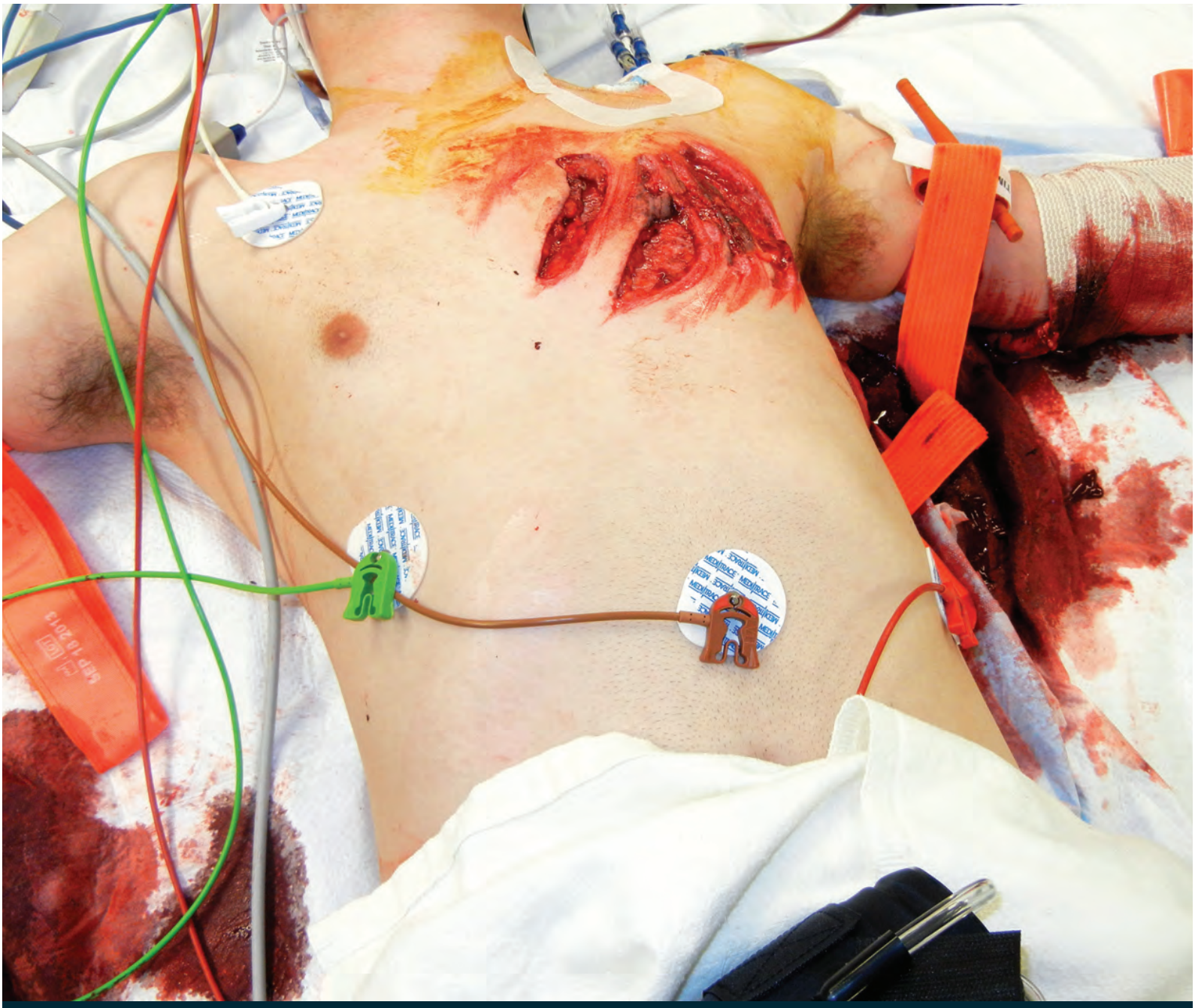


3 SHOCK

The first step in the initial management of shock is to recognize its presence.

