Shock, Sepsis, and Multiple Organ Dysfunction Syndrome

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CONCEPTUAL FOCUS

Fluid and Electrolytes
Gas Exchange
Inflammation
Perfusion

LEARNING OUTCOMES

1. Relate the pathophysiology to the clinical manifestations of the different types of shock.
2. Compare the effects of shock, sepsis, systemic inflammatory response syndrome, and multiple organ dysfunction syndrome (MODS) on the major body systems.
3. Compare the interprofessional care, drug therapy, and nursing management of patients with different types of shock.
4. Describe the interprofessional care and nursing management of a patient with MODS.

KEY TERMS

cardiogenic shock
hypovolemic shock
multiple organ dysfunction syndrome (MODS)
neurogenic shock
obstructive shock
sepsis
septic shock
shock
systemic inflammatory response syndrome (SIRS)

Shock, systemic inflammatory response syndrome (SIRS), sepsis, and multiple organ dysfunction syndrome (MODS) are serious and interrelated problems (Fig. 42.1). **Shock** is a syndrome characterized by decreased tissue perfusion and impaired cellular metabolism. This results in an imbalance between the supply of and demand for O_2 and nutrients. The exchange of O_2 and nutrients at the cellular level is essential to life. When cells are hypoperfused, the demand for O_2 and nutrients exceeds the supply at the microcirculatory level. Ischemia can occur, leading to cell injury and death. Thus shock is life-threatening. This chapter gives an overview of the different types of shock, SIRS, sepsis, and MODS and their related management.

SHOCK

Classification of Shock

The 4 main categories of shock are cardiogenic, hypovolemic, distributive, and obstructive (Table 42.1). Although the cause, initial presentation, and management vary for each type, the physiologic responses of cells to hypoperfusion are similar.

Cardiogenic Shock

Cardiogenic shock occurs when either systolic or diastolic dysfunction of the heart's pumping action results in reduced cardiac output (CO), stroke volume (SV), and BP. These changes compromise myocardial perfusion, further depress myocardial function, and decrease CO and perfusion. Causes of cardiogenic shock are shown in Table 42.1. It is the leading cause of death from acute myocardial infarction (MI). Although the in-hospital mortality has improved due to aggressive intervention, the 6- to 12-month mortality remains 50%.[1]

Fig. 42.2 shows the pathophysiology of cardiogenic shock. The heart's inability to pump the blood forward is called *systolic dysfunction*. This inability results in a low CO (less than 4 L/min) and *cardiac index* (less than 2.5 L/min/m^2). Systolic dysfunction primarily affects the left ventricle because systolic pressure is greater on the left side of the heart. The most common cause of systolic dysfunction is acute MI. When systolic dysfunction affects the right side of the heart, blood flow through the pulmonary circulation is reduced. Decreased filling of the heart results in decreased SV. Table 42.1 lists causes of diastolic dysfunction.

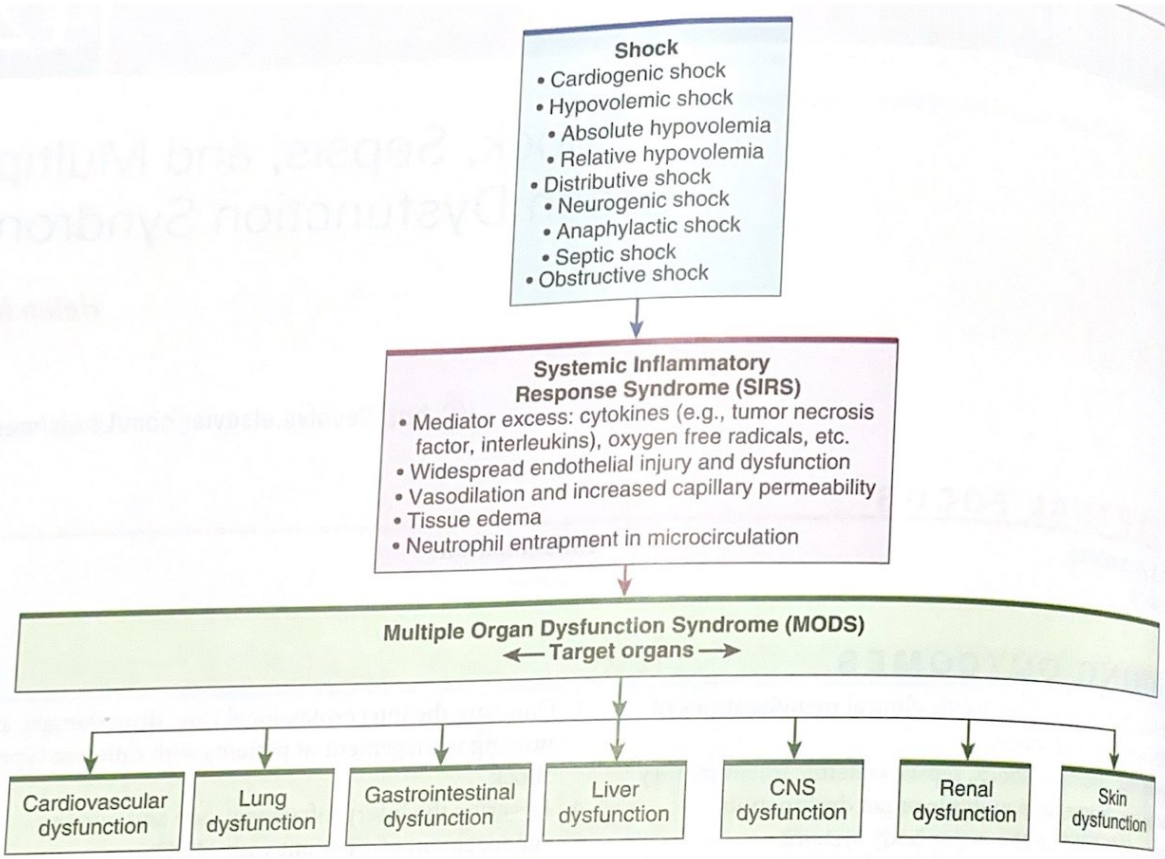

Fig. 42.1 Relationship of shock, systemic inflammatory response syndrome, and multiple organ dysfunction syndrome.

TABLE 42.1 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, latex 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	
• 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
• Infection	Pneumonia, peritonitis, urinary tract, invasive procedures, indwelling lines and catheters
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

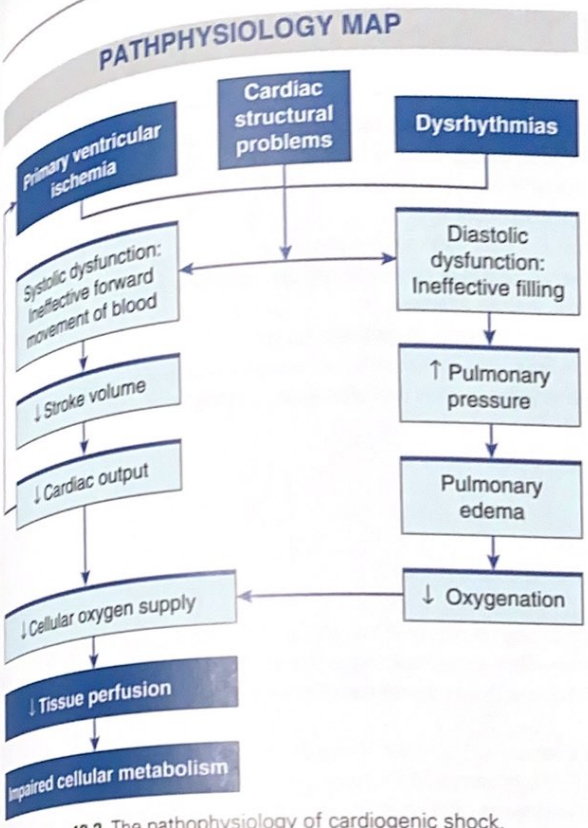

Fig. 42.2 The pathophysiology of cardiogenic shock.

