TABLE 11-3 Common Causes of Bradyasystolic Arrest
Myocardial ischemia or infarction
Sick sinus syndrome
Asphyxiation (including near-drowning)
Hypoxia
Hypercarbia
Stroke
Opiates, β-blockers, calcium channel blockers, adenosine, or parasympathetics

Primary bradyasystole occurs when the heart's electrical system fails to generate and/or propagate an adequate number of ventricular depolarizations per minute to sustain consciousness and other vital functions. Secondary bradyasystole is present when factors external to the heart's electrical system cause it to fail (e.g., hypoxia). Common causes of bradyasystolic arrest are listed in **Table 11-3.**

One of the most baffling mysteries of bradyasystolic cardiac arrest relates to myocardial mechanics. Bradyasystole, unlike ventricular fibrillation, is accompanied by very little myocardial oxygen consumption in animal models. Because of this, myocardial high-energy phosphate stores should decay relatively slowly during bradyasystole. This should theoretically result in a high incidence of return of spontaneous circulation after restoration of a more normal rhythm (e.g., with the early use of electrical pacing). It is unclear why conventional treatment of bradyasystolic cardiac arrest with atropine, epinephrine, or electrical pacemakers rarely results in survival to hospital discharge. Return of spontaneous circulation is infrequent, and long-term neurologically intact survival is rare in bradyasystolic cardiac arrest.

Other factors must play a determining role in the pathophysiology and subsequent outcome of bradyasystolic cardiac arrest. Bradyasystolic arrest is not just a disorder of rhythm generation or propagation; it is a perplexing syndrome characterized by such rhythm disturbances accompanied, in many cases, by profound depression of myocardial and vascular function. Suspected causes include endogenous myocardial depressants (including down-regulation of catecholamine receptors and/or toxic influences of intense sympathetic stimulation), neurogenic influences, postischemic myocardial stunning, and/or free radical injury.

It is important to differentiate pulseless electrical activity from conditions in which the rescuer is unable to detect a pulse, but there is unmistakable evidence that there is adequate blood pressure and cardiac output to maintain vital organ perfusion (e.g., a conscious patient with profound vasoconstriction caused by hypothermia, or "pseudo pulseless electrical activity").[43]

TABLE 11-4 Conditions That Cause Pulseless Electrical Activity
Hypovolemia
Tension pneumothorax
Pericardial tamponade
Pulmonary embolism
Massive myocardial dysfunction due to ischemia or infarction, myocarditis, cardiotoxins, etc.
Drug toxicity (e.g., β-blockers, calcium channel blockers, tricyclic antidepressants)
Profound shock
Hypoxia
Acidosis
Severe hypercarbia
Auto positive end-expiratory pressure
Hypothermia
Hyperkalemia
Pseudo pulseless electrical activity

The underlying physiologic cause of pulseless electrical activity is a marked reduction in cardiac output due to either profound myocardial depression or mechanical factors that reduce venous return or impede the flow of blood through the cardiovascular system. Common conditions that can cause pulseless electrical activity are shown in **Table 11-4**. The management of patients with pulseless electrical activity is directed at identifying and treating the underlying cause or causes.

REFERENCES

The complete reference list is available online at www.TintinalliEM.com.

CHAPTER 12

Approach to Shock

Bret A. Nicks
John Gaillard

EPIDEMIOLOGY

The exact number of cases of shock that present to the ED in the United States is difficult to ascertain due to the insensitivity of clinical parameters, current definitions, and lack of a central database repository. Previous estimates propose that more than 1 million cases of shock are seen in the ED each year in the United States.[1] These estimates are largely based on the assumption that hypotension, defined as a systolic blood pressure <90 mm Hg, is consistent with shock in adults. Using this definition, the incidence of patients with hypotension that present to American EDs is approximately 5.6 million cases per year.[2]

Mortality depends on the inciting event. Septic shock has an estimated mortality of 40% to 60%.[3] Cardiogenic shock has an estimated mortality of 36% to 56%.[4] Approximately 30% to 45% of patients with septic shock and 60% to 90% of patients with cardiogenic shock die within 1 month of presentation.[3,4] With a greater recognition and improved treatment, mortality from neurogenic shock has been reduced significantly. The definition of and treatment approach to shock continue to evolve, but the initial approach to a patient in shock follows similar principles, regardless of the inciting factors or cause.

Patients present to the ED in varying stages of critical illness and shock. These stages are confounded by age, comorbidities, and delays in presentation. A focus on early recognition, rapid diagnosis, and empiric resuscitation is essential. Therapy and patient stabilization may need to occur simultaneously with evaluation.

PATHOPHYSIOLOGY

Shock is a state of circulatory insufficiency that creates an imbalance between tissue oxygen supply (delivery) and oxygen demand (consumption) resulting in end-organ dysfunction. Reduction in effective perfusion may be due to a local or global delivery deficiency or utilization deficiency with suboptimal substrate at the cellular or subcellular level.[5] The mechanisms that can result in shock are frequently divided into four categories: (1) hypovolemic, (2) cardiogenic, (3) distributive, and (4) obstructive.