CHAPTER 3 OUTLINE

OBJECTIVES

After reading this chapter and comprehending the knowledge components of the ATLS provider course, you will be able to:

1. Define shock.
2. Describe the likely causes of shock in trauma patients.
3. Describe the clinical signs of shock and relate them to the degree of blood loss.
4. Explain the importance of rapidly identifying and controlling the source of hemorrhage in trauma patients.
5. Describe the proper initial management of hemorrhagic shock in trauma patients.
6. Describe the rationale for ongoing evaluation of fluid resuscitation, organ perfusion, and tissue oxygenation in trauma patients.
7. Explain the role of blood replacement in managing shock.
8. Describe special considerations in diagnosing and treating shock related to advanced age, athleticism, pregnancy, medications, hypothermia, and presence of pacemakers and implantable cardioverter-defibrillators.

The first step in managing shock in trauma patients is to recognize its presence. Once shock is identified, initiate treatment based on the probable cause. The definition of shock—an abnormality of the circulatory system that results in inadequate organ perfusion and tissue oxygenation—also guides the trauma team in the diagnosis and treatment. Diagnosing shock in a trauma patient relies on a synthesis of clinical findings and laboratory tests. No single vital sign and no laboratory test, on its own, can definitively diagnose shock. Trauma team members must quickly recognize inadequate tissue perfusion by recognizing the clinical findings that commonly occur in trauma patients.

The second step in managing shock is to identify the probable cause of shock and adjust treatment accordingly. In trauma patients, this process is related to the mechanism of injury. Most injured patients in shock have hypovolemia, but they may suffer from cardiogenic, obstructive, neurogenic, and/or, rarely, septic shock. For example, tension pneumothorax can reduce venous return and produce obstructive shock. Cardiac tamponade also produces obstructive shock, as blood in the pericardial sac inhibits cardiac contractility and cardiac output. Trauma team members should consider these diagnoses in patients with injuries above the diaphragm. Neurogenic shock results from extensive injury to the cervical or upper thoracic spinal cord caused by a loss of sympathetic tone and subsequent vasodilation. Shock does not result from an isolated brain injury unless the brainstem is involved, in which case the prognosis is poor. Patients with spinal cord injury may initially present in shock resulting from both vasodilation and hypovolemia, especially if there are multiple other injuries. Septic shock is unusual, but must be considered in patients whose arrival at the emergency facility was delayed for many hours. In the elderly, the underlying reason or precipitating cause of traumatic injury may be an unrecognized infection, commonly a urinary tract infection.

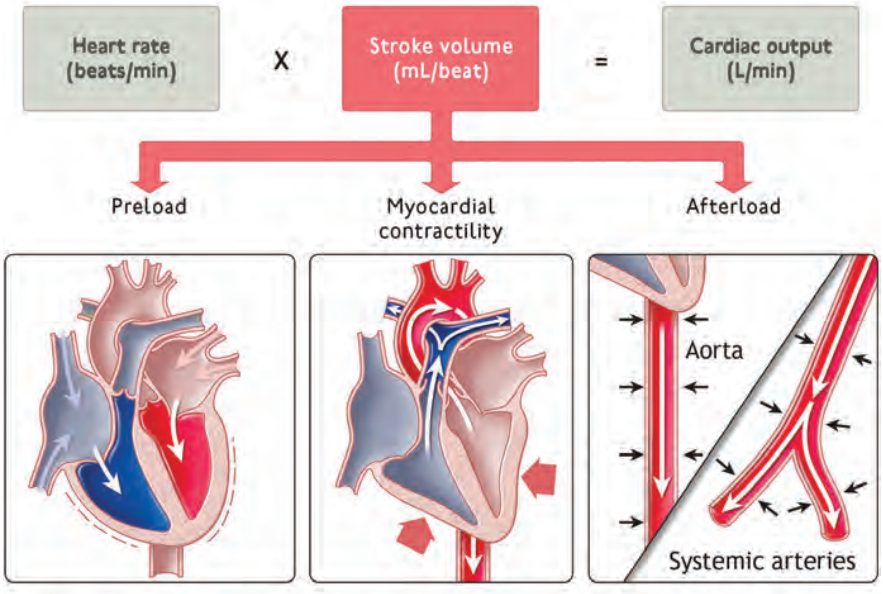
Patient management responsibilities begin with recognizing the presence of shock. Initiate treatment immediately and identify the probable cause. The patient's response to initial treatment, coupled with the findings of the primary and secondary surveys, usually provides sufficient information to determine the cause of shock. Hemorrhage is the most common cause of shock in trauma patients.

