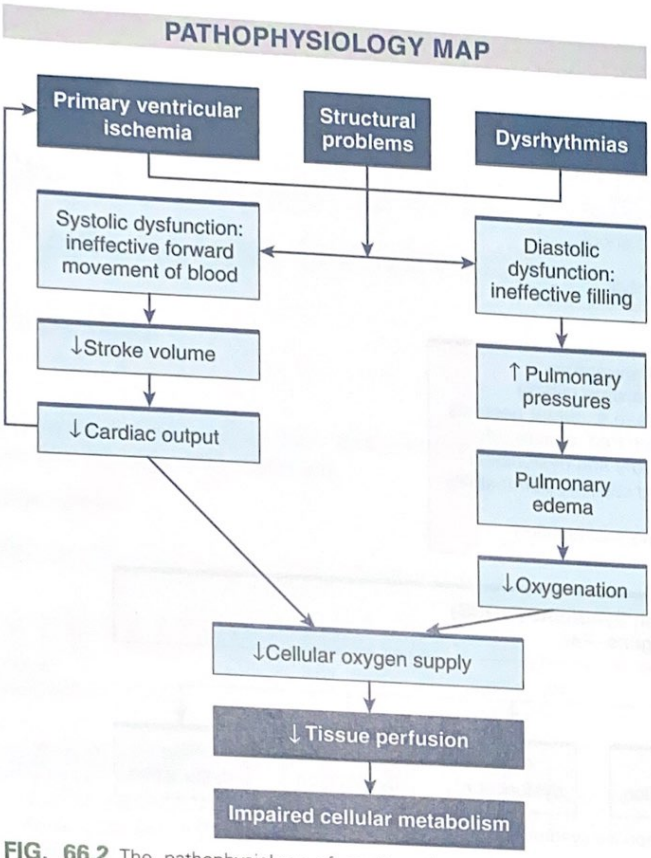


FIG. 66.2 The pathophysiology of cardiogenic shock. (Modified from Urden LD, Stacy KM, Lough ME: *Critical care nursing: Diagnosis and management*, ed 6, St Louis, 2010, Mosby.)

problem (e.g., valvular disorder, ventricular septal rupture), or dysrhythmias, the physiologic responses are similar. The patient has impaired tissue perfusion and cellular metabolism.

The early presentation of a patient with cardiogenic shock is similar to that of a patient with acute decompensated heart failure (HF) (see Chapter 34). The patient may have tachycardia and hypotension. Pulse pressure may be narrowed due to the heart's inability to pump blood forward during systole and increased volume during diastole. An increase in systemic vascular resistance (SVR) increases the workload of the heart. This increases myocardial O_2 consumption.

On assessment, the patient is tachypneic and has crackles on auscultation of breath sounds because of pulmonary congestion. The hemodynamic profile shows an increase in the pulmonary artery wedge pressure (PAWP), stroke volume variation (SVV), and pulmonary vascular resistance.

Signs of peripheral hypoperfusion (e.g., cyanosis, pallor, diaphoresis, weak peripheral pulses, cool and clammy skin, delayed capillary refill) occur. Decreased renal blood flow results in sodium and water retention and decreased urine output. Anxiety, confusion, and agitation may develop with impaired cerebral perfusion. Tables 66.2 and 66.3 describe the laboratory findings and clinical presentation of a patient with cardiogenic shock.

Hypovolemic Shock. **Hypovolemic shock** occurs from inadequate fluid volume in the intravascular space to support adequate perfusion (Table 66.1).[2] The volume loss may be either an absolute or a relative volume loss. *Absolute hypovolemia* results when fluid is lost through hemorrhage, gastrointestinal (GI) loss (e.g., vomiting, diarrhea), fistula drainage, diabetes insipidus, or diuresis. In *relative hypovolemia,* fluid volume moves out of the vascular space into the extravascular space (e.g., intracavitary space). We call this type of fluid shift *third spacing.* One example of relative volume loss is fluid leaking from the vascular space to the interstitial space from increased capillary permeability, as seen in burns (see Chapter 24).

Whether the loss of intravascular volume is absolute or relative, the physiologic consequences are similar. The reduced intravascular volume results in a decreased venous return to the heart, decreased preload, decreased SV, and decreased CO. A cascade of events results in decreased tissue perfusion and impaired cellular metabolism, the hallmarks of shock (Fig. 66.3).

The patient's response to acute volume loss depends on several factors, including extent of injury, age, and general state of health. The clinical presentation of hypovolemic shock is consistent (Table 66.3). An overall assessment of physiologic reserves may indicate the patient's ability to compensate. A patient may compensate for a loss of up to 15% of the total blood volume (around 750 mL). Further loss of volume (15% to 30%) results in a sympathetic nervous system (SNS)–mediated response.[2] This response results in an increase in heart rate, CO, and respiratory rate and depth. The decreased circulating blood volume causes decreases in SV, central venous pressure (CVP), and PAWP.[3]

The patient may appear anxious. Urine output begins to decrease. If hypovolemia is corrected by crystalloid fluid replacement at this time, tissue dysfunction is generally reversible. If volume loss is greater than 30%, compensatory mechanisms may fail and immediate replacement with blood products should be started. Loss of autoregulation in the microcirculation and irreversible tissue destruction occur with loss of more than 40% of the total blood volume. Common laboratory studies and assessments that are done include serial measurements of hemoglobin and hematocrit levels, electrolytes, lactate, blood gases, mixed central venous O_2 saturation (SvO_2), and hourly urine outputs (Table 66.2).

Distributive Shock

Neurogenic Shock. **Neurogenic shock** is a hemodynamic phenomenon that can occur within 30 minutes of a spinal cord injury and last up to 6 weeks. Neurogenic shock related to spinal cord injuries is generally associated with a cervical or high thoracic injury. The injury results in a massive vasodilation without compensation because of the loss of SNS vasoconstrictor tone.[4] This massive vasodilation leads to a pooling of blood in the blood vessels, tissue hypoperfusion, and impaired cellular metabolism (Fig. 66.4).

In addition to spinal cord injury, spinal anesthesia can block transmission of impulses from the SNS. Depression of the vasomotor center of the medulla from drugs (e.g., opioids, benzodiazepines) can decrease the vasoconstrictor tone of the peripheral blood vessels, resulting in neurogenic shock (Table 66.1).

The classic manifestations are hypotension (from the massive vasodilation) and bradycardia (from unopposed parasympathetic stimulation).[4] The patient may not be able to regulate body temperature. Combined with massive vasodilation, the inability to regulate temperature promotes heat loss. At first, the patient's skin is warm due to the massive vasodilation. As the heat disperses, the patient is at risk for hypothermia. Later, the patient's skin may be cool or warm depending on the ambient temperature (*poikilothermia,* taking on the temperature of the environment). In either case, the skin is usually dry. Tables 66.2 and 66.3 further describe the laboratory findings and clinical presentation of a patient with neurogenic shock.