FACTORS AFFECTING CARDIAC OUTPUT

An understanding of the mechanisms of oxygen delivery and consumption is foundational to the treatment of shock. While the physiology is complex, familiarity with the basic principles, equations, and their interactions is essential (**Tables 12-1 and 12-2**). As noted in the cardiac output (CO) equation, CO is determined by heart rate and stroke volume. Stroke volume is dependent on preload, afterload, and contractility. The mean arterial pressure (MAP) demonstrates the impact that CO has on MAP (which can also be estimated with the formula: 2 × diastolic blood pressure + systolic blood pressure/3). This is important because there is a MAP threshold below which oxygen delivery is decreased. Systemic

TABLE 12-1 Physiologic Equations

Parameter	Equation Specifics
Cardiac output	Cardiac Output = Heart Rate × Stroke Volume CO = HR × SV
Mean arterial pressure	Mean Arterial Pressure = Cardiac Output × Systemic Vascular Resistance MAP = CO × SVR
Oxygen delivery	Oxygen Delivery = Cardiac Output × Arterial Oxygen Content $Do_2 = CO \times [(1.39 \times Hb \times Sao_2) + (Pao_2 \times 0.0031)]$ Do_2 is the amount of O_2 delivered to the tissues per minute. A normal value is 1000 mL O_2 per minute.
Arterial oxygen content	Arterial Oxygen Content = Amount of Oxygen in the Blood $Cao_2 = (1.39 \times Hb \times Sao_2) + (Pao_2 \times 0.0031)$
Oxygen consumption	Oxygen Consumption = Cardiac Output × (Arterial O_2 Content – Venous O_2 Content) $\dot{V}o_2 = CO \times (Cao_2 - Cvo_2)$ Alternative equation: $\dot{V}o_2 = CO \times Hb \times 1.39 \times (Sao_2 - Smvo_2)$ The amount of O_2 consumed by tissues each minute is equal to the difference in O_2 delivered to tissues and the O_2 returning from tissues to the heart. A normal value is about 250 mL O_2 per minute. Note that this formula ignores the small contribution from dissolved oxygen.
Shock index	Shock Index = Heart Rate ÷ Systolic Blood Pressure SI = HR/SBP A normal value is 0.5–0.7. A persistent elevation of the shock index (>1.0) indicates an impaired left ventricular function (as a result of blood loss or cardiac depression) and carries a high mortality rate.

Abbreviations and units: Hb = hemoglobin in grams/dL; HR = heart rate per min; Pao_2 = partial pressure of oxygen in arterial blood in mm Hg; Sao_2 = oxygen saturation expressed as a fraction of 1.0, rather than a percentage; SBP = systolic blood pressure in mm Hg; $Smvo_2$ = mixed venous oxygen saturation expressed as a fraction of 1.0, rather than a percentage; SV = stroke volume in mL; SVR = systemic vascular resistance in mm Hg·min/L.

TABLE 12-2 Abbreviations and Definitions of Hemodynamic Parameters

Cao_2	Arterial oxygen content
CI	Cardiac index (cardiac output/body surface area)
CO	Cardiac output
Cvo_2	Venous oxygen content
CVP	Central venous pressure
Do_2	Systemic oxygen delivery
DBP	Diastolic blood pressure
Hb	Hemoglobin
MAP	Mean arterial pressure
MODS	Multiorgan dysfunction syndrome
$Paco_2$	Partial pressure of arterial carbon dioxide
Pao_2	Partial pressure of arterial oxygen
Sao_2	Arterial oxygen saturation
$Scvo_2$	Central venous oxygen saturation from the superior vena cava
$Smvo_2$ (Svo_2)	Mixed venous oxygen saturation from the pulmonary artery
SBP	Systolic blood pressure
SI	Shock index
SIRS	Systemic inflammatory response syndrome
SVR	Systemic vascular resistance
$\dot{V}o_2$	Systemic oxygen consumption

vascular resistance (SVR) directly impacts MAP, but also impacts afterload and thus CO. The physiologic mechanism of oxygen delivery to peripheral tissues (Do_2) is described in the oxygen delivery equation. Recognize that blood pressure is not represented in this equation. Patients in shock may initially have normal blood pressures (cryptic shock), yet have other objective signs of shock (see "Clinical Features" below). It is from these basic equations that the concept of preload influencing stroke volume, which itself influences CO and Do_2, has become fundamental in shock management.

Tissue oxygenation is predicated on CO being sufficient enough to deliver oxygenated hemoglobin to the tissues. CO is dependent on the interplay of cardiac inotropy (speed and shortening capacity of myocardium), chronotropy (heart contraction rate), and lusitropy (ability to relax and fill heart chambers). Determinants of inotropy include autonomic input from sympathetic activation, parasympathetic inhibition, circulating catecholamines, and short-lived responses to an increase in afterload (Anrep effect) or heart rate (Bowditch effect). Increases in the inotropic state help to maintain stroke volume at high heart rates.[6] Under certain conditions, such as shock states, higher levels of epinephrine will be produced and reinforce adrenergic tone. Epinephrine levels are significantly elevated during induced hemorrhagic shock, but these levels subsequently reduce to almost normal levels after adequate blood pressure is restored.[7] Previous studies have also shown that an acidotic milieu, which is common in shock, further compromises ventricular contractile force and blood pressure.[8] Chronotropy and lusitropy are both influenced by sympathetic input. Norepinephrine interacts with cardiac β_1-receptors, resulting in increased cyclic adenosine monophosphate. This leads to a process of intracellular signaling with an increased chronotropy and sequestration of calcium, leading to myocardial relaxation.[6]