The early presentation of a patient with cardiogenic shock is similar to that of a patient with acute decompensated heart failure (HF) (see Chapter 38). 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. There are crackles on auscultation of breath sounds because of pulmonary congestion. The hemodynamic profile shows an increase in the pulmonary artery (PA) wedge pressure (PAWP), stroke volume variation (SVV), and pulmonary vascular resistance.

Signs of peripheral hypoperfusion (e.g., cyanosis, pallor, 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 42.2 and 42.3 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 42.1).[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 26).

Whether the intravascular volume loss is absolute or relative, the 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. 42.3).

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 42.3). 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 (approximately 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.

The patient may appear anxious. Urine output begins to decrease. If hypovolemia is quickly corrected by crystalloid fluid replacement, 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 42.2).

Distributive Shock

Distributive shock has many causes. It is a condition of relative hypovolemia due to intravascular volume redistribution from loss of vascular tone or disordered permeability. The 3 subsets are neurogenic, anaphylactic, and septic.[3]

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. 42.4).

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 vasoconstrictor tone of the peripheral blood vessels, resulting in neurogenic shock (Table 42.1).

The classic manifestations are hypotension (from the massive vasodilation) and bradycardia (from unopposed parasympathetic stimulation).[4] The patient may not be able to regulate

TABLE 42.2 Diagnostic Studies

Shock

Study	Finding	Significance of Finding
Arterial blood gases	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)
• D-dimer	↑	
• Fibrin split products (FSPs)	↑	
• Fibrinogen level	↓	
• INR	↑	
• Platelet count	↓	
• PTT and PT	↑	
• Thrombin time	↑	
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; *BUN,* blood urea nitrogen; *DIC,* disseminated intravascular coagulation; *GGT,* γ-glutamyl transferase; *INR,* international normalized ratio; *PT,* prothrombin time; *PTT,* partial thromboplastin time.

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). The skin is usually dry. Tables 42.2 and 42.3 further describe the laboratory findings and clinical presentation of a patient with neurogenic shock.

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 65). 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, leading to anaphylactic shock, after contact, inhalation, ingestion, or injection with an antigen (allergen) to which the person has previously been sensitized (Table 42.1). IV administration of the antigen (allergen) is the route most likely to cause anaphylaxis. However, oral, topical, and inhalation routes can cause

TABLE 42.3 Clinical Presentation of Types of Shock

	Cardiogenic Shock	Hypovolemic Shock	Neurogenic Shock	Anaphylactic Shock	Septic Shock	Obstructive 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
Gastrointestinal System ↓Bowel sounds Nausea, vomiting	Absent bowel sounds	Bowel dysfunction	Abdominal pain Cramping Diarrhea Nausea Vomiting	GI bleeding Paralytic ileus	↓ To absent bowel sounds
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
Renal System ↑ Na^+ and H_2O retention ↓Renal blood flow ↓Urine output	↓ Urine output	Bladder dysfunction	Incontinence	↓ Urine output	↓ Urine output
Respiratory System Tachypnea Crackles Cyanosis	Tachypnea → bradypnea (late)	Dysfunction related to level of injury	Shortness of breath Edema of larynx and epiglottis Stridor Wheezing	Hyperventilation Crackles Respiratory alkalosis → respiratory acidosis Hypoxemia Respiratory failure ARDS Pulmonary hypertension	Tachypnea → bradypnea (late) Shortness of breath
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
Diagnostic Findings (see Table 42.2) ↑ b-Type natriuretic peptide (BNP) ↑ Blood glucose ↑ BUN ↑ Cardiac biomarkers Chest x-ray (e.g., pulmonary infiltrates) EKG (e.g., dysrhythmias) Echocardiogram (e.g., left ventricular dysfunction)	Electrolyte changes ↓ Hematocrit ↓ Hemoglobin ↑ Lactate ↑ Urine specific gravity		History of allergies Exposure to contrast media	↑ Blood glucose ↑ Lactate ↓ Platelets Positive blood cultures ↑ Procalcitonin ↑ Urine specific gravity ↓ Urine Na^+ ↑/↓ WBC	Specific to cause of obstruction

PATHPHYSIOLOGY MAP

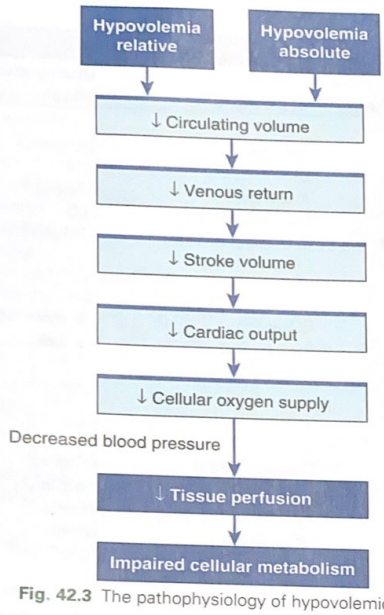

Fig. 42.3 The pathophysiology of hypovolemic shock.

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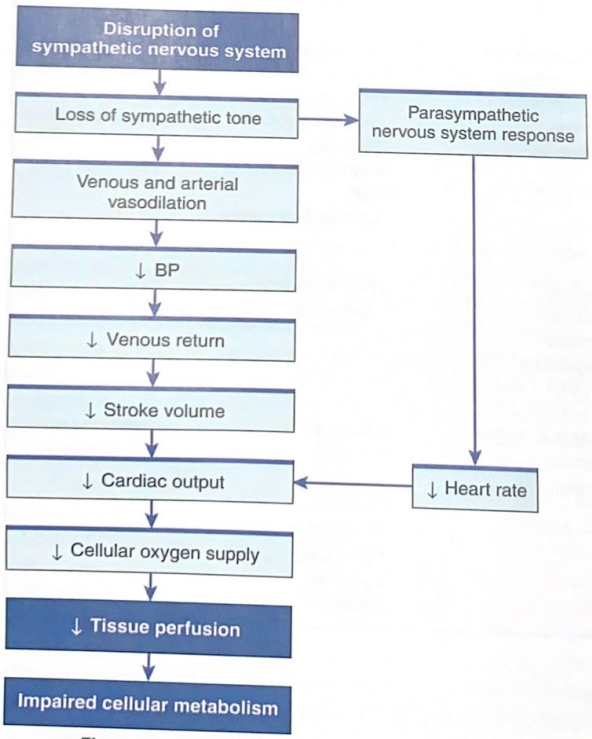

Fig. 42.4 The pathophysiology of neurogenic shock.

TABLE 42.4 Diagnostic Criteria

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])
- SBP ≤100 mm Hg
- Significant edema or positive fluid balance (>20 mL/kg over 24 hr)
- Tachypnea (respiratory rate ≥22/min)

Inflammatory Variables
- ↑ C-reactive protein
- Leukocytosis (WBC count >12,000/μL)
- Leukopenia (WBC count <4000/μL)
- Normal WBC count with >10% immature forms (bands)
- ↑ 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 s)
- 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, ↓ capillary refill

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

anaphylactic reactions. Tables 42.2 and 42.3 describe the laboratory findings and clinical presentation of a patient in anaphylactic shock. Quick action is critical to prevent an allergic reaction from progressing to anaphylactic shock. Anaphylaxis is discussed in Chapter 14.