TABLE 66.2 Diagnostic Studies

Shock

Study	Finding	Significance of Finding
Arterial blood gasses	Respiratory alkalosis	Found in early shock due to hyperventilation
	Metabolic acidosis	Occurs later in shock when lactate accumulates in blood from anaerobic metabolism
Base deficit	>−6	Acid production due to hypoxia
Blood cultures	Growth of organisms	May grow organisms in patients who are in septic shock
BUN	↑	Impaired kidney function caused by hypoperfusion from severe vasoconstriction, or occurs due to cell catabolism (e.g., trauma, infection)
Creatine kinase	↑	Trauma, MI in response to cellular damage and/or hypoxia
Creatinine	↑	Impaired kidney function caused by hypoperfusion because of severe vasoconstriction
DIC screen		Acute DIC can develop within hours to days after an initial assault on the body (e.g., shock)
• Fibrin split products (FSP)	↑	
• Fibrinogen level	↓	
• Platelet count	↓	
• PTT and PT	↑	
• INR	↑	
• Thrombin time	↑	
• D-dimer	↑	
Glucose	↑	Found in early shock because of release of liver glycogen stores in response to sympathetic nervous system stimulation and cortisol. Insulin insensitivity develops
	↓	Depleted glycogen stores with liver dysfunction possible as shock progresses
Electrolytes (serum)		
• Sodium	↑	Found in early shock because of ↑ secretion of aldosterone, causing renal retention of sodium
	↓	May be iatrogenic if excess hypotonic fluid is given after fluid loss
• Potassium	↑	Results when dead cells release potassium. Occurs in acute kidney injury and acidosis
	↓	Found in early shock because of ↑ secretion of aldosterone, causing renal excretion of potassium
Lactate level	↑	Usually ↑ once significant hypoperfusion and impaired O_2 use at the cellular level have occurred. By-product of anaerobic metabolism
Liver enzymes (ALT, AST, GGT)	↑	Liver cell destruction in progressive stage of shock
Procalcitonin (PCT)	↑	Biomarker released in response to bacterial infections
RBC count, hematocrit, hemoglobin	Normal	Remains within normal limits in shock because of relative hypovolemia and pump failure and in hemorrhagic shock before fluid resuscitation
	↓	Hemorrhagic shock after fluid resuscitation when fluids other than blood are used
	↑	Nonhemorrhagic shock caused by actual hypovolemia and hemoconcentration
Troponin	↑	MI
White blood cell count	↑, ↓	Infection, septic shock

ALT, Alanine aminotransferase; *AST,* aspartate aminotransferase; *GGT,* γ-glutamyl transferase; *INR,* international normalized ratio; *PT,* prothrombin time; *PTT,* partial thromboplastin time.

Although spinal shock and neurogenic shock often occur in the same patient, they are not the same disorder. *Spinal shock* is a transient condition that is present after an acute spinal cord injury (see Chapter 60). The patient with spinal shock has an absence of all voluntary and reflex neurologic activity below the level of the injury.

Anaphylactic Shock. **Anaphylactic shock** is an acute, life-threatening hypersensitivity (allergic) reaction to a sensitizing substance (e.g., drug, chemical, vaccine, food, insect venom).[5] The reaction quickly causes massive vasodilation, release of vasoactive mediators, and an increase in capillary permeability. As capillary permeability increases, fluid leaks from the vascular space into the interstitial space.

Anaphylactic shock can lead to respiratory distress due to laryngeal edema or severe bronchospasm and circulatory failure from the massive vasodilation. The patient has a sudden onset of symptoms, including dizziness, chest pain, incontinence, swelling of the lips and tongue, wheezing, and stridor. Skin changes include flushing, pruritus, urticaria, and angioedema. The patient may be anxious and confused and have a sense of impending doom.

A patient can have a severe allergic reaction, possibly leading to anaphylactic shock, after contact, inhalation, ingestion, or injection with an antigen (allergen) to which the person has previously been sensitized (Table 66.1). IV administration of the antigen (allergen) is the route most likely to cause anaphylaxis. However, oral, topical, and inhalation routes can cause anaphylactic reactions. Tables 66.2 and 66.3 describe the laboratory findings and clinical presentation of a patient in anaphylactic shock. Quick and decisive action is critical to prevent an allergic reaction from progressing to anaphylactic shock. (Anaphylaxis is discussed in Chapter 13.)

Septic Shock. **Sepsis** is a life-threatening syndrome in response to an infection. It is characterized by a dysregulated patient response along with new organ dysfunction related to the infection (Table 66.4).[6] In as many as 30% of patients with sepsis, the causative organism is not identified. Sepsis and septic shock have a high incidence worldwide, with a mortality rate of 25% or higher.[7]

Septic shock is a subset of sepsis. It has an increased mortality risk due to profound circulatory, cellular, and metabolic abnormalities. Septic shock is characterized by persistent hypotension, despite adequate fluid resuscitation, and inadequate tissue perfusion that results in tissue hypoxia.[6,7] The main organisms that cause sepsis are gram-negative and gram-positive bacteria.