SHOCK PATHOPHYSIOLOGY

An overview of basic cardiac physiology and blood loss pathophysiology is essential to understanding the shock state.

BASIC CARDIAC PHYSIOLOGY

Cardiac output is defined as the volume of blood pumped by the heart per minute. This value is determined by multiplying the heart rate by the stroke volume (the amount of blood that leaves the heart with each cardiac contraction). Stroke volume is classically determined by preload, myocardial contractility, and afterload (■ FIGURE 3-1).



■ FIGURE 3-1 Cardiac output is the volume of blood pumped by the heart per minute, determined by multiplying the heart rate by the stroke volume (i.e., the amount of blood that leaves the heart with each cardiac contraction). Stroke volume is classically determined by preload, myocardial contractility, and afterload.

Preload, the volume of venous blood return to the left and right sides of the heart, is determined by venous capacitance, volume status, and the difference between mean venous systemic pressure and right atrial pressure. This pressure differential determines venous flow. The venous system can be considered a reservoir, or capacitance, system in which the volume of blood is divided into two components:

1. The first component represents the volume of blood that would remain in this capacitance circuit if the pressure in the system were zero. This component does not contribute to the mean systemic venous pressure.
2. The second component represents the venous volume that contributes to the mean systemic venous pressure. Nearly 70% of the body's total blood volume is estimated to be located in the venous circuit. Compliance of the venous system involves a relationship between venous volume and venous pressure. This pressure gradient drives venous flow and therefore the volume of venous return to the heart. Blood loss depletes this component of venous volume and reduces the pressure gradient; consequently, venous return is reduced.

The volume of venous blood returned to the heart determines myocardial muscle fiber length after ventricular filling at the end of diastole. According to Starling's law, muscle fiber length is related to the contractile properties of myocardial muscle. Myocardial contractility is the pump that drives the system.

Afterload, also known as peripheral vascular resistance, is systemic. Simply stated, afterload is resistance to the forward flow of blood.

BLOOD LOSS PATHOPHYSIOLOGY

Early circulatory responses to blood loss are compensatory and include progressive vasoconstriction of cutaneous, muscular, and visceral circulation to preserve blood flow to the kidneys, heart, and brain. The usual response to acute circulating volume depletion is an increase in heart rate in an attempt to preserve cardiac output. In most cases, tachycardia is the earliest measurable circulatory sign of shock. The release of endogenous catecholamines increases peripheral vascular resistance, which in turn increases diastolic blood pressure and reduces pulse pressure. However, this increase in pressure does little to increase organ perfusion and tissue oxygenation.

For patients in early hemorrhagic shock, venous return is preserved to some degree by the compensatory mechanism of contraction of the volume of blood in the venous system. This compensatory mechanism is limited. The most effective method of restoring adequate cardiac output, end-organ perfusion, and tissue oxygenation is to restore venous return to normal by locating and stopping the source of bleeding. Volume repletion will allow recovery from the shock state only when the bleeding has stopped.

At the cellular level, inadequately perfused and poorly oxygenated cells are deprived of essential substrates for normal aerobic metabolism and energy production. Initially, compensation occurs by shifting to anaerobic metabolism, resulting in the formation of lactic acid and development of metabolic acidosis. If shock is prolonged, subsequent end-organ damage and multiple organ dysfunction may result.

Administration of an appropriate quantity of isotonic electrolyte solutions, blood, and blood products helps combat this process. Treatment must focus on reversing the shock state by stopping the bleeding and providing adequate oxygenation, ventilation, and appropriate fluid resuscitation. Rapid intravenous access must be obtained.

Definitive control of hemorrhage and restoration of adequate circulating volume are the goals of treating hemorrhagic shock. Vasopressors are contraindicated as a first-line treatment of hemorrhagic shock because they worsen tissue perfusion. Frequently monitor the patient's indices of perfusion to detect any deterioration in the patient's condition as early as possible so it can be reversed. Monitoring also allows for evaluation of the patient's response to therapy. Reassessment helps clinicians identify patients in compensated shock and those who are unable to mount a compensatory response before cardiovascular collapse occurs.