Septic shock. **Sepsis** is a life-threatening syndrome in response to infection. It is characterized by a dysregulated patient response along with new organ dysfunction related to the infection (Table 42.4).[6] In as many as 30% of patients with sepsis, we never identify the causative agent. 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.[8] The main organisms that cause sepsis are gram-negative and gram-positive bacteria. Parasites

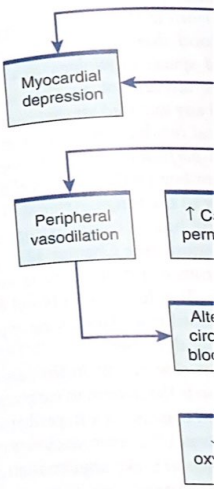

Fig. 42.5

fungi, and viruses can also cause
42.5 presents the pathophysiolog

When a microorganism enter
immune and inflammatory resp
septic shock, the body's respons
gerated. Both proinflammatory
are activated. Coagulation incre
Endotoxins from the microor
release of cytokines. These incl
interleukin-1 (IL-1), and other
act through secondary mediato
tor, IL-6, and IL-8.[9] (See Chap
matory response.) The release
in the formation of microthro
tion. The combined effects of
damage, vasodilation, increas
trophil and platelet aggregatio

Septic shock has 3 major
tion, maldistribution of bloo
Patients may be euvolemic,
and shifting of fluids out
hypovolemia and hypotens
circulation is decreased, c
hypoxia. We think the com
in sepsis-induced myocard
(EF) is decreased for the
Because of a decreased EF
SV. The EF typically impr
over 7 to 10 days. Persist
24 hours is an ominous

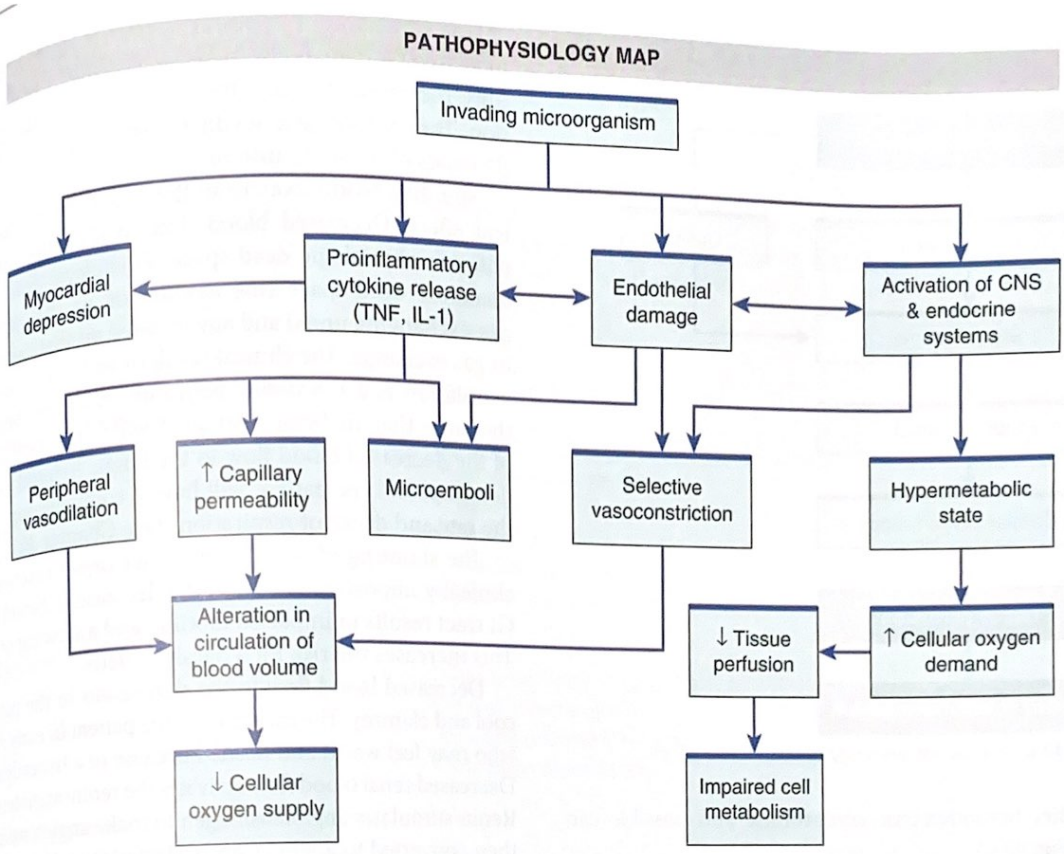

Fig. 42.5 The pathophysiology of septic shock. *IL,* Interleukin; *TNF,* tumor necrosis factor.

fungi, and viruses can also cause sepsis and septic shock.[8] Fig. 42.5 presents the pathophysiology of septic shock.

When a microorganism enters the body, this triggers normal immune and inflammatory responses. 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.[9] (See Chapter 12 for more about the inflammatory response.) The release of platelet-activating factor results in the formation of microthrombi and microvasculature obstruction. The combined effects of the mediators result in endothelial damage, 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 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; 40% develop acute respiratory distress syndrome (ARDS) (see Chapter 32). These patients may need intubation with mechanical ventilation.

Other signs include changes in neurologic status, decreased urine output, and GI dysfunction, such as GI bleeding and paralytic ileus. Table 42.3 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. 42.6). This can be caused by restricted diastolic filling of the right ventricle from compression (e.g., cardiac tamponade, tension pneumothorax, superior vena cava syndrome).[10] 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 PA. 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

PATHOPHYSIOLOGY MAP

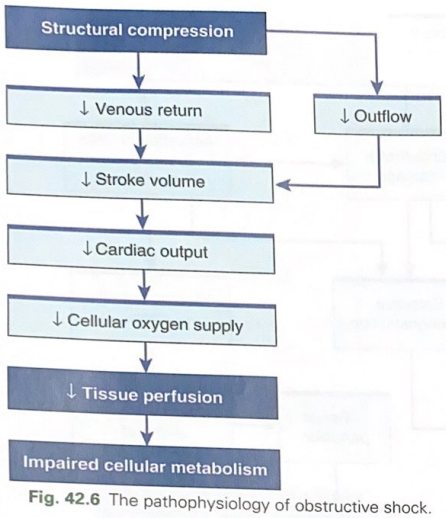

Fig. 42.6 The pathophysiology of obstructive shock.

prevent further hemodynamic compromise and possible cardiac arrest (Fig. 42.6).

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 shock into 4 overlapping stages: (1) initial stage, (2) compensatory stage, (3) progressive stage, and (4) refractory stage.[5]

Initial Stage

The *initial stage* of shock 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.[7] 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 42.5).

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 increased 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 demands of the myocardium.

Shunting blood away from the lungs has an important clinical effect. 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 32).

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 renal blood flow activates the renin-angiotensin system. Renin stimulates angiotensinogen to make angiotensin I, which is then converted to angiotensin II. 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). 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 now profoundly affected. CO begins to fall, causing a decrease in BP and coronary artery, cerebral, and peripheral perfusion. Decreased cellular perfusion continues. Altered capillary permeability occurs, allowing fluid and protein to leak out of the vascular space into the surrounding interstitial space. Circulating volume decreases with 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, GI tract, lungs) and peripheral tissues by further decreasing perfusion.