LACTIC ACID

When compensatory mechanisms fail to correct the imbalance between tissue supply and demand, anaerobic metabolism occurs and results in the formation of lactic acid. Lactic acid is rapidly buffered, resulting in the formation of measured serum lactate. Normal venous lactate levels are less than 2.0 mmol/L. Most cases of lactic acidosis are a result of inadequate oxygen delivery, but lactic acidosis occasionally can develop from an excessively high oxygen demand (e.g., status epilepticus). In other cases, lactic acidosis occurs because of impaired tissue oxygen utilization (e.g., septic shock or the postresuscitation phase of cardiac arrest). Elevated lactate is a marker of impaired oxygen delivery or utilization and correlates with short-term prognosis of critically ill patients in the ED.[7]

COMPENSATORY MECHANISMS AND THEIR FAILURE

Shock provokes a myriad of autonomic responses, many of which serve to maintain perfusion pressure to vital organs. Stimulation of the carotid baroreceptor stretch reflex activates the sympathetic nervous system triggering (1) arteriolar vasoconstriction, resulting in redistribution of blood flow from the skin, skeletal muscle, kidneys, and splanchnic viscera; (2) an increase in heart rate and contractility that increases CO; (3) constriction of venous capacitance vessels, which augments venous return; (4) release of the vasoactive hormones epinephrine, norepinephrine, dopamine, and cortisol to increase arteriolar and venous tone; and (5) release of antidiuretic hormone and activation of the renin-angiotensin axis to enhance water and sodium conservation to maintain intravascular volume.[8]

These compensatory mechanisms attempt to maintain Do_2 to the most critical organs (heart and brain), but blood flow to other organs, such as the kidneys and GI tract, may be compromised. The cellular response to decreased Do_2 (adenosine triphosphate depletion) leads to ion-pump dysfunction, influx of sodium, efflux of potassium, and reduction in membrane resting potential. As shock progresses, the loss of cellular integrity and the breakdown in cellular homeostasis result in cellular death. These pathologic events give rise to a cascade of metabolic features including hyperkalemia, hyponatremia, azotemia, hyper- or hypoglycemia, and lactic acidosis.

TABLE 12-3 Clinical Features of Systemic Inflammatory Response Syndrome (SIRS)

Two or more of the following features are required to make a diagnosis of SIRS:
Temperature >38°C (100.4°F) or <36°C (96.8°F)
Heart rate >90 beats/min
Respiratory rate >20 breaths/min (or carbon dioxide tension <32 mm Hg)
WBC count >12.0 × 10^9/L, <4.0 × 10^9/L, or >10% immature forms or bands

In the early phases of shock, these physiologic changes may produce a clinical syndrome called the systemic inflammatory response syndrome or SIRS (**Table 12-3**).

As systemic inflammatory response syndrome progresses, shock ensues, followed by the multiorgan dysfunction syndrome, which is manifested by renal failure, respiratory failure, myocardial depression, liver failure, and then disseminated intravascular coagulation. The fulminant progression from systemic inflammatory response syndrome to multiorgan dysfunction syndrome is determined by the balance of anti-inflammatory and proinflammatory mediators and the level of inadequate tissue perfusion (**Figure 12-1**).

CATEGORIES OF SHOCK

The four categories of shock can be described in terms of their respective physiologic changes and common causes, recognizing that overlap is common (**Table 12-4**). **Hypovolemic shock** occurs when decreased intravascular fluid or decreased blood volume causes decreased preload, stroke volume, and CO. Severe blood loss (hemorrhage) can cause decreased myocardial oxygenation, which decreases contractility and CO. This action may lead to an autonomic increase in the SVR.

TABLE 12-4 Categories of Shock

Type	Hemodynamic Changes	Etiologies
Hypovolemic	Decreased preload, increased SVR, decreased CO	Hemorrhage, capillary leak, GI losses, burns
Cardiogenic	Increased preload, increased afterload, increased SVR, decreased CO	MI, dysrhythmias, heart failure, valvular disease
Obstructive	Decreased preload, increased SVR, decreased CO	PE, pericardial tamponade, tension PTX
Distributive	Decreased preload, increased SVR, mixed CO	Sepsis, neurogenic shock, anaphylaxis

Abbreviations: CO = cardiac output; MI = myocardial infarction; PE = pulmonary embolism; PTX = pneumothorax; SVR = systemic vascular resistance.

Hypovolemic shock can also occur due to volume loss from other etiologies. In **cardiogenic shock**, the left ventricle fails to deliver oxygenated blood to peripheral tissues due to variances in contractility, as well as preload and afterload. Myocardial infarction is the most common cause of cardiogenic shock. Dysrhythmias are another common cause because they can lead to a decreased CO. Bradyarrhythmias result in low CO, and tachyarrhythmias can result in decreased preload and stroke volume. **Obstructive shock** is due to a decrease in venous return or cardiac compliance due to an increased left ventricular outflow obstruction or marked preload decrease. Cardiac tamponade and tension pneumothorax are common causes. In **distributive shock**, there is relative intravascular volume depletion due to marked systemic vasodilatation. This is most commonly seen in septic shock. Compensatory responses to decreased SVR may include increased CO (increased contractility and heart rate) and tachycardia. The concurrent decreased SVR results in a decreased preload and may hinder CO