Most injured patients who are in hemorrhagic shock require early surgical intervention or angioembolization to reverse the shock state. The presence of shock in a trauma patient warrants the immediate involvement of a surgeon. Strongly consider arranging for early transfer of these patients to a trauma center when they present to hospitals that are not equipped to manage their injuries.

INITIAL PATIENT ASSESSMENT

Optimally, clinicians recognize the shock state during the initial patient assessment. To do so, they must be familiar with the clinical differentiation of causes of shock—chiefly, hemorrhagic and non-hemorrhagic shock.

RECOGNITION OF SHOCK

Profound circulatory shock, as evidenced by hemodynamic collapse with inadequate perfusion of the skin, kidneys, and central nervous system, is simple to recognize. After ensuring a patent airway and adequate ventilation, trauma team members must carefully evaluate the patient's circulatory status for early manifestations of shock, such as tachycardia and cutaneous vasoconstriction.

Relying solely on systolic blood pressure as an indicator of shock can delay recognition of the condition, as compensatory mechanisms can prevent a measurable fall in systolic pressure until up to 30% of the patient's blood volume is lost. Look closely at pulse rate, pulse character, respiratory rate, skin perfusion, and pulse pressure (i.e., the difference between systolic and diastolic pressure). In most adults, tachycardia and cutaneous vasoconstriction are the typical early physiologic responses to volume loss.

Any injured patient who is cool to the touch and is tachycardic should be considered to be in shock until proven otherwise. Occasionally, a normal heart rate or even bradycardia is associated with an acute reduction of blood volume; other indices of perfusion must be monitored in these situations.

The normal heart rate varies with age. Tachycardia is diagnosed when the heart rate is greater than 160 beats per minute (BPM) in an infant, 140 BPM in a preschool-aged child, 120 BPM in children from school age to puberty, and 100 BPM in adults. Elderly patients may not exhibit tachycardia because of their limited cardiac response to catecholamine stimulation or the concurrent use of medications, such as ß-adrenergic blocking agents. The body's ability to increase the heart rate also may be limited by the presence of a pacemaker. A narrowed pulse pressure suggests significant blood loss and involvement of compensatory mechanisms.

Massive blood loss may produce only a slight decrease in initial hematocrit or hemoglobin concentration. Thus, a very low hematocrit value obtained shortly after injury suggests either massive blood loss or a preexisting anemia, and a normal hematocrit does not exclude significant blood loss. Base deficit and/or lactate levels can be useful in determining the presence and severity of shock. Serial measurements of these parameters to monitor a patient's response to therapy are useful.

CLINICAL DIFFERENTIATION OF CAUSE OF SHOCK

Shock in a trauma patient is classified as hemorrhagic or non-hemorrhagic shock. A patient with injuries above the diaphragm may have evidence of inadequate organ perfusion and tissue oxygenation due to poor cardiac performance from blunt myocardial injury, cardiac tamponade, or a tension pneumothorax that produces inadequate venous return (preload). To recognize and manage all forms of shock, clinicians must maintain a high level of suspicion and carefully observe the patient's response to initial treatment.

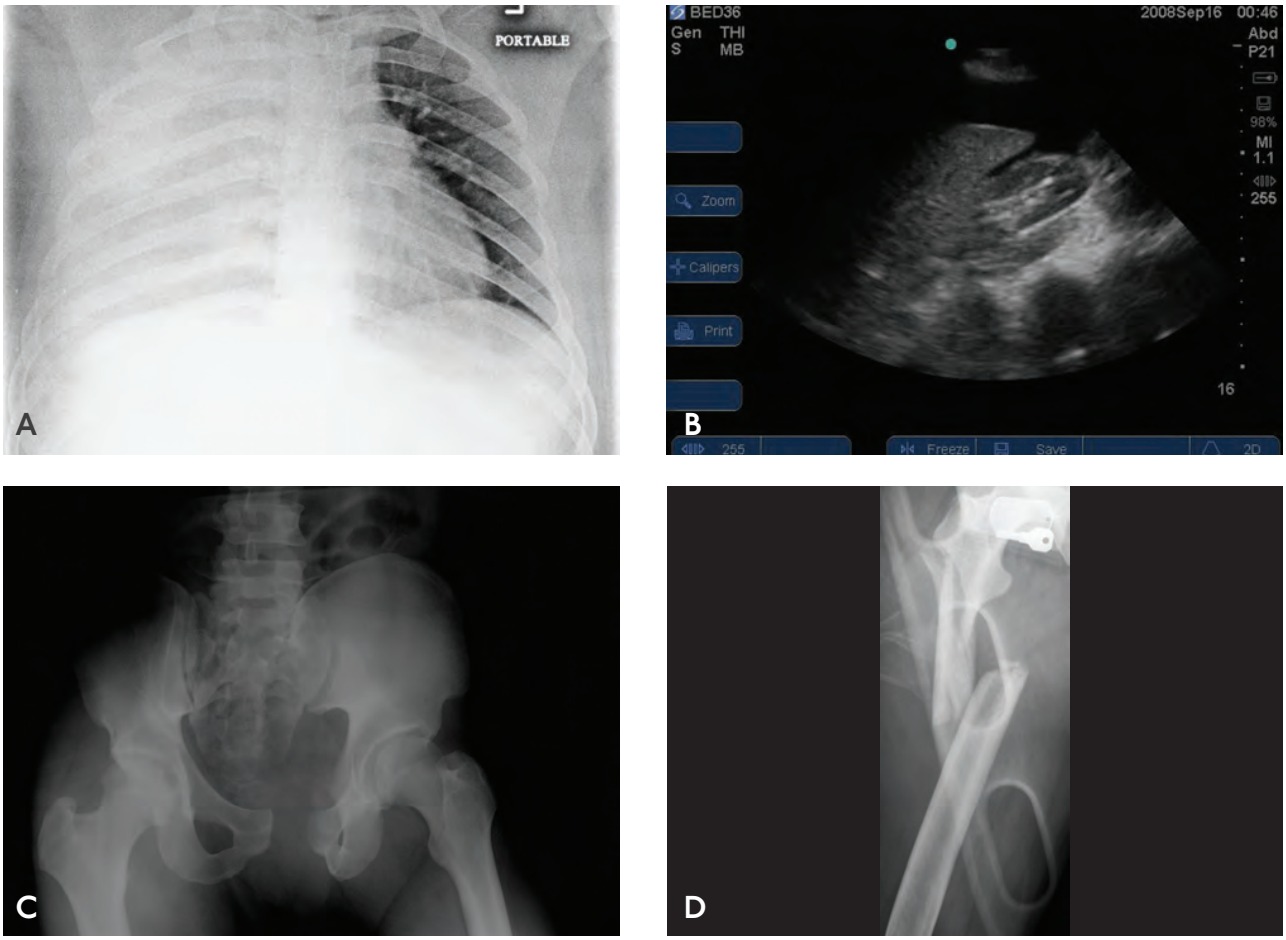
Initial determination of the cause of shock requires an appropriate patient history and expeditious, careful physical examination. Selected additional tests, such as chest and pelvic x-rays and focused assessment with sonography for trauma (FAST) examinations, can confirm the cause of shock, but should not delay appropriate resuscitation. (See ***FAST video on MyATLS mobile app.***)

Overview of Hemorrhagic Shock

Hemorrhage is the most common cause of shock after injury, and virtually all patients with multiple injuries have some degree of hypovolemia. Therefore, if signs of shock are present, treatment typically is instituted as if the patient were hypovolemic. However, while instituting treatment, it is important to identify the small number of patients whose shock has a different cause (e.g., a secondary condition, such as cardiac tamponade, tension pneumothorax, spinal cord injury, or blunt cardiac injury), which complicates the presentation of hemorrhagic shock.

The treatment of hemorrhagic shock is described later in this chapter, but the primary focus is to promptly identify and stop hemorrhage. Sources of potential blood loss—chest, abdomen, pelvis, retroperitoneum, extremities, and external bleeding—must be quickly assessed by physical examination and appropriate adjunctive studies. Chest x-ray, pelvic x-ray, abdominal

■ **FIGURE 3-2** Using ultrasound (FAST) to search for the cause of shock.



■ **FIGURE 3-3** Assessment of circulation includes rapidly determining the site of blood loss. In addition to the floor, blood may be in four other places ("on the floor plus four more"): **A.** the chest; **B.** the abdomen; **C.** the pelvis and retroperitoneum; and **D.** major long bones and soft tissues.

assessment with either FAST or diagnostic peritoneal lavage (DPL), and bladder catheterization may all be necessary to determine the source of blood loss (■ **FIGURES 3-2** and **3-3**).

Overview of Non-hemorrhagic Shock

The category of non-hemorrhagic shock includes cardiogenic shock, cardiac tamponade, tension pneumothorax, neurogenic shock, and septic shock. Even without blood loss, most non-hemorrhagic shock states transiently improve with volume resuscitation.

Cardiogenic Shock

Myocardial dysfunction can be caused by blunt cardiac injury, cardiac tamponade, an air embolus, or, rarely, myocardial infarction. Suspect a blunt cardiac injury when the mechanism of injury to the thorax involves rapid deceleration. All patients with blunt thoracic trauma need continuous electrocardiographic (ECG) monitoring to detect injury patterns and dysrhythmias. (See *Chapter 4: Thoracic Trauma*.) The shock state may be secondary to myocardial infarction in the elderly and other high-risk patients, such as those with cocaine intoxication. Therefore, cardiac enzyme levels may assist in diagnosing and treating injured patients in the emergency department (ED), as acute myocardial ischemia may be the precipitating event.

Cardiac Tamponade

Although cardiac tamponade is most commonly encountered in patients with penetrating thoracic trauma, it can result from blunt injury to the thorax. Tachycardia, muffled heart sounds, and dilated, engorged neck veins with hypotension and insufficient response to fluid therapy suggest cardiac tamponade.

However, the absence of these classic findings does not exclude the presence of this condition.

Tension pneumothorax can mimic cardiac tamponade, with findings of distended neck veins and hypotension in both. However, absent breath sounds and hyperresonant percussion are not present with tamponade. Echocardiography may be useful in diagnosing tamponade and valve rupture, but it is often not practical or immediately available in the ED. FAST performed in the ED can identify pericardial fluid, which suggests cardiac tamponade as the cause of shock. Cardiac tamponade is best managed by formal operative intervention, as pericardiocentesis is at best only a temporizing maneuver. (See *Chapter 4: Thoracic Trauma*.)

Tension Pneumothorax

Tension pneumothorax is a true surgical emergency that requires immediate diagnosis and treatment. It develops when air enters the pleural space, but a flap-valve mechanism prevents its escape. Intrapleural pressure rises, causing total lung collapse and a shift of the mediastinum to the opposite side, with subsequent impairment of venous return and a fall in cardiac output. Spontaneously breathing patients often manifest extreme tachypnea and air hunger, while mechanically ventilated patients more often manifest hemodynamic collapse. The presence of acute respiratory distress, subcutaneous emphysema, absent unilateral breath sounds, hyperresonance to percussion, and tracheal shift supports the diagnosis of tension pneumothorax and warrants immediate thoracic decompression without waiting for x-ray confirmation of the diagnosis. Needle or finger decompression of tension pneumothorax temporarily relieves this life-threatening condition. Follow this procedure by placing a chest tube using appropriate sterile technique. (See *Appendix G: Breathing Skills* and *Chest Tube video on MyATLS mobile app.*)

Neurogenic Shock

Isolated intracranial injuries do not cause shock, unless the brainstem is injured. Therefore, the presence of shock in patients with head injury necessitates the search for another cause. Cervical and upper thoracic spinal cord injuries can produce hypotension due to loss of sympathetic tone, which compounds the physiologic effects of hypovolemia. In turn, hypovolemia compounds the physiologic effects of sympathetic denervation. The classic presentation of neurogenic shock is hypotension without tachycardia or cutaneous vasoconstriction. A narrowed pulse pressure is not seen in neurogenic shock. Patients who have sustained a spinal cord injury often have concurrent torso trauma; therefore, patients with known or suspected neurogenic shock are treated initially for hypovolemia. The failure of fluid resuscitation to restore organ perfusion and tissue oxygenation suggests either continuing hemorrhage or neurogenic shock. Advanced techniques for monitoring intravascular volume status and cardiac output may be helpful in managing this complex problem. (See *Chapter 7: Spine and Spinal Cord Trauma*.)

Septic Shock

Shock due to infection immediately after injury is uncommon; however, it can occur when a patient's arrival at the ED is delayed for several hours. Septic shock can occur in patients with penetrating abdominal injuries and contamination of the peritoneal cavity by intestinal contents. Patients with sepsis who also have hypotension and are afebrile are clinically difficult to distinguish from those in hypovolemic shock, as patients in both groups can have tachycardia, cutaneous vasoconstriction, impaired urinary output, decreased systolic pressure, and narrow pulse pressure. Patients with early septic shock can have a normal circulating volume, modest tachycardia, warm skin, near normal systolic blood pressure, and a wide pulse pressure.

HEMORRHAGIC SHOCK

Hemorrhage is the most common cause of shock in trauma patients. The trauma patient's response to blood loss is made more complex by fluid shifts among the fluid compartments in the body, particularly in the extracellular fluid compartment. Soft tissue injury, even without severe hemorrhage, can result in shifts of fluid to the extracellular compartment. The response to blood loss must be considered in the context of these fluid shifts. Also consider the changes associated with severe, prolonged shock and the pathophysiologic results of resuscitation and reperfusion.

DEFINITION OF HEMORRHAGE

Hemorrhage is an acute loss of circulating blood volume. Although it can vary considerably, normal adult blood volume is approximately 7% of body weight. For example, a 70-kg male has a circulating blood volume of approximately 5 L. The blood volume

of obese adults is estimated based on their ideal body weight, because calculation based on actual weight can result in significant overestimation. The blood volume for a child is calculated as 8% to 9% of body weight (70–80 mL/kg). (See *Chapter 10: Pediatric Trauma*.)

PHYSIOLOGIC CLASSIFICATION

The physiologic effects of hemorrhage are divided into four classes, based on clinical signs, which are useful for estimating the percentage of acute blood loss. The clinical signs represent a continuum of ongoing hemorrhage and serve only to guide initial therapy. Subsequent volume replacement is determined by the patient's response to therapy. The following classification system is useful in emphasizing the early signs and pathophysiology of the shock state:

- Class I hemorrhage is exemplified by the condition of an individual who has donated 1 unit of blood.
- Class II hemorrhage is uncomplicated hemorrhage for which crystalloid fluid resuscitation is required.
- Class III hemorrhage is a complicated hemorrhagic state in which at least crystalloid infusion is required and perhaps also blood replacement.
- Class IV hemorrhage is considered a preterminal event; unless aggressive measures are taken, the patient will die within minutes. Blood transfusion is required.

■ **TABLE 3-1** outlines the estimated blood loss and other critical measures for patients in each classification of shock.

Class I Hemorrhage: <15% Blood Volume Loss

The clinical symptoms of volume loss with class I hemorrhage are minimal. In uncomplicated situations, minimal tachycardia occurs. No measurable changes occur in blood pressure, pulse pressure, or respiratory rate. For otherwise healthy patients, this amount of blood loss does not require replacement, because transcapillary refill and other compensatory mechanisms will restore blood volume within 24 hours, usually without the need for blood transfusion.

TABLE 3-1 SIGNS AND SYMPTOMS OF HEMORRHAGE BY CLASS

PARAMETER	CLASS I	CLASS II (MILD)	CLASS III (MODERATE)	CLASS IV (SEVERE)
Approximate blood loss	<15%	15–30%	31–40%	>40%
Heart rate	↔	↔/↑	↑	↑/↑↑
Blood pressure	↔	↔	↔/↓	↓
Pulse pressure	↔	↓	↓	↓
Respiratory rate	↔	↔	↔/↑	↑
Urine output	↔	↔	↓	↓↓
Glasgow Coma Scale score	↔	↔	↓	↓
Base deficit[a]	0 to −2 mEq/L	−2 to −6 mEq/L	−6 to −10 mEq/L	−10 mEq/L or less
Need for blood products	Monitor	Possible	Yes	Massive Transfusion Protocol

[a] Base excess is the quantity of base (HCO_3^-, in mEq/L) that is above or below the normal range in the body. A negative number is called a base deficit and indicates metabolic acidosis.

Data from: Mutschler A, Nienaber U, Brockamp T, et al. A critical reappraