CHAPTER

11

Shock, Sepsis, and Multiple Organ Dysfunction Syndrome

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INTRODUCTION

Shock is a clinical syndrome characterized by inadequate tissue perfusion that results in cellular, metabolic, and hemodynamic derangements. Impaired tissue perfusion occurs when there is an imbalance between cellular oxygen supply and cellular oxygen demand. Shock can result from ineffective cardiac function, inadequate blood volume, or inadequate vascular tone. The effects of shock are not isolated to one organ system; instead, all body systems may be affected. Shock can progress to organ failure and death unless compensatory mechanisms reverse the process, or clinical interventions are successfully implemented. Shock frequently results in systemic inflammatory response syndrome (SIRS) and multiple organ dysfunction syndrome (MODS). Shock is associated with many causes and a variety of clinical manifestations. Patient responses to shock and treatment strategies vary, thus presenting a challenge to the healthcare team in assessment and management.

This chapter discusses the various clinical conditions that create the shock state including hypovolemia, cardiogenic shock, distributive shock (anaphylactic, neurogenic, and septic shock), and obstructive shock. The progression of shock to SIRS and MODS is also described. The pathophysiology, clinical presentation, and definitive and supportive management of each type of shock state are reviewed.

REVIEW OF ANATOMY AND PHYSIOLOGY

The cardiovascular system is a closed, interdependent system composed of the heart, blood, and vascular bed. Arteries, arterioles, capillaries, venules, and veins make up the vascular bed. The microcirculation, the portion of the vascular bed between the arterioles and the venules, is the most significant portion of the circulatory system for cell survival. Its functions are the delivery of oxygen and nutrients to cells, the removal of waste products of cellular metabolism, and the regulation of blood volume. In addition, the vessels of the microcirculation constrict or dilate selectively to regulate blood flow to cells in need of oxygen and nutrients.

The structure of the microcirculation differs according to the function of the tissues and organs it supplies; however, all of the vascular beds have common structural characteristics (Figure 11-1). As oxygenated blood leaves the left side of the heart and enters the aorta, it flows through progressively smaller arteries until it flows into an arteriole. Arterioles are lined with smooth muscle, which allows these small vessels to change diameter and, as a result, to direct and adjust blood flow to the capillaries. From the arteriole, blood enters a metarteriole, a smaller vessel that branches from the arteriole at right angles. Metarterioles are partially lined with smooth muscle, which also allows them to adjust diameter size and to regulate blood flow into capillaries.

Blood next enters the capillary network by passing through a muscular precapillary sphincter. Capillaries are narrow, thin-walled vascular networks that branch off the metarterioles. This network configuration increases the surface area to allow for greater fluid and nutrient exchange. It also decreases the velocity of the blood flow to prolong transport time through the capillaries. Capillaries have no contractile ability and are not responsive to vasoactive chemicals, electrical or mechanical stimulation, or pressure across their walls. The precapillary sphincter is the only means of regulating blood flow into a capillary. When the

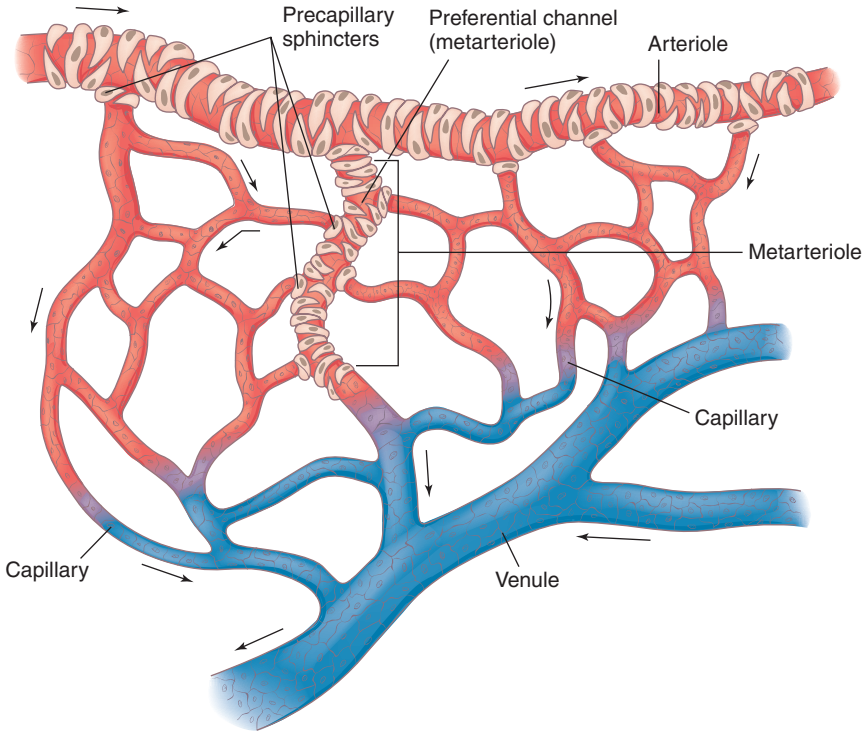

FIGURE 11-1 Microcirculation. (From McCance KL, Huether SE. *Pathophysiology. The Biologic Basis for Disease in Adults and Children.* 6th ed. St. Louis: Mosby; 2010.)

precapillary sphincter constricts, blood flow is diverted away from a capillary bed and directed to one that supplies tissues in need of oxygen and nutrients. The capillary bed lies close to the cells of the body, a position that facilitates the delivery of oxygen and nutrients to the cells.

Once nutrients are exchanged for cellular waste products in the capillaries, blood enters a venule. These small muscular vessels are able to dilate and constrict, offering postcapillary resistance for the regulation of blood flow through capillaries. Blood then flows from the venule and enters the larger veins of the venous system. Another component of the microcirculation consists of the arteriovenous anastomoses that connect arterioles directly to venules. These muscular vessels are able to shunt blood away from the capillary circulation and send it directly to tissues in need of oxygen and nutrients.

Blood pressure is determined by cardiac output and systemic vascular resistance (SVR). Blood pressure is decreased whenever there is a decrease in cardiac output (hypovolemic, cardiogenic, or obstructive shock) or SVR (neurogenic, anaphylactic, or septic shock). Changing pressures within the vessels as blood moves from an area of high pressure within the arteries and passes to the venous system, which has lower pressures, facilitate the flow of blood. The force of resistance opposes blood flow; thus as resistance increases, blood flow decreases. Resistance is determined by three factors: (1) vessel length, (2) blood viscosity, and (3) vessel diameter. Increased resistance occurs with increased vessel length, increased blood viscosity, and decreased blood vessel diameter. Vessel diameter is the most important determinant of resistance.

As the pressure of blood within the vessel decreases, the diameter of the vessel decreases, resulting in decreased blood flow. The critical closing pressure and the resultant cessation of blood flow occur when blood pressure decreases to a point at which it is no longer able to keep the vessel open.

The delivery of oxygen to tissues and cells is required for the production of cellular energy (adenosine triphosphate [ATP]). The delivery of oxygen (DO_2) requires an adequate hemoglobin level to carry oxygen, adequate functioning of the lungs to oxygenate the blood and saturate the hemoglobin (SaO_2), and adequate cardiac functioning (cardiac output) to transport the oxygenated blood to the tissues and cells. Any impairment in the DO_2, or any increase in the consumption of oxygen by the tissues (VO_2), causes a decrease in oxygen reserve (as indicated by the mixed venous oxygen saturation [SvO_2]), which may result in tissue hypoxia, depletion of the supply of ATP, lactic acidosis, organ dysfunction, and potentially death.

Pathophysiology

Diverse events can initiate the shock syndrome. Shock begins when the cardiovascular system fails to function properly because of an alteration in at least one of the four essential circulatory components: blood volume, myocardial contractility, blood flow, or vascular resistance. Under healthy circumstances, these components function together to maintain circulatory homeostasis. When one of these components fails, the others compensate. However, as compensatory mechanisms fail, or if more than one of the

TABLE 11-1 CLASSIFICATION OF SHOCK

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

circulatory components is affected, a state of shock ensues. Shock states are classified according to which one of these components is adversely affected (Table 11-1).

Shock is not a single clinical entity but a life-threatening response to alterations in circulation resulting in impaired tissue perfusion. As the delivery of adequate oxygen and nutrients decreases, impaired cellular metabolism occurs. Cells convert from aerobic to anaerobic metabolism. Less energy in the form of ATP is produced. Lactic acid, a by-product of anaerobic metabolism, causes tissue acidosis. Cells in all organ systems require energy to function, and this resultant tissue acidosis impairs cellular metabolism. Shock is not selective in its effects—all cells, tissues, and organ systems suffer as a result of the physiological response to the stress of shock and decreased tissue perfusion. The end result is organ dysfunction because of decreased blood flow through the capillaries that supply the cells with oxygen and nutrients (Figure 11-2).

Stages of Shock

Although the response to shock is highly individualized, a pattern of stages progresses at unpredictable rates. If each stage of shock is not recognized and treated promptly, progression to the next stage occurs. The pathophysiological events and associated clinical findings for each stage are summarized in Table 11-2.

Stage I: Initiation

The process of shock is initiated by subclinical hypoperfusion that is caused by inadequate DO_2, inadequate extraction of oxygen, or both. No obvious clinical indications of hypoperfusion are noted in this stage although hemodynamic alterations, such as a decrease in cardiac output, are noted if invasive hemodynamic monitoring is used for patient assessment.

Stage II: Compensatory Stage

The sustained reduction in tissue perfusion initiates a set of neural, endocrine, and chemical compensatory mechanisms in an attempt to maintain blood flow to vital organs and to restore homeostasis. During this stage, symptoms become apparent, but shock may still be reversed with minimal morbidity if appropriate interventions are initiated.

Neural compensation. Baroreceptors (which are sensitive to pressure changes) and chemoreceptors (which are sensitive to chemical changes) located in the carotid sinus and aortic arch detect the reduction in arterial blood pressure. Impulses are relayed to the vasomotor center in the medulla oblongata, stimulating the sympathetic branch of the autonomic nervous system to release epinephrine and norepinephrine from the adrenal medulla. In response to this catecholamine release, both heart rate and contractility increase to improve cardiac output. Dilation of the coronary arteries occurs to increase perfusion to the myocardium to meet the increased demands for oxygen. Systemic vasoconstriction and redistribution of blood occurs. Arterial vasoconstriction improves blood pressure, whereas venous vasoconstriction augments venous return to the heart, increasing preload and cardiac output. Blood is shunted from the kidneys, gastrointestinal tract, and skin to the heart and brain. Bronchial smooth muscles relax, and respiratory rate and depth are increased, improving gas exchange and oxygenation. Additional catecholamine effects include increased blood glucose levels as the liver is stimulated to convert glycogen to glucose for energy production; dilation of pupils; and peripheral vasoconstriction and increased sweat gland activity resulting in cool, moist skin.

Endocrine compensation. In response to the reduction in blood pressure, messages are also relayed to the hypothalamus, which stimulates the anterior and posterior pituitary gland. The anterior pituitary gland releases adrenocorticotropic hormone (ACTH), which acts on the adrenal cortex to release glucocorticoids and mineralocorticoids (e.g., aldosterone). Glucocorticoids increase the blood glucose level by increasing the conversion of glycogen to glucose (glycogenolysis) and causing the conversion of fat and protein to glucose (gluconeogenesis). Mineralocorticoids act on the renal tubules causing the reabsorption of sodium and water, resulting in increased intravascular volume and blood pressure. The renin-angiotensin-aldosterone system (Figure 11-3) is stimulated by a reduction of pressure in the renal arterioles of the kidneys and/or by a decrease in sodium levels as sensed by the kidney's juxtaglomerular apparatus. In response to decreased renal perfusion, the juxtaglomerular apparatus releases renin. Renin circulates in the blood and reacts with angiotensinogen to produce angiotensin I. Angiotensin I circulates through the lungs, where it forms angiotensin II, a potent arterial and venous vasoconstrictor that increases blood pressure and improves venous return to the heart. Angiotensin II also activates the adrenal cortex to release aldosterone.

Antidiuretic hormone (ADH) is released by the posterior pituitary gland in response to the increased osmolality of the blood that occurs in shock. The overall effects of endocrine compensation result in an attempt to combat shock by providing the body with glucose for energy and by increasing the intravascular blood volume.

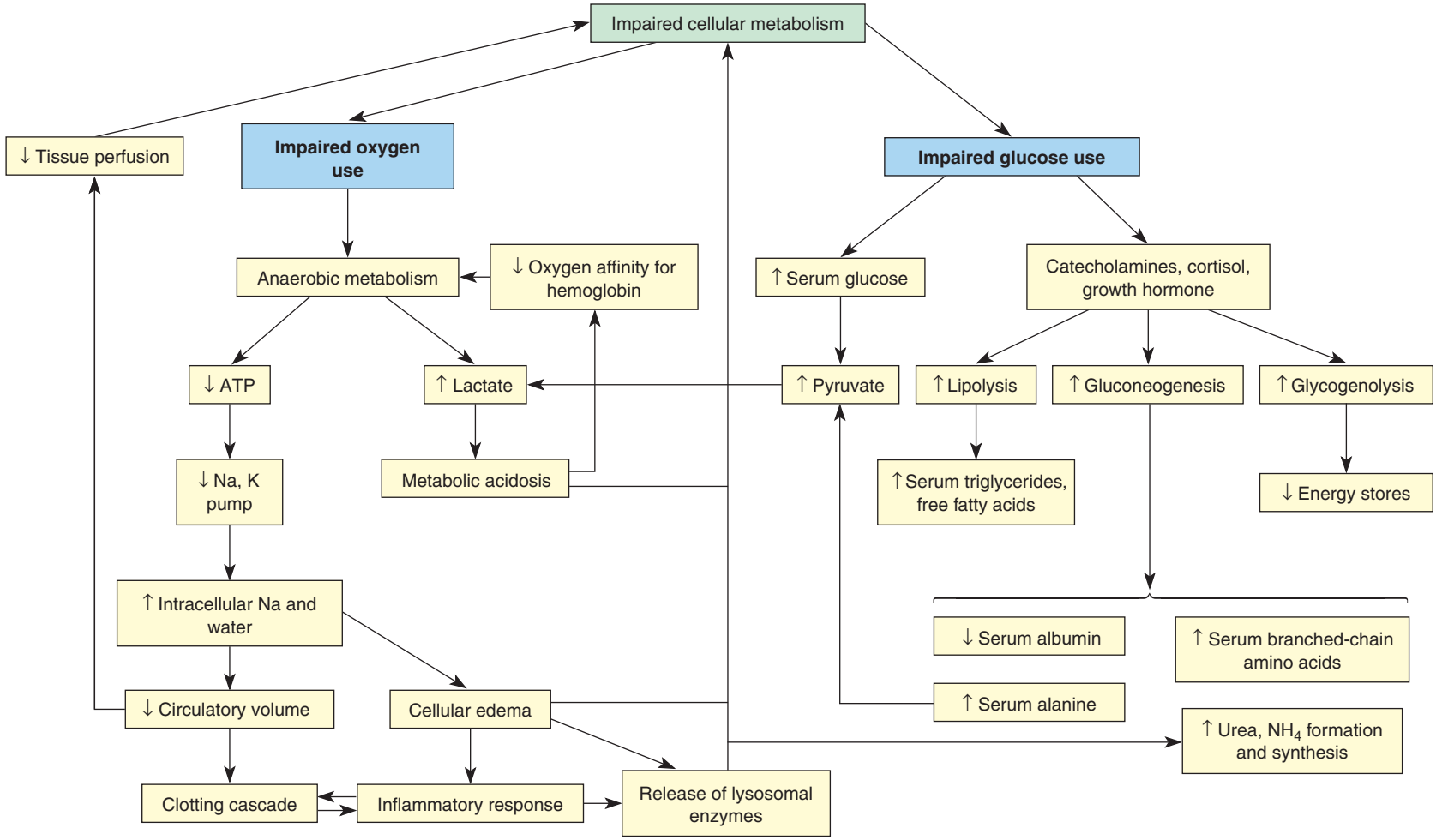

FIGURE 11-2 Impairment of cellular metabolism by shock. *ATP,* Adenosine triphosphate; *K,* potassium; *Na,* sodium; *NH_4,* ammonia. (From McCance KL, Huether SE. *Pathophysiology. The Biologic Basis for Disease in Adults and Children*. 6th ed. St. Louis: Mosby; 2010.)

TABLE 11-2 STAGES OF SHOCK

STAGE OF SHOCK	PHYSIOLOGICAL EVENTS	CLINICAL PRESENTATION
I: Initiation	↓ Tissue oxygenation caused by: ↓ Intravascular volume (hypovolemic) ↓ Myocardial contractility (cardiogenic) Obstruction of blood flow (obstructive) ↓ Vascular tone (distributive) Septic (mediator release) Anaphylactic (histamine release) Neurogenic (suppression of SNS)	No observable clinical indications ↓ CO may be noted with invasive hemodynamic monitoring
II: Compensatory	**Neural compensation by SNS** ↑ Heart rate and contractility Vasoconstriction Redistribution of blood flow from nonessential to essential organs Bronchodilation **Endocrine compensation** (RAAS, ADH, glucocorticoids release) Renal reabsorption of sodium, chloride, and water Vasoconstriction Glycogenolysis and gluconeogenesis **Chemical compensation**	↑ Heart rate (except neurogenic) Narrowed pulse pressure Rapid, deep respirations causing respiratory alkalosis Thirst Cool, moist skin Oliguria Diminished bowel sounds Restlessness progressing to confusion Hyperglycemia ↑ Urine specific gravity and ↓ creatinine clearance
III: Progressive	Progressive tissue hypoperfusion Anaerobic metabolism with lactic acidosis Failure of sodium-potassium pump Cellular edema	Dysrhythmias ↓ BP with narrowed pulse pressure Tachypnea Cold, clammy skin Anuria Absent bowel sounds Lethargy progressing to coma Hyperglycemia ↑ BUN, creatinine, and potassium Respiratory and metabolic acidosis
IV: Refractory	Severe tissue hypoxia with ischemia and necrosis Worsening acidosis SIRS MODS	Life-threatening dysrhythmias Severe hypotension despite vasopressors Respiratory and metabolic acidosis Acute respiratory failure Acute respiratory distress syndrome Disseminated intravascular coagulation Hepatic dysfunction/failure Acute kidney injury Myocardial ischemia/infarction/failure Cerebral ischemia/infarction

ADH, Antidiuretic hormone; *BP,* blood pressure; *BUN,* blood urea nitrogen; *CO,* cardiac output; *MODS,* multiple organ dysfunction syndrome; *RAAS,* renin-angiotensin-aldosterone system; *SIRS,* systemic inflammatory response syndrome; *SNS,* sympathetic nervous system.

Chemical compensation. As pulmonary blood flow is reduced, ventilation-perfusion imbalances occur. Initially, alveolar ventilation is adequate, but the perfusion of blood through the alveolar capillary bed is decreased. Chemoreceptors located in the aorta and carotid arteries are stimulated in response to this low oxygen tension in the blood. Consequently, the rate and depth of respirations increase. As the patient hyperventilates, carbon dioxide is excreted and respiratory alkalosis occurs. A reduction in carbon dioxide levels and the alkalotic state cause vasoconstriction of cerebral blood vessels. This vasoconstriction, coupled with the reduced oxygen tension, may lead to cerebral hypoxia and ischemia. The overall effects of chemical compensation result in an attempt to combat shock by increasing oxygen supply; however, cerebral perfusion may decrease.