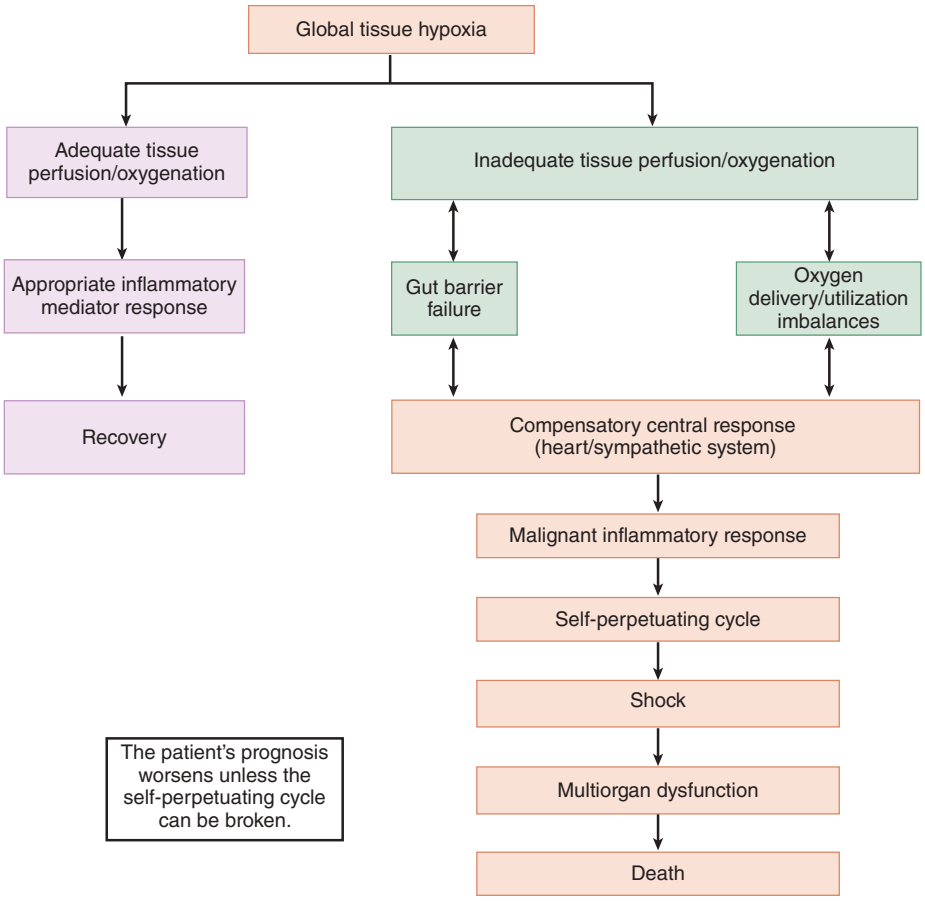

FIGURE 12-1. The pathophysiology of shock, the systemic inflammatory response syndrome, and multiorgan dysfunction.

overall. In sepsis, up to 40% of patients may have a transient cardiomyopathy characterized by decreased contractility and increased mortality.[3,4] Anaphylaxis, adrenal insufficiency and neurogenic shock are additional causes of distributive shock.

CLINICAL FEATURES

HISTORY AND COMORBIDITIES

While the clinical presentation of a patient in shock and the underlying cause may be quite apparent (e.g., acute myocardial infarction, anaphylaxis, or hemorrhage), it may be difficult to obtain a history from patients in shock. Assistance with medical history from EMS, family, or other sources may help determine the cause of shock, especially if the patient has comorbidities. Some patients in shock may have few symptoms other than generalized weakness, lethargy, or altered mental status. If the patient is unresponsive, consider trauma as a primary or secondary complication.

PHYSICAL EXAMINATION

Shock is usually associated with systemic arterial hypotension—systolic blood pressure <90 mm Hg. Blood pressure is the product of flow and resistance (MAP = CO × SVR). Blood pressure may not drop if there is an increase in peripheral vascular resistance in the presence of decreased CO with inadequate tissue hypoperfusion, making blood pressure an insensitive marker for global tissue hypoperfusion. **Shock may occur with a normal blood pressure, and hypotension may occur without shock.** No single vital sign is diagnostic of shock, and blood pressure is particularly insensitive in the presence of peripheral vascular disease, tachycardia with a small pulse pressure, or cardiac dysrhythmias. Composite physical findings are useful in the assessment of shock (**Table 12-5**).

TABLE 12-5 Composite Physical Examination Findings in Shock

Temperature	Hyperthermia or hypothermia may be present. Endogenous hypothermia (hypometabolic shock) must be distinguished from exogenous environmental hypothermia.
Heart rate	Usually elevated; however, paradoxical bradycardia can be seen in shock states due to hypoglycemia, β-blocker use, and preexisting cardiac disease.
Systolic blood pressure	May actually increase slightly when cardiac contractility increases in early shock and then fall as shock advances.
Diastolic blood pressure	Correlates with arteriolar vasoconstriction and may rise early in shock and then fall when cardiovascular compensation fails.
Pulse pressure	Increases early in shock and decreases before systolic pressure begins to drop.
Mean arterial blood pressure	Often low, <65 mm Hg.
CNS	Acute delirium or brain failure, restlessness, disorientation, confusion, and coma secondary to a decrease in cerebral perfusion pressure
Skin/capillary refill	Pallor, pale, dusky, clammy, cyanosis, sweating, altered temperature, and increased capillary refill time of >2–3 s.
Cardiovascular	Neck vein distention or flattening depending on the type of shock. Tachycardia and arrhythmias. An S_3 may result from high-output states. Decreased coronary perfusion pressures can lead to ischemia, decreased ventricular compliance, increased left ventricular diastolic pressure, and pulmonary edema.
Respiratory	Tachypnea, increased minute ventilation, increased dead space, bronchospasm, and hyper- or hypocapnia with progression to respiratory failure.
Splanchnic organs	Ileus, GI bleeding, pancreatitis, acalculous cholecystitis, and mesenteric ischemia can occur due to low flow states.
Renal	Reduced glomerular filtration rate. Renal blood flow redistributes from the renal cortex toward the renal medulla leading to oliguria.
Metabolic	Hyperglycemia, hypoglycemia, and hyperkalemia. As shock progresses, metabolic acidosis occurs with concurrent attempted respiratory compensation.

DIAGNOSIS

LABORATORY EVALUATION

No single laboratory value is sensitive or specific for shock. Laboratory studies are driven by the clinical presentation and the presumptive cause. Common studies are listed in **Table 12-6**. Arterial blood gases are useful to assess acid–base status and ventilation and oxygenation concerns, whereas a venous blood gas is limited to acid–base information. A rise in serum lactate correlates with mortality in many shock states;[7] typically this is due to anaerobic metabolism, but nonhypoxic causes of lactic acidosis due to cellular dysfunction occur in shock states. Serial lactate assessments may be indicated because lactate clearance is associated with improved outcomes in septic shock and may assist with resuscitation.[9] A wide range of laboratory abnormalities may be encountered in shock, but most abnormal values merely point to the particular organ system that is contributing to, or being affected by, the shock state.

TABLE 12-6 Initial Diagnostic Studies to Evaluate a Patient in Shock*

CBC with differential
Electrolytes, glucose, calcium, magnesium, phosphorus
BUN, creatinine
Serum lactate
ECG
Urinalysis
Chest radiograph
Coagulation studies: prothrombin time, PTT, INR
Arterial blood gas (measured pH, carbon dioxide, and oxygen levels)
Hepatic function panel
Cultures: blood, urine, suspicious wounds, quantitative sputum culture
Culture
Cortisol level
Pregnancy test
CT of chest/abdomen/pelvis as indicated history, physical exam

*Ordering of the listed tests should be individualized by patient presentation and history.

IMAGING

Chest X-Ray The portable anteroposterior view chest x-ray is often used in the evaluation of unstable patients to avoid transporting the patient during resuscitation. While limitations exist, evaluation of the heart size, presence of pulmonary edema, free air under the diaphragm, pneumothorax, infiltrates, or effusions may provide useful clinical information.

US Bedside US assessment is an important tool for developing a differential diagnosis, assessing volume status, defining cardiac function, and assisting with procedures. Various US methods are described to determine overall volume status by assessing right-sided filling pressures, including measuring inferior vena cava respiratory variation, end-expiratory vena cava respiratory variation, and other methods. Defining the degree of hypovolemia is inexact.

Bedside cardiac US to assess left ventricular ejection fraction can assist with determining the cause of shock. Emergency physicians trained in focused bedside cardiac US can provide an estimated ejection fraction with high relative correlation to cardiologists.[10]

US may also be used to assess for vascular emergencies. Identifying an abdominal aortic aneurysm on US may lead to further evaluation.

Additional US protocols, such as the Abdominal and Cardiac Evaluation with Sonography in Shock protocol and the Rapid Ultrasound in Shock protocol, have been formulated using many of the aforementioned concepts. The Abdominal and Cardiac Evaluation with Sonography in Shock protocol looks at cardiac function, inferior vena cava dynamics, pulmonary congestion, sliding and consolidation, abdominal free fluid, abdominal aortic aneurysm, and leg venous thrombosis to assist in differential diagnosis generation or narrowing.[11] The Rapid Ultrasound in Shock exam involves a three-part bedside physiologic assessment simplified as the pump (cardiac), the tank (volume status), and the pipes (arterial and venous).[12,13] However, as with any US intervention, operator competency and available resources are essential.

CT Although CT is an accurate and noninvasive approach for detecting internal pathology, patients must travel from the ED to the radiology suite, which may be unadvisable in unstable shock. The potential benefits of CT must be weighed against the associated risks, including concerns about renal function due to hypovolemia and contrast-induced nephropathy. CT scans without IV contrast will add some information to the clinical picture, although not to the degree of a scan with IV contrast.

HEMODYNAMIC MONITORING

Hemodynamic monitoring helps assess the severity of shock and the response to treatment. Monitoring capabilities should initially include pulse oximetry, electrocardiographic monitoring, and noninvasive blood pressure monitoring. In the critical care arena, intra-arterial blood pressure monitoring, end-tidal carbon dioxide monitoring, central venous pressure, and central venous oxygen saturation from the superior vena cava ($Scvo_2$) monitoring are frequently used. When obtaining central access, the average access time, number of attempts, and mechanical complications are reduced when a US-assisted approach is used.[14]

EARLY TREATMENT

Comprehensive and timely ED care can significantly decrease the predicted mortality of critically ill patients in as little as 6 hours of treatment.[15] Application of an algorithmic approach to optimize hemodynamic end points with early goal-directed therapy in the ED reduced mortality by 16% in patients with severe sepsis or septic shock in 2001.[16] That original study, the Surviving Sepsis Campaign that followed,[17] and other algorithmic efforts[18] have changed the approach to sepsis and shock care on a worldwide basis. Two large, multicenter, randomized controlled trials published in 2014 failed to show additional benefits to a rigid algorithmic approach.[19,20] However, we attempt to present below the most beneficial aspects of shock care demonstrated by the medical progress of the last 15 years. **The *ABCDE* tenets of shock resuscitation are establishing *a*irway, controlling the work of *b*reathing, optimizing the *c*irculation, assuring adequate oxygen *d*elivery, and achieving *e*nd points of resuscitation.[21]**

ESTABLISHING THE AIRWAY

Airway control is best obtained through endotracheal intubation. Sedatives used to facilitate intubation may cause arterial vasodilatation, venodilation, or myocardial suppression and may result in hypotension. Positive-pressure ventilation reduces preload and CO. The combination of sedative agents and positive-pressure ventilation will often lead to hemodynamic collapse. To avoid this unwanted situation, initiate volume resuscitation and vasoactive agents before intubation and positive-pressure ventilation.

CONTROLLING THE WORK OF BREATHING

Control of breathing is required when significant tachypnea accompanies shock. Respiratory muscles are significant consumers of oxygen during shock and contribute to lactate production. Mechanical ventilation and sedation allow for adequate oxygenation, improvement of hypercapnia, and assisted, controlled, synchronized ventilation. All of these treatments decrease the work of breathing and improve survival. When starting mechanical ventilation on a patient, it is essential to consider the patient's compensatory minute ventilation prior to intubation to ensure appropriate initial settings are selected. After a patient is placed on mechanical ventilation, obtain an arterial blood gas to evaluate acid–base status, oxygenation, and ventilation. Neuromuscular blocking agents should be considered to further decrease respiratory muscle oxygen consumption and preserve Do_2 to vital organs, especially if patients are severely hypoxemic due to acute respiratory distress syndrome.[22]

OPTIMIZING THE CIRCULATION

Fluids Circulatory or hemodynamic stabilization begins with intravascular access through large-bore peripheral venous lines. The Trendelenburg position does not improve cardiopulmonary performance compared with the supine position. It may worsen pulmonary gas exchange and predispose to aspiration. Passive leg raising above the level of the heart with the patient supine may be effective. If passive leg raising results in an increase in blood pressure or CO, fluid resuscitation is indicated.[23]

Fluid resuscitation should begin with isotonic crystalloid.[24] The amount and rate of infusion are determined by an estimate of the hemodynamic abnormality. Most patients in shock have either an absolute or relative volume deficit. The exception is the patient in cardiogenic shock with pulmonary edema. Administer fluid rapidly (over 5 to 20 minutes), in set quantities of 500 or 1000 mL of normal saline, and reassess the patient after each bolus. Patients with a modest degree of hypovolemia usually require an initial 20 to 30 mL/kg of isotonic crystalloid, as is suggested in the 2012 Surviving Sepsis Campaign Guidelines; however, there are few data to support this uniform recommendation, and fluid volume should be individualized to each patient.[25] More fluids are needed for profound volume deficits. It is common for patients in septic shock to receive 6 L of crystalloid in the first 24 hours of hospital care. For large fluid volumes, consider using lactated ringer's or plasmalyte to avoid hyperchloremic metabolic acidosis.[26,27]

Central venous access may aid in assessing volume status (preload) and monitoring $Scvo_2$. It is also the preferred route for the long-term administration of certain vasopressor therapy. However, there is no need for universal central access in patients with septic shock, and the need for central access should be individually determined.[20]

Vasopressors Vasopressors are used when there has been an inadequate response to volume resuscitation or if there are contraindications to volume infusion.[25] Vasopressors are most effective when the vascular space is "full" and least effective when the vascular space is depleted. Patients with chronic hypertension may be at greater risk of renal injury at lower blood pressures; however, in others, there appears to be no mortality benefit in raising MAP above the 65 to 70 mm Hg range.[28,29]

Vasopressor agents have variable effects on the α-adrenergic, β-adrenergic, vasopressin, and dopaminergic receptors (**Table 12-7**). Although vasopressors improve perfusion pressure in the large vessels, they may decrease capillary blood flow in certain tissue beds, especially the GI tract and peripheral vasculature. If multiple vasopressors are used, they should be simplified as soon as the best therapeutic agent is identified. In addition to a vasopressor, an inotrope may be needed to directly increase CO by increasing contractility and stroke volume.

ASSURING ADEQUATE OXYGEN DELIVERY

Control of oxygen consumption ($\dot{V}o_2$) is important in restoring the balance of oxygen supply and demand to the tissue (oxygen consumption equation). A hyperadrenergic state results from the compensatory response to shock, physiologic stress, pain, and anxiety. Shivering frequently results when a patient is unclothed for examination and then left inadequately covered in a cold resuscitation room. The combination of these variables increases $\dot{V}o_2$. Pain further suppresses myocardial function, further impairing Do_2 and $\dot{V}o_2$. Providing analgesia,

TABLE 12-7 Commonly Used Vasoactive Agents (all vasopressors increase myocardial oxygen demand; most should be titrated to desired effect)

Drug	Dose	Action	Cardiac Contractility	Vasoconstriction	Vasodilation	Cardiac Output
Dobutamine	2.0–20.0 micrograms/kg/min	β_1, some β_2 and α_1 in large dosages	++++	+	++	Increases
Side effects and comments	Inotrope only; Causes tachydysrhythmias, occasional GI distress, hypotension in volume-depleted patients; has less peripheral vasoconstriction than dopamine; can cause fewer arrhythmias than isoproterenol					
Dopamine	0.5–20 micrograms/kg/min	α, β, and dopaminergic	++ at 2.5–5 micrograms/kg/min	++ at 5–20 micrograms/kg/min	+ at 0.5–2.0 micrograms/kg/min	Usually increases
Side effects and comments	Tachydysrhythmias; a cerebral, mesenteric, coronary, and renal vasodilator at low doses; Surviving Sepsis Campaign second line, lot of overlap with α/β/dopaminergic receptors and dose; can be given through a peripheral IV					
Epinephrine	2–10 micrograms/min	α and β	++++ at 0.5–8 micrograms/kg/min	++++ at >8 micrograms/kg/min	+++	Increases
Side effects and comments	Causes tachydysrhythmia, leukocytosis; increases myocardial oxygen consumption; may increase lactate; no real maximum dose					
Isoproterenol	0.01–0.05 micrograms/kg/min	β_1 and some β_2	++++	0	++++	Increases
Side effects and comments	Inotrope; causes tachydysrhythmia, facial flushing, hypotension in hypovolemic patients; increases myocardial oxygen consumption; never use alone in shock					
Norepinephrine	0.5–50 micrograms/min	Primarily α_1, some β_1	++	++++	0	Slightly increases
Side effects and comments	Useful when loss of venous tone predominates; first-line agent for most situations; should be given through a central line					
Phenylephrine	10–200 micrograms/min	Pure α	0	++++	0	Decreases
Side effects and comments	Reflex bradycardia, headache, restlessness, excitability, rarely arrhythmias; can be used on patients in shock with tachycardia or supraventricular arrhythmias; not good comparatively for septic shock					
Vasopressin	0.01–0.04 units/min	Directly stimulates V_1 receptor on smooth muscle	0	++++	0	0
Side effects and comments	Primarily vasoconstriction; usually started at max dose and not titrated					

Note: 0 = no effect; + = mild effect; ++ = moderate effect; +++ = marked effect; ++++ = very marked effect.

muscle relaxation, warm covering, anxiolytics, and even paralytic agents, when appropriate, decreases this inappropriate systemic oxygen consumption.

Once blood pressure is stabilized through optimization of preload and afterload, Do_2 can be assessed and further manipulated. Restore arterial oxygen saturation to ≥91%. In shock states, consider a transfusion of packed red blood cells to maintain hemoglobin ≥7 to 9 grams/dL.[25] If CO can be assessed, it should be increased using volume infusion or inotropic agents in incremental amounts until venous oxygen saturation (mixed venous oxygen saturation [$Smvo_2$] or $Scvo_2$) and lactate are normalized.

Sequential examination of lactate and $Smvo_2$ or $Scvo_2$ is a method to assess adequacy of a patient's resuscitation. Continuous measurement of $Smvo_2$ or $Scvo_2$ can be used in the ED, although recent literature questions the need for this in resuscitation management.[20] A variety of technologic tools may be used to assess tissue perfusion during resuscitation.[30–35] These technologies may be available in some EDs, but it is essentially standard of care in intensive care units. Transfer of the patient to the intensive care unit should not be delayed so that monitoring devices can be placed in the ED.[15,36]

END POINTS OF RESUSCITATION

The goal of resuscitation is to use hemodynamic and physiologic values to guide therapy in order to maximize survival and minimize morbidity. No therapeutic end point is universally effective, and only a few have been tested in prospective trials, with mixed results.[20,28,29] Hypotension at ED presentation is associated with poor outcomes.[37] Noninvasive parameters, such as blood pressure, heart rate, and urine output, may underestimate the degree of remaining hypoperfusion and oxygen debt, so the use of additional physiologic end points may be informative.[20,28,29,37] A goal-directed approach of MAP >65 mm Hg, central venous pressure of 8 to 12 mm Hg, $Scvo_2$ >70%, and urine output >0.5 mL/kg/h during ED resuscitation of septic shock has been shown to decrease mortality, but which of the metrics accounts for the mortality decrease remains in question.[16,19,20,25] Source control, whether with infection, hemorrhage, or other state of shock, is essential in the initial stages of management. If shock or hypotension persists, reassessment at the patient's bedside is essential while considering the important issues in **Table 12-8**.

CONTROVERSIES OF TREATMENT

FLUID THERAPY

Rapid restoration of fluid deficits modulates inflammation and, if the condition progresses to shock, decreases the need for subsequent vasopressor therapy, steroid administration, and invasive monitoring (e.g., pulmonary artery catheterization and arterial line placement).[25,38] Although there is general agreement that volume therapy is an integral component of early resuscitation, there is a lack of consensus for the type of fluid, standards of volume assessment, and end points. **Table 12-9** compares the most commonly used fluid therapies.[39-43]

Colloids are high-molecular-weight solutions that increase plasma oncotic pressure. Colloids can be classified as either natural (albumin) or artificial (starches, dextrans, and gelatins). Due to their higher molecular weight, colloids stay in the intravascular space significantly longer than crystalloids. The intravascular half-life of albumin is 16 hours versus 30 to 60 minutes for normal saline and lactated

TABLE 12-8 Questions to Answer if There Is Persistent Shock or Hypotension

Equipment and monitoring
Is the patient appropriately monitored?
Is there an equipment malfunction, such as dampening of the arterial line or disconnection from the transducer?
Is the IV tubing into which the vasopressors are running connected appropriately?
Are the vasopressor infusion pumps working?
Are the vasopressors mixed adequately and in the correct dose?
Patient assessment
Do mentation and clinical appearance match the degree of hypotension?
Is the patient adequately volume resuscitated?
Does the patient have a pneumothorax after placement of central venous access?
Has the patient been adequately assessed for an occult penetrating injury (a bullet hole or stab wound)?
Is there hidden bleeding from a ruptured spleen, large-vessel aneurysm, or ectopic pregnancy?
Does the patient have adrenal insufficiency? The incidence of adrenal dysfunction can be as high as 30% in this subset of patients.
Is the patient allergic to the medication just given or taken before arrival?
Is there cardiac tamponade in the dialysis patient or cancer patient?
Is there associated acute myocardial infarction, aortic dissection, or pulmonary embolus?

ringer's solution.[24,39-41] Resuscitation with **crystalloids** requires two to four times more volume than colloids.[24,38,39] The outcome advantage between crystalloid and colloids continues to remain unresolved in sepsis, despite multiple studies.[24,38-41] Due to the equivalency and the higher cost of colloids, crystalloids would seem to be a better choice for resuscitation in the ED.

BICARBONATE USE IN SHOCK

Bicarbonate administration shifts the oxygen-hemoglobin dissociation curve to the left, impairs tissue unloading of hemoglobin-bound oxygen, and may worsen intracellular acidosis. However, many clinicians remain uncomfortable withholding bicarbonate if the pH is <7.00. Animal studies of profound acidosis demonstrate decreased ventricular contractility and systolic blood pressure.[7] In settings of a low pH and when evidence of decreased contractility (despite ongoing resuscitative efforts) or development of a dysrhythmia, partially correct the metabolic acidosis, either with sodium bicarbonate boluses or a drip. Recognize the risk of paradoxical intracerebral intracellular acidosis in the process. Consider situations, such as end-stage renal disease and renal tubular acidosis, that cannot reclaim bicarbonate through normal renal processes and whether bicarbonate may be indicated.

TABLE 12-9 Fluid Therapy

Crystalloids	
Normal saline (NS)	Slightly hyperosmolar containing 154 mEq/L of both sodium and chloride. Risk of inducing hyperchloremic metabolic acidosis when given in large amounts due to relatively high chloride concentration.
Lactated ringer's (LR)	Lactate can accept a proton and subsequently be metabolized to carbon dioxide and water by the liver, leading to release of carbon dioxide in the lungs and excretion of water by the kidneys. LR results in a buffering of the acidemia that is advantageous over NS. Theoretical risk of inducing hyperkalemia in patients with renal insufficiency or renal failure due to small potassium content (very small amount).
Colloids	
Albumin	Derived from human plasma. Available in varying strengths from 4% to 25%. Multiple studies have shown that there is no outcome difference whether colloids or crystalloids are used. Colloids cost significantly more than crystalloids. One study actually showed an increase in mortality in trauma patients complicated by head injury.
Hydroxyethyl starch	Synthetic colloid derived from hydrolyzed amylopectin. Hydroxyethyl starch should be avoided in sepsis. Many harmful effects: renal impairment at recommended doses and impairing long-term survival at high doses, coagulopathy and bleeding complications from reduced factor VIII and von Willebrand factor levels, impaired platelet function.

DISPOSITION AND TRANSITION TO THE INTENSIVE CARE UNIT

Early recognition, treatment, and subsequent transfer of critically ill patients to the intensive care unit improves patient outcomes and improves ED throughput.[15,36] Communicate and document all ED resuscitative efforts to the critical care team. Even when resuscitation is systematic and thoughtful, miscommunication can undo the benefits of initial ED treatment. Ideally, before transfer, verbally communicate and document a system-oriented problem list with an assessment and plan, including all procedures and complications. For prolonged or "boarded" ED stays, constantly reassess the critically ill patient and ensure that care plans are continuing. Often, this will entail ordering tests that are not commonly performed on ED patients or ordering subsequent doses of medicines, particularly antibiotics.

PROGNOSIS

Some clinical variables are associated with poor outcome, such as severity of shock, temporal duration, underlying cause, preexisting vital organ dysfunction, and reversibility. Early recognition, intervention, source control, and smooth transitions of care help ensure the most ideal outcomes. While associated morbidity and mortality remain high for patients with shock, integration of protocol-based care pathways, with ongoing refinement in response to new information, may lead to continued reductions over time.[15,20,25,36] Additional outcome predictions related to physiologic scoring systems, ED-based shock interventions, and the balance between invasive and noninvasive or minimally invasive strategies are still being studied.[15,44,45]

REFERENCES

The complete reference list is available online at www.TintinalliEM.com.

CHAPTER 13

Fluid and Blood Resuscitation in Traumatic Shock

David M. Somand
Kevin R. Ward

INTRODUCTION

Circulatory shock has a high mortality. Severe hemorrhage after injury carries a mortality rate of 30% to 40% and is responsible for almost 50% of deaths occurring within 24 hours of injury.[1,2] Septic shock has a mortality of up to 50%.[3] Resuscitation, starting in the prehospital setting and continuing throughout the victim's care in the ED and on into the hospital, has the goal of restoring the necessary level of tissue perfusion and oxygenation for survival while simultaneously limiting further volume loss.

Intravascular volume depletion is a common feature of circulatory shock; crystalloids, colloids, and blood products (packed red blood cells