TABLE 66.3 Clinical Presentation of Types of Shock

Cardiogenic Shock	Hypovolemic Shock	DISTRIBUTIVE SHOCK			Obstructive Shock
		Neurogenic Shock	Anaphylactic Shock	Septic Shock	
Cardiovascular System					
Tachycardia ↓ BP ↓ SV, CO ↑ SVR, PAWP, CVP ↓ Capillary refill	Tachycardia ↓ Preload ↓ CO, CVP, PAWP ↑ SVR ↓ Capillary refill	Bradycardia ↓ BP ↓ CO, CVP, SVR ↓/↑ Temperature	Tachycardia ↑ CO ↓ CVP, PAWP Chest pain Third spacing of fluid	Tachycardia ↓/↑ Temperature Myocardial dysfunction Biventricular dilation ↓ Ejection fraction	Tachycardia ↓ BP ↓ Preload ↓ CO ↑ SVR, CVP
Respiratory System					
Tachypnea Crackles Cyanosis	Tachypnea → bradypnea (late)	Dysfunction related to level of injury	Shortness of breath Edema of larynx and epiglottis Wheezing Stridor Rhinitis	Hyperventilation Crackles Respiratory alkalosis → respiratory acidosis Hypoxemia Respiratory failure ARDS Pulmonary hypertension	Tachypnea → bradypnea (late) Shortness of breath
Renal System					
↑ Na^+ and H_2O retention ↓ Renal blood flow ↓ Urine output	↓ Urine output	Bladder dysfunction	Incontinence	↓ Urine output	↓ Urine output
Skin					
Pallor Cool, clammy	Pallor Cool, clammy	↓ Skin perfusion Cool or warm Dry	Flushing Pruritus Urticaria Angioedema	Warm and flushed → cool and mottled (late)	Pallor Cool, clammy
Neurologic System					
↓ Cerebral perfusion: • Anxiety • Confusion • Agitation	↓ Cerebral perfusion: • Anxiety • Confusion • Agitation	Flaccid paralysis below the level of the lesion Loss of reflex activity	Anxiety Feeling of impending doom Confusion ↓ LOC Metallic taste	Change in mental status (e.g., confusion) Agitation Coma (late)	↓ Cerebral perfusion: • Anxiety • Confusion • Agitation
Gastrointestinal System					
↓ Bowel sounds Nausea, vomiting	Absent bowel sounds	Bowel dysfunction	Cramping Abdominal pain Nausea Vomiting Diarrhea	GI bleeding Paralytic ileus	↓ To absent bowel sounds
Diagnostic Findings*					
↑ Cardiac biomarkers ↑ b-Type natriuretic peptide (BNP) ↑ Blood glucose ↑ BUN ECG (e.g., dysrhythmias) Echocardiogram (e.g., left ventricular dysfunction Chest x-ray (e.g., pulmonary infiltrates)	↓ Hematocrit ↓ Hemoglobin ↑ Lactate ↑ Urine specific gravity Changes in electrolytes		Sudden onset History of allergies Exposure to contrast media	↑/↓ WBC ↓ Platelets ↑ Lactate ↑ Blood glucose ↑ Procalcitonin ↑ Urine specific gravity ↓ Urine Na^+ Positive blood cultures	Specific to cause of obstruction

*Also see Table 66.2.

Parasites, fungi, and viruses can also cause sepsis and septic shock.[6] Fig. 66.5 presents the pathophysiology of septic shock.

When a microorganism enters the body, the normal immune or inflammatory responses are triggered. However, in sepsis and septic shock the body's response to the microorganism is exaggerated. Both proinflammatory and antiinflammatory responses are activated, coagulation increases, and fibrinolysis decreases.[6] Endotoxins from the microorganism cell wall stimulate the release of cytokines. These include tumor necrosis factor (TNF), interleukin-1 (IL-1), and other proinflammatory mediators that act through secondary mediators, such as platelet-activating factor, IL-6, and IL-8.[6] (See Chapter 11 for discussion of the inflammatory response.) The release of platelet-activating factor results in

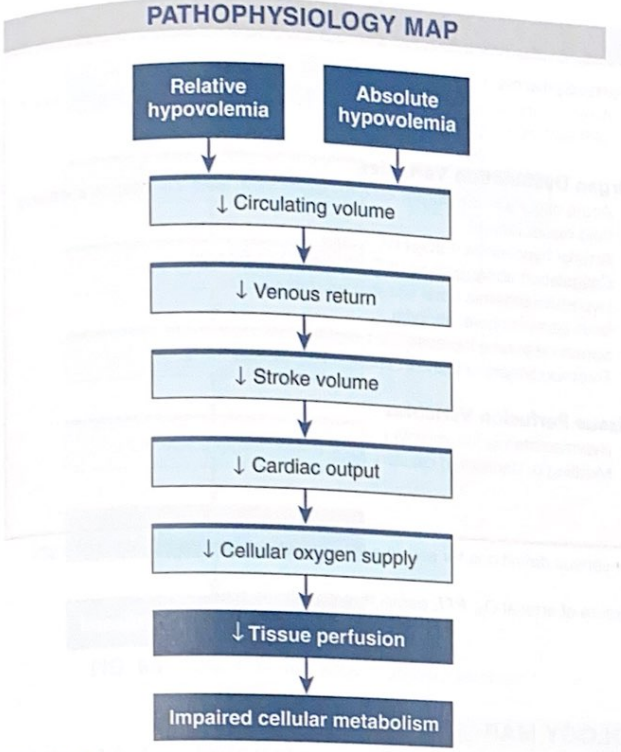


FIG. 66.3 The pathophysiology of hypovolemic shock. (Modified from Urden LD, Stacy KM, Lough ME: *Critical care nursing: Diagnosis and management*, ed 6, St Louis, 2010, Mosby.)

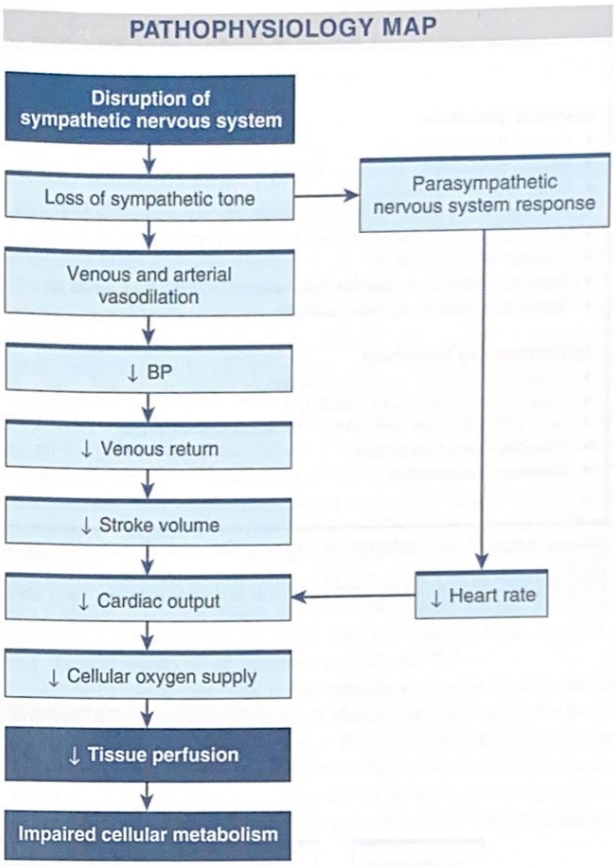


FIG. 66.4 The pathophysiology of neurogenic shock. (Modified from Urden LD, Stacy KM, Lough ME: *Critical care nursing: Diagnosis and management*, ed 6, St Louis, 2010, Mosby.)

the formation of microthrombi and obstruction of the microvasculature. The combined effects of the mediators result in damage to the endothelium, vasodilation, increased capillary permeability, and neutrophil and platelet aggregation and adhesion to the endothelium.

Septic shock has 3 major pathophysiologic effects: vasodilation, maldistribution of blood flow, and myocardial depression. Patients may be euvolemic, but because of acute vasodilation and shifting of fluids out of the intravascular space, relative hypovolemia and hypotension occur. Blood flow in the microcirculation is decreased, causing poor O_2 delivery and tissue hypoxia. We think the combination of TNF and IL-1 has a role in sepsis-induced myocardial dysfunction. The ejection fraction (EF) is decreased for the first few days after the initial insult. Because of a decreased EF, the ventricles dilate to maintain the SV. The EF typically improves, and ventricular dilation resolves over 7 to 10 days. Persistent high CO and a low SVR beyond 24 hours is an ominous finding. It is often associated with an increased development of hypotension and MODS. Coronary artery perfusion and myocardial O_2 metabolism are not primarily altered in septic shock.

Respiratory failure is common. The patient initially hyperventilates as a compensatory mechanism, causing respiratory alkalosis. Once the patient can no longer compensate, respiratory acidosis develops. Respiratory failure develops in 85% of patients with sepsis, and 40% develop acute respiratory distress syndrome (ARDS) (see Chapter 67). These patients may need to be intubated and mechanically ventilated.

Other signs of septic shock include changes in neurologic status, decreased urine output, and GI dysfunction, such as GI bleeding and paralytic ileus. Table 66.3 gives the clinical presentation of a patient with septic shock.

Obstructive Shock. **Obstructive shock** develops when a physical obstruction to blood flow occurs with a decreased CO (Fig. 66.6). This can be caused by restricted diastolic filling of the right ventricle from compression (e.g., cardiac tamponade, tension pneumothorax, superior vena cava syndrome). Other causes include *abdominal compartment syndrome*, in which increased abdominal pressures compress the inferior vena cava. This decreases venous return to the heart. Pulmonary embolism and right ventricular thrombi cause an outflow obstruction as blood leaves the right ventricle through the pulmonary artery. This leads to decreased blood flow to the lungs and decreased blood return to the left atrium.

Patients have a decreased CO, increased afterload, and variable left ventricular filling pressures depending on the obstruction. Other signs include jugular venous distention and pulsus paradoxus. Rapid assessment and treatment are important to prevent further hemodynamic compromise and possible cardiac arrest (Fig. 66.6).

Stages of Shock

In addition to understanding the underlying pathogenesis of the type of shock the patient has, management is guided by knowing where the patient is on the shock "continuum." We categorize

TABLE 66.4 Diagnostic Criteria for Sepsis

Infection, documented or suspected, and some of the following:

General Variables
- Altered mental status
- Fever (temperature >100.9°F [38.3°C])
- Heart rate >90 beats/min
- Hyperglycemia (blood glucose >140 mg/dL) in the absence of diabetes
- Hypothermia (core temperature <97.0°F [36°C])
- Systolic BP ≤100 mm Hg
- Significant edema or positive fluid balance (>20 mL/kg over 24 hr)
- Tachypnea (respiratory rate ≥22/min)

Inflammatory Variables
- Leukocytosis (WBC count >12,000/μL)
- Leukopenia (WBC count <4000/μL)
- Normal WBC count with >10% immature forms (bands)
- Elevated C-reactive protein
- Elevated procalcitonin

Hemodynamic Variables
- Arterial hypotension (SBP <90 mm Hg, MAP <70 mm Hg, or a decrease in SBP >40 mm Hg)

Organ Dysfunction Variables
- Acute oliguria (urine output <0.5 mL/kg/hr for at least 2 hr despite adequate fluid resuscitation)
- Arterial hypoxemia (PaO_2/FIO_2 <300)
- Coagulation abnormalities (INR >1.5 or PTT >60 sec)
- Hyperbilirubinemia (total bilirubin >4 mg/dL)
- Ileus (absent bowel sounds)
- Serum creatinine increase >0.5 mg/dL
- Thrombocytopenia (platelet count <100,000/μL)

Tissue Perfusion Variables
- Hyperlactatemia (>1 mmol/L)
- Mottling or decreased capillary refill

Source: Singer M, Deutschman CS, Seymour CW, et al: The third international consensus definitions for sepsis and septic shock (Sepsis-3), *JAMA* 315:801, 2016.

FIO_2, Fraction of inspired O_2; *INR*, international normalized ratio; *PaO_2*, partial pressure of arterial O_2; *PTT*, partial thromboplastin time.

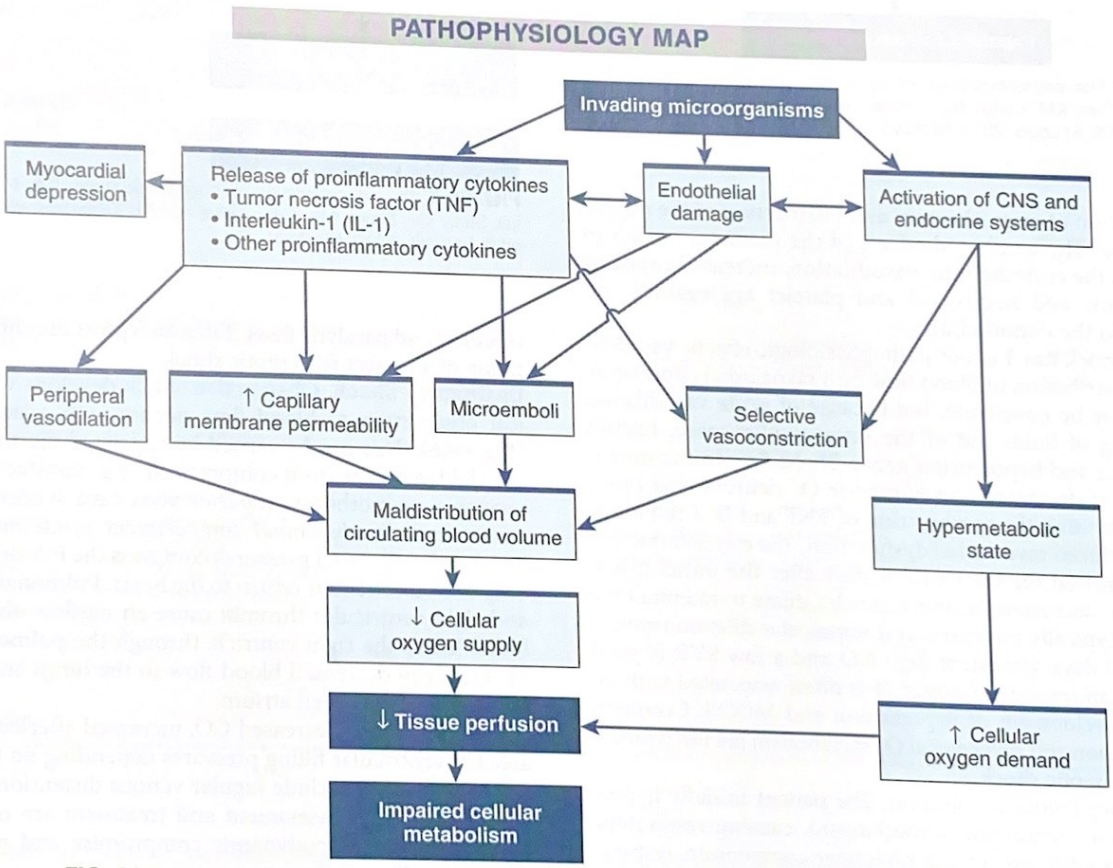


FIG. 66.5 The pathophysiology of septic shock. (Modified from Urden LD, Stacy KM, Lough ME: *Critical care nursing: Diagnosis and management*, ed 6, St Louis, 2010, Mosby.)

PATHOPHYSIOLOGY MAP

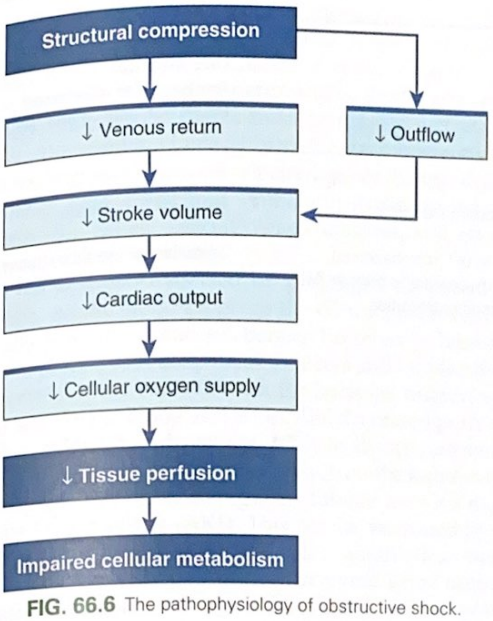


FIG. 66.6 The pathophysiology of obstructive shock.

shock into 4 overlapping stages: (1) initial stage, (2) compensatory stage, (3) progressive stage, and (4) refractory stage.[6]

Initial Stage. The continuum begins with the *initial stage* of shock that occurs at a cellular level. This stage is usually not clinically apparent. Metabolism changes at the cellular level from aerobic to anaerobic, causing lactic acid buildup. Lactic acid is a waste product that is removed by the liver. However, this process requires O_2, which is unavailable because of the decrease in tissue perfusion.

Compensatory Stage. In the *compensatory stage* the body activates neural, hormonal, and biochemical compensatory mechanisms to try to overcome the increasing consequences of anaerobic metabolism and maintain homeostasis. The patient's clinical presentation begins to reflect the body's responses to the imbalance in O_2 supply and demand (Table 66.5).

A classic sign of shock is a drop in BP. This occurs because of a decrease in CO and a narrowing of the pulse pressure. The baroreceptors in the carotid and aortic bodies immediately respond by activating the SNS. The SNS stimulates vasoconstriction and the release of the potent vasoconstrictors epinephrine and norepinephrine. Blood flow to the heart and brain is maintained. Blood flow to the nonvital organs, such as kidneys, GI tract, skin, and lungs, is diverted or shunted.

The myocardium responds to the SNS stimulation and the increase in O_2 demand by increasing the heart rate and contractility. Increased contractility increases myocardial O_2 consumption. The coronary arteries dilate to try to meet the increased O_2 demands of the myocardium.

Shunting blood away from the lungs has an important clinical effect in the patient in shock. Decreased blood flow to the lungs increases the patient's physiologic dead space. *Physiologic dead space* is the anatomic dead space (the amount of air that will not reach gas-exchanging units) and any inspired air that cannot take part in gas exchange. The clinical result of an increase in dead space ventilation is a *ventilation-perfusion mismatch*. Some areas of the lungs that are being ventilated will not be perfused because of the decreased blood flow to the lungs. Arterial O_2 levels will decrease, and the patient will have a compensatory increase in the rate and depth of respirations (see Chapter 67).

The shunting of blood from other organ systems results in clinically important changes. The decrease in blood flow to the GI tract results in impaired motility and a slowing of peristalsis. This increases the risk for a paralytic ileus.

Decreased blood flow to the skin results in the patient feeling cool and clammy. The exception is the patient in early septic shock who may feel warm and flushed because of a hyperdynamic state. Decreased blood flow to the kidneys activates the renin-angiotensin system. Renin stimulates angiotensinogen to make angiotensin I, which is then converted to angiotensin II (see Fig. 44.4). Angiotensin II is a potent vasoconstrictor that causes both arterial and venous vasoconstriction. The net result is an increase in venous return to the heart and an increase in BP. Angiotensin II stimulates the adrenal cortex to release aldosterone. This results in sodium and water reabsorption and potassium excretion by the kidneys. The increase in sodium reabsorption raises the serum osmolality and stimulates the release of antidiuretic hormone (ADH) from the posterior pituitary gland. ADH increases water reabsorption by the kidneys, further increasing blood volume. The increase in total circulating volume results in an increase in CO and BP.

A multisystem response to decreasing tissue perfusion starts during the compensatory stage of shock. At this stage, the body can compensate for the changes in tissue perfusion. If the cause of the shock is corrected, the patient will recover with little or no residual effects. If the cause of the shock is not corrected and the body is unable to compensate, the patient enters the progressive stage of shock.

Progressive Stage. The *progressive stage* of shock begins as compensatory mechanisms fail. Changes in the patient's mental status are important findings in this stage. Patients must be moved to the intensive care unit (ICU), if not already there, for advanced monitoring and treatment.

The cardiovascular system is profoundly affected in the progressive stage of shock. CO begins to fall, resulting in a decrease in BP and coronary artery, cerebral, and peripheral perfusion. Continued decreased cellular perfusion and resulting altered capillary permeability are the distinguishing features of this stage. Altered capillary permeability allows fluid and protein to leak out of the vascular space into the surrounding interstitial space. In addition to the decrease in circulating volume, there is an increase in systemic interstitial edema. The patient may have *anasarca* (diffuse profound edema). Fluid leakage from the vascular space affects the solid organs (e.g., liver, spleen, GI tract, lungs) and peripheral tissues by further decreasing perfusion.

Sustained hypoperfusion results in weak peripheral pulses, and ischemia of the distal extremities eventually occurs. Myocardial dysfunction from decreased perfusion results in dysrhythmias, myocardial ischemia, and possibly MI. The result is a complete deterioration of the cardiovascular system.

The pulmonary system is often the first system to display signs of critical dysfunction. During the compensatory stage, blood flow to the lungs is already reduced. In response to the decreased blood flow and SNS stimulation, the pulmonary arterioles constrict, resulting in increased pulmonary artery (PA) pressure. As the pressure within the pulmonary vasculature increases, blood flow to the pulmonary capillaries decreases and ventilation-perfusion mismatch worsens.

TABLE 66.5 Manifestations of Stages of Shock*

Compensatory Stage	Progressive Stage	Refractory Stage
Neurologic System Oriented to person, place, time Restless, apprehensive, confused Change in level of consciousness	↓ Cerebral perfusion pressure ↓ Cerebral blood flow ↓ Responsiveness to stimuli Delirium	Unresponsive Areflexia (loss of reflexes) Pupils nonreactive and dilated
Cardiovascular System Sympathetic nervous system response: • Release of epinephrine/norepinephrine (vasoconstriction) • ↑ MVO_2 • ↑ Contractility • ↑ HR Coronary artery dilation Narrowed pulse pressure ↓ BP	↑ Capillary permeability → systemic interstitial edema ↓ CO → ↓ BP and ↑ HR MAP <60 mm Hg (or 40 mm Hg drop in BP from baseline) ↓ Coronary perfusion → dysrhythmias, myocardial ischemia, MI ↓ Peripheral perfusion → ischemia of distal extremities, ↓ pulses, ↓ capillary refill	Profound hypotension ↓ CO Bradycardia, irregular rhythm ↓ BP inadequate to perfuse vital organs
Respiratory System ↓ Blood flow to the lungs: • ↑ Physiologic dead space • ↑ Ventilation-perfusion mismatch • Hyperventilation • ↑ Minute ventilation (V_E) • Tachypnea	ARDS: • ↑ Capillary permeability • Pulmonary vasoconstriction • Pulmonary interstitial edema • Alveolar edema • Diffuse infiltrates • Tachypnea • ↓ Compliance • Moist crackles	Severe refractory hypoxemia Respiratory failure
Gastrointestinal System ↓ Blood supply ↓ GI motility Hypoactive bowel sounds ↑ Risk for paralytic ileus	Vasoconstriction and ↓ perfusion → ischemic gut (e.g., stomach, small and large intestines, gallbladder, pancreas): • Erosive ulcers • GI bleeding • Translocation of GI bacteria • Impaired absorption of nutrients	Ischemic gut
Renal System ↓ Renal blood flow ↑ Renin resulting in release of angiotensin (vasoconstrictor) ↑ Aldosterone resulting in Na^+ and H_2O reabsorption ↑ Antidiuretic hormone resulting in H_2O reabsorption	Renal tubules become ischemic → acute tubular necrosis ↓ Urine output ↑ BUN-to-creatinine ratio ↑ Urine sodium ↓ Urine osmolality and specific gravity ↓ Urine potassium Metabolic acidosis	Anuria
Hepatic System	Failure to metabolize drugs and waste products Cell death (↑ liver enzymes) Jaundice (↓ clearance of bilirubin) ↑ NH_3 (ammonia) and lactate	Metabolic changes from accumulation of waste products (e.g., NH_3, lactate, CO_2)
Hematologic System	DIC: • Thrombin clots in microcirculation • Consumption of platelets and clotting factors	DIC progresses
Temperature Normal or abnormal	Hypothermia or hyperthermia	Hypothermia
Skin Pale and cool Warm and flushed	Cold and clammy	Mottled, cyanotic

*The shock continuum begins with the *initial stage* of shock. This stage occurs at the cellular level and is usually not clinically apparent. Also see Table 66.2 and Table 66.3.

MVO_2, myocardial O_2 consumption.

Another key response in the lungs is the movement of fluid from the pulmonary vasculature into the interstitial space. As capillary permeability increases, the movement of fluid to the interstitial spaces results in interstitial edema, bronchoconstriction, and a decrease in functional residual capacity. With further increases in capillary permeability, fluid moves into the alveoli, causing alveolar edema and a decrease in surfactant production. The combined effects of pulmonary vasoconstriction and bronchoconstriction are impaired gas exchange, decreased compliance, and worsening ventilation-perfusion mismatch. Clinically, the patient has tachypnea, crackles, and an overall increased work of breathing.

The GI system is affected by prolonged decreased tissue perfusion. As the blood supply to the GI tract is decreased, the normally protective mucosal barrier becomes ischemic. This ischemia predisposes the patient to ulcers and GI bleeding (see Chapter 41). It increases the risk for bacterial migration from the GI tract to the blood and lungs. The decreased perfusion to the GI tract leads to a decreased ability to absorb nutrients.

The effect of prolonged hypoperfusion on the kidneys is renal tubular ischemia. The resulting acute tubular necrosis may lead to acute kidney injury (AKI). This can be worsened by nephrotoxic drugs (e.g., certain antibiotics, anesthetics, diuretics) (see Chapter 46). The patient has decreased urine output and increased blood urea nitrogen (BUN) and serum creatinine. Metabolic acidosis occurs from the kidneys' inability to excrete acids (especially lactic acid) and reabsorb bicarbonate.

The sustained hypoperfusion in the progressive stage of shock greatly affects other organs. The loss of the functional ability of the liver leads to a failure of the liver to metabolize drugs and waste products (e.g., lactate, ammonia). Jaundice results from an accumulation of bilirubin. As the liver cells die, liver enzymes increase. The liver loses its ability to function as an immune organ. Kupffer cells no longer destroy bacteria from the GI tract. Instead, they are released into the bloodstream, increasing the possibility of bacteremia.

Dysfunction of the hematologic system adds to the complexity of the clinical picture. The patient is at risk for disseminated intravascular coagulation (DIC). The consumption of the platelets and clotting factors with secondary fibrinolysis results in clinically significant bleeding from many orifices. These include the GI tract, lungs, and puncture sites (see Chapter 30). Altered laboratory values in DIC are shown in Table 66.3.

In this stage, aggressive interventions are needed to prevent the development of MODS.

Refractory Stage. In the last stage of shock, the *refractory stage,* decreased perfusion from peripheral vasoconstriction and decreased CO worsen anaerobic metabolism. The accumulation of lactic acid contributes to increased capillary permeability and dilation. Increased capillary permeability allows fluid and plasma proteins to leave the vascular space and move to the interstitial space. Blood pools in the capillary beds due to the constricted venules and dilated arterioles. The loss of intravascular volume worsens hypotension and tachycardia and decreases coronary blood flow. Decreased coronary blood flow leads to worsening myocardial depression and a further decline in CO. Cerebral blood flow cannot be maintained and cerebral ischemia results.

The patient in this stage of shock has profound hypotension and hypoxemia. The failure of the liver, lungs, and kidneys results in an accumulation of waste products, such as lactate, urea, ammonia, and CO_2. The failure of 1 organ system affects several other organ systems. Recovery is unlikely in this stage. The organs are in failure and the body's compensatory mechanisms are overwhelmed (Table 66.5).

Diagnostic Studies

There is no single diagnostic study to determine whether a patient is in shock. The diagnosis starts with a history and physical examination. Obtaining a thorough medical and surgical history and a history of recent events (e.g., surgery, chest pain, trauma) gives valuable data.

Decreased tissue perfusion in shock leads to an increased lactate with a base deficit (the amount needed to bring the pH back to normal). These laboratory changes reflect an increase in anaerobic metabolism.[8] Table 66.2 outlines laboratory findings seen in shock.

Other diagnostic studies include a 12-lead electrocardiogram (ECG), continuous ECG monitoring, chest x-ray, continuous pulse oximetry, and invasive and noninvasive hemodynamic monitoring. Chapter 65 discusses hemodynamic monitoring)

Interprofessional Care

Critical factors in the successful management of a patient in shock relate to the early recognition and treatment of the shock state. Prompt intervention in the early stages of shock may prevent the decline to the progressive or irreversible stage. Successful management of the patient in shock includes (1) identification of patients at risk for the development of shock; (2) integration of the patient's history, physical examination, and clinical findings to establish a diagnosis; (3) interventions to control or eliminate the cause of the decreased perfusion; (4) protecting target and distal organs from dysfunction; and (5) providing multisystem supportive care.

Table 66.6 provides an overview of the initial assessment findings and interventions for the emergency care of patients in shock. General management strategies begin with ensuring that the patient is responsive and has a patent airway. Once the airway is established, either naturally or with an endotracheal tube, O_2 delivery must be optimized. Supplemental O_2 and mechanical ventilation may be needed to maintain an arterial O_2 saturation of 90% or more (PaO_2 greater than 60 mm Hg) to avoid hypoxemia (see Chapter 65). The mean arterial pressure (MAP) and circulating blood volume are optimized with fluid replacement and drug therapy.

Oxygen and Ventilation. O_2 delivery depends on CO, available hemoglobin, and arterial O_2 saturation (SaO_2). Methods to optimize O_2 delivery are directed at increasing supply and decreasing demand. Supply is increased by (1) optimizing the CO with fluid replacement and/or drug therapy, (2) increasing the hemoglobin through transfusion of whole blood or packed red blood cells (RBCs), and/or (3) increasing the arterial O_2 saturation with supplemental O_2 and mechanical ventilation.

Plan care to avoid disrupting the balance of O_2 supply and demand. Space activities that increase O_2 consumption (e.g., endotracheal suctioning, position changes) appropriately for O_2 conservation. Intermittent or continuous monitoring of $ScvO_2$ by a central venous catheter or mixed venous O_2 saturation (SvO_2) may be helpful. Both reflect the dynamic balance between O_2 supply and demand. Assess these values along with related hemodynamic measures (e.g., arterial pressure–based cardiac output [APCO], O_2 consumption, hemoglobin) to evaluate the patient's response to treatments and activities (see Chapter 65).

dysfunction also contributes to metabolic acidosis. Hypocalcemia, hypomagnesemia, and hypophosphatemia are common.

Clinical Manifestations of SIRS and MODS

The clinical manifestations of SIRS and MODS are described in Table 66.10.

NURSING AND INTERPROFESSIONAL MANAGEMENT: SIRS AND MODS

The prognosis for the patient with MODS is poor, with mortality rates of 40% to 60%.[22] Mortality increases as more organ systems fail. The most common cause of death continues to be sepsis. Survival improves with early, goal-directed therapy. So, the most important goal is to prevent SIRS from progressing to MODS.

A critical part of your role is vigilant assessment and ongoing monitoring to detect early signs of deterioration or organ dysfunction. Interprofessional care for patients with SIRS and MODS focuses on (1) prevention and treatment of infection, (2) maintaining tissue oxygenation, (3) nutritional and metabolic support, and (4) appropriate support of individual failing organs. Table 66.10 outlines the management for patients with SIRS and MODS.

TABLE 66.10 Manifestations and Management of SIRS and MODS

Manifestations	Management
Respiratory System	
Development of ARDS (see Chapter 67): • Bilateral fluffy infiltrates on chest x-ray • Decreased compliance • Dyspnea (severe) • Increased minute ventilation • PaO_2/FIO_2 ratio <200 • PAWP <18 mm Hg • Pulmonary hypertension • Refractory hypoxemia • Tachypnea • Ventilation-perfusion (V/Q) mismatch	Optimize O_2 delivery and minimize O_2 consumption Mechanical ventilation (see Chapter 65) • Positive end-expiratory pressure • Lung protective modes (e.g., pressure-control inverse ratio ventilation, low tidal volumes) • Permissive hypercapnia • Positioning (e.g., continuous lateral rotation therapy, prone positioning)
Cardiovascular System	
Biventricular failure ↓ BP, MAP, SVR ↑ HR, CO, SV Massive vasodilation Myocardial depression Systolic, diastolic dysfunction	Volume management to ↑ preload Hemodynamic monitoring Arterial pressure monitoring to maintain MAP >65 mm Hg Vasopressors Intermittent or continuous $ScvO_2$ or SvO_2 monitoring Balance O_2 supply and demand Continuous ECG monitoring Circulatory assist devices VTE prophylaxis
Central Nervous System	
Acute change in neurologic status Confusion, disorientation, delirium Fever Hepatic encephalopathy Seizures	Evaluate for hepatic or metabolic encephalopathy Optimize cerebral blood flow ↓ Cerebral O_2 requirements Prevent secondary tissue ischemia Calcium channel blockers (reduce cerebral vasospasm)
Endocrine System	
Hyperglycemia → hypoglycemia	Provide continuous infusion of insulin and glucose to maintain blood glucose 140–180 mg/dL (7.77–10.0 mmol/L)
Renal System	
Prerenal: renal hypoperfusion • BUN/creatinine ratio >20:1 • ↓ Urine Na^+ <20 mEq/L • ↑ Urine osmolality • Urine specific gravity >1.020	Diuretics • Loop diuretics (e.g., furosemide [Lasix]) • May need to ↑ dosage due to ↓ glomerular filtration rate Continuous renal replacement therapy (see Chapter 46)
Intrarenal: acute tubular necrosis • BUN/creatinine ratio <10:1–15:1 • ↑ Urine Na^+ >20 mEq/L • ↓ Urine osmolality • Urine specific gravity ~1.010	
GI System	
GI bleeding Hypoperfusion → ↓ peristalsis, paralytic ileus Mucosal ischemia • ↓ Intramucosal pH • Potential translocation of gut bacteria • Potential abdominal compartment syndrome Mucosal ulceration on endoscopy	Stress ulcer prophylaxis • Antacids (e.g., Maalox) • Proton pump inhibitors (e.g., omeprazole [Prilosec]) • sucralfate (Carafate) Monitor abdominal distention, intraabdominal pressures Dietitian consult Enteral nutrition Stimulate mucosal activity Provide essential nutrients and optimal calories
Hepatic System	
Bilirubin >2 mg/dL (34 μmol/L) Hepatic encephalopathy Jaundice ↑ Liver enzymes (ALT, AST, GGT) ↓ Serum albumin, prealbumin, transferrin ↑ Serum NH_3 (ammonia)	Maintain adequate tissue perfusion Provide nutritional support (e.g., enteral nutrition) Careful use of drugs metabolized by liver
Hematologic System	
↑ Bleeding times, ↑ PT, ↑ PTT ↑ D-dimer ↑ Fibrin split products ↓ Platelet count (thrombocytopenia)	Observe for bleeding from obvious and/or occult sites Replace factors being lost (e.g., platelets) Minimize traumatic interventions (e.g., IM injections, multiple venipunctures)

ALT, Alanine aminotransferase; *AST,* aspartate aminotransferase; *GGT,* γ-glutamyl transferase; *PA,* pulmonary artery; *PAWP,* pulmonary artery wedge pressure; *PT,* prothrombin time; *PTT,* partial thromboplastin time; *$ScvO_2$,* O_2 saturation in venous blood; *SvO_2,* O_2 saturation in mixed venous blood; *SVR,* systemic vascular resistance.

Prevention and Treatment of Infection

Aggressive infection control strategies are essential to decrease the risk for HAIs. Early, aggressive surgery is recommended to remove necrotic tissue (e.g., early debridement of burn tissue) that can provide a culture medium for microorganisms. Aggressive pulmonary management, including early mobilization, can reduce the risk for infection. Strict asepsis can decrease infections related to intraarterial lines, endotracheal tubes, indwelling urinary catheters, IV lines, and other invasive devices or procedures. Daily assessment of the ongoing need for invasive lines and other devices is an important strategy to prevent or limit HAIs.

Despite aggressive strategies, infection may develop. Once an infection is suspected, begin interventions to treat the cause. Send appropriate cultures and start broad-spectrum antibiotic therapy, as ordered. Adjust therapy based on the culture results, if needed.

Maintenance of Tissue Oxygenation

Hypoxemia often occurs because patients have greater O_2 needs and decreased O_2 supply to the tissues. Interventions that decrease O_2 demand and increase O_2 delivery are essential. Sedation, mechanical ventilation, analgesia, and rest may decrease O_2 demand and should be considered. Treating fever, chills, and pain decrease O_2 demand. O_2 delivery may be optimized by using individualized tidal volumes with positive end-expiratory pressure, increasing preload (e.g., fluids) or myocardial contractility to enhance CO, or reducing afterload to increase CO.

Nutritional and Metabolic Needs

Hypermetabolism can result in profound weight loss, cachexia, and further organ failure. Protein-calorie malnutrition is a key sign of hypermetabolism. Total energy expenditure is often increased 1.5 to 2.0 times the normal metabolic rate. Because of their short half-life, monitor plasma transferrin and prealbumin levels to assess hepatic protein synthesis.

The goal of nutritional support is to preserve organ function. Providing early and optimal nutrition decreases morbidity and mortality rates. EN is preferred. If it cannot be used, PN should be considered. (Chapter 39 discusses EN and PN.) Provide glycemic control with a goal of ≤180 mg/dL with insulin infusions in these patients.[21]

Support of Failing Organs

Support of any failing organ is a goal of therapy. For example, the patient with ARDS requires aggressive O_2 therapy and mechanical ventilation (see Chapter 67). DIC should be treated appropriately (e.g., blood products) (see Chapter 30). Renal failure may require dialysis. Continuous renal replacement therapy is better tolerated than hemodialysis, especially in a patient with hemodynamic instability (see Chapter 46).

A final consideration may be that further interventions are futile. It is important to maintain communication between the health care team and the patient's caregiver about realistic goals and likely outcomes for the patient with MODS. Withdrawal of life support and starting end-of-life care may be the best options for the patient.

CASE STUDY

Shock

(© Thinkstock.)

Patient Profile

K.L., a 25-yr-old Korean American, was not wearing his seat belt when he was driving a motor vehicle involved in a crash. The windshield was broken, and K.L. was found 10 ft from his car. He was face down, conscious, and moaning. His wife and daughter were in the car with their seat belts on. They sustained minor injuries and were very frightened and upset. All passengers were taken to the ED. This information pertains to K.L.

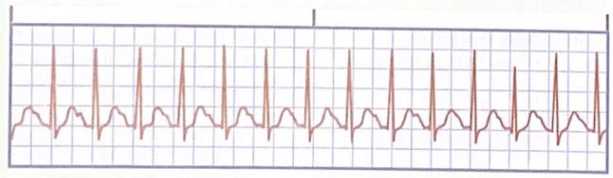
Subjective Data

- States, "I can't breathe"
- Cries out when abdomen is palpated

Objective Data

Physical Examination

- *Cardiovascular:* BP 80/56 mm Hg; apical pulse 138 but no palpable radial or pedal pulses; carotid pulse 1+. ECG is below:



- *Respiratory:* respiratory rate 35 breaths/min; labored breathing with shallow respirations; asymmetric chest wall movement; absence of breath sounds on left side. Trachea deviated slightly to the right
- *Abdomen:* slightly distended and left upper quadrant painful on palpation
- *Musculoskeletal:* open compound fracture of the lower left leg

Diagnostic Studies

- Chest x-ray: hemothorax and 6 rib fractures on left side
- Hematocrit: 28%

Interprofessional Care (in the ED)

- Intraosseous access in right proximal tibia placed prehospital
- Left chest tube placed, draining bright red blood
- Fluid resuscitation started with crystalloids
- High-flow O_2 via nonrebreather mask

Emergency Surgical Procedures

- Splenectomy
- Repair of torn intercostal artery
- Repair of compound fracture

Discussion Questions

1. What types of shock is K.L. experiencing? What clinical manifestations did he display that support your answer?
2. What were the causes of K.L.'s shock states? What are other causes of these types of shock?
3. ***Priority Decision:*** What are the priority nursing responsibilities for K.L.?
4. ***Priority Decision:*** What ongoing nursing assessment parameters are essential for this patient?
5. What are his potential complications?
6. ***Patient-Centered Care:*** K.L.'s parents arrive. English is their second language. They are very anxious and asking about their son. What can you do to provide culturally competent family-centered care?
7. ***Priority Decision:*** Based on the assessment data presented, what are the priority nursing diagnoses?
8. ***Collaboration:*** Identify the tasks that could be delegated to unlicensed assistive personnel (UAP).
9. ***Evidence-Based Practice:*** You are orienting a new graduate RN. He asks you why crystalloids are used for fluid resuscitation. What is your response?

Answers available at *http://evolve.elsevier.com/Lewis/medsurg*.