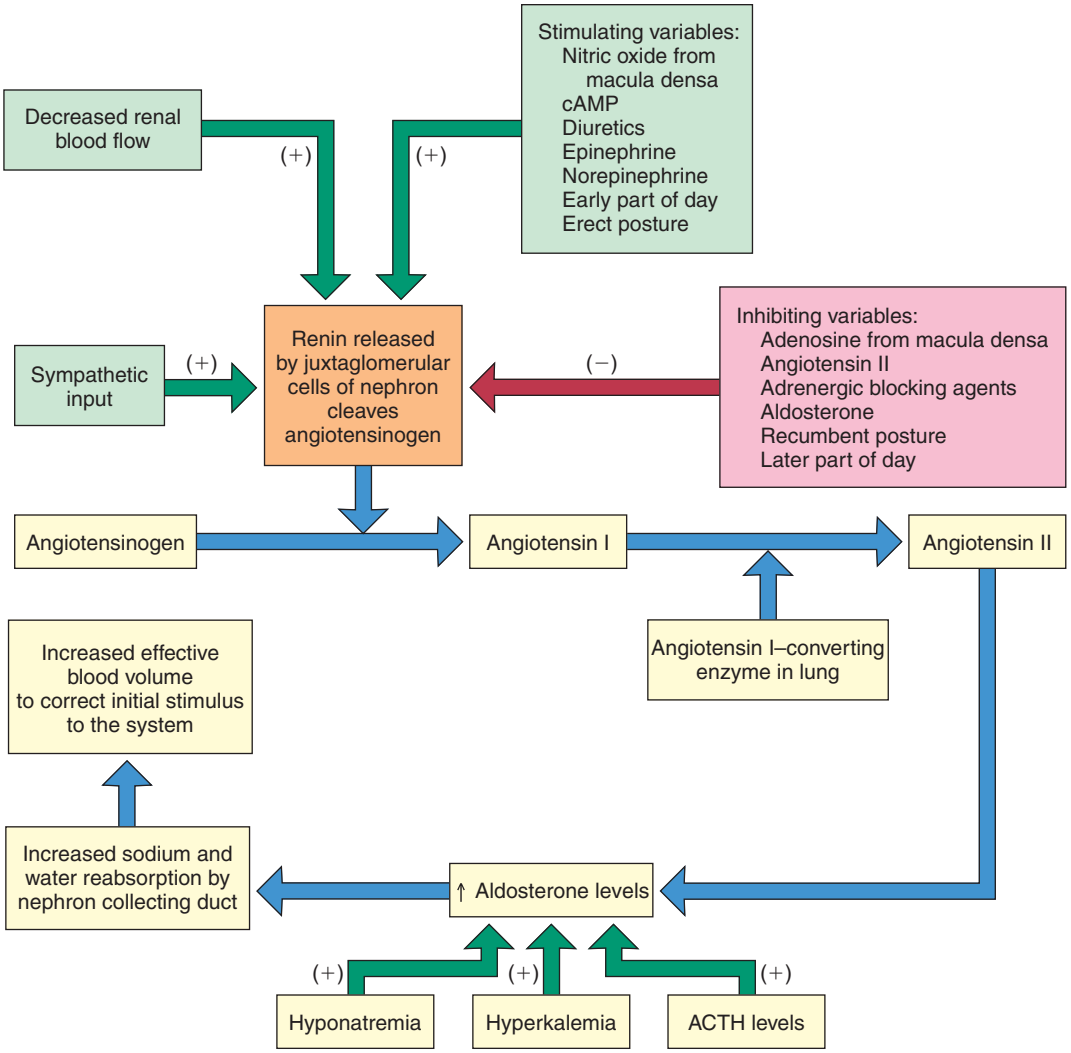

FIGURE 11-3 The feedback mechanisms regulating aldosterone secretion. *ACTH,* Adrenocorticotropic hormone; *cAMP,* cyclic adenosine monophosphate. (From McCance KL, Huether SE. *Pathophysiology. The Biologic Basis for Disease in Adults and Children.* 6th ed. St. Louis: Mosby; 2010.)

Stage III: Progressive Stage

If the cause of hypoperfusion is not corrected or if the compensatory mechanisms continue without reversing the shock, profound hypoperfusion results with further patient deterioration. Vasoconstriction continues in the systemic circulation. Although this effect shunts blood to vital organs, the decrease in blood flow leads to ischemia in the extremities, weak or absent pulses, and altered body defenses. Prolonged vasoconstriction results in decreased capillary blood flow and cellular hypoxia. The cells convert to anaerobic metabolism, producing lactic acid, which leads to metabolic acidosis. Anaerobic metabolism produces less ATP than aerobic metabolism, which reduces the energy available for cellular metabolism. The lack of ATP also causes failure of the sodium-potassium pump. Sodium and water accumulate within the cell, resulting in cellular swelling and a further reduction in cellular function.

The microcirculation exerts the opposite effect and dilates to increase the blood supply to meet local tissue needs. Whereas the arterioles remain constricted in an attempt to keep vital organs perfused, the precapillary sphincters relax, allowing blood to flow into the capillary bed. Meanwhile, postcapillary sphincters remain constricted. As a result, blood flows freely into the capillary bed but accumulates in the capillaries as blood flow exiting the capillary bed is impeded. Capillary hydrostatic pressure increases, and fluid is pushed from the capillaries into the interstitial space, causing interstitial edema. This intravascular to interstitial fluid shift is further aggravated by the release of histamine and other inflammatory mediators that increase capillary permeability,

along with the loss of proteins through enlarged capillary pores, which decreases capillary oncotic pressure. As intravascular blood volume decreases, the blood becomes more viscous and blood flow is slowed. This situation causes capillary sludging as red blood cells, platelets, and proteins clump together. The loss of intravascular volume and capillary pooling further reduce venous return to the heart and cardiac output.

Coronary artery perfusion pressure is decreased. Myocardial depressant factor (MDF) is released by the ischemic pancreas, causing a decrease in myocardial contractility. Cardiac output, blood pressure, and tissue perfusion continue to decrease, contributing to worsening cellular hypoxia. At this point, the patient shows classic signs and symptoms of shock. This phase of shock responds poorly to fluid replacement alone and requires aggressive interventions if it is to be reversed.

Stage IV: Refractory Stage

Prolonged inadequate tissue perfusion that is unresponsive to therapy ultimately contributes to multiple organ dysfunction and death. A large volume of the blood remains pooled in the capillary bed, and the arterial blood pressure is too low to support perfusion of the vital organs.

Dysrhythmias occur because of the failure of the sodium-potassium pump, resulting from decreased ATP, hypoxemia, ischemia, and acidosis. Cardiac failure may occur because of ischemia, acidosis, and the effects of MDF.

Endothelial damage in the capillary bed and precapillary arterioles, along with damage to the type II pneumocytes, which make surfactant, leads to acute respiratory distress syndrome (ARDS). Hypoxemia causes hypoxemic vasoconstriction of the pulmonary circulation and pulmonary hypertension. Ventilation-perfusion mismatch occurs because of disturbances in both ventilation and perfusion. Pulmonary edema may result from disruption of the alveolar-capillary membrane, ARDS, heart failure, or overaggressive fluid resuscitation.

When cerebral perfusion pressure is significantly impaired, loss of autoregulation occurs, resulting in brain ischemia. Cerebral infarction may occur. Sympathetic nervous system dysfunction results in massive vasodilation, depression of cardiac and respiratory centers results in bradycardia and bradypnea, and impaired thermoregulation results in poikilothermism.

Renal vasoconstriction and hypoperfusion of the kidney decreases the glomerular filtration rate. Prolonged ischemia causes acute kidney injury with acute tubular necrosis. Metabolic acids accumulate in the blood, worsening the metabolic acidosis caused by lactic acid production during anaerobic metabolism.

Hypoperfusion damages the reticuloendothelial cells, which recirculate bacteria and cellular debris, thereby predisposing the patient to bacteremia and sepsis. Damage to hepatocytes causes the liver to be unable to detoxify drugs, toxins, and hormones, conjugate bilirubin, or synthesize clotting factors. Hepatic dysfunction causes a decreased ability to mobilize carbohydrate, protein, and fat stores, which results in hypoglycemia.

Pancreatic enzymes are released by the ischemic and damaged pancreas. Pancreatic ischemia causes the release of MDF, which impairs cardiac contractility. Hyperglycemia may occur because of endogenous corticosteroids, exogenous corticosteroids, or insulin resistance. This hyperglycemia results in dehydration and electrolyte imbalances related to osmotic diuresis; impairment of leukocyte function causing decreased phagocytosis and increased risk of infection; depression of the immune response; impairment in gastric motility; shifts in substrate availability from glucose to free fatty acids or lactate; negative nitrogen balance; and decreased wound healing.

Ischemia and increased gastric acid production caused by glucocorticoids increase the risk of stress ulcer development. Prolonged vasoconstriction and ischemia lead to the inability of the intestinal walls to act as intact barriers to prevent the migration of bacteria out of the gastrointestinal tract. This may result in the translocation of bacteria from the gastrointestinal tract into the lymphatic and vascular beds, increasing the risk for sepsis.

Hypoxia and release of inflammatory cytokines impair blood flow and result in microvascular thrombosis. Sluggish blood flow, massive tissue trauma, and consumption of clotting factors may cause disseminated intravascular coagulation (DIC). The bone marrow mobilizes the release of white blood cells, causing leukocytosis early in shock and then leukopenia as depletion of white blood cells in blood and in bone marrow occurs. Massive tissue injury caused by widespread ischemia stimulates the development of a systemic inflammatory response syndrome (SIRS) with a massive release of mediators of the inflammatory process.

Poor renal function, respiratory failure, and impaired cellular function aggravate the existing state of acidosis, which contributes to further fluid shifts, loss of vasomotor tone, and relative hypovolemia. Alterations in the cardiovascular system and continued acidosis cause a reduction in heart rate, impaired myocardial contractility, and a further decrease in cardiac output and tissue perfusion. Cerebral ischemia occurs because of the reduction in cerebral blood flow. Consequently, the sympathetic nervous system is stimulated, an effect that aggravates the existing vasoconstriction, increasing afterload and decreasing cardiac output. Prolonged cerebral ischemia eventually causes the loss of sympathetic nervous system response, and vasodilation and bradycardia result. The patient's decreasing blood pressure and heart rate cause a lethal decrease in tissue perfusion, multisystem organ failure that is unresponsive to therapy, and ultimately brain death and cardiopulmonary arrest.

Systemic Inflammatory Response Syndrome

SIRS is widespread inflammation that can occur in patients with diverse disorders such as infection, trauma, shock, pancreatitis, or ischemia.[8] It may result from or lead to MODS. SIRS is most frequently associated with sepsis. Sepsis is defined as infection associated with SIRS.[8]

The inflammatory cascade maintains homeostasis through a balance between proinflammatory and antiinflammatory processes. Inflammation is normally a localized process; SIRS is a systemic response associated with the release of mediators. These mediators cause an increase in the permeability of the endothelial wall, shifting fluid from the intravascular space into extravascular spaces, including the interstitial space. Intravascular volume is reduced, resulting in a condition of relative hypovolemia. Other mediators cause microvascular clotting, impaired fibrinolysis, and widespread vasodilation.

Effects of Aging

The effects of aging diminish the body's ability to tolerate shock states. As the body ages, the left ventricular wall thickens, ventricular compliance decreases, and calcification and fibrosis of the heart valves occur. Stroke volume and, resultantly, cardiac output are reduced. There is a decreased sensitivity of the baroreceptors and a diminished heart rate response to sympathetic nervous system stimulation in the early stage of shock. Older adults are more likely to be prescribed beta-blockers, which also decrease the heart rate response. Arterial walls lose elasticity causing an increase in SVR, which increases the myocardial oxygen demand and decreases the responsiveness of the arterial system to the effects of catecholamines.

Aging causes decreased lung elasticity, decreased alveolar perfusion, decreased alveolar surface area, and thickening of the alveolar-capillary membrane. These changes limit the body's ability to increase blood oxygen levels during shock states. The ability of the kidney to concentrate urine decreases with age, which limits the body's ability to conserve water when required.

The immune system loses effectiveness with age, referred to as immunosenescence. This increases the risk of infection and sepsis, especially with illness, injury, or surgery. Older adults are also at greater risk for anaphylaxis since they have been exposed to more antigens and therefore have antibodies to more antigens.

ASSESSMENT

An understanding of the pathophysiology of shock and identification of patients at risk are essential for the prevention of shock. Assessment focuses on three areas: history, clinical presentation, and laboratory studies. The logical approach is to review the history of the patient and then assess the systems most sensitive to a lack of oxygen and nutrients. The patient's history may include an identifiable predisposing factor or cause of the shock state.

Clinical Presentation

Multiple body systems are affected by the shock syndrome. The clinical presentation specific to each classification of shock is discussed later (also see boxes, "Clinical Alert," and "Geriatric Considerations").

! CLINICAL ALERT

Shock

ASSESSMENT	SIGNIFICANCE
Change in vital signs, hemodynamic parameters, sensorium	Secondary to decreased tissue perfusion and initiation of compensatory mechanisms
Decreased urine output, rising BUN and creatinine levels	Secondary to initiation of compensatory mechanisms and decreased renal perfusion
Tachypnea, hypoxemia, worsening chest x-ray	Related to development of acute respiratory distress syndrome secondary to hypoperfusion
Petechiae, ecchymosis, bleeding from puncture sites, overt or occult blood in urine, stool, gastric aspirate, tracheal aspirate	Related to development of disseminated intravascular coagulation secondary to shock, SIRS
Hypoglycemia, increase in liver enzymes	Related to hepatic dysfunction secondary to hypoperfusion

BUN, Blood urea nitrogen; *SIRS,* systemic inflammatory response syndrome.

GERIATRIC CONSIDERATIONS

- Decreased skin turgor makes assessment of fluid status more difficult
- Dehydration is common, and may increase the risk for hypovolemia
- Infection is common in the elderly and is one of the top causes of death; several factors contribute to infection:
  - Changes in skin and mucous membranes
  - Increased risk for influenza, pneumonia, cancer, and autoimmune diseases
  - Nutritional deficits associated with poor nutrition, weight loss, low albumin levels, poor oral hygiene, and altered mental status
  - Medications that affect the immune system

Central Nervous System

The central nervous system is the most sensitive to changes in the supply of oxygen and nutrients. It is the first system affected by changes in cellular perfusion. Initial responses of the central nervous system to shock include restlessness, agitation, and anxiety. As the shock state progresses, the patient becomes confused and lethargic because of the decreased perfusion to the brain. As shock progresses, the patient becomes unresponsive.

Cardiovascular System

A major focus of assessment is blood pressure. It is important for the nurse to know the patient's baseline blood pressure.

During the compensatory stage, innervation of the sympathetic nervous system results in an increase in myocardial contractility and vasoconstriction, which results in a normal or slightly elevated systolic pressure, an increased diastolic pressure, and a narrowed pulse pressure. As the shock state progresses, the systolic blood pressure decreases, but the diastolic pressure remains normal, resulting in a narrowed pulse pressure. This narrowed pulse pressure may precede changes in heart rate.[10]

Definitions vary, but a decrease in systolic blood pressure to less than 90 mm Hg is considered hypotensive. If the patient is hypertensive, a decrease in systolic pressure of 40 mm Hg from the usual systolic pressure is considered severely hypotensive. Auscultated blood pressure in shock may be significantly inaccurate because of peripheral vasoconstriction. If blood pressure is not audible, the approximate systolic pressure can be assessed by palpation or ultrasound (Doppler) devices. If the brachial pulse is readily palpable, the approximate systolic pressure is 80 mm Hg. Corresponding blood pressure for palpation of the femoral and carotid pulses is 70 and 60 mm Hg, respectively. Intra-arterial pressure monitoring may be indicated to directly measure blood pressure to obtain accurate readings and guide therapy.

The rate, quality, and character of major pulses (i.e., carotid, radial, femoral, dorsalis pedis, and posterior tibial) are evaluated. In shock states, the pulse is often weak and thready. The pulse rate is increased, usually greater than 100 beats per minute, through stimulation of the sympathetic nervous system as a compensatory response to the decreased cardiac output and increased demand of the cells for oxygen. In later stages of shock, the pulse slows, possibly from release of MDF.

Normal compensatory responses to shock may be altered if the patient is taking certain medications. Negative inotropic agents, such as propranolol and metoprolol, are widely used in the treatment of angina, hypertension, and dysrhythmias. These agents work primarily by blocking the effects of the beta branch of the sympathetic nervous system, and cause a decrease in heart rate and cardiac output. A patient who is taking these medications has an altered ability to respond to the stress of shock and may not exhibit the typical signs and symptoms such as tachycardia and anxiety.

Assessment of the jugular veins provides information regarding the volume and pressure in the right side of the heart. It is an indirect method of evaluating the central venous pressure. Neck veins are distended in patients with obstructive or cardiogenic shock and are flat in hypovolemic shock.

Capillary refill assesses the ability of the cardiovascular system to maintain perfusion to the periphery. The normal response to pressure on the nail beds is blanching; the color returns to a normal pink hue 1 to 2 seconds after the pressure is released. A delay in the return of color indicates peripheral vasoconstriction. Capillary refill provides a quick assessment of the patient's overall cardiovascular status, but this assessment is not reliable in a patient who is hypothermic or has peripheral circulatory problems.

A central venous catheter may be inserted to aid in the differential diagnosis of shock, to administer and monitor therapies, and to evaluate the preload of the heart. Normally, the central venous pressure (or right atrial pressure [RAP]) is 2 to 6 mm Hg. When blood volume decreases (hypovolemic shock), or the vascular capacitance increases (distributive shock), the central venous pressure decreases. In cardiogenic shock, the central venous pressure is increased because of poor myocardial contractility and high filling pressure in the ventricles. In obstructive shock secondary to cardiac tamponade or tension pneumothorax, the central venous pressure is high.

A pulmonary artery (PA) catheter is a useful tool for diagnosing and treating the patient in shock. The risks associated with catheter insertion and central line-associated bloodstream infection must be weighed against the clinical information obtained from this invasive diagnostic device (see box, "QSEN Exemplar"). The PA catheter can give information regarding cardiac dynamics, fluid balance, and effects of vasoactive agents. Preload, which is measured by RAP for the right ventricle and by the pulmonary artery occlusion pressure (PAOP) for the left ventricle, is used to assess fluid balance. Cardiac output and index, afterload, and stroke work indices can also be assessed with a PA catheter (refer to Chapter 8). Table 11-3 describes hemodynamic values and alterations in each classification of shock. Critical care management involves optimizing cardiac

QSEN EXEMPLAR

Quality Improvement

Central line–associated bloodstream infections (CLABSI) and ventilator-associated pneumonia (VAP) are common causes of morbidity and mortality in critically ill patients. The Rhode Island Collaborative is a partnership of 11 hospitals including 23 critical care units that sought to study outcomes of bundled infection prevention interventions. Best practice protocols were selected based upon a Michigan-based quality improvement project. Interventions associated with CLABSI prevention included: hand washing, full barrier precautions during central line insertion, chlorhexidine skin cleansing, avoidance of femoral insertion sites, and timely removal of unnecessary central line catheters. Head-of-the-bed elevation, deep vein thrombosis prophylaxis, gastric ulcer prophylaxis, daily assessment for weaning appropriateness, and appropriate management of sedation comprised VAP preventative strategies. Additionally, a comprehensive safety program was implemented in each participating unit. In an effort to promote a culture of safety, practitioners were empowered to stop procedures where safety was potentially compromised. The bundled intervention protocols were associated with significant, sustained statewide declines in CLABSI and VAP rates, thus reducing the risk for sepsis.

Reference

DePalo VA, McNicoll L, Cornell M, Rocha JM, Adams L, & Pronovost PJ. The Rhode Island collaborative: a model for reducing central line-associated bloodstream infection and ventilator-assisted pneumonia statewide. *Quality and Safety in Healthcare,* 2010;19, 555-561.