TABLE 42.5 Manifestations

Compensatory Stage

Cardiovascular System
Sympathetic nervous system response:
- Epinephrine/norepinephrine (vasoconstrict
- ↑ Myocardial O_2 consumption
- ↑ Contractility
- ↑ HR
- Coronary artery dilation
- Narrowed pulse pressure
- ↓ BP

Gastrointestinal System
↓ Blood supply
↓ GI motility
Hypoactive bowel sounds
↑ Risk for paralytic ileus

Hepatic System

Hematologic System

Neurologic System
Oriented to person, place, time
Restless, apprehensive, confused
Change in level of consciousness

Renal System
↓ Renal blood flow
↑ Renin resulting in release of angiote
↑ Aldosterone causing Na^+ and H_2O re
↑ Antidiuretic hormone causing H_2O re

Respiratory System
- ↓ Blood flow to the lungs:
- ↑ Physiologic dead space
- ↑ Ventilation-perfusion mismatch
- Hyperventilation
- ↑ Minute ventilation (V_E)
- Tachypnea

Skin
Pale and cool
Warm and flushed

Temperature
Normal or abnormal

TABLE 42.5 Manifestations of Stages of Shock

Compensatory Stage	Progressive Stage	Refractory Stage
Cardiovascular System Sympathetic nervous system response: • Epinephrine/norepinephrine (vasoconstriction) release • ↑ Myocardial O_2 consumption • ↑ 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
Gastrointestinal System ↓ Blood supply ↓ GI motility Hypoactive bowel sounds ↑ Risk for paralytic ileus	Vasoconstriction and ↓ perfusion → ischemic gut (e.g., stomach, intestines, gallbladder, pancreas): • Erosive ulcers • GI bleeding • Translocation of GI bacteria • Impaired absorption of nutrients	Ischemic gut
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
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
Renal System ↓ Renal blood flow ↑ Renin resulting in release of angiotensin (vasoconstrictor) ↑ Aldosterone causing Na^+ and H_2O reabsorption ↑ Antidiuretic hormone causing 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
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
Skin Pale and cool Warm and flushed	Cold and clammy	Mottled, cyanotic
Temperature Normal or abnormal	Hypothermia or hyperthermia	Hypothermia

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

The pulmonary system is often the first system to show signs of critical dysfunction. Blood flow to the lungs is reduced. In response to the decreased blood flow and SNS stimulation, the pulmonary arterioles constrict. This increases PA pressure. As the pressure within the pulmonary vasculature increases, blood flow to the pulmonary capillaries decreases and ventilation-perfusion mismatch worsens.

Another key response in the lungs is fluid movement from the pulmonary vasculature into the interstitial space. As capillary permeability increases, fluid moving into the interstitial spaces results in interstitial edema, bronchoconstriction, and decreased 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 46). It increases the risk for bacterial migration from the GI tract to the blood and lungs. There is a decreased ability to absorb nutrients.

The loss of the liver's functional ability leads to a failure of the liver to metabolize drugs and waste products (e.g., lactate, ammonia). Jaundice results from the 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 risk of bacteremia.

Prolonged hypoperfusion on the kidneys causes 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 51). 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.

Hematologic dysfunction adds to the complexity of the clinical picture. The patient is at risk for disseminated intravascular coagulation (DIC). The consumption of 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 34). Table 42.3 shows laboratory values in DIC.

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

Refractory Stage

In this last stage of shock, decreased perfusion from peripheral vasoconstriction and decreased CO worsen anaerobic metabolism. Lactic acid accumulation 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 now 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 an 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 42.7).

Diagnostic Studies

There is no single diagnostic study to determine whether a patient is in shock. The diagnosis starts with a history and physical assessment. A thorough medical and surgical history and a history of recent events (e.g., surgery, chest pain, trauma) give 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.[7] Table 42.2 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 hemodynamic monitoring. Chapter 35 discusses hemodynamic monitoring.

Interprofessional Care

Successful management of a patient in shock depends on the early recognition and treatment of shock. Prompt intervention in the early stages of shock may prevent the decline to the progressive or irreversible stage. Successful management includes (1) identifying patients at risk for developing shock; (2) integrating the patient's history, physical assessment, and clinical findings to establish a diagnosis; (3) interventions to control or eliminate the cause; (4) protecting target and distal organs from dysfunction; and (5) providing multisystem supportive care.

Table 42.6 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 28). The mean arterial pressure (MAP) and circulating blood volume are optimized with fluid replacement and drug therapy.

TABLE 42.6 EMERGENCY MANAGEMENT

Shock

Common Causes

Medical
- Addisonian crisis
- Dehydration
- Diabetes
- Diabetes insipidus
- MI
- Pulmonary embolus
- Sepsis

Surgical
- Aortic dissection
- GI bleeding
- Postoperative bleeding
- Ruptured ectopic pregnancy or ovarian cyst
- Ruptured organ or vessel
- Vaginal bleeding

Trauma
- Fractures, spinal injury
- Multiorgan injury
- Ruptured or lacerated vessel or organ (e.g., spleen)

Assessment Findings
- Anxiety
- Chills
- Confusion
- Cool, clammy skin (warm skin in early onset of septic and neurogenic shock)
- Cyanosis
- Dysrhythmias
- Extreme thirst
- Feeling of impending doom
- Hypotension
- ↓ Level of consciousness
- Narrowed pulse pressure
- Nausea and vomiting
- Obvious hemorrhage or injury
- ↓ O_2 saturation
- Pallor
- Rapid, weak, thready pulses
- Restlessness
- Tachypnea, dyspnea, or shallow, irregular respirations
- Temperature dysregulation
- Weakness

Interventions

Initial
- If unresponsive, assess circulation, airway, and breathing (CAB).
- If responsive, monitor airway, breathing, and circulation (ABC).
- Stabilize cervical spine as appropriate.
- Control any external bleeding with direct pressure or pressure dressing.
- Give high-flow O_2 (100%) by nonrebreather mask or bag-valve-mask.
- Anticipate need for intubation and mechanical ventilation.
- Establish IV access with 2 large-bore catheters (14- to 16-gauge) or an intraosseous access device; aid with central line insertion.
- Begin fluid resuscitation with crystalloids (e.g., 30 mL/kg repeated until hemodynamic improvement is seen).
- Draw blood for laboratory studies (e.g., blood cultures, lactate, WBC).
- Assess for life-threatening injuries (e.g., cardiac tamponade, liver laceration, tension pneumothorax).
- Consider vasopressor therapy if hypotension persists after fluid resuscitation.
- Insert an indwelling urinary catheter and nasogastric tube.
- Start antibiotic therapy after blood cultures if sepsis is suspected.
- Obtain 12-lead ECG and treat dysrhythmias.

Ongoing Monitoring
- ABCs
- Level of consciousness
- Vital signs, including pulse oximetry; peripheral pulses, capillary refill, skin color and temperature
- Respiratory status
- Heart rate and rhythm
- Urine output

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 blood transfusions, 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 Chapters 28 and 35).

Fluid Resuscitation

The cornerstone of therapy for septic, hypovolemic, and anaphylactic shock is volume expansion with fluid administration (Table 42.7).[8] The goal for fluid resuscitation is to restore tissue perfusion. Fluid resuscitation should start using 1 or 2 large-bore (e.g., 14- to 16-gauge) IV catheters, an intraosseous (IO) access device, or a central venous catheter.

! SAFETY ALERT

Intraosseous (IO) Access

- Use an IO access device for emergency resuscitation when IV access cannot be obtained.
- Insertion sites include the sternum, proximal and distal tibia, and proximal and distal humerus.
- Remove IO devices within 24 hr of insertion or as soon as possible after peripheral or central IV access is obtained.
- Monitor for complications: extravasation of drugs and fluids into the soft tissue, fractures caused during insertion, and osteomyelitis.

The choice of fluid is based on the type and volume of fluid lost and the patient's clinical status. The ideal choice of fluid is controversial.[11–13] Currently, normal saline is most often used in the initial resuscitation of shock. Large-volume resuscitation with normal saline can lead to hyperchloremic metabolic acidosis. Lactated Ringer solution can cause serum lactate levels to increase because the failing liver cannot convert lactate to bicarbonate.[13] Transfusions of RBCs may be given to treat hypovolemic shock due to bleeding. Colloids (4% to 5%) have not been shown to improve patient outcomes.[12,13]

Fluid responsiveness is determined by clinical assessment. Although BP helps to determine whether the patient's CO is adequate, an assessment of end-organ perfusion (e.g., urine output, neurologic function, peripheral pulses) provides more

TABLE 42.7 Fluid Therapy in Shock

Fluid Type	Mechanism of Action	Type of Shock	Nursing Implications
Crystalloids			
Isotonic			
• 0.9% NaCl, normal saline solution (NSS) • Lactated Ringer (LR) solution	Fluid primarily stays in the intravascular space, ↑ intravascular volume.	Used for initial volume replacement in most types of shock.	Monitor patient closely for circulatory overload. Do not use LR in patients with liver failure. LR may be used if hyperchloremic acidosis develops from use of NSS in fluid resuscitation.
Hypertonic			
• 1.8%, 3%, 5% NaCl	Fluid stays in the intravascular space, increases serum osmolarity, shifts fluid volume from intracellular space to extracellular space to intravascular space.	May be used for initial volume expansion in hypovolemic shock.	Monitor patient closely for signs of hypernatremia (e.g., disorientation, seizures). Central line preferred for infusing saline solutions ≥3%, since these may damage veins.
Blood or Blood Products			
Packed red blood cells Fresh frozen plasma Platelets	Replaces blood loss, increases O_2-carrying capability. Replaces coagulation factors. Helps control bleeding caused by thrombocytopenia.	All types.	Same precautions as any blood administration (see Chapter 34).
Colloids			
Human serum albumin (5% or 25%)	Can increase plasma colloid osmotic pressure. Rapid volume expansion.	All types except cardiogenic and neurogenic shock.	Use 5% solution in hypovolemic patients. Use 25% solution in patients with fluid and sodium restrictions. Monitor for circulatory overload. Mild side effects of chills, fever, and urticaria may develop. More expensive than crystalloids. Increases risk for bleeding. Monitor patient for allergic reactions and acute kidney injury. Has maximum volume recommendations per manufacturer.
dextran (dextran 40)	Hyperosmotic glucose polymer.	Limited use because of side effects, including reducing platelet adhesion, diluting clotting factors.	

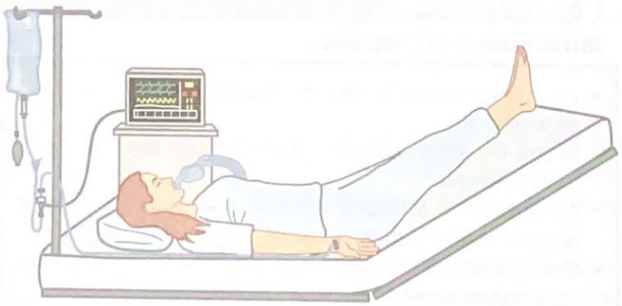

Fig. 42.7 Passive leg raise challenge in a patient with septic shock.

relevant data. Monitor vital signs, cerebral and abdominal perfusion pressures, capillary refill, neurologic status, and skin temperature. Evaluate trends in BP with an automatic BP cuff or an arterial catheter. Use an indwelling urinary catheter to monitor urine output during resuscitation. Hemodynamic parameters, such as SVV or CO, are also used.

Other interventions we use to monitor fluid response include a passive leg raise (PLR) challenge and inferior vena cava evaluation.[14,15] A PLR challenge provides a transient increase in fluid volume of 150 to 500 mL by placing the patient supine and raising the legs to 45 degrees (Fig. 42.7). Response is monitored within 1 to 2 minutes by measuring CO, CI, SV, SVV, or other parameters for improvement. If the response is positive, the patient is fluid responsive and should receive more fluids.

! SAFETY ALERT

Complications of Fluid Resuscitation

- Warm crystalloid and colloid solutions during massive fluid resuscitation to prevent hypothermia.
- When giving large volumes of packed RBCs, remember that they do not contain clotting factors.
- Assess for hypocalcemia and disseminated intravascular coagulation (DIC).
- Replace clotting factors based on the clinical status and laboratory studies.

Drug Therapy

The goal of drug therapy is to correct decreased tissue perfusion. Decisions on which drug to use should be based on the physiologic goal. We give IV drugs used to improve perfusion via an infusion pump and central venous line. Many of these drugs have vasoconstrictor properties that are harmful if the drug leaks into the tissues while being infused peripherally (Table 42.8).

Sympathomimetic drugs. Drugs that mimic the action of the SNS are called *sympathomimetic*. Their effects are mediated through their binding to α- or β-adrenergic receptors. They differ in their relative α- and β-adrenergic effects.[16]

Many of these drugs cause peripheral vasoconstriction and are called *vasopressor drugs* (e.g., norepinephrine, dopamine, phenylephrine). These drugs can cause severe peripheral vasoconstriction and an increase in SVR, further risking

TABLE 42.8 Drug Therapy

Shock

Drug[a]	Mechanism of Action	Type of Shock	Nursing Implications
angiotensin II (Giapreza)	↑ BP, ↑ MAP ↑ SVR	Septic and other distributive shock	Give via central line. Monitor for thromboembolic events. Provide VTE prophylaxis.
dobutamine	↑ Myocardial contractility ↓ Ventricular filling pressures ↓ SVR, PAWP ↑ CO, SV, CVP ↑/↓ HR	Used in cardiogenic shock with severe systolic dysfunction Used in septic shock to increase O_2 delivery and raise $ScvO_2$ or SvO_2 to 70% if Hgb >7 g/dL or Hct ≥30% Cardiogenic shock	Give via central line (infiltration leads to tissue sloughing). Do not give in same line with $NaHCO_3$. Monitor HR, BP (hypotension may worsen, requiring addition of a vasopressor). Stop infusion if tachydysrhythmias develop.
dopamine	Positive inotropic effects: ↑ Myocardial contractility ↑ Automaticity ↑ Atrioventricular conduction ↑ HR, CO ↑ BP, ↑ MAP ↑ MVO_2 Can cause progressive vasoconstriction at high doses		Give via central line (infiltration leads to tissue sloughing). Do not give in same line with $NaHCO_3$. Monitor for tachydysrhythmias. Monitor for peripheral vasoconstriction (e.g., paresthesias, coldness in extremities) at moderate to high doses.
epinephrine (Adrenalin)	*Low doses:* β-Adrenergic agonist (cardiac stimulation, bronchodilation, peripheral vasodilation) ↑ HR, contractility, CO ↓ SVR *High doses:* α-Adrenergic agonist (peripheral vasoconstriction) ↑ SV, SVR ↑ Systolic/↓ diastolic BP, widened pulse pressure ↑ CVP, PAWP	Cardiogenic shock Anaphylactic shock Septic shock, if 2nd agent needed after norepinephrine Cardiac arrest, pulseless ventricular tachycardia, ventricular fibrillation, asystole	Monitor for HR >110 beats/min. Monitor for dyspnea, pulmonary edema. Monitor for chest pain, dysrhythmias from ↑ MVO_2. Monitor for renal failure due to ischemia.
hydrocortisone (Solu-Cortef)	↓ Inflammation, reverses ↑ capillary permeability ↑ BP, HR	Septic shock requiring vasopressor therapy (despite fluid resuscitation) to maintain adequate BP Anaphylactic shock if hypotension persists after initial therapy	Monitor for hypokalemia, hyperglycemia. Consider use as continuous infusion.
nitroglycerin	Venous dilation Dilates coronary arteries ↓ Preload, MVO_2, SVR, BP	Cardiogenic shock	Continuously monitor BP and HR, since reflex tachycardia may occur. Glass bottle recommended for infusion.
norepinephrine (Levophed)	β_1-Adrenergic agonist (cardiac stimulation) α-Adrenergic agonist (peripheral vasoconstriction) Renal and splanchnic vasoconstriction ↑ BP, MAP, CVP, PAWP, SVR ↑/↓ CO	Cardiogenic shock after MI Septic shock—first drug of choice for BP unresponsive to adequate fluid resuscitation	Give via central line (infiltration leads to tissue sloughing). Monitor for dysrhythmias due to ↑ MVO_2 requirements.
phenylephrine	α-Adrenergic agonist (peripheral vasoconstriction) Renal, mesenteric, splanchnic, cutaneous, and pulmonary blood vessel constriction ↑ HR, BP, SVR ↑/↓ CO	Neurogenic shock	Monitor for reflex bradycardia, headache, restlessness. Monitor for renal failure from ↓ renal blood flow. Give via central line (infiltration leads to tissue sloughing).
sodium nitroprusside	Arterial and venous vasodilation ↓ Preload, afterload ↓ CVP, PAWP ↑/↓ CO ↓ BP	Cardiogenic shock with ↑ SVR	Continuously monitor BP. Protect solution from light. Wrap infusion bottle with opaque covering. Give with D_5W only. Monitor serum cyanide levels and for signs of cyanide toxicity (e.g., metabolic acidosis, tachycardia, altered level of consciousness, seizures, coma, almond smell on breath).
vasopressin	Antidiuretic hormone Nonadrenergic vasoconstrictor ↑ MAP ↑ Urine output	Shock states (most often septic shock) refractory to other vasopressors	Given with norepinephrine and in low doses. Infusions are not titrated. Monitor hemodynamic pressures and urine output.

[a]Consult agency guidelines, pharmacist, pharmacology references, and drug manufacturer's materials for more information and dosing recommendations.

CVP, Central venous pressure; *MVO_2*, myocardial O_2 consumption; *PAWP*, pulmonary artery wedge pressure; *SVR*, systemic vascular resistance.

tissue perfusion. The increased SVR increases the workload of the heart and myocardial O_2 demand. It can harm a patient in cardiogenic shock by causing further myocardial damage and increasing the risk for dysrhythmias.[17] Use of vasopressor drugs is limited to patients who do not respond to fluid resuscitation. Adequate fluid resuscitation must be achieved before starting vasopressors because the vasoconstrictor effects in patients with low blood volume will further reduce tissue perfusion. Typically, if hypotension persists after adequate fluid resuscitation, we then give a vasopressor (e.g., norepinephrine, dopamine) and/or an inotrope (e.g., dobutamine).

The goal of vasopressor therapy is to achieve and maintain a MAP of greater than 65 mm Hg.[16] Continuously monitor end-organ perfusion (e.g., urine output, level of consciousness) and serum lactate levels (e.g., every 3 hours for the first 6 hours) to ensure that tissue perfusion is adequate.

Vasodilator drugs. Patients in cardiogenic shock have decreased myocardial contractility. Vasodilators may be needed to decrease afterload. This reduces myocardial workload and O_2 requirements. Although generalized sympathetic vasoconstriction is a useful compensatory mechanism for maintaining BP, excessive constriction can reduce tissue blood flow and increase the workload of the heart. The reason for using vasodilator therapy is to break the harmful cycle of widespread vasoconstriction causing a decrease in CO and BP, resulting in further sympathetic-induced vasoconstriction.

The goal of vasodilator therapy, as in vasopressor therapy, is to maintain the MAP greater than 65 mm Hg. Monitor hemodynamic parameters (e.g., CVP, CO, $ScvO_2/SvO_2$, SV, PA pressures) and assessment findings so that fluids can be increased, or vasodilator therapy decreased if a serious fall in CO or BP occurs. The vasodilator agent most often used for the patient in cardiogenic shock is nitroglycerin. Vasodilation may be enhanced with nitroprusside or nitroglycerin in noncardiogenic shock.

Nutrition Therapy

Protein-calorie malnutrition is common because of hypermetabolism. Nutrition is vital to reducing mortality.[18] Enteral nutrition (EN) should be started within the first 24 hours. However, full calorie replacement is not recommended for previously well-nourished adults early in a critical illness.[18] Start the patient on a *trophic feeding*. This is a small amount of EN (e.g., 10 mL/hr). Early EN enhances perfusion of the GI tract and helps maintain the integrity of the gut mucosa. Advance feedings as tolerated and as prescribed. We use parenteral nutrition (PN) only if EN is contraindicated. Chapter 44 discusses EN and PN.

Weigh the patient daily on the same scale at the same time of day. If the patient has a significant weight loss, rule out dehydration before adding more calories. Large weight gains are common because of third spacing of fluids. Monitor serum protein, total albumin, prealbumin, BUN, serum glucose, and serum electrolytes to assess nutrition status.

Measures Specific to Type of Shock

Cardiogenic Shock

For a patient in cardiogenic shock, the overall goal is to restore heart function and the balance between O_2 supply and demand in the myocardium. Cardiac catheterization is done as soon as possible after the initial insult.[1] Specific measures to restore blood flow include angioplasty with stenting, emergency revascularization, and valve replacement. Until these interventions are done, we must support the heart to optimize SV and CO to achieve optimal perfusion (Tables 42.8 and 42.9).[12]

Hemodynamic management aims to reduce the workload of the heart through drug therapy and/or mechanical intervention. Drug choice is based on the clinical goal. Drugs can decrease the workload of the heart by dilating coronary arteries (e.g., nitrates) and reducing preload (e.g., diuretics), afterload (e.g., vasodilators), and heart rate and contractility (e.g., β-adrenergic blockers).

The patient may benefit from a circulatory assist device (e.g., intraaortic balloon pump, ventricular assist device [VAD]) (see Chapter 39). The goals are to decrease SVR and left ventricular workload so that the heart can heal. A VAD may be used as a temporary measure for the patient in cardiogenic shock who is awaiting heart transplantation. Heart transplantation is an option for a small, select group of patients with cardiogenic shock.

Hypovolemic Shock

The underlying principles of managing hypovolemic shock focus on stopping the fluid loss and restoring circulating volume. We often calculate the initial fluid resuscitation using a 3:1 rule (3 mL of isotonic crystalloid for every 1 mL of estimated blood loss). Table 42.7 describes fluids used for volume resuscitation, their mechanism of action, and specific nursing implications.

Septic Shock

Patients in septic shock need large amounts of fluid replacement (Fig. 42.8). The overall goal of fluid resuscitation is to restore the intravascular volume and organ perfusion. We achieve initial volume resuscitation by giving 30 mL/kg of an isotonic crystalloid solution. Albumin 4% to 5% is an option when patients need substantial volume.

A fluid challenge technique (e.g., a minimum of 30 mL/kg of crystalloids) may be done and repeated until hemodynamic improvement (e.g., increase in MAP and/or CVP) is seen. To optimize and evaluate large-volume fluid resuscitation, hemodynamic monitoring is needed. Table 42.9 shows predetermined end points of fluid resuscitation along with methods to reassess volume status.

If the patient is hypotensive after initial volume resuscitation and no longer fluid responsive, we may add vasopressors. The first drug of choice is norepinephrine.[16] Vasodilation and low CO, or vasodilation alone, can cause low BP despite adequate fluid resuscitation. Vasopressin may be added for those who are refractory to initial vasopressor therapy.[17] Exogenous vasopressin can replace the stores of physiologic vasopressin that are often depleted in septic shock.

DRUG ALERT

Vasopressin

- Given along with norepinephrine.
- Infuse at low doses (e.g., 0.03 units/min) using an IV pump.
- Do not titrate infusion.
- Use cautiously in patients with coronary artery disease.

TABLE 42.9 Interprofessional Care

Shock / Oxygenation	Circulation	Drug Therapies	Supportive Therapies
Cardiogenic Shock • Provide supplemental O_2 (e.g., nasal cannula, nonrebreather mask) • Intubation and mechanical ventilation, if needed • Monitor $ScvO_2$ or SvO_2	• Restore blood flow with angioplasty with stenting, emergent coronary revascularization • Reduce workload of heart with circulatory assist devices: IABP, VAD	• Nitrates (e.g., nitroglycerin) • Inotropes (e.g., dobutamine) • Diuretics (e.g., furosemide) • β-Adrenergic blockers (contraindicated with ↓ ejection fraction)	• Treat dysrhythmias
Hypovolemic Shock • Provide supplemental O_2 • Monitor $ScvO_2$ or $ScvO_2$	• Rapid fluid replacement using 2 large-bore (14–16 gauge) peripheral IV lines, an intraosseous access device, or central venous catheter • Restore fluid volume (e.g., blood or blood products, crystalloids) • End points of fluid resuscitation: • CVP 15 mm Hg • PAWP 10–12 mm Hg	• No specific drug therapy	• Correct the cause (e.g., stop bleeding, GI losses) • Use warmed IV fluids, including blood products (if appropriate)
Septic Shock • Provide supplemental O_2 • Intubation and mechanical ventilation, if needed • Monitor $ScvO_2$ or SvO_2	• Aggressive fluid resuscitation (e.g., 30 mL/kg of crystalloids repeated if hemodynamic improvement is noted) • End points of fluid resuscitation are based on: • Focused physical assessment including vital signs, cardiopulmonary assessment, capillary refill, peripheral pulses, and skin or any 2 of the following: • $ScvO_2$ >70 or SvO_2 >65 • CVP 8–12 mm Hg • Cardiovascular ultrasound • Fluid responsiveness with passive leg raise or fluid challenge	• Antibiotics as ordered • Vasopressors (e.g., norepinephrine) • Inotropes (e.g., dobutamine) • Anticoagulants (e.g., low-molecular-weight heparin)	• Obtain cultures (e.g., blood, wound) before beginning antibiotics • Monitor temperature • Control blood glucose • Stress ulcer prophylaxis
Neurogenic Shock • Maintain patent airway • Provide supplemental O_2 • Intubation and mechanical ventilation (if needed)	• Cautious administration of fluids	• Vasopressors (e.g., phenylephrine) • Atropine (for bradycardia)	• Minimize spinal cord trauma with stabilization • Monitor temperature
Anaphylactic Shock • Maintain patent airway • Optimize oxygenation with supplemental O_2 • Intubation and mechanical ventilation, if needed	• Aggressive fluid resuscitation with colloids	• Epinephrine (IM or IV) • Antihistamines (e.g., diphenhydramine) • Histamine (H_2)-receptor blockers • Bronchodilators: nebulized (e.g., albuterol) • Corticosteroids (if hypotension persists)	• Identify and remove offending cause • Prevent via avoidance of known allergens • Premedicate with history of prior sensitivity (e.g., contrast media)
Obstructive Shock • Maintain patent airway • Provide supplemental O_2 • Intubation and mechanical ventilation, if needed	• Restore circulation by treating cause of obstruction • Fluid resuscitation may provide temporary improvement in CO and BP	• No specific drug therapy	• Treat cause of obstruction (e.g., pericardiocentesis for cardiac tamponade, needle decompression or chest tube insertion for tension pneumothorax, embolectomy for pulmonary embolism)

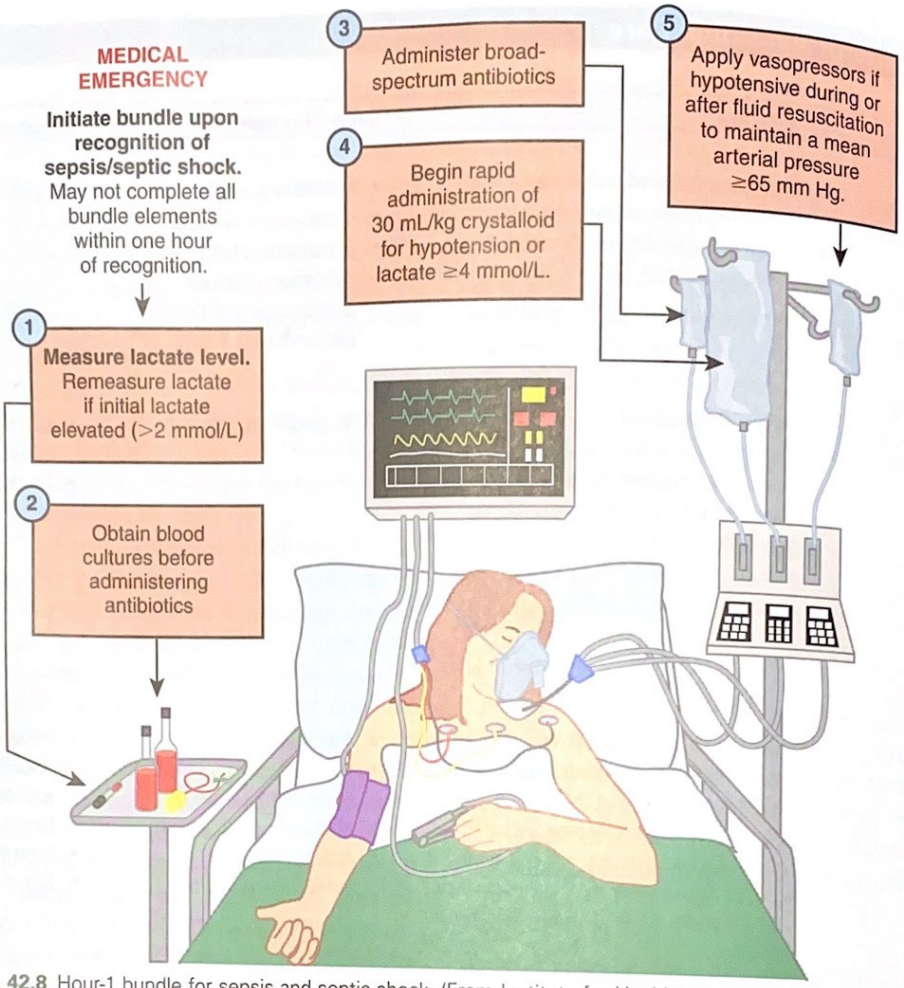

Fig. 42.8 Hour-1 bundle for sepsis and septic shock. (From Institute for Healthcare Improvement: Surviving Sepsis campaign.)

Vasopressor drugs may increase BP but can decrease SV. Giving an inotropic agent (e.g., dobutamine) can offset the decrease in SV and increase tissue perfusion (Table 42.8). IV corticosteroids may be considered for patients in septic shock who cannot maintain an adequate BP despite vasopressor therapy and fluid resuscitation.

To try to meet the increasing tissue demands coupled with a low SVR, the patient initially has a normal or high CO. If the patient is unable to achieve and maintain an adequate CO and has unmet tissue O_2 demands, CO may have to be increased using drug therapy (e.g., dopamine). $ScvO_2$ or SvO_2 monitoring is used to assess the balance between O_2 delivery and consumption, and adequacy of the CO (see Chapter 35). If balance is maintained, tissue demands will be met.

Broad-spectrum antibiotics are an important and early part of therapy. They should be started within the first hour of sepsis or septic shock.[19] Obtain cultures (e.g., blood, wound, urine, stool, sputum) before starting antibiotics. However, this should not delay the start of antibiotics within the first hour. Specific antibiotics may be ordered after we identify the organism.

Glucose levels should be maintained below 180 mg/dL (10.0 mmol/L) for patients in shock.[7] Monitor glucose levels according to agency policy. Stress ulcer prophylaxis with proton pump inhibitors (e.g., pantoprazole) and VTE prophylaxis (e.g., heparin, enoxaparin [Lovenox]) are recommended.[7]

Neurogenic Shock

The specific treatment of neurogenic shock is based on the cause. If the cause is spinal cord injury, we use general measures to promote spinal stability (e.g., spinal precautions, cervical stabilization with a collar). Once the spine is stabilized, treating hypotension and bradycardia are essential to prevent further spinal cord damage. Treatment involves vasopressors (e.g., phenylephrine) to maintain BP and organ perfusion (Table 42.8). Bradycardia may be treated with atropine. Infuse fluids cautiously because hypotension is not related to fluid loss. The patient with a spinal cord injury is monitored for hypothermia caused by hypothalamic dysfunction (Table 42.9).

Anaphylactic Shock

The first strategy in managing patients at risk for anaphylactic shock is prevention. A thorough history is key to avoiding risk factors for anaphylaxis (Table 42.1). The clinical presentation of anaphylactic shock is dramatic. Immediate intervention is required. Epinephrine is the drug of choice to treat anaphylactic

shock.* It causes peripheral vasoconstriction and bronchodilation and opposes the effect of histamine. Diphenhydramine and histamine receptor blockers (e.g., famotidine) are given as adjunctive therapies to block the ongoing release of histamine from the allergic reaction.

Maintaining a patent airway is important. The patient can quickly develop airway compromise from laryngeal edema or bronchoconstriction. Nebulized bronchodilators are highly effective. Aerosolized epinephrine can reduce treat laryngeal edema. Endotracheal intubation may be needed to secure and maintain a patent airway.

Hypotension results from fluid leaking out of the intravascular space into the interstitial space due to increased capillary permeability and vasodilation. Aggressive fluid resuscitation, usually with crystalloids, is needed. IV corticosteroids may be helpful in anaphylactic shock if significant hypotension persists after 1 to 2 hours of aggressive therapy (Tables 42.8 and 42.9).

Obstructive Shock

The main strategy in treating obstructive shock is early recognition and treatment to relieve or manage the obstruction (Table 42.1). Mechanical decompression for pericardial tamponade, tension pneumothorax, and hemopneumothorax may be done by needle or tube insertion. A pulmonary embolism requires immediate anticoagulation therapy, thrombolytic therapy, or pulmonary embolectomy. Superior vena cava syndrome, a compression or obstruction of the outflow tract of the mediastinum, may be treated by radiation, debulking, or removal of the mass or cause. A decompressive laparotomy may be done for abdominal compartment syndrome for patients with high intraabdominal pressures and hemodynamic instability.

NURSING MANAGEMENT: SHOCK

Assessment

Focus your assessment on the ABCs: airway, breathing, and circulation. Next, assess for tissue perfusion. This includes evaluating vital signs, level of consciousness, peripheral pulses, capillary refill, skin (e.g., temperature, color, moisture), and urine output. As shock progresses, the patient's neurologic status declines, urine output decreases, skin becomes cooler and mottled, and peripheral pulses decrease.

To understand the complexity of the patient's clinical status, integrate all the assessment data. Obtain a brief history from the patient or caregiver. Include a description of the events leading to the shock state, time of onset and duration of symptoms, and health history (e.g., medications, allergies). Obtain details about any care the patient received before hospitalization.

Clinical Problems

Clinical problems for the patient in shock may include:

- Impaired cardiac function
- Impaired respiratory function
- Altered BP

More information on nursing diagnoses and interventions for the patient with shock is presented in eNursing Care Plan 42.1 (available on the website for this chapter).

Planning

The overall goals for a patient in shock include (1) adequate tissue perfusion, (2) restoration of normal or baseline BP, (3) recovery of organ function, (4) avoiding complications from prolonged states of hypoperfusion, and (5) preventing health care–associated complications.

Implementation

Health Promotion

You play a key role in the prevention of shock by identifying patients at risk. In general, patients who are older, are immunocompromised, or have chronic illnesses are at an increased risk. Any person who has surgery or trauma is at risk for shock from hemorrhage, spinal cord injury, sepsis, and other problems (Table 42.1).

Planning is essential to help prevent shock after you identify an at-risk patient. For example, a patient with an acute anterior wall MI is at high risk for cardiogenic shock.[1] The main goal for this patient is to limit the infarct size. This is done by restoring coronary blood flow. Rest, analgesics, and sedation can reduce the myocardial demand for O_2. Modify the ICU environment to provide care at intervals that will not increase the patient's O_2 demand. For example, if the patient becomes tired with bathing, perform this care at a time that does not interfere with tests or other activities that may increase O_2 demand.

A person with certain severe allergies, such as to drugs, shellfish, insect bites, and latex, is at increased risk for anaphylactic shock. This risk can be decreased by carefully assessing the patient for allergies.

! SAFETY ALERT

Preventing Allergic Reactions

- Always confirm the patient's allergies before giving drugs or starting diagnostic procedures (e.g., CT scan with contrast media).
- Premedicate (e.g., diphenhydramine, methylprednisolone) patients who need a drug to which they are at high risk for an allergic reaction (e.g., contrast media).
- Encourage patients with allergies to obtain and wear a medical alert device and report all allergies to their HCPs.
- Teach patients about kits that contain equipment and drugs (e.g., epinephrine) for the treatment of acute allergic reactions.

Careful monitoring of fluid balance can help prevent hypovolemic shock. Ongoing monitoring of intake and output and daily weights is important. Identifying trends in the patient's condition is more meaningful than any single piece of clinical information.

Carefully monitor all patients for infection. Progression from an infection to sepsis and septic shock depends on the patient's defense mechanisms. Patients who are immunocompromised are at high risk for opportunistic infections. Strategies to decrease the risk for health care–associated