TABLE 11-3 HEMODYNAMIC ALTERATIONS IN SHOCK STATES

HEMODYNAMIC PARAMETER, NORMAL VALUE	HYPOVOLEMIC	CARDIOGENIC	OBSTRUCTIVE	DISTRIBUTIVE		
				SEPTIC	ANAPHYLACTIC	NEUROGENIC
Heart rate 60-100 beats/min	High	High	High	High	High	Normal or low
Blood pressure	Normal → Low	Normal → Low	Normal → Low	Normal → Low	Normal → Low	Normal → Low
Cardiac output 4-8 L/min	Low	Low	Low	High then low	Normal → Low	Normal → Low
Cardiac index 2.5-4.0 L/min/m^2	Low	Low	Low	High then low	Normal → Low	Normal → Low
RAP 2-6 mm Hg	Low	High	High	Low to variable	Low	Low
PAOP 8-12 mm Hg or PADP 8-15 mm Hg	Low	High	High if impaired diastolic filling or high LV afterload; Low if high RV afterload	Low to variable	Low	Low
SVR 770-1500 dynes/sec/cm^{-5}	High	High	SVR Low PVR High	Low to variable	Low	Low
SvO_2 60%-75%	Low	Low	Low	High then low	Low	Low

LV, Left ventricular; *PADP,* pulmonary artery diastolic pressure; *PAOP,* pulmonary artery occlusion pressure; *PVR,* pulmonary vascular resistance; *RAP,* right atrial pressure; *RV,* right ventricular; *SvO_2,* mixed venous oxygen saturation; *SVR,* systemic vascular resistance.

output, and minimizing myocardial oxygen consumption. If an oximetric PA catheter is inserted, mixed venous oxygen saturation (SvO_2) is measured. The SvO_2 reflects the amount of oxygen bound to hemoglobin in the venous circulation and reflects the balance between oxygen delivery (DO_2) and consumption (VO_2). If the SvO_2 is less than 60%, either the DO_2 is inadequate or the VO_2 is excessive. The SvO_2 is decreased in all forms of shock except in early septic shock, where the poor oxygen extraction causes SvO_2 to be high. The SvO_2 is useful in identifying the type of shock and in evaluating the effectiveness of treatment. Continuous measurement of central venous oxygenation ($ScvO_2$) can also be obtained using a fiberoptic central venous catheter rather than an oximetric PA catheter. $ScvO_2$ correlates to SvO_2 and is easier to obtain in emergent situations.[30]

Respiratory System

In the early stage of shock, respirations are rapid and deep. The respiratory center responds to shock and metabolic acidosis with an increase in respiratory rate to eliminate carbon dioxide. Direct stimulation of the medulla by chemoreceptors alters the respiratory pattern. As the shock state progresses, metabolic wastes accumulate and cause generalized muscle weakness, resulting in shallow breathing with poor gas exchange.

Although pulse oximetry is commonly used to measure arterial oxygen saturation (SpO_2), it must be used with caution in patients in shock because decreased peripheral circulation may result in inaccurate readings. Arterial blood gas analysis provides a more accurate assessment of oxygenation.

Renal System

Renal hypoperfusion and decreased glomerular filtration rate cause oliguria (urine output <0.5 mL/kg/hr). The renin-angiotensin-aldosterone system is activated, which promotes the retention of sodium and the reabsorption of water in the kidneys, further decreasing urinary output. This prerenal cause of acute kidney injury is manifested by concentrated urine and an increased blood urea nitrogen level, while the serum creatinine level remains normal. If the decreased perfusion is prolonged, acute tubular necrosis, a form of intrarenal failure, occurs and creatinine levels increase.

Gastrointestinal System

Hypoperfusion of the gastrointestinal system results in a slowing of intestinal activity with decreased bowel sounds, distention, nausea, and constipation. Paralytic ileus and ulceration with bleeding may occur with prolonged hypoperfusion. Damage to the microvilli allows translocation of bacteria from the gastrointestinal tract to the lymphatic and systemic circulation, increasing the risk of infection and sepsis in the already compromised critically ill patient.

Hypoperfusion of the liver leads to decreased function and alterations in liver enzyme levels such as lactate dehydrogenase (LDH) and aspartate aminotransferase (AST). If decreased perfusion persists, the liver is not able to produce coagulation factors, detoxify drugs, or neutralize invading microorganisms. Clotting disorders, drug toxicity concerns, and increased susceptibility for infection occur.

Hematological System

The interaction between inflammation and coagulation enhances clotting and inhibits fibrinolysis, leading to clotting in the microcirculatory system and bleeding. An increased consumption of platelets and clotting factors occurs, causing a consumptive coagulopathy. The inability of the liver to manufacture clotting factors also contributes to the coagulopathy. A decreased platelet count, decreased clotting factors, and prolonged clotting times are seen with coagulopathy. Petechiae and ecchymosis may occur, along with blood in the urine, stool, gastric aspirate, and/or tracheal secretions. The clotting in the microcirculation causes peripheral ischemia manifested by acrocyanosis and necrosis of digits and extremities. Leukocytosis frequently occurs, especially in early septic shock. Leukopenia occurs later because of consumption of white blood cells.

Integumentary System

Skin color, temperature, texture, turgor, and moisture level are evaluated. Cyanosis may be present; however, it is a late and unreliable sign. The patient may exhibit central cyanosis, seen in the mucous membranes of the mouth and nose; or peripheral cyanosis, evident in the nails and earlobes. While turgor is frequently used to determine the presence of interstitial dehydration, elderly adults have decreased skin elasticity, making this evaluation misleading.

Laboratory Studies

Laboratory studies assist in the differential diagnosis of the patient in shock (see box, "Laboratory Alert"). However, by the time many of the laboratory values are altered, the patient is in the later stages of shock. The clinical picture is often more useful for early diagnosis and immediate treatment.

! LABORATORY ALERT

Shock

DIAGNOSTIC STUDY	CRITICAL VALUE	SIGNIFICANCE
Chemistry Studies		
Glucose	<70 or >100 mg/dL	Frequently ↑ early shock, ↓ late shock ↑ Impairs immune response
Blood urea nitrogen	>20 mg/dL	↑ Hypoperfusion (prerenal failure) ↑ Gastrointestinal bleeding and catabolism
Creatinine	>1.2 mg/dL	↑ Acute kidney injury
Sodium	<136 or >145 mEq/L	↓ Hemodilution from replacement of excessive hypotonic fluid
		↑ Hemoconcentration from fluid loss
Chloride	>108 mEq/L	↑ Excess infusion of normal saline; may cause hyperchloremic acidosis
Potassium	<3.5 or >5.3 mEq/L	↓ Excessive loss of potassium
		↑ Impaired elimination from acute kidney injury
		Observe for cardiac dysrhythmias
Lactate	>2.2 mEq/L	↑ Hypoxia leading to anaerobic metabolism and production of lactic acid
AST	>20 units/L	↑ Hepatic impairment
LDH	>102 units/L	↑ Hepatic impairment, renal impairment, intestinal ischemia, or myocardial infarction
Hematology Studies		
WBCs	<4500 or >11,000/microliter	↑ Stress response; significant increase indicates infection ↓ Late shock due to consumption of WBCs
Hemoglobin	<12 g/dL	↓ Blood loss
Hematocrit	<35%	↓ Blood loss ↑ Dehydration and hemoconcentration
Arterial Blood Gases		
pH	<7.35 or >7.45	↑ Early shock—respiratory alkalosis due to hyperventilation ↓ Late shock—metabolic acidosis due to lactic acidosis
$PaCO_2$	<35 or >45 mm Hg	↓ Early shock—respiratory alkalosis due to hyperventilation
PaO_2	<80 mm Hg	↓ Hypoxemia; may indicate pulmonary edema or ARDS
HCO_3^-	<22 mEq/L	↓ Late shock—metabolic acidosis caused by hypoxia, anaerobic metabolism, and lactic acidosis

ARDS, Acute respiratory distress syndrome; *AST,* aspartate aminotransferase; *HCO_3^-,* bicarbonate; *LDH,* lactate dehydrogenase; *$PaCO_2$,* partial pressure of arterial carbon dioxide; *PaO_2,* partial pressure of arterial oxygen; *WBCs,* white blood cells.

Serum lactate level is a measure of the overall state of shock, regardless of the cause. The lactate level is an indicator of decreased oxygen delivery to the cells and of the adequacy of treatment. Elevated lactate levels produce an acidic environment and decreased arterial pH. The serum lactate level correlates with the degree of hypoperfusion.

MANAGEMENT

Management of the patient in shock consists of identifying and treating the cause of the shock as rapidly as possible. Care is directed toward correcting or reversing the altered circulatory component (e.g., blood volume, myocardial contractility, obstruction, or vascular resistance) and reversing tissue hypoxia. A combination of fluid, pharmacological, and mechanical therapies are implemented to maintain tissue perfusion and improve oxygen delivery. These interventions include increasing the cardiac output and cardiac index, increasing the hemoglobin level, and increasing the arterial oxygen saturation. Efforts are also aimed toward minimizing oxygen consumption. Specific management for each classification of shock is discussed later. Nursing interventions are summarized in the box, "Evidence-Based Practice."

Maintenance of Circulating Blood Volume and Adequate Hemoglobin Level

Regardless of the cause, shock produces profound alterations in fluid balance. Therefore patients experiencing absolute hypovolemia (hypovolemic shock) or relative hypovolemia (distributive shock) require the administration of intravenous (IV) fluids to restore intravascular volume, maintain oxygen-carrying capacity, and establish the hemodynamic stability necessary for optimal tissue perfusion. The choice of fluid and the volume and rate of infusion depend on the type of fluid lost, the patient's hemodynamic status, and coexisting conditions.

EVIDENCE-BASED PRACTICE

Nursing Interventions for Shock

Problem

Severe sepsis and septic shock result in mortality rates greater than 20%.[1] Evidence-based practice guidelines have been published by the Surviving Sepsis Campaign (SSC) to facilitate medical management.[2,3] Patients also require expert nursing knowledge and skill to recognize the possible development of sepsis and the delivery of competent care of the patient with sepsis to improve their survival and quality of life after hospitalization.[1]

Clinical Question

What nursing interventions are recommended in caring for patients with severe sepsis or septic shock?

Evidence

1. Early enteral nutrition started in the first 24 to 48 hours after admission to a critical care unit **(Level of Evidence: A).**

Patients require nutrition that not only meets higher caloric requirements, but also supports the immune system and promotes cellular repair.[6] Early enteral nutrition assists the intestinal mucosa in maintaining its barrier function and can reduce the risk for infection by as much as 30% to 40%.[7]

2. A plan for pressure ulcer prevention and management **(Level of Evidence: D).**

The hemodynamic alterations associated with shock and sepsis, and use of vasoactive medications, combined with comorbid conditions, limited mobility, and altered sensation place the patient at higher risk for the development of pressure ulcers.[4] Frequent turning, reduction of friction and shear, pressure redistribution through the use of special mattress surfaces are all within the control of the bedside nurse.[5]

Implications for Nursing

These two basic nursing interventions should be a regular feature of nursing care in the ICU patient with sepsis or multiple organ dysfunction. The interventions may require the nurse to initiate a multiprofessional collaboration with other team members—dieticians, wound care specialists, pharmacists, physicians—to fully implement them; but it is the nurse who provides the attentiveness in initiating and maintaining these interventions.

References

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Benefits of IV fluid administration include increased intravascular volume, increased venous return to the right side of the heart, optimal stretching of the ventricle, improved myocardial contractility, and increased cardiac output. However, these effects may be dangerous to the patient in cardiogenic shock because large volumes of fluid overwork an already failing heart. Instead, cardiogenic shock is managed primarily with medications that reduce both preload and afterload.

Fluid administration is adjusted based on changes in blood pressure, urine output, hemodynamic values, diagnostic test results, and the clinical picture of the patient's response to treatment. Values obtained from hemodynamic monitoring also assist in monitoring effects of treatment. Generally, volume replacement continues until an adequate mean arterial pressure (65 to 70 mm Hg) is achieved and evidence of end-organ tissue perfusion is reestablished, as evidenced by improvement in the level of consciousness, urinary output, and peripheral perfusion.

Patients in severe shock may require immediate, rapid volume replacement. The IV infusion rate can be increased by using a blood pump to administer fluids under pressure, by using large-bore infusion tubing, or by using a rapid-infusion device. Infusion pumps are used to rapidly and accurately administer large volumes of fluids. Administration of large volumes of room-temperature fluids can rapidly drop core body temperature and cause hypothermia. Fluid-related hypothermia causes alterations in cardiac contractility and coagulation. For this reason, large volumes of fluids should be infused through warming devices (Figure 11-4).

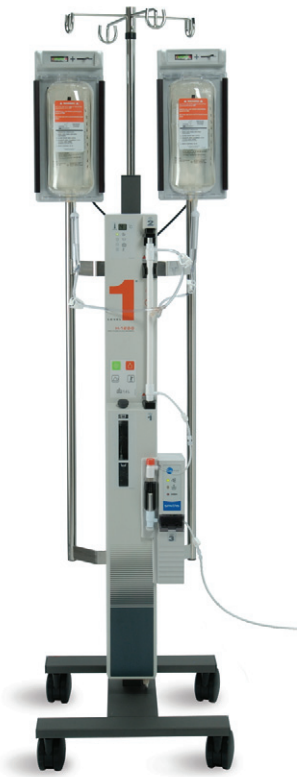

FIGURE 11-4 Level 1 rapid infuser. (Courtesy Level 1, Inc., Rockland, Massachusetts.)

Intravenous Access

IV access is needed to administer fluids and medications. The patient in shock requires a minimum of two IV catheters, one in a peripheral vein and one in a central vein. Peripheral access via a large-gauge catheter (14- or 16-gauge) in a large vein in the antecubital fossa provides a route for rapid administration of fluids and medications. Establishing IV routes in a patient in shock is challenging because peripheral vasoconstriction and venous collapse make access difficult.

A central venous catheter is inserted for large-volume replacement and is also used to monitor central venous pressures. Central venous catheters are commonly inserted into the subclavian, internal jugular, or femoral veins. An upper extremity insertion site is preferred over the femoral vein.[25] Multilumen catheters, which provide multiple access ports, allow the concurrent administration of fluid, medication, and blood products. A PA catheter may be inserted to monitor hemodynamic pressures and guide fluid replacement.

Fluid Challenge

Once IV access is established, a fluid challenge may be performed to assess the patient's hemodynamic response to fluid administration. Various methods for administering a fluid challenge exist. Typically, a rapid infusion of 250 mL (up to 2 L) of a crystalloid solution is initiated first. Nursing responsibilities include obtaining the baseline hemodynamic measurements, administering the fluid challenge, and assessing the patient's response. A fluid challenge algorithm is helpful in guiding fluid resuscitation (Figure 11-5).

Types of Fluids

The choice of fluids depends on the cause of the volume deficit, the patient's clinical status, and the physician's preference. Although the nurse is not responsible for selecting the infusion or transfusion, an understanding of the rationale for the prescribed fluid and the expected effects is needed to assess patient outcomes. The nurse carefully monitors the patient's response to fluid therapy.

Blood, blood products, crystalloids, and colloids are used alone, or in combination, to restore intravascular volume. Crystalloids are infused until diagnostic testing and blood typing and crossmatching are completed. Colloids are avoided in situations where there is an increase in capillary permeability, as in sepsis and septic shock, anaphylactic shock, and early burn injury. A systematic review of 30 randomized controlled trials found no benefit in giving colloids over crystalloids, and recommended against the administration of colloids in most patient populations. In critically ill patients with hypovolemia, administration of albumin was associated with a higher risk of death.[9] A Cochrane Review demonstrated no difference in mortality between the use of crystalloid solution or colloids in the resuscitation of critically ill patients.[26]

Crystalloids are inexpensive and readily available. Crystalloids are classified by tonicity. Isotonic solutions have approximately the same tonicity as plasma (osmolality, 250 to 350 mOsm/L). Lactated Ringer's (LR) solution and 0.9% normal

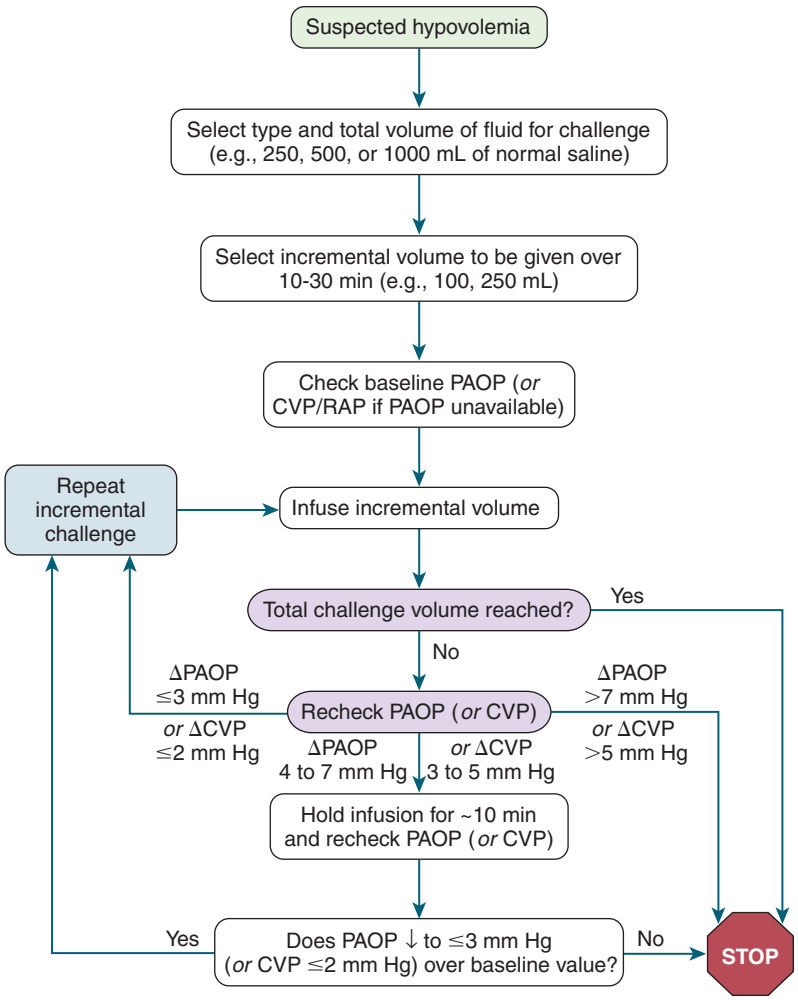

FIGURE 11-5 Fluid challenge algorithm. *CVP,* Central venous pressure; *PAOP,* pulmonary artery occlusion pressure. (Adapted from Kruse JA, Fink MP, Carlson RW. *Saunders Manual of Critical Care.* Philadelphia: Saunders; 2003.)

saline are isotonic solutions that are commonly infused. These solutions move freely from the intravascular space into the tissues. Traditionally, 3 mL of crystalloid solution is administered to replace each 1 mL of blood loss. LR solution closely resembles plasma and may be the only fluid replacement required if blood loss is less than 1500 mL. LR solution contains lactate, which is a salt that the liver converts to bicarbonate, so it counteracts metabolic acidosis if the liver function is normal. LR should not be infused in patients with impaired liver function or severe lactic acidosis. Although 0.9% normal saline is an isotonic solution, its side effects include hypernatremia, hypokalemia, and hyperchloremic metabolic acidosis. Solutions of 5% dextrose in water and 0.45% normal saline are hypotonic and are not used for fluid resuscitation. Hypotonic solutions rapidly leave the intravascular space, causing interstitial and intracellular edema.

When large volumes of crystalloids are infused, the patient is at risk of developing hemodilution of red blood cells and plasma proteins. Hemodilution of red blood cells impairs oxygen delivery if the hematocrit value is decreased and the cardiac output cannot increase enough to compensate. Hemodilution of plasma proteins decreases colloid osmotic pressure and places the patient at risk of developing pulmonary edema. Elderly patients are at increased risk of developing pulmonary edema and may require invasive hemodynamic monitoring to guide fluid resuscitation.

Colloids contain proteins that increase osmotic pressure. Osmotic pressure holds and attracts fluid into blood vessels, thereby expanding plasma volume. Because colloids remain in the intravascular space longer than crystalloids, smaller volumes of colloids are given in shock states. Albumin and plasma protein fraction (Plasmanate) are naturally occurring colloid solutions that are infused when the volume loss is caused by a loss of plasma rather than blood, such as in burn injury (see Chapter 20), peritonitis, and bowel obstruction. Typing and crossmatching of albumin and plasma protein fraction are not required. Pulmonary edema is a potential complication of colloid administration, resulting from increased pulmonary

capillary permeability or increased capillary hydrostatic pressure in the pulmonary vasculature created by rapid plasma expansion.

Hetastarch (Hespan) is a synthetic colloid solution that acts as a plasma expander but carries less risk for pulmonary edema. Side effects include altered prothrombin time (PT) and activated partial thromboplastin time (aPTT) and the potential for circulatory overload. No more than 1 L should be administered in a 24-hour period.[10]

Blood products, packed red blood cells, fresh frozen plasma, and platelets are administered to treat major blood loss. Typing and crossmatching of these products are performed to identify the patient's blood type (A, B, AB, O) and Rh factor, and to ensure compatibility with the donor blood to prevent transfusion reactions. In extreme emergencies, the patient may be transfused with type-specific or O-negative blood, which is considered the universal donor blood type.

Transfusions require an IV access with at least a 20-gauge, preferably an 18-gauge or larger, catheter (a 22- or 23-gauge needle or catheter may be used in adults with small veins). Solutions other than 0.9% normal saline are not infused with blood because they cause red blood cells to aggregate, swell, and burst. In addition, IV medications are never infused in the same port with blood. Appropriate patient and blood identification is necessary before starting any transfusion.

Transfusions are administered with a blood filter to trap debris and clots. Frequent patient assessment is necessary during a blood transfusion to monitor for adverse reactions. In the event of a reaction, the transfusion is stopped, the transfusion tubing is disconnected from the IV access site, and the vein is kept open with an IV of 0.9% normal saline solution. The patient is assessed, and the physician and laboratory are notified. All transfusion equipment (bag, tubing, and remaining solutions) and any blood or urine specimens obtained are sent to the laboratory according to hospital policy. The events of the reaction, interventions used, and patient response to treatment are documented.

The transfusion administration time varies with the particular blood product used and the individual patient circumstances. Documentation of the transfusion includes the blood product administered, baseline vital signs, start and completion time of the transfusion, volume of blood and fluid, assessment of the patient during the transfusion, and any nursing actions taken.

Packed red blood cells increase the blood volume and therefore provide more oxygen-carrying capability to the tissues. One unit of packed red blood cells increases the hematocrit value by about 3% and the hemoglobin value by 1 g/dL. Typing and crossmatching of packed red blood cells are required. Red blood cells tend to aggregate because of the fibrinogen coating; therefore, washed red blood cells may be given. Acidosis, hyperkalemia, and coagulation problems are associated with transfusions of banked blood older than 24 hours. Massive transfusion (approximately 10 units) is associated with decreased 2,3-diphosphoglycerate (2,3-DPG), causing a shift of the oxyhemoglobin dissociation curve to the left, which impairs the delivery of oxygen to the tissues.

Fresh frozen plasma is administered to replace all clotting factors except platelets. When massive transfusions are infused, fresh frozen plasma is given rapidly to restore coagulation factors. One unit of fresh frozen plasma is given for every 4 to 5 units of packed red blood cells transfused. Typing and crossmatching of fresh frozen plasma are required.

Platelets are given rapidly to help control bleeding caused by low platelet counts (usually <50,000/microliter). Typing of platelets, but not crossmatching, is required.

Maintenance of Arterial Oxygen Saturation and Ventilation

Airway maintenance is the top priority. The airway is maintained by proper head position, use of oral or nasopharyngeal airways, or intubation, depending on the patient's condition. Suctioning and chest physical therapy facilitate secretion removal and help maintain a patent airway.

Oxygen is administered to elevate the arterial oxygen tension, thereby improving tissue oxygenation. Oxygen is administered by methods ranging from nasal cannula to mechanical ventilation, depending on the patient's condition.

Mechanical ventilation is used to maintain adequate ventilation as reflected by a normal partial pressure of arterial carbon dioxide ($PaCO_2$) level. Another benefit of mechanical ventilation in a patient with shock is to reduce the work of breathing and the associated increase in oxygen consumption. Tidal volumes and inspiratory pressures are kept low to prevent ventilator-induced lung injury. Tidal volumes are generally between 6 and 8 mL/kg of ideal body weight, and the inspiratory plateau pressures are maintained at less than 30 cm H_2O.[35] Positive end-expiratory pressure (PEEP) is used to maintain alveolar recruitment and may protect against ventilator-induced lung injury by preventing the repetitive opening and collapsing of the alveoli.[29] Newer ventilator modes, such as the pressure-regulated volume-controlled mode, aid in keeping inspiratory pressures low.

Sedation or neuromuscular blockade is considered to reduce oxygen consumption. Arterial blood gases, pulse oximetry, and hemodynamic monitoring aid in the evaluation of oxygen consumption and delivery.

Pharmacological Support

Pharmacological management of shock is based on the manipulation of the determinants of cardiac output: heart rate, preload, afterload, and contractility. Figure 11-6 describes therapies used to manipulate these parameters. These drugs are preferably administered through a central venous catheter. Hemodynamic monitoring is often used to assess the effectiveness of medications. Older adults are particular sensitive to the physiological impact of medications and the deleterious effects of polypharmacy. Table 11-4 describes medications that are commonly administered in shock.

Cardiac Output

Low or high heart rates and dysrhythmias decrease cardiac output. Chronotropic drugs and antidysrhythmic agents are given as indicated. In neurogenic shock, sinus bradycardia

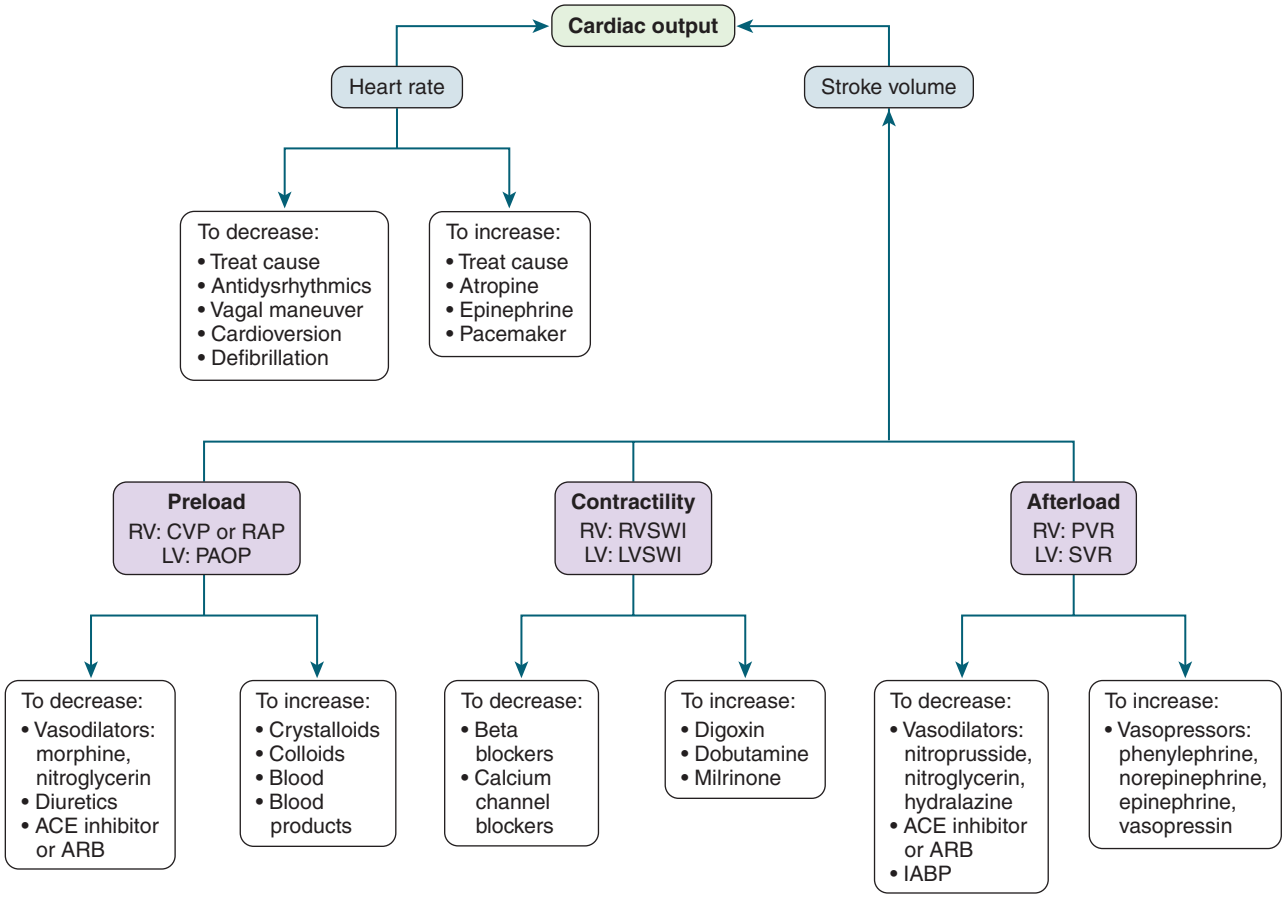

FIGURE 11-6 Therapeutic manipulation of cardiac output and myocardial oxygen consumption. *ACE,* Angiotensin-converting enzyme; *ARB,* angiotensin receptor blocker; *CVP,* central venous pressure; *IABP,* intra-aortic balloon pump; *LV,* left ventricle; *LVSWI,* left ventricular stroke work index; *PAOP,* pulmonary artery occlusion pressure; *PVR,* pulmonary vascular resistance; *RAP,* right atrial pressure; *RV,* right ventricle; *RVSWI,* right ventricular stroke work index; *SVR,* systemic vascular resistance. (Adapted from Dennison RD. *Pass CCRN!* 3rd ed. St. Louis: Mosby; 2007.)

TABLE 11-4 PHARMACOLOGY

Medications Commonly Used in Shock

MEDICATION	ACTION/USE	DOSE/ROUTE	SIDE EFFECTS	NURSING IMPLICATIONS
Dobutamine (Dobutrex)	Stimulates primarily $beta_1$ receptors to ↑ contractility and ↑ heart rate and cause vasodilation in low CO states	2-20 mcg/kg/min IV infusion Central venous catheter preferred	Tachycardia Dysrhythmias Hypotension Nausea, vomiting Dyspnea Headache Anxiety Paresthesia Palpitations Chest pain	Monitor BP, HR, ECG, PAP, PAOP, SVR, CO, and CI Use cautiously in patients with hypertension, myocardial ischemia, or ventricular dysrhythmias Replace volume before initiation of infusion Do not administer with alkaline solutions
Dopamine (Intropin)	Used in low CO states or vasodilatory states (distributive shock) to restore vascular tone Dose-dependent effect At 2-10 mcg/kg/min stimulates $beta_1$ receptors to ↑ contractility and ↑ HR	2-20 mcg/kg/min IV infusion and titrated upward as necessary to (maximum of 50 mcg/kg/min) Central venous catheter preferred	Tachycardia Dysrhythmias Nausea, vomiting Dyspnea Headache Palpitations Chest pain in patients with coronary artery disease Tissue necrosis if extravasation occurs	Monitor HR, BP, ECG, PAP, PAOP, SVR, CO, CI, and urine output Treat cause of ↓ BP before initiating (e.g., hypovolemia treated with fluid resuscitation) Wean slowly Treat extravasation with phentolamine (Regitine) Do not administer with alkaline solutions

Continued

TABLE 11-4 PHARMACOLOGY—cont'd

Medications Commonly Used in Shock

MEDICATION	ACTION/USE	DOSE/ROUTE	SIDE EFFECTS	NURSING IMPLICATIONS
Dopamine (Intropin)—cont'd	At 10-20 mcg/kg/min stimulates alpha receptors to cause vasoconstriction and increased SVR			
Norepinephrine (Levophed)	Stimulates alpha receptors to cause vasoconstriction Used in vasodilatory states (distributive shock) to restore vascular tone Stimulation of beta receptors to ↑ contractility and ↑ HR	2-12 mcg/min IV infusion and titrated upward as needed to a maximum of 30 mcg/min Central venous catheter preferred	Tachycardia Ventricular dysrhythmias Hypertension Anxiety Headache Tremor Dizziness Chest pain Metabolic (lactic) acidosis Tissue necrosis if extravasation occurs	Monitor BP, HR, ECG, urine output, and neurological status Treat extravasation with phentolamine (Regitine) Do not administer with alkaline solutions
Phenylephrine (Neosynephrine)	Stimulates alpha receptors to cause vasoconstriction Used in vasodilatory states (distributive shock) to restore vascular tone	2-10 mcg/kg/min IV infusion Central venous catheter preferred	Reflex bradycardia Ventricular dysrhythmias Hypertension Nausea, vomiting Paresthesia Palpitations Anxiety Restlessness Headache Tremor Chest pain	Monitor HR, BP, and ECG Treat reflex bradycardia with atropine
Vasopressin	Vasoconstriction via smooth muscle contraction of all parts of capillaries, arterioles, and venules Used in vasodilatory states (distributive shock) to restore vascular tone	0.01 to 0.03 unit/min IV infusion Central venous catheter preferred	↓ HR ↑ BP Fever Hyponatremia Abdominal cramps Tremor Headache Seizures Coma Chest pain and myocardial ischemia	
Nitroglycerin	Vasodilation by direct smooth muscle relaxation, predominantly venous Used in preload and/or afterload reduction (cardiogenic shock) Dose-dependent effect Arterial dilation only if infusion >1 mcg/kg/min	Initial dose 5-10 mcg/min IV infusion; increase by 5-10 mcg/min every 5 minutes until desired results are achieved (control of chest pain and decreased preload)	↑ or ↓ HR ↑ or ↓ BP Palpitations Weakness Apprehension Flushing Dizziness Syncope Headache	Monitor HR, BP, and urine output Monitor RAP, PAP, PAOP, SVR, CO, and CI if pulmonary artery catheter present Use cautiously in hypotension Administer in glass bottle via non–polyvinyl chloride tubing

TABLE 11-4 PHARMACOLOGY—cont'd

Medications Commonly Used in Shock

MEDICATION	ACTION/USE	DOSE/ROUTE	SIDE EFFECTS	NURSING IMPLICATIONS
Nitroprusside (Nipride)	Vasodilation by direct smooth muscle relaxation, predominantly arterial Used in preload and/or afterload reduction (cardiogenic shock)	0.5-10 mcg/kg/min IV infusion	Nausea, vomiting, abdominal pain Headache Tinnitus Dizziness Diaphoresis Apprehension Hypotension Tachycardia Palpitations Hypoxemia (nitroprusside-induced intrapulmonary thiocyanate toxicity)	Monitor HR, BP, urine output, and neurological status Monitor for patient thiocyanate toxicity (metabolic acidosis, confusion, hyperreflexia, and seizures) Serum thiocyanate levels drawn daily if drug is used longer than 72 hours Treatment includes amyl nitrate, sodium nitrate, and/or sodium thiosulfate Protect from light by wrapping with opaque material (e.g., aluminum foil)

All medications should be administered via volumetric infusion pump.
BP, Blood pressure; *CI,* cardiac index; *CO,* cardiac output; *D_5W,* 5% dextrose in water; *ECG,* electrocardiogram; *HR,* heart rate; *IV,* intravenous; *NS,* normal (0.9%) saline; *PAOP,* pulmonary artery occlusion pressure; *PAP,* pulmonary artery pressure; *RAP,* right atrial pressure; *SVR,* systemic vascular resistance.

secondary to cervical spinal cord injury does not usually require therapy. However, if the bradycardia is significant and results in decreased perfusion, atropine or a temporary pacemaker may be required.

Preload

In hypovolemic and distributive shock, fluid administration is the primary treatment to increase preload. In cardiogenic shock, the myofibrils are overstretched and the preload needs to be reduced. Venous vasodilators or diuretics are administered to reduce preload.

Afterload

Afterload is low in distributive shock. In this situation, agents that cause vasoconstriction are administered to increase vascular tone and tissue perfusion pressure. Examples of vasoconstrictive drugs include phenylephrine, norepinephrine, epinephrine, or vasopressin. These drugs increase blood pressure and SVR. A negative effect of drugs that increase afterload is an increase in the myocardial oxygen demand. Accurate measurement/calculation of SVR and PVR via a pulmonary artery catheter assists in assessment.

Vasopressors should not be administered in hypovolemic shock because these patients require volume replacement. Administration of vasopressors in hypovolemia causes vasoconstriction and further diminishes tissue perfusion.

In cardiogenic shock, the afterload needs to be reduced. The use of arterial vasodilators to reduce afterload may be limited by the patient's blood pressure. In situations where hypotension prevents the use of arterial vasodilators, an intraaortic balloon pump is used to decrease afterload.

Contractility

Drugs that increase contractility, such as dobutamine, may be administered in cardiogenic shock. Although drugs that decrease contractility (e.g., beta-blockers) may be used to decrease myocardial oxygen consumption in patients with coronary artery disease, they are contraindicated in a patient in shock.

Other Medications

Other drugs used to manage shock include sedatives, analgesics, insulin, corticosteroids, and antibiotics. Although respiratory acidosis is treated by improving ventilation, metabolic acidosis caused by lactic acidosis is best treated by improving the aspects of DO_2: SaO_2, hemoglobin level, and cardiac output. Arterial blood gas analysis and serum lactate levels are used to guide treatment.

Hyperglycemia is common in patients in shock, especially patients with septic shock. Data suggest that intensive insulin therapy to maintain serum glucose levels less than 150 mg/dL reduces morbidity and mortality in critically ill patients.[37]

Low–molecular weight heparin is frequently prescribed for deep vein thrombosis prophylaxis. An H_2-receptor antagonist (ranitidine [Zantac] or proton pump inhibitor (pantoprazole [Protonix]) is frequently prescribed for peptic ulcer prophylaxis.

Maintenance of Body Temperature

The patient's temperature is monitored frequently. Care is directed toward maintaining normal body temperature. Hypothermia depresses cardiac contractility and impairs cardiac output and oxygen delivery. Hypothermia also impairs the

coagulation pathway, which can result in a significant coagulopathy. Hypothermia is anticipated when fluids are infused rapidly, and use of a fluid warmer should be considered. Patients should be kept warm and comfortable, but not overly warmed. Excessive warmth increases the oxygen demand on an already stressed cardiovascular system.

Nutritional Support

Nutritional support is essential for patient survival. The goals of nutritional support are to initiate enteral intake as soon as possible and to maintain sufficient caloric intake to assist in the healing process. Early enteral feeding decreases hypermetabolism, minimizes bacterial translocation, decreases diarrhea, and decreases length of stay. Nutritional requirements of the patient in shock are highly variable depending on the degree of hemodynamic stability, the cause of shock, and the patient's age, gender, and preexisting diseases. Enteral feeding is the preferred method, and immune-boosting formulas may be prescribed. Administration of enteral nutrition may be limited by paralytic ileus, gastric dilation, or both, which are common in shock. Total parenteral nutrition is given if patients are unable to tolerate enteral feeding (see Chapter 6).[3]

Maintenance of Skin Integrity

The decreased perfusion seen in shock can precipitate injury to the skin. Meticulous skin care is required to promote skin integrity. The patient is turned at frequent intervals, and lotion is applied. Pressure-relieving devices, such as therapeutic beds or mattresses, may be indicated. Therapeutic beds with automatic rotation features do not substitute for the pressure relief afforded by manual turning and positioning. Lotion moisturizes the skin to reduce the effects of shear and friction generated when the patient is repositioned in the bed. The heels should be elevated off the surface of the bed with pillows or with pressure-relief boots.

Psychological Support

Nursing interventions also focus on identifying the impact of the illness on the patient and the family. Nursing interventions include providing information, which is essential for the psychological well-being of the patient and the family, and may help to give them a sense of understanding and control of the situation. Since shock has a high mortality, a discussion should be initiated regarding life-sustaining therapies (see Chapters 2 and 3).

NURSING DIAGNOSIS

The primary nursing diagnosis for all patients in shock is altered tissue perfusion. This diagnosis may be related to decreased tissue perfusion, myocardial contractility, vascular resistance, obstruction, or a combination of these. The nurse provides care to support tissue perfusion of the patient in shock until definitive care is underway. Supportive care is aimed at maintenance of organ function (see box, "Nursing Care Plan for the Patient in Shock").

NURSING CARE PLAN

for the Patient in Shock

NURSING DIAGNOSIS

Altered tissue perfusion related to decreased blood volume (hypovolemic shock); decreased myocardial contractility (cardiogenic shock); impaired circulatory blood flow (obstructive shock); and widespread vasodilatation (septic, anaphylactic, or neurogenic shock)

PATIENT OUTCOMES

Adequate Tissue Perfusion

- Alert and responsive
- Skin warm and dry with good turgor
- Vital signs and hemodynamic parameters within normal limits (see Table 11-3)
- Systolic and diastolic blood pressure within 20 mm Hg of baseline
- Heart rate 60 to 100 beats per minute
- Oxygen saturation 90% or greater
- Normal body temperature
- Strong peripheral pulses
- Balanced intake and output
- Stable body weight
- Urine output at least 0.5 mL/kg/hr
- Normal serum and urine laboratory values and ABG results
- Adequate pain management
- Absence of complications (ARDS, DIC, acute kidney injury, hepatic failure, MODS)

NURSING CARE PLAN—cont'd

for the Patient in Shock

NURSING INTERVENTIONS	RATIONALES
• Monitor for early symptoms of shock (see Table 11-2)	• Initiate early support to improve outcomes and reduce risk of complications, organ failure, and death
• Establish or maintain patent airway	• Provide adequate gas exchange
• Monitor oxygenation: pulse oximetry, ABGs, SvO_2; administer oxygen to maintain SpO_2 at least 90%	• Assess for need for supplemental oxygen; ensure adequate oxygen delivery to the tissues
• Prepare for intubation and mechanical ventilation as needed	• Mechanical ventilation is frequently required to ensure adequate ventilation and reduce the work of breathing
• Establish intravenous access; use large-bore catheters (14 or 16 gauge); obtain central venous access, if possible	• Provide rapid fluid administration; central IV access allows for fluid and drug administration without the concerns of peripheral infiltration and irritation
• Control bleeding through the application of pressure or surgical intervention	• Prevent blood loss
• Administer fluids as ordered (crystalloids, colloids, blood products)	• Maintain tissue perfusion
• Consider warming fluids before infusing	• Reduce hypothermia and its complications
• Replace blood components as indicated; obtain laboratory specimen for type and crossmatch	• Replace volume loss associated with blood loss; prevent transfusion reaction
• Evaluate patient's response to fluid challenges and blood product administration: improved vital signs, level of consciousness, urinary output, hemodynamic values, and serum and urine laboratory values	• Monitor response to treatment
• Monitor for clinical indications of fluid overload (↑ HR, ↑ RR, dyspnea, crackles) when fluids are administered rapidly	• Assess for signs of volume overload in response to treatment
• Monitor cardiopulmonary status: HR, RR, BP, MAP, skin color, temperature, and moisture, capillary refill, hemodynamic values, cardiac rhythm, neck veins, lung sounds	• Monitor response to treatment
• Monitor level of consciousness	• Assess perfusion of the central nervous system
• Monitor gastrointestinal status: abdominal distention, bowel sounds, gastric pH, vomiting, large enteral feeding residual	• Assess perfusion of the gastrointestinal system and prevent potential complications
• Monitor fluid balance: I&O, daily weights, amount and type of drainage (chest tube, nasogastric, wounds)	• Evaluate need for continued fluid volume support
• Monitor serial serum values: Hct, Hgb, WBC, PT, aPTT, D-dimer, platelets, ABGs, chemistry profile, lactate, cultures	• Evaluate response to treatment
• Assess and treat pain and discomfort: monitor pain level; administer analgesics; implement comfort and relaxation measures (turning, repositioning, skin care, music); maintain appropriate room temperature; evaluate patient's response	• Promote patient comfort and evaluate response to pain management
• Administer medications as prescribed and specific for the classification of shock (see Table 11-4)	• Improve outcomes and reduce complications
• Provide wound care as indicated and evaluate healing	• Promote wound healing and prevent infection
• Provide adequate nutritional support; collaborate with dietitian about patient's nutritional needs; promote early enteral feed (if tolerated)	• Promote optimum cell function and healing; reduce complications
• Provide psychological support for patient, family, and others	• Reduce the stress response and physiological demand; promote family functioning
• Evaluate patient response to interventions and adjust treatments accordingly; monitor for complications	• Monitor patient response to determine need for modification of treatment and/or nursing care

ABG, Arterial blood gas; *aPTT,* activated partial thromboplastin time; *ARDS,* acute respiratory distress syndrome; *BP,* blood pressure; *DIC,* disseminated intravascular coagulation; *Hct,* hematocrit; *Hgb,* hemoglobin; *HR,* heart rate; *I&O,* intake and output; *IV,* intravenous; *MAP,* mean arterial pressure; *MODS,* multiple organ dysfunction syndrome; *PT*, prothrombin time; *RR,* respiratory rate; *SpO_2,* oxygen saturation by pulse oximetry; *SvO_2,* mixed venous oxygen saturation; *WBC*, white blood cell.

Based on data from Gulanick M and Myers JL. *Nursing Care Plans: Diagnoses, Interventions, and Outcomes,* 7th edition, St. Louis: Mosby; 2011.

SPECIFIC CLASSIFICATIONS OF SHOCK

Table 11-5 provides a summary of the classifications of shock.

Hypovolemic Shock

Hypovolemic shock occurs when the circulating blood volume is inadequate to fill the vascular network. Intravascular volume deficits may be caused by external or internal losses of either blood or fluid. In these situations, the intravascular blood volume is depleted and unavailable to transport oxygen and nutrients to tissues. The severity of hypovolemic shock is dependent upon the volume deficit, the acuity of volume loss, the type of fluid lost, and the age and preinjury health status of the patient.

External volume deficits include loss of blood, plasma, or body fluids. The most common cause of hypovolemic shock is hemorrhage. External loss of blood may occur after traumatic injury, surgery, or obstetrical delivery or with coagulation alterations (hemophilia, thrombocytopenia, DIC, and anticoagulant medications). External plasma losses may be seen in patients with burn injuries who have significant fluid shifts from the intravascular space to the interstitial space (see Chapter 20. Excessive external loss of fluid may occur through the gastrointestinal tract via suctioning, upper gastrointestinal bleeding, vomiting, diarrhea, reduction in oral fluid intake, or fistulas; through the genitourinary tract as a result of excessive diuresis, diabetes mellitus with polyuria, diabetes insipidus, or Addison disease; or through the skin secondary to diaphoresis without fluid and electrolyte replacement.

Blood or body fluids may be sequestered within the body outside the vascular bed. Internal sequestration of blood may be seen in patients with a ruptured spleen or liver, hemothorax, hemorrhagic pancreatitis, fractures of the femur or pelvis, and dissecting aneurysm. Internal sequestration of body fluids includes ascites, peritonitis, and peripheral edema. Fluid sequestration is also seen in patients with intestinal obstruction, which causes fluid to leak from the intestinal capillaries into the lumen of the intestine.

Fluid losses may be obvious or subtle. Assessment includes weighing dressings; measuring drainage from chest or nasogastric tubes; monitoring potential sites for bleeding, such as surgical wounds, or IV or intraarterial catheter sites after removal; and considering insensible losses, such as perspiration. Abdominal girth is measured periodically in patients in whom occult bleeding may be suspected or in those with ascites. Daily weights are obtained by using the same scale with the patient wearing the same clothing at approximately the same time each day. Evaluation of the hematocrit is useful in determining whether blood or fluid was lost. In a patient with blood loss, the hematocrit will be decreased, whereas in a patient with fluid loss, the hematocrit will be increased.

TABLE 11-5 SUMMARY OF CLASSIFICATIONS OF SHOCK

CLASSIFICATION	POSSIBLE CAUSES	CLINICAL PRESENTATION	MANAGEMENT
Hypovolemic shock	*External loss of blood:* GI hemorrhage Surgery Trauma *External loss of fluid:* Diarrhea Diuresis Burns *Internal sequestration of blood fluid:* Hemoperitoneum Retroperitoneal hemorrhage Hemothorax Hemomediastinum Dissecting aortic aneurysm Femur or pelvic fracture Ascites Pleural effusion	↑↓ HR ↓ BP Tachypnea Oliguria Cool, pale skin Decreased mental status Flat neck veins ↓ CO, CI, RAP, PAP, PAOP ↑ SVR ↓ SvO_2 ↑ *Hematocrit:* if from dehydration ↓ *Hematocrit:* if from blood loss	Eliminate and treat the cause Replace lost volume with appropriate fluid
Cardiogenic shock	Myocardial infarction Myocardial contusion Cardiomyopathy Myocarditis Severe heart failure Dysrhythmias Valvular dysfunction	↑ HR Dysrhythmias ↓ BP Chest pain Tachypnea Oliguria Cool, pale skin ↓ Mentation	Improve contractility with inotropic agents Mechanical support Emergency revascularization Reduce preload Reduce afterload Prevent/treat dysrhythmias

TABLE 11-5 SUMMARY OF CLASSIFICATIONS OF SHOCK—cont'd

CLASSIFICATION	POSSIBLE CAUSES	CLINICAL PRESENTATION	MANAGEMENT
Cardiogenic shock—cont'd	Ventricular septal rupture	Left ventricular failure Right ventricular failure ↓ CO, CI ↑ RAP, PAP, PAOP, SVR ↓ SvO_2	
Obstructive shock	*Impaired diastolic filling:* Cardiac tamponade Tension pneumothorax Constrictive pericarditis Compression of great veins *Increased right ventricular afterload:* Pulmonary embolism (PE) Severe pulmonary hypertension Increased intrathoracic pressure *Increased left ventricular afterload:* Aortic dissection Systemic embolization Aortic stenosis Abdominal hypertension	↑ HR Dysrhythmias ↓ BP Chest pain Dyspnea Oliguria Cool, pale skin Decreased mental status Jugular venous distention *Cardiac tamponade:* muffled heart sounds, pulsus paradoxus *Tension pneumothorax:* diminished breath sounds on affected side, tracheal shift away from affected side *Pulmonary embolism:* right ventricular failure *Aortic dissection:* ripping chest pain, pulse differences between left and right side, widened mediastinum ↓ CO, CI ↑ or normal RAP, PAP, PAOP ↑ PVR, ↓ SVR ↓ SvO_2	Eliminate source of obstruction or compression Pericardiocentesis for cardiac tamponade Fibrinolytics, anticoagulants for PE Emergency decompression for tension pneumothorax
Anaphylactic shock	*Foods:* fish, shellfish, eggs, milk, wheat, strawberries, peanuts, tree nuts (pecans, walnuts), food additives *Drugs:* antibiotics, ACE inhibitors, aspirin, local anesthetics, narcotics, barbiturates, contrast media, blood and blood products, allergic extracts *Bites or stings:* venomous snakes, wasps, hornets, spiders, jellyfish, stingrays, deer flies, fire ants *Chemicals:* latex, lotions, soap, perfumes, iodine-containing solutions	↑ HR; dysrhythmias ↓ BP Chest pain Tachypnea Flushed, warm to hot skin Oliguria Restlessness, change in LOC, seizures Nausea, vomiting, abdominal cramping, diarrhea Dyspnea, cough, stridor, wheezing, dysphagia Urticaria, angioedema, hives ↓ CO, CI ↓ RAP, PAP, PAOP, SVR ↓ SvO_2 ↑ IgE	Remove offending agent or slow absorption: remove stinger; apply ice to sting or bite; discontinue drug, dye, blood; lavage stomach if antigen ingested; flush skin with water Maintain airway, oxygenation, and ventilation; intubation may be necessary Modify or block the effects of mediators: epinephrine, antihistamines, steroids Maintain MAP
Neurogenic shock	General or spinal anesthesia Epidural block Cervical spinal cord injury *Drugs:* barbiturates, phenothiazines, sympathetic blocking agents	↓ HR ↓ BP Hypothermia Warm, dry, flushed skin Oliguria Neurological deficit ↓ CO, CI ↓ RAP, PAP, PAOP, SVR ↓ SvO_2	Eliminate and treat the cause Maintain MAP Maintain adequate heart rate VTE prophylaxis

Continued

TABLE 11-5 SUMMARY OF CLASSIFICATIONS OF SHOCK—cont'd

CLASSIFICATION	POSSIBLE CAUSES	CLINICAL PRESENTATION	MANAGEMENT
Septic shock	*Immunosuppression:* Extremes of age Malnutrition Alcoholism or drug abuse Malignancy History of splenectomy Chronic health problems Immunosuppressive therapies *Significant bacteremia:* Invasive procedures and devices Traumatic wounds or burns GI infection or untreated disease Peritonitis Food poisoning Prolonged hospitalization Translocation of GI bacteria (associated with NPO status)	*Early, hyperdynamic, warm:* ↑ HR Normal or ↓ BP ↑ Pulse pressure Skin warm, flushed Confusion Oliguria ↑ CO, CI ↓ RAP, PAP, PAOP, SVR ↑ SvO_2 *Late, hypodynamic, cold:* ↑ HR ↓ BP ↓ Pulse pressure Skin cool, pale ↓ LOC Anuria Hypothermia ↓ CO, CI Variable RAP, PAP, PAOP, SVR ↓ SvO_2 Positive culture	Good hand-washing techniques Avoid invasive procedures Identify source of infection Meticulous oral and airway care Meticulous catheter and wound care Avoid NPO status: initiate and maintain enteral nutrition Antibiotics as indicated by culture results Control hyperthermia Maintain MAP

ACE, Angiotensin-converting enzyme; *BP,* blood pressure; *CI,* cardiac index; *CO,* cardiac output; *GI,* gastrointestinal; *HR,* heart rate; *IgE,* immunoglobulin; *LOC,* level of consciousness; *MAP,* mean arterial pressure; *NPO,* nothing by mouth; *PAOP,* pulmonary artery occlusion pressure; *PAP,* pulmonary artery pressure; *PE,* pulmonary embolism; *PVR,* pulmonary vascular resistance; *RAP,* right atrial pressure; SvO_2, mixed venous oxygen saturation; *SVR,* systemic vascular resistance; *VTE,* venous thromboembolism.

Hypovolemic shock results in a reduction of intravascular volume and a decrease in venous return to the right side of the heart. Ventricular filling pressures (preload) are reduced, resulting in a decrease in stroke volume and cardiac output. As the cardiac output decreases, blood pressure decreases and tissue perfusion decreases. Figure 11-7 summarizes the pathophysiology of hypovolemic shock.

Patients with hypovolemic shock present with signs and symptoms as a result of poor organ perfusion, including altered mentation ranging from lethargy to unresponsiveness; rapid, deep respirations; cool, clammy skin with weak, thready pulses; tachycardia; and oliguria. Hypovolemic shock resulting from hemorrhage is classified according to the volume of blood lost and the resultant effects on the level of consciousness, vital signs, and urine output (Table 11-6).

An increase in abdominal girth may be an indicator of abdominal bleeding or fluid loss into the abdomen. Ultrasonography is performed to determine the presence of abdominal bleeding or fluid loss. If bleeding or fluid loss is found, computed tomography may be obtained to pinpoint sources of bleeding, (hypovolemic shock) or locate possible abscesses, which can cause sepsis.

Management of hypovolemic shock focuses on identifying, treating, and eliminating the cause of the hypovolemia and replacing lost fluid. Examples of treating the cause include surgery, antidiarrheal medication for diarrhea, and insulin for hyperglycemia. The type of fluid lost is considered when determining fluid replacement. Isotonic crystalloids such as normal saline are generally used first, although blood and blood products may be administered if the patient is bleeding. The 3-for-1 rule is used which recommends the replacement of 300 mL of isotonic solution for every 100 mL of blood lost. Hemodynamic monitoring provides objective data to guide fluid replacement. Patients receiving blood replacement are likely to require less than 3 times the lost volume.[2] Hypertonic saline (3%) expands the intravascular volume by creating an osmotic effect that displaces water from the intracellular space. Administration of hypertonic saline is an alternative in trauma patients because less volume is required.[2]

Cardiogenic Shock

Cardiogenic shock can occur when the heart fails to act as an effective pump. A decrease in myocardial contractility results in decreased cardiac output and impaired tissue perfusion. Cardiogenic shock is one of the most difficult types of shock to treat and carries a hospital mortality of 67%.[13]

The most common cause of cardiogenic shock is an extensive left ventricular myocardial infarction. A correlation exists between the amount of myocardial damage and the likelihood of cardiogenic shock. If 40% or more of the left ventricle is damaged, the likelihood of cardiogenic shock increases. Other causes of cardiogenic shock include

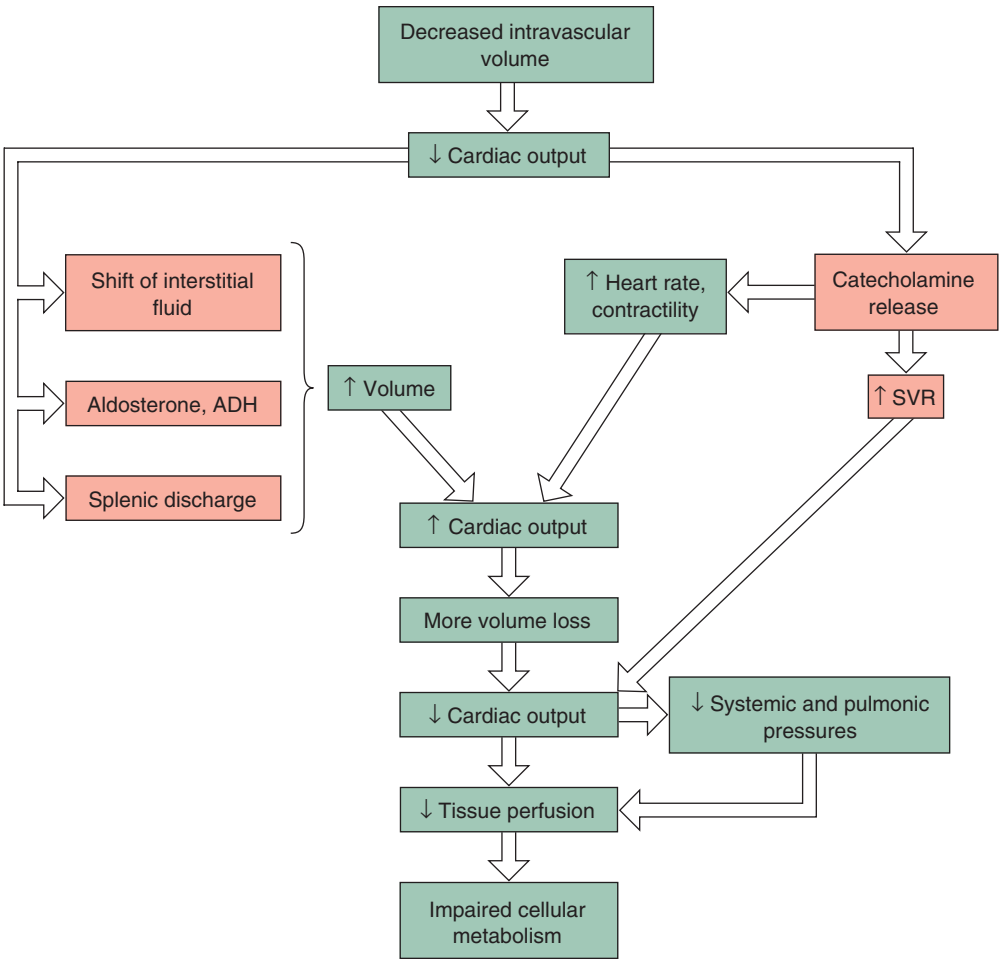

FIGURE 11-7 Hypovolemic shock. This type of shock becomes life threatening when compensatory mechanisms *(orange boxes)* are overwhelmed by continuous loss of intravascular volume. *ADH,* Antidiuretic hormone; *SVR,* systemic vascular resistance. (From McCance KL, Huether SE. *Pathophysiology. The Biologic Basis for Disease in Adults and Children*. 6th ed. St. Louis: Mosby; 2010.)

TABLE 11-6 SEVERITY OF HEMORRHAGIC SHOCK

	CLASS			
INDICATORS	I	II	III	IV
Blood loss (% blood volume)	<15%	15%-30%	30%-40%	>40%
Blood loss (mL)	<750	750-1500	1500-2000	>2000
Heart rate per minute	<100	100-120	120-140	>140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate per minute	14-20	20-30	30-40	>35
Urine output (mL/hr)	>30	20-30	<20	Negligible
CNS/mental status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargy
Fluid replacement	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and blood

CNS, Central nervous system.
Adapted from American College of Surgeons' Committee on Trauma. (2008). *Advanced trauma life support (ATLS) program for doctors student manual* (8th ed.). Chicago: American College of Surgeons.

dysrhythmias, cardiomyopathy, myocarditis, valvular dysfunction, severe heart failure, and structural disorders.[13]

The pathophysiology of cardiogenic shock can be understood by reviewing cardiac dynamics of cardiac output and stroke volume. When damage to the myocardium occurs, contractile force is reduced and stroke volume decreases. Ventricular filling pressures increase because blood remains in the cardiac chambers. Cardiac output and ejection fraction decrease causing hypotension. This hypotension brings about a reflex compensatory peripheral vasoconstriction and increased afterload. At the same time, backup of blood into the pulmonary circulation causes decreased oxygen perfusion across alveolar membranes, thus reducing the oxygen tension in the blood and decreasing cellular metabolism. Figure 11-8 summarizes the pathophysiology of cardiogenic shock.

An increased demand is placed on the myocardium as it attempts to increase perfusion to the cells. The heart rate increases as a compensatory mechanism, resulting in an increased oxygen demand on an overworked myocardium. In patients with cardiogenic shock secondary to acute myocardial infarction, the increased demand may increase infarction size.

The clinical presentation of cardiogenic shock includes manifestations of left ventricular failure (S_3 heart sound, crackles, dyspnea, hypoxemia) and right ventricular failure (jugular venous distention, peripheral edema, hepatomegaly). A pulmonary artery catheter is useful in trending hemodynamic parameters. In cardiogenic shock, cardiac output and cardiac index decrease; however, RAP, pulmonary artery pressure (PAP), and PAOP increase as pressure and volume back up into the pulmonary circulation and the right side of the heart.

Prevention and treatment of cardiogenic shock is aimed at promoting myocardial contractility, decreasing the myocardial oxygen demand, and increasing the oxygen supply to the damaged tissue. Aggressive management after a myocardial infarction includes percutaneous coronary interventions, intracoronary stent placement, or both, fibrinolytic agents when primary percutaneous coronary intervention is not available, glycoprotein IIb/IIIa inhibitors, and beta-blockers to limit the size of the infarction. Pain relief and rest reduce the workload of the heart and the infarct size. Oxygen administration increases oxygen delivery to the ischemic muscle and may help save myocardial tissue.[18]

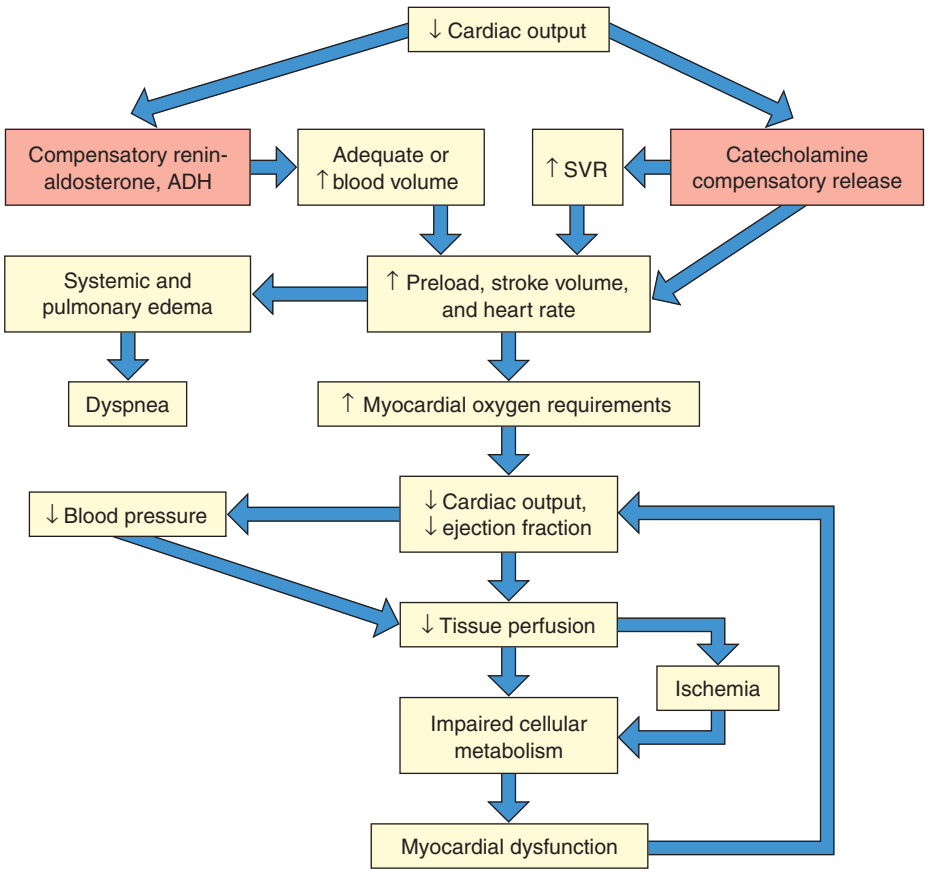

FIGURE 11-8 Cardiogenic shock. *ADH,* Antidiuretic hormone; *SVR,* systemic vascular resistance. Shock becomes life threatening when compensatory mechanisms *(orange boxes)* cause increased myocardial oxygen requirements. (From McCance KL, Huether SE. *Pathophysiology. The Biologic Basis for Disease in Adults and Children.* 6th ed. St. Louis: Mosby; 2010.)

Pharmacological agents are administered to decrease preload (RAP, PAOP), decrease afterload (SVR), increase stroke volume, increase cardiac index, and increase contractility (see Table 11-4). Diuretics (e.g., furosemide) and venous vasodilators (e.g., morphine, nitroglycerin, nitroprusside) reduce preload and venous return to the heart. Nitroglycerin at low doses (<1 mcg/kg/min) causes venous vasodilation to decrease preload. At higher doses (>1 mcg/kg/min) arterial vasodilation decreases afterload. These drugs must be used cautiously because they may cause hypotension, thereby contributing to further cellular hypoperfusion.

Positive inotropic agents (e.g., dobutamine) are given to increase the contractile force of the heart. As contractility increases, ventricular emptying improves, filling pressures decrease (RAP, PAOP), and stroke volume improves. The improved stroke volume increases cardiac output and improves tissue perfusion. However, positive inotropic agents also increase myocardial oxygen demand and must be used cautiously in patients with myocardial ischemia.

Afterload reduction may be achieved by the cautious administration of arterial vasodilators (e.g., nitroprusside) to decrease SVR, increase stroke volume, and increase cardiac index. Blood pressure must be carefully monitored to keep the mean arterial pressure above 65 mm Hg to ensure organ perfusion. Significant hypotension may limit the use of arterial vasodilators, as coronary artery perfusion pressure may be reduced and worsen myocardial ischemia. In this situation, afterload reduction is achieved through the insertion of an intraaortic balloon pump (IABP).

The *IABP* is a cardiac assist device that provides *counterpulsation therapy* concurrently with pharmacological support. IABP therapy is initiated by inserting a dual-chambered balloon into the descending thoracic aorta via the femoral artery. The balloon is inserted percutaneously at the patient's bedside or under fluoroscopy. The tip of the balloon is positioned just distal to the left subclavian artery (Figure 11-9). Correct placement is verified by chest x-ray.

The IABP improves coronary artery perfusion, reduces afterload, and improves perfusion to vital organs. The balloon is inflated mechanically with helium. Inflation and deflation are automatically timed with the cardiac cycle. The IABP inflates during diastole when the aortic valve is closed. The inflation cycle displaces blood backward and forward simultaneously. The backward flow increases perfusion to the coronary arteries, and the forward flow increases perfusion to vital organs. Balloon deflation occurs just before systole and left ventricular ejection. This sudden deflation, along with the displacement of blood that occurred during diastole, reduces the pressure in the aorta and decreases afterload and myocardial oxygen demand. Desired outcomes for a patient in cardiogenic shock with an IABP include decreased SVR, diminished symptoms of myocardial ischemia (chest pain, ST-segment elevation), and increased stroke volume and cardiac output.

Counterpulsation therapy with an IABP requires a high degree of nursing skill because of the complexity of the equipment and the need for frequent monitoring. Many institutions require nurses to be credentialed in managing the patient with an IABP. Limb ischemia and embolic phenomena are potential complications that must be assessed. Other complications include dissection of the aorta, infection, ineffective pumping, and technical problems. Use of the IABP is contraindicated in patients with aortic valve insufficiency or aortic aneurysm.

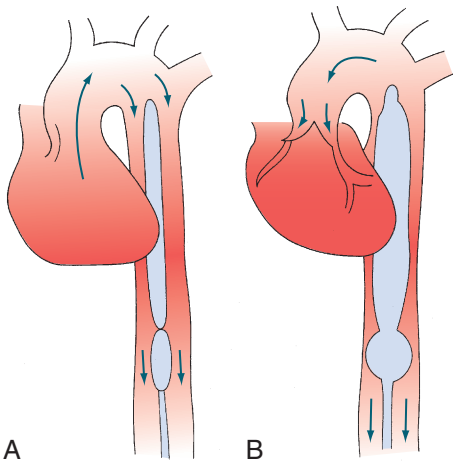

FIGURE 11-9 Intraaortic balloon pump. The balloon is deflated during systole **(A)** and inflated during diastole **(B).**

Ventricular assist devices (VADs) may be used temporarily to support a failing ventricle that has not responded to IABP therapy and pharmacological therapy. VADs are used to treat cardiogenic shock by allowing the ventricle to recover or to support the patient awaiting cardiac transplant as a bridge to transplant. They can be used to support the left ventricle, the right ventricle, or both ventricles. VADs vary in design and technology. In general, they consist of an external pump, which diverts blood from the failing ventricle or ventricles and pumps it back into the aorta (left VAD [LVAD]), the pulmonary artery (right VAD [RVAD]), or both great vessels (Bi-VAD). The use of VADs requires extensive training and advanced nursing care. These devices are not typically available in community hospitals.

Obstructive Shock

Obstructive shock (also known as extracardiac obstructive shock) occurs when there is a physical impairment to adequate circulatory blood flow. Causes of obstructive shock include impaired diastolic filling (cardiac tamponade, tension pneumothorax, constrictive pericarditis, compression of the great veins), increased right ventricular afterload (pulmonary embolism, severe pulmonary hypertension, increased intrathoracic pressures), and increased left ventricular afterload (aortic dissection, systemic embolization, aortic stenosis). Obstruction of the heart or great vessels either impedes venous return to the right side of the heart or prevents effective pumping action of the heart. This results in decreased

cardiac output, hypotension, decreased tissue perfusion, and impaired cellular metabolism (Figure 11-10).

Common clinical findings in obstructive shock include chest pain, dyspnea, jugular venous distention, and hypoxia. Other findings are dependent on the cause. Cardiac tamponade is manifested by muffled heart sounds, hypotension, and pulsus paradoxus. *Pulsus paradoxus* is a decrease in systolic blood pressure of more than 10 mm Hg during inspiration. Tension pneumothorax is manifested by diminished breath sounds on the affected side and tracheal shift away from the affected side. Massive pulmonary embolism is manifested by clinical indications of right ventricular failure (jugular venous distention, peripheral edema, hepatomegaly). Aortic dissection is manifested by complaints of ripping chest pain that radiates to the back, pulse differences between the left and right side, and a widened mediastinum on chest x-ray, echocardiogram, or computed tomography scan.

Obstructive shock may be prevented or treated, or both, by aggressive interventions to relieve the source of the compression or obstruction. Cardiac tamponade may be relieved by a *pericardiocentesis,* or the removal of fluid from the pericardial sac. A tension pneumothorax from blunt or penetrating chest injuries may be relieved by a needle *thoracentesis* to remove the accumulated intrathoracic pressure. The risk of pulmonary embolism may be reduced by early surgical reduction of long bone fractures, devices to enhance circulation in immobile patients (e.g., sequential compression devices), range-of-motion exercises, and prophylactic anticoagulant therapy.

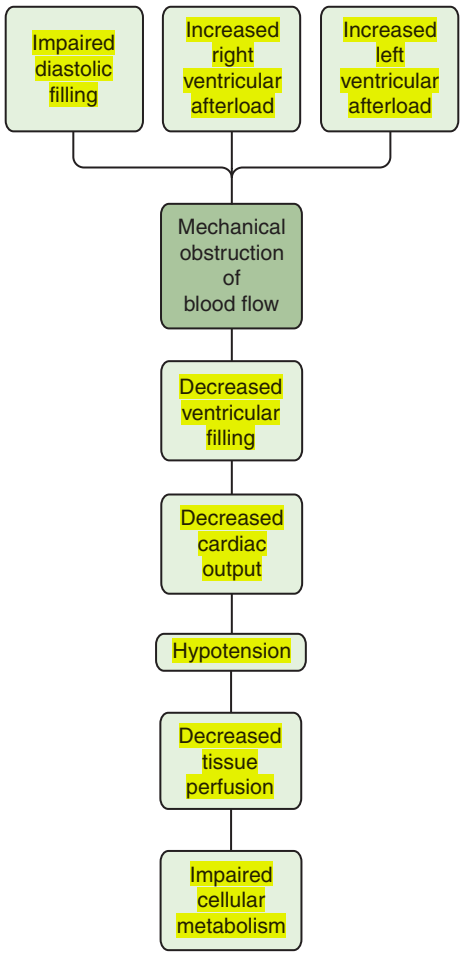

FIGURE 11-10 Obstructive shock.

Distributive Shock

Distributive shock, also known as vasogenic shock, describes several different types of shock that present with widespread vasodilation and decreased SVR. Neurogenic, anaphylactic, and septic shock are forms of distributive shock. Vasodilation increases the vascular capacity; however, the blood volume is unchanged, resulting in a relative hypovolemia. This causes a decrease in venous return to the right side of the heart and a reduction in ventricular filling pressures. Anaphylactic shock and septic shock are also complicated by an increase in capillary permeability, which decreases intravascular volume, further compromising venous return. Eventually, in all forms of distributive shock, stroke volume, cardiac output, and blood pressure decrease, resulting in decreased tissue perfusion and impaired cellular metabolism.

Neurogenic Shock

Neurogenic shock occurs when a disturbance in the nervous system affects the vasomotor center in the medulla. In healthy persons, the vasomotor center initiates sympathetic stimulation of nerve fibers that travel down the spinal cord and out to the periphery. There, they innervate the smooth muscles of the blood vessels to cause vasoconstriction. In neurogenic shock, there is an interruption of impulse transmission or a blockage of sympathetic outflow resulting in vasodilation, inhibition of baroreceptor response, and impaired thermoregulation. Consequently, these reactions create vasodilation with decreased SVR, venous return, preload, and cardiac output and a relative hypovolemia. Figure 11-11 summarizes the pathophysiology of neurogenic shock.

Causes of neurogenic shock include injury or disease of the upper spinal cord, spinal anesthesia, nervous system damage, administration of ganglionic and adrenergic blocking agents, and vasomotor depression. Patients who have a cervical spinal cord injury may experience a permanent or temporary interruption in sympathetic nerve stimulation. Spinal anesthesia may extend up the spinal cord and may block sympathetic nerve impulses from the vasomotor center. Vasomotor depression may be seen with deep general anesthesia, injury to the medulla, administration of drugs, severe pain, and hypoglycemia.

The most profound features of neurogenic shock are bradycardia with hypotension from the decreased sympathetic activity. The skin is frequently warm, dry, and flushed. Hypothermia develops from uncontrolled heat loss. Venous pooling in the lower extremities promotes the formation of deep vein thrombosis, which can result in pulmonary embolism. A neurological deficit may be evident.

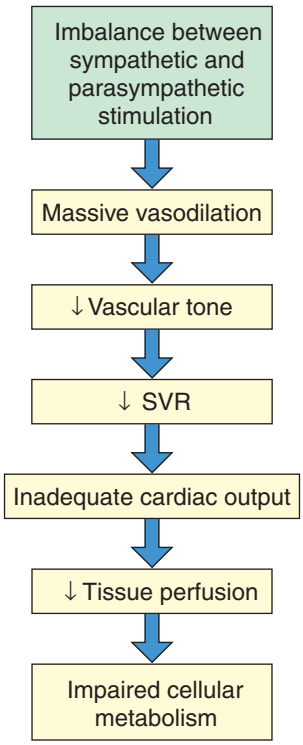

FIGURE 11-11 Neurogenic shock. *SVR,* Systemic vascular resistance. (From McCance KL, Huether SE. *Pathophysiology. The Biologic Basis for Disease in Adults and Children.* 6th ed. St. Louis: Mosby; 2010.)

Management focuses on treating the cause, including reversal of offending drugs or glucose administration for hypoglycemia. Immobilization of spinal injuries with traction devices (halo brace to maintain alignment) or surgical intervention to stabilize the injury assists in preventing severe neurogenic shock. For patients receiving spinal anesthesia, elevating the head of the bed may prevent the progression of the spinal blockade up the cord. IV fluids are infused to treat hypotension; however, they must be given cautiously to prevent fluid overload and cerebral or spinal cord edema.[6] Vasopressors are frequently required to maintain perfusion. Alpha- and beta-adrenergic agents, such as dopamine or norepinephrine, are preferred because pure alpha-adrenergic agents, such as phenylephrine, are associated with persistent bradycardia.[17] Hypothermia is common so the patient is rewarmed slowly, because rapid rewarming may cause vasodilation and worsen the patient's hemodynamic status. Atropine is used for symptomatic bradycardia; however, a temporary or permanent pacemaker may be required.

Anaphylactic Shock

A severe allergic reaction can precipitate a second form of distributive shock known as anaphylactic shock. Antigens, which are foreign substances to which someone is sensitive, initiate an antigen-antibody response. Table 11-5 lists some common antigens causing anaphylaxis.

Once an antigen enters the body, antibodies (immunoglobulin E [IgE]) are produced that attach to mast cells and basophils. The greatest concentrations of mast cells are found in the lungs, around blood vessels, in connective tissue, and in the uterus. Mast cells are also found to a lesser extent in the kidneys, heart, skin, liver, and spleen and in the omentum of the gastrointestinal tract. Basophils circulate in the blood. Both mast cells and basophils contain histamine and histamine-like substances, which are potent vasodilators.

The initial exposure (primary immune response) to the antigen does not usually cause any harmful effects; however, subsequent exposures to the antigen may cause an anaphylactic reaction (secondary immune response). The antigen-antibody reaction causes cellular breakdown and the release of powerful vasoactive mediators from the mast cells and basophils. These mediators cause bronchoconstriction, excessive mucus secretion, vasodilation, increased capillary permeability, inflammation, gastrointestinal cramps, and cutaneous reactions that stimulate nerve endings, causing itching and pain. Figure 11-12 summarizes the pathophysiology of anaphylactic shock. The combined effects result in decreased blood pressure, relative hypovolemia caused by the vasodilation and fluid shifts, and symptoms of anaphylaxis that primarily affect the skin, respiratory, and gastrointestinal systems.

Obtaining a thorough history of allergies and drug reactions, especially reactions to drugs with similar structures, is an important strategy to prevent anaphylactic shock. For example, if patients are allergic to penicillin, they are likely to have a reaction to ampicillin (Principen), carbenicillin (Geopen), or nafcillin sodium. The response to IV administration of medications, particularly antibiotics, is monitored. Injecting small amounts of a drug before the entire dose is given is recommended to assist in detecting a possible reaction. Care is taken during the transfusion of blood or blood products, which can result in allergic reactions. The patient receiving any of these products is observed closely for any signs of an allergic reaction.

The clinical presentation of anaphylactic shock includes flushing, pruritus, urticaria, and angioedema (swelling of eyes, lips, tongue, hands, feet, genitalia). Cough, runny nose, nasal congestion, hoarseness, dysphonia, and dyspnea are common because of upper airway obstruction from edema of the larynx, epiglottis, or vocal cords. Stridor may occur as a result of laryngeal edema. Lower airway obstruction may result from diffuse bronchoconstriction and cause wheezing and chest tightness. Tachycardia and hypotension occur, and the patient may show signs of pulmonary edema. Gastrointestinal symptoms of nausea, vomiting, cramping, abdominal pain, and diarrhea may also occur. Neurological symptoms include lethargy and decreased consciousness. Elevated levels of IgE are seen on laboratory analysis.

Goals of therapy are to remove the antigen, reverse the effects of the mediators, and promote adequate tissue perfusion. If the anaphylactic reaction results from medications, contrast dye, or blood or blood products, the infusion is immediately stopped. Airway, ventilation, and circulation are

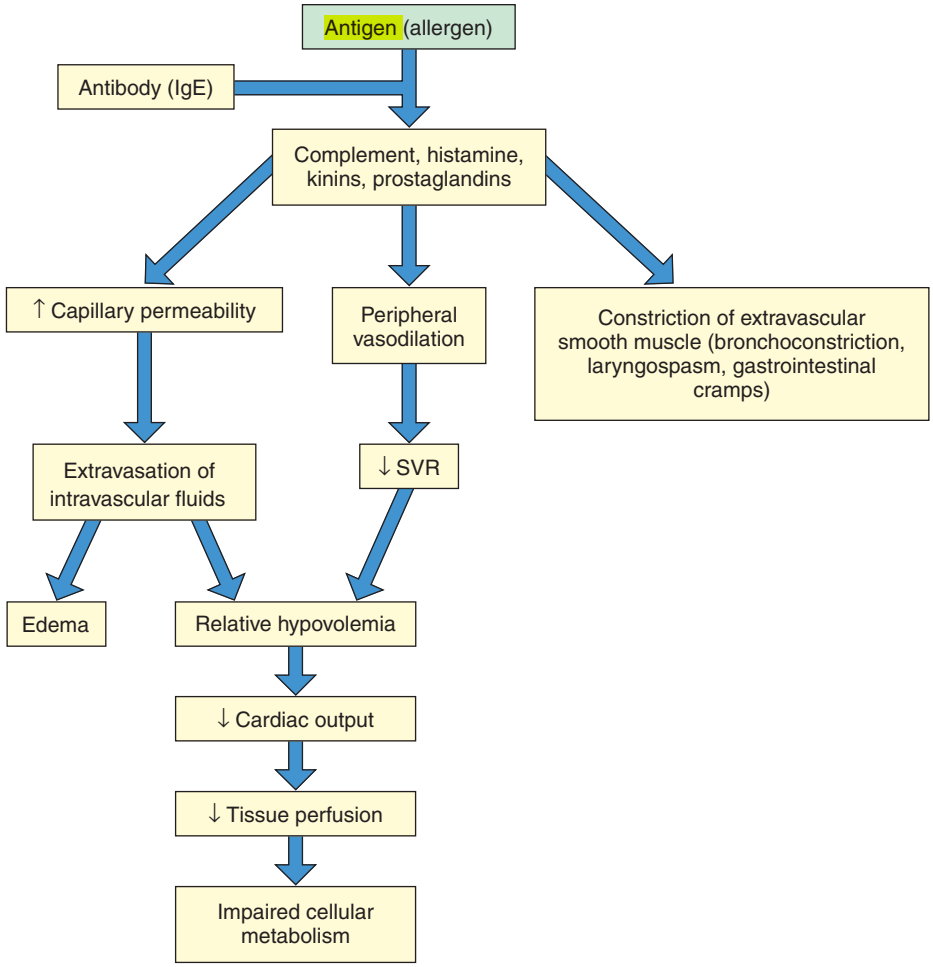

FIGURE 11-12 Anaphylactic shock. *IgE,* Immunoglobulin E; *SVR,* systemic vascular resistance. (From McCance KL, Huether SE. *Pathophysiology. The Biologic Basis for Disease in Adults and Children*. 6th ed. St. Louis: Mosby; 2010.)

supported. Laryngeal edema may be severe enough to require intubation or cricothyrotomy if swelling is so severe that an endotracheal tube cannot be placed. Oxygen is administered to keep the SpO_2 greater than 90%. Removal of the offending agent is achieved by removing the stinger, administering antivenom, stopping the drug, performing gastric lavage, or flushing the skin.[31]

Epinephrine is the drug of choice for treating anaphylactic shock. Epinephrine is an adrenergic agent that promotes bronchodilation and vasoconstriction. For mild reactions, epinephrine 0.1 to 0.25 mg (1.0 to 2.5 mL of a 1:10,000 solution) is administered intramuscularly or subcutaneously. The dose may be repeated at 20- to 30-minute intervals until anaphylaxis is resolved. To block histamine release, diphenhydramine (Benadryl), an H_1-receptor blocker, or ranitidine, an H_2-receptor blocker, may decrease some of the cutaneous symptoms of anaphylaxis, but both are considered second-line treatment.[31] Corticosteroids such as methylprednisolone (Solu-Medrol) are used to reduce inflammation. Fluid replacement, positive inotropic agents, and vasopressors may be required.

Septic Shock

Septic shock is one component of a continuum of progressive clinical insults including SIRS, sepsis, and MODS. In the past, there has been confusion about what these various syndromes represented. Because of this confusion and the complexity of these syndromes, consensus definitions were identified in 1992[8] and were reviewed in 2001.[21] The 2001 consensus group took the definitions a step further by identifying diagnostic criteria for sepsis. The intent of the criteria is to provide a tool to recognize and diagnose sepsis quickly, prompt the search for an infectious source, and to initiate the appropriate therapy. None of the diagnostic criteria are specific for sepsis because these parameters can be altered by other conditions. The definitions and diagnostic criteria are presented in Table 11-7. Invasion of the host by a microorganism or an infection begins the process that may progress to sepsis, followed by severe sepsis and septic shock, which progresses to MODS.

Once a microorganism has invaded a host, an inflammatory response is initiated to restore homeostasis. SIRS occurs, leading to release of inflammatory mediators or cytokines,

TABLE 11-7 CLINICAL CONDITION, DIAGNOSTIC CRITERIA, AND MANAGEMENT IN THE CONTINUUM OF SEPSIS

CLINICAL CONDITION AND DEFINITION	DIAGNOSTIC CRITERIA	MANAGEMENT
Infection: Inflammatory response to microorganisms	Fever	Administer antibiotics Surgical excision or drainage of source of infection
SIRS: Systemic inflammatory response to a clinical insult including infection, pancreatitis, ischemia, trauma, or hemorrhagic shock	Tachycardia (HR ≥90 beats/min) Respiratory rate >20 breaths/min or $PaCO_2$ <32 mm Hg Temperature >38° C (hyperthermia) or <36° C (hypothermia)	Administer antibiotics Remove source of infection Maintain adequate ventilation and oxygenation Replace fluid
Sepsis: Systemic response to infection manifested by two or more of the symptoms noted with SIRS	Leukocytosis (WBC count >12,000 cells/microliter) or leukopenia (WBC count <4000 cells/microliter) or >10% immature bands	Antipyretics
Severe sepsis: Sepsis associated with organ dysfunction.	As above with evidence of impaired systemic perfusion and organ function, possibly including lactic acidosis, oliguria, or acute change in mental status	Administer antibiotics Remove source of infection Maintain adequate ventilation and oxygenation Maximize oxygen delivery; minimize oxygen demand Replace fluid Administer vasoactive medications Correct acid-base abnormalities Monitor and support organ function Consider hydrocortisone if poor response to fluids and vasoactive medication[12]
Septic shock: Sepsis with hypotension despite adequate fluid resuscitation, along with perfusion abnormalities	Hypotension Lactic acidosis, oliguria, acute change in mental status Patients receiving inotropic agents or vasopressors may not exhibit hypotension	Administer antibiotics Maintain adequate ventilation and oxygenation Maximize oxygen delivery; minimize oxygen demand Replace fluid Administer vasoactive medications Correct acid-base abnormalities Monitor and support organ function Consider hydrocortisone if poor response to fluids and vasoactive medication[12]
MODS: Altered organ function in acutely ill patients	See Table 11-9	Maintain adequate ventilation and oxygenation Maximize oxygen delivery; minimize oxygen demand Perform dialysis Monitor and support organ function Monitor clotting studies and bleeding

HR, Heart rate; *MODS,* multiple organ dysfunction syndrome; *$PaCO_2$,* partial pressure of arterial carbon dioxide; *SIRS,* systemic inflammatory response syndrome; *WBC,* white blood cell.

Definitions modified from Dellinger RP, Levy MM, Carlet JM, Bion J, Parker M, Jaeschke R, et al. Surviving Sepsis Campaign: International guidelines for management of severe sepsis and septic shock: 2008. *Critical Care Medicine,* 2008;36(1), 298-327.

which are produced by the white blood cells. SIRS can also occur as a result of trauma, shock, pancreatitis, or ischemia.[12] For reasons not completely understood, SIRS may progress to septic shock and MODS (Figure 11-13). Cytokines are proinflammatory or antiinflammatory. Proinflammatory cytokines including tumor necrosis factor, interleukin-1α, and interleukin-β produce pyrogenic responses and initiate the hepatic response to infection. Antiinflammatory cytokines including nitric oxide, lipopolysaccharide, and interleukin-1–receptor antagonist are compensatory, ensuring that the effect of the proinflammatory mediators does not become destructive. In sepsis, continued activation of proinflammatory

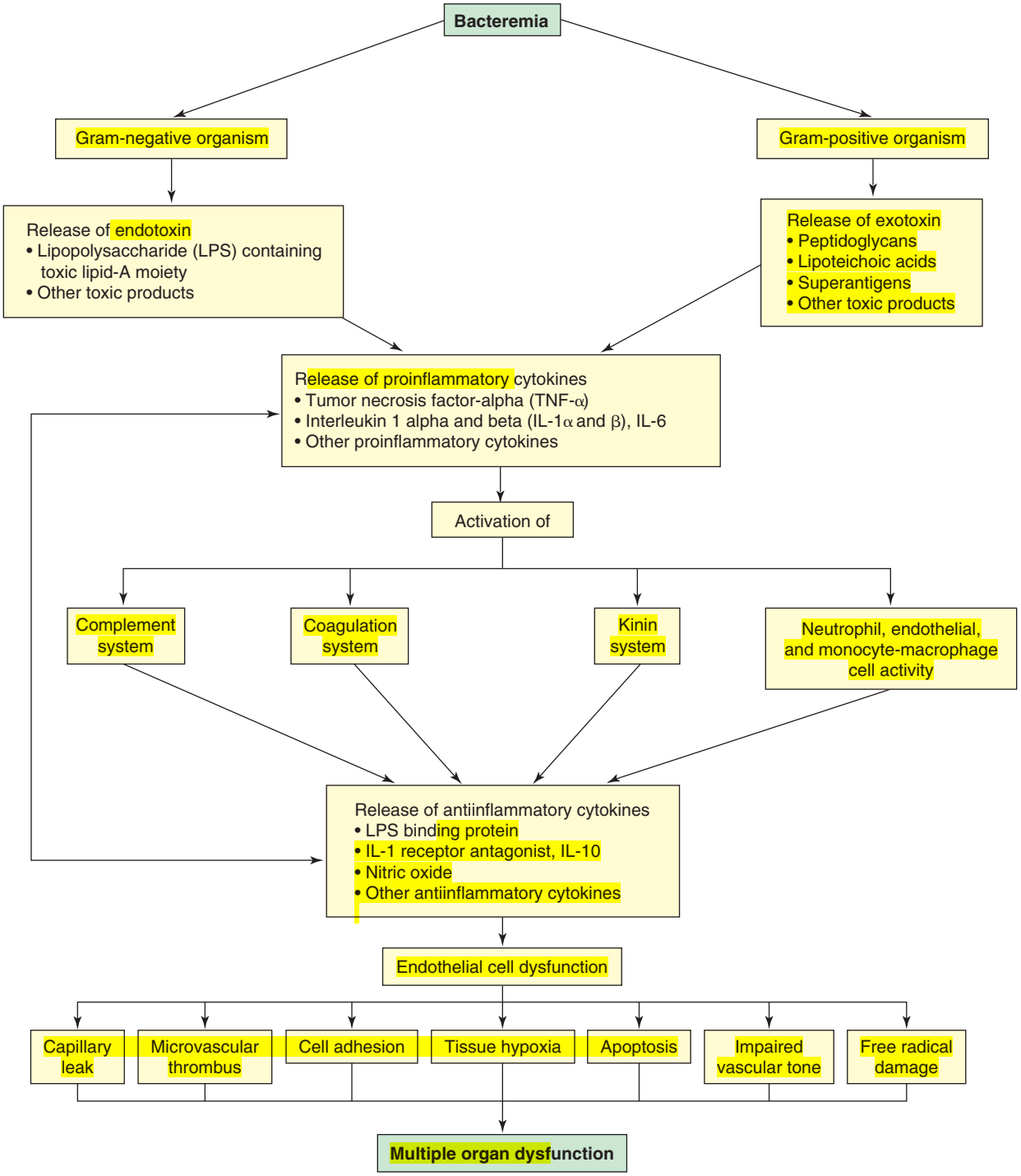

FIGURE 11-13 Sepsis and septic shock pathophysiology. (Modified from McCance KL, Huether SE. *Pathophysiology. The Biologic Basis for Disease in Adults and Children*. 6th ed. St. Louis: Mosby; 2010.)

cytokines overwhelms the antiinflammatory cytokines and excessive systemic inflammation results.

A state of enhanced coagulation occurs through stimulation of the coagulation cascade, with a reduction in the levels of activated protein C and antithrombin III. This results in the generation of thrombin and the formation of microemboli that impair blood flow and organ perfusion. Fibrinolysis is activated in response to the activation of the coagulation cascade to promote clot breakdown. However, activation is followed by inhibition, further promoting coagulopathy. This imbalance among inflammation, coagulation, and fibrinolysis results in systemic inflammation, widespread coagulopathy, and microvascular thrombi that impair tissue perfusion, leading to MODS.

TABLE 11-8 STAGES OF SEPTIC SHOCK

EARLY (HYPERDYNAMIC; LOOKS LIKE INFECTION)	LATE (HYPODYNAMIC; LOOKS LIKE SHOCK)
Clinical Presentation	
Tachycardia	Tachycardia
Pulses bounding	Pulses weak and thready
Blood pressure: normal or low	Hypotension
Wide pulse pressure	Narrow pulse pressure
Skin warm, flushed	Skin cool, pale
Hyperpnea	Bradypnea or tachypnea
Change in mental status (irritability and confusion)	↓ Level of consciousness (lethargy or coma)
Oliguria	Anuria
Hyperthermia	Hypothermia
Hemodynamic Parameters	
↑ CO/CI	↓ CO/CI
↓ RAP/PAP/PAOP	RAP/PAP/PAOP variable
↓ SVR	SVR variable
↑ SvO_2	↓ SvO_2
Diagnostic Findings	
ABGs: respiratory alkalosis with hypoxemia	ABGs: metabolic acidosis with hypoxemia
↑ PT and aPTT	↑ PT and aPTT
↓ Platelets	↓ Platelets
↑ WBC count	↓ WBC count
↑ Glucose	↓ Glucose
	↑ BUN, creatinine
	↑ Serum arterial lactate
	↑ Amylase, lipase
	↑ AST, ALT, LDH

ABGs, Arterial blood gases; *ALT,* alanine aminotransferase; *aPTT,* activated partial thromboplastin time; *AST,* aspartate aminotransferase; *BUN,* blood urea nitrogen; *CI,* cardiac index; *CO,* cardiac output; *LDH,* lactic dehydrogenase; *PAOP,* pulmonary artery occlusion pressure; *PAP,* pulmonary artery pressure; *PT,* prothrombin time; *RAP,* right atrial pressure; *SvO_2,* oxygen saturation of venous blood; *SVR,* systemic vascular resistance; *WBC,* white blood cell.

These inflammatory mediators also damage the endothelial cells that line blood vessels, producing profound vasodilation and increased capillary permeability. Initially, this results in tachycardia, hypotension, and low SVR. Although norepinephrine and the renin-angiotensin-aldosterone system are activated in response to this clinical state, they are unable to enter the cells, and hypotension and vasodilation persist. In contrast, the plasma levels of the antidiuretic hormone (ADH or vasopressin) are low despite the presence of hypotension. The exact mechanism that creates this low concentration is not known; however, administering a continuous vasopressin infusion significantly increases blood pressure in septic shock.[20]

Once sepsis is present, it can progress to septic shock. Septic shock is sepsis with hypotension that is unresponsive to fluid resuscitation along with signs of inadequate organ perfusion such as metabolic acidosis, acute encephalopathy, oliguria, hypoxemia, or coagulation disorders. The clinical course of septic shock is frequently differentiated between the early (warm, hyperdynamic) phase and the late (cold, hypodynamic) phase (Table 11-8).

Factors that increase the risk of developing sepsis are categorized as either situations that cause immunosuppression or situations that cause significant bacteremia (see Table 11-5). Sepsis is infection with SIRS and is the systemic response to infection. SIRS is present if two or more of the clinical manifestations of SIRS are identified (see Table 11-7). Sepsis can advance to severe sepsis with hypotension, chills, decreased urine output, decreased skin perfusion, poor capillary refill, skin mottling, decreased platelets, petechiae, hyperglycemia, and unexplained changes in mental status.[14]

Prevention of sepsis is promoted by preventing infections, including proper hand washing, use of aseptic technique, and awareness of the patient at risk. The critically ill patient is debilitated and has many potential portals of entry for bacterial invasion. Meticulous technique is required during procedures such as suctioning, dressing changes, and wound care and when handling catheters or tubes. Frequent assessment of temperature, wounds, and laboratory results including white blood cell count, differential counts, and cultures is important for the identification of infection.

Gram-negative bacteria such as *Escherichia coli*, *Klebsiella* species, or *Pseudomonas* species are a common cause of infections in adults. Common sites of infection include the pulmonary system, urinary tract, gastrointestinal system,

and wounds. Urinary tract infection is an often overlooked cause of secondary bloodstream infections. Minimizing the use of indwelling catheters by assessing daily their need and promptly removing unnecessary catheters is recommended.[25,28]

Gram-positive bacteria such as *Staphylococcus aureus* can also lead to sepsis and septic shock. These bacteria release a potent toxin that exerts its effects within hours. Gram-positive infection has been associated with the use of tampons in menstruating women (known as toxic shock syndrome); however, it is also seen after vaginal and cesarean delivery and in patients with surgical wounds, abscesses, infected burns, abrasions, insect bites, herpes zoster, cellulitis, septic abortion, and osteomyelitis. In addition, the bacteria may be transmitted from mother to newborn. Management includes antimicrobial therapy, removal of the source of infection if one is found, fluid resuscitation, and vasoactive medication to improve cardiac performance.

Pneumonia is a common trigger for sepsis. Ventilator-associated pneumonia (VAP) is a significant risk factor for the development of sepsis. Several strategies have been identified that reduce the risk of ventilator-associated pneumonia and are easily implemented. These include providing regular oral care with chlorhexidine-based antiseptic to intubated patients, and reducing the number of ventilator circuit changes.[1,3,12] VAP prevention measures also include venous thromboembolism (VTE) prophylaxis, stress ulcer prophylaxis, and ventilator weaning trials. This capacity is assessed with a "sedation vacation," a planned holding of sedation to evaluate the patient's ability to wean from the ventilator. Another strategy is the use of an endotracheal tube with a dorsal lumen to allow continuous suction of secretions from the subglottic area.[15]

Timely identification of the causative organism and the initiation of appropriate antibiotics improve survival of patients with sepsis or septic shock.[16] Any catheter suspected to be a source of infection should be removed. Surgery may be required to locate the source of infection, drain an abscess, and/or debride any necrosis.

Before antibiotic therapy is initiated, culture and sensitivity tests of blood, urine, sputum, wound, tip of a catheter, and any suspicious site are obtained. This helps to identify the source of the infection, the type of organisms, and which antibiotics should be used.[27] However, the need for early administration of antibiotics, preferably within 1 hour, requires the initial antibiotic selection be directed toward the most likely organism, and frequently, empirical and broad-spectrum antibiotics are initiated.[12] Antibiotics may be changed after Gram stain results (approximately 4 hours) or culture and sensitivity results (approximately 72 hours) are available. Antibiotics are discontinued if the cause of the sepsis is not bacterial. Unfortunately, antibiotics do not act on the immune response to infection and do not directly improve tissue perfusion.

Early goal-directed therapy has been shown to decrease mortality in patients with severe sepsis and septic shock and is advocated for the first 6 hours of sepsis resuscitation.[29,36] Early goal-directed therapy includes administration of IV fluids to keep the central venous pressure at 8 mm Hg or greater (but not >15 mm Hg) and the heart rate at less than 110 beats per minute, administration of vasopressors to keep the mean arterial pressure at 65 mm Hg or greater, and administration of dobutamine, packed red blood cells, or both to keep the central venous oxygen saturation ($ScvO_2$) at 70% or greater.[12,36,40]

Isotonic crystalloid solutions are infused for fluid resuscitation. Colloids are likely to leak out of the vascular bed into the interstitium because of increased capillary permeability. Vasopressors, frequently norepinephrine or dopamine, are used to increase SVR and mean arterial pressure. Vasopressin may be added to norepinephrine, especially when high doses of norepinephrine are required.[23] Advantages of vasopressin include decreasing exogenous catecholamines and increasing the release of cortisol and ACTH.[20] In addition, vasopressin causes vasoconstriction without the adverse effects of tachycardia and ventricular ectopy seen with catecholamines such as dopamine or norepinephrine. Dobutamine may be used to increase the myocardial contractility and improve the cardiac index and DO_2 in patients with a decreased $ScvO_2$. If the patient's hematocrit is less than 30%, the administration of packed red blood cells is advocated to increase DO_2.[32]

Elevated cardiac troponin levels and elevated brain natriuretic peptide (BNP) indicate left ventricular dysfunction and a poor prognosis in patients with sepsis and septic shock.[22,38] ACTH-stimulated cortisol levels may be measured because poor ACTH-cortisol responses are associated with a high mortality.[11] Corticosteroids have been shown to reduce mortality for those patients with sepsis or septic shock who have an inadequate response to the ACTH stimulation test.[5] Routine use of corticosteroids, however, is not recommended because of the effects on glucose homeostasis, the risk for infection, and the potential for myopathy.[34]

In severe sepsis, the patient has excessive coagulation, inflammation, and impaired fibrinolysis. Recombinant human activated protein C (drotrecogin alfa [Xigris]) is an antiinflammatory, antithrombotic, and profibrinolytic agent that reduces the inflammatory, clotting, and bleeding responses to sepsis. It has been shown to reduce mortality in patients with severe sepsis with dysfunction of two organ systems.[7] Continued evaluation has failed to show a survival benefit for patients with severe sepsis and septic shock. The manufacturer announced a voluntary recall from the market in October 2011 (www.fda.gov/Drugs/DrugSafety/ucm277114.htm).

Hyperglycemia and insulin resistance are common in the patient with sepsis. The effect is even more significant in patients with MODS caused by sepsis.[37] Guidelines published in 2008[12] recommend frequent glucose testing and intensive IV insulin protocols to maintain blood glucose levels at less than 150 mg/dL. Data from the NICE-SUGAR trial[24] suggest that the target be less than 180 mg/dL. On the

basis of those results, normal blood glucose levels may not be the clinical goal.

Although pyrogens (polypeptides that produce fever) aid in activation of the immune response, temperature reduction is considered for core body temperatures of 41° C or higher because of the significant increase in oxygen consumption. Treatment of fever includes physiological cooling (ice packs, tepid baths, cooling blanket, or misting) along with administration of antipyretics (acetaminophen, ibuprofen, or aspirin). Care must be taken to avoid overcooling because hypothermia adversely affects oxygen delivery and may result in shivering, which increases oxygen consumption.

Many experimental therapies have been advocated for sepsis and septic shock. Plasmapheresis may remove endotoxin and other harmful substances produced by either the infective organism or the inflammatory process.[19] Immunoglobulins may also be prescribed, especially in patients who are immunocompromised.[19]

MULTIPLE ORGAN DYSFUNCTION SYNDROME

MODS is the progressive dysfunction of two or more organ systems as a result of an uncontrolled inflammatory response to severe illness or injury. Organ dysfunction can progress to organ failure and death. The most common causes of MODS are sepsis and septic shock; however, MODS can occur after any severe injury or disease process that activates a massive systemic inflammatory response including any classification of shock. The immune system and the body's response to stress can cause maldistribution of circulating volume, global tissue hypoxia, and metabolic alterations that result in damage to organs. Failure of two or more organs is associated with an estimated 45% to 55% mortality, 80% mortality when three or more organ systems fail, and 100% mortality if three or more organ systems fail for longer than 4 days.[4]

Damage to organs may be primary or secondary. In *primary MODS* there is direct injury to an organ from shock, trauma, burn injury, or infection with impaired perfusion that results in dysfunction. Decreased perfusion may be localized or systemic. As a result of this insult, the stress response and inflammatory response are activated with the release of catecholamines and activation of mediators that affect cellular activity (Figure 11-14).

Secondary MODS is a consequence of widespread systemic inflammation that results in dysfunction of organs not involved with the initial insult. It occurs in response to altered regulation of the acute immune and inflammatory responses. Failure to control the inflammatory response leads to excessive production of inflammatory cells and biochemical mediators that cause widespread damage to vascular endothelium and organ damage. The interaction of injured organs then leads to self-perpetuating inflammation with maldistribution of blood flow and hypermetabolism.

Maldistribution of blood flow refers to the uneven distribution of flow to various organs and between the large vessels and capillary beds. It is caused by vasodilation, increased capillary permeability, selective vasoconstriction, and impaired microvascular circulation. This impaired blood flow leads to impaired tissue perfusion and a decreased oxygen supply to the cells. The organs most severely affected are the lungs, splanchnic bed, liver, and kidneys.

Hypermetabolism with altered carbohydrate, fat, and lipid metabolism is initially compensatory to meet the body's increased demands for energy. Eventually, hypermetabolism becomes detrimental, placing tremendous demands on the heart as cardiac output increases up to twice the normal value. Hyperglycemia occurs as gluconeogenesis by the liver increases and glucose use by the cells decreases.

The decreased oxygen delivery to the cells (from maldistribution of blood flow) and increased oxygen needs of the cells (from hypermetabolism) create an imbalance in oxygen supply and demand. In MODS, the amount of oxygen consumed becomes dependent on the amount of oxygen that can be delivered to the cells. Hypoxemia, cellular acidosis, and impaired cellular function result with the development of multiple organ failure.

The clinical presentation of MODS is caused by inflammatory mediator damage, tissue hypoxia, and hypermetabolism. Damage to the organs is usually sequential rather than simultaneous. The first system frequently affected is the pulmonary system, with acute ARDS developing within 12 to 24 hours after the initial insult. Coagulopathy frequently develops, followed by renal, hepatic, and intestinal impairment.[33] Failure of the cardiovascular system, neurological system, or both, are frequently fatal events. MODS progresses from minor dysfunction of one or multiple organs to multiple organs requiring support.

Criteria used in the diagnoses of organ dysfunction are described in Table 11-9. Pulmonary dysfunction is manifested by tachypnea, hypoxemia despite high levels of supplemental oxygen, and chest x-ray changes. Hematological dysfunction is manifested by petechiae, bleeding, thrombocytopenia, prolonged PT and aPTT, increased fibrin split products, and a positive D-dimer. The earliest sign of hepatic dysfunction is hypoglycemia, which is followed by jaundice, increased liver enzymes and bilirubin, prolonged PT, and decreased albumin. The first indication of intestinal dysfunction is frequently intolerance of enteral feedings with abdominal distention and increased retention volumes. Renal dysfunction is evidenced by oliguria to anuria, increased blood urea nitrogen and creatinine, and fluid and electrolyte imbalance. Tachycardia (frequently with dysrhythmias), hypotension, and hemodynamic alterations indicate cardiovascular dysfunction. Finally, cerebral dysfunction is manifested by a change in level of consciousness, confusion, and focal neurological signs such as hemiparesis. The final response to MODS is hypotension that is unresponsive to fluids and vasopressors, and cardiac arrest.

Management of MODS focuses on prevention and support. The initial source of inflammation must be eliminated or controlled. A secondary insult must be avoided. Potential

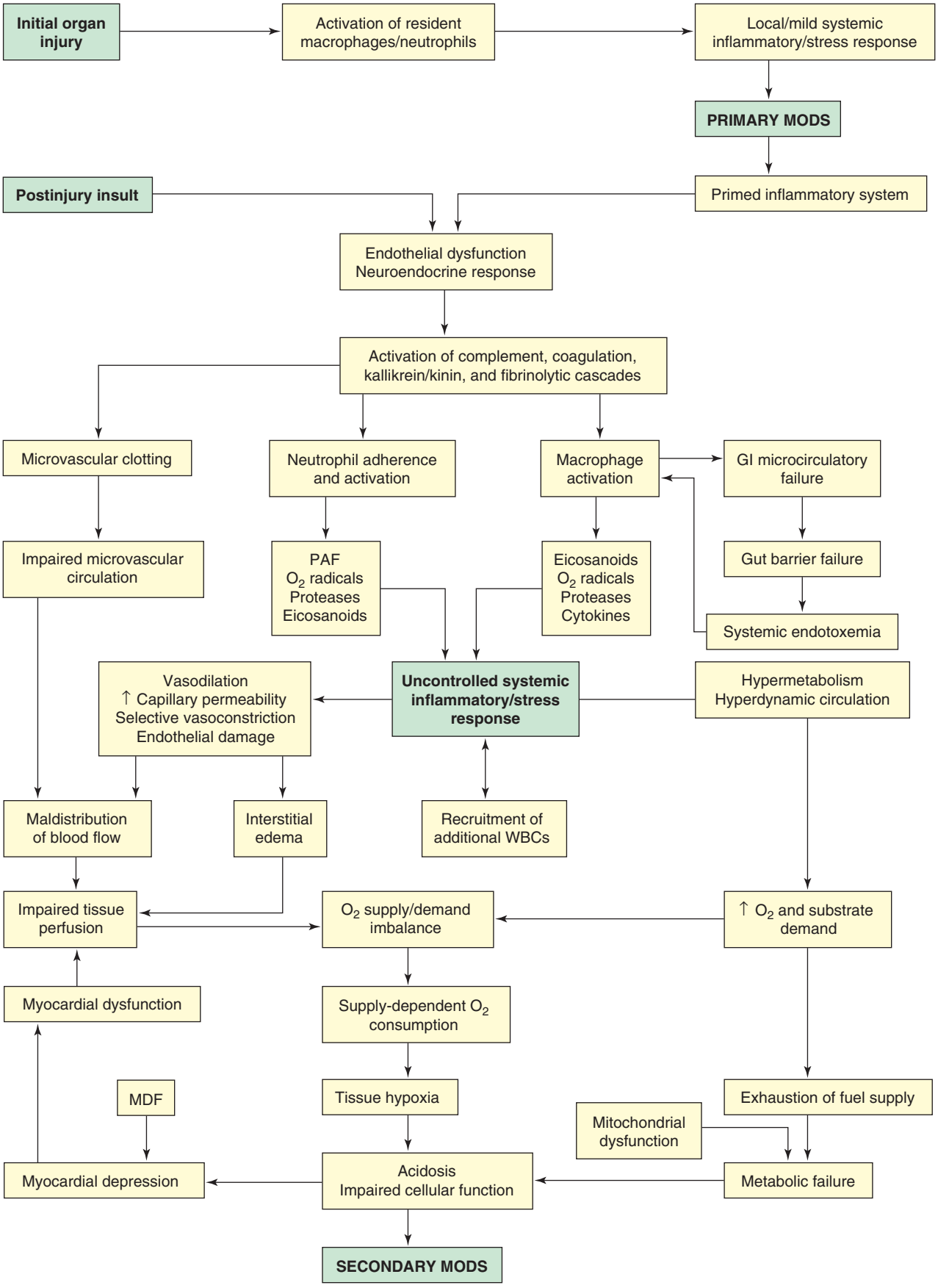

FIGURE 11-14 Pathogenesis of multiple organ dysfunction syndrome. *GI,* Gastrointestinal; *MDF,* myocardial depressant factor; *MODS,* multiple organ dysfunction syndrome; *PAF,* platelet activating factor; *WBCs,* white blood cells. (Modified from McCance KL, Huether SE. *Pathophysiology. The Biologic Basis for Disease in Adults and Children.* 6th ed. St. Louis: Mosby; 2010.)

TABLE 11-9 MULTIPLE ORGAN DYSFUNCTION SYNDROME

SYSTEM	DYSFUNCTION	CLINICAL PRESENTATION
Pulmonary	Acute respiratory distress syndrome	Predisposing factor such as shock or sepsis Unexplained hypoxemia (↓ PaO_2, ↓ SaO_2) Dyspnea Tachypnea PaO_2/FiO_2 ratio <300 for acute lung injury and <200 for ARDS Bilateral pulmonary infiltrates on chest x-ray PAOP <18 mm Hg
Cardiovascular	Hyperdynamic or hypodynamic	See Table 11-8
Hematological	Disseminated intravascular coagulation	Fibrin split products >1:40 or D-dimer >2 mg/L Thrombocytopenia Prolonged PT and aPTT INR >1.5 Bleeding Petechiae
Renal	Acute tubular necrosis	Oliguria ↑ Serum creatinine, ↑ BUN Urinary sodium >20 mEq/L
Liver	Hepatic dysfunction/failure	↑ Serum bilirubin ↑ AST, ALT, LDH Jaundice Hepatomegaly ↑ Serum ammonia ↓ Serum albumin
Central nervous system	Cerebral ischemia/infarction	Lethargy Altered level of consciousness Fever
Metabolic	Lactic acidosis	↑ Serum lactate level

ALT, Alanine transaminase; *aPTT*, activated partial thromboplastin time; *AST*, aspartate transaminase; *BUN*, blood urea nitrogen; *FiO_2*, fraction of inspired oxygen; *INR*, international normalized ratio; *LDH*, lactic dehydrogenase; *PaO_2*, partial pressure of arterial oxygen; *PAOP*, pulmonary artery occlusion pressure; *PT*, prothrombin time; *SaO_2*, arterial oxygen saturation.

sites of infection are removed, including debriding necrotic tissue, draining abscesses, reducing the number of invasive procedures performed, and removing hematomas. Goals are to control infection, provide adequate tissue oxygenation, restore intravascular volume, and support organ function. Antibiotics are administered. SpO_2 is maintained between 88% and 92%, hemoglobin levels should be above 7 to 9 g/dL, and an SvO_2 greater than 70% is desired. Aggressive fluid therapy with isotonic crystalloid solutions is initiated early during systemic vasodilation to promote oxygen delivery to the tissues.

Support for each organ must be provided. Respiratory failure is managed with mechanical ventilation with low tidal volumes, high oxygen concentrations, and positive end-expiratory pressures (see Chapter 14). Adequate nutrition and metabolic support is provided with enteral feedings (see Chapter 6). Acute kidney injury is managed with continuous renal replacement therapies or hemodialysis (see Chapter 15). Inotropic drugs (low-dose dopamine or dobutamine) or vasopressors (norepinephrine or vasopressin) may be needed to maximize cardiac contractility and maintain cardiac output.

PATIENT OUTCOMES

The expected outcome for the patient in shock is that the patient will have improved tissue perfusion. Specific patient outcomes include alertness and orientation; normotension; warm, dry skin; adequate urine output; hemodynamic and laboratory values within normal limits; absence of infection; and intact skin. The patient should be resting quietly.

CASE STUDY

Mr. R., a 33-year-old man, was involved in a motor vehicle crash in which he sustained chest injuries. Mr. R., the driver, was not wearing his seat belt, and the steering wheel was bent. At the scene, Mr. R. was unresponsive. After placing a cervical collar to stabilize his neck, the paramedics performed endotracheal intubation and provided ventilation with 100% oxygen via a bag-valve device. Vital signs included a palpable systolic blood pressure (BP) of 60 mm Hg and a heart rate of 136 beats per minute. Mr. R.'s skin was pale, cold, and clammy with a delay in capillary refill. Peripheral pulses were weak and thready. Two 14-gauge peripheral intravenous catheters were inserted, and lactated Ringer's solution was infused at a wide open rate. He was transported to the emergency department on a backboard. The initial assessment in the emergency department noted that his palpable BP had increased to 90 mm Hg, and heart rate was 125 beats per minute. He was restless in response to pain, with no other purposeful responses. Pupils were equal and reactive to light. Chest expansion was unequal, and breath sounds were markedly diminished on the right side. A chest x-ray documented a 70% hemopneumothorax on the right side, and a 36-French chest tube was inserted at the eighth intercostal space at the right midaxillary line. Immediately, 2000 mL of blood was drained from the chest, and an additional 500 mL of drainage was recorded in the next 30 minutes. Initial laboratory results were:

Hemoglobin: 9 g/dL
Prothrombin time: 15 seconds
Hematocrit: 31%
Partial thromboplastin time: 47 seconds
Platelets: 274,000/microliter
Red blood cells: 2.9 million/microliter
White blood cells: 5300/microliter

An indwelling urinary catheter was inserted, and 80 mL of clear, yellow urine immediately drained. Fluid resuscitation was continued to maintain a systolic BP at 90 to 100 mm Hg. Mr. R. was taken immediately to the operating room, where a right thoracotomy was performed, with repair of the right axillary artery. In the operating room, his vital signs remained stable with continued fluid resuscitation of crystalloids, blood, and fresh frozen plasma.

After surgery, he was admitted to the critical care unit, where his BP was 116/70 mm Hg, heart rate was 90 beats per minute, and respiration rate was 24 breaths per minute on the ventilator (assist/control mode with a rate of 20 breaths per minute). He was responsive to commands and denied pain. He was medicated with morphine, 4 mg intravenous push every hour for pain. Laboratory results were:

Hemoglobin: 11 g/dL
Prothrombin time: 18.7 seconds
Hematocrit: 34%
Partial thromboplastin time: 71.7 seconds
Platelets: 180,000/microliter
Fibrinogen: 76 mg/dL
Red blood cells: 4.8 million/microliter
White blood cells: 5300/microliter

Arterial blood gases (on assisted ventilation with FiO_2 0.60):

pH: 7.30
$PaCO_2$: 40 mm Hg
PaO_2: 90 mm Hg
SaO_2: 92%
HCO_3^-: 17 mEq/L

Questions

1. What type of shock did Mr. R. demonstrate at the scene, and what components of his assessment supported this diagnosis?
2. Mr. R.'s initial assessment indicates that he is in which stage of shock?
3. In the emergency department, Mr. R. received lactated Ringer's solution for fluid resuscitation. Is this the appropriate solution at this time?
4. Explain Mr. R.'s arterial blood gas results. What treatment is indicated?
5. Describe the nursing care Mr. R. will receive in the first 24 hours after his surgery.
6. Describe the risk factors Mr. R. has for developing sepsis.

SUMMARY

The risk of shock is a common threat for all patients. Its causes are many, and treatment for shock is varied and complex. Complications of shock are related to the metabolic and tissue changes that result. If the normal compensatory mechanisms are not supported by effective therapeutic interventions, the pathological consequences perpetuate a vicious cycle of shock. The cycle is initiated by ischemia to the cells. Ischemia results in anaerobic metabolism, which leads to an accumulation of lactic acid and metabolic acidosis. This acidosis potentially leads to irreversible changes in the cells, organ failure, multiorgan failure, and death.

Prevention is the primary goal; it is accomplished through the identification of high-risk patient conditions and early interventions. Successful management relies on accurate nursing assessments, data analysis, implementation of definitive interventions, and evaluation of patient response to treatment. Shock is a crisis for the patient, family, nurse, and healthcare team. A multiprofessional approach of clinical expertise combined with caring assists the patient in reaching a positive outcome.

CRITICAL THINKING EXERCISES

1. Several people are admitted to the critical care unit, including (1) a 79-year-old man with a small anterior myocardial infarction and no prior cardiac history, (2) a 47-year-old man being given contrast media during a diagnostic procedure, (3) a 17-year-old adolescent with a cervical spine injury after a diving accident, and (4) a 72-year-old woman who was admitted with a bowel perforation caused by intestinal malignancy. Discuss what additional assessment information is needed to determine which of these patients has the potential to develop shock and the rationale for your decision.
2. A patient was admitted from the emergency department after a motorcycle crash in which he sustained blunt abdominal trauma. IV access was established in the internal jugular and left antecubital veins, and lactated Ringer's solution was infused. The results of initial computed tomography scan of the abdomen were negative. On admission to the critical care unit, you review the results of the hematological profile. Explain the rationale for the alterations in these values.
   a. Hemoglobin: 9.1 g/dL
   b. Hematocrit: 31.1%
   c. Platelets: 274,000/microliter
   d. Red blood cells: 2.9 million/microliter
   e. White blood cells: 9800/microliter
   f. Prothrombin time: 15 seconds
   g. Activated partial thromboplastin time: 38 seconds
3. Describe factors in the critically ill patient that increase susceptibility to the development of severe sepsis and septic shock. Describe how these can be prevented.
4. Differentiate between the early, hyperdynamic phase and the late, hypodynamic phase of septic shock.
5. Which type of shock is associated with the following hemodynamic changes?
   a. Bradycardia, decreased SVR, decreased SvO_2
   b. Increased RAP, PAP, PAOP, increased SVR, increased SvO_2
   c. Tachycardia, decreased SVR, increased SvO_2
   d. Decreased RAP, PAP, PAOP, increased SVR, decreased SvO_2
   e. Tachycardia, decreased SVR, decreased SvO_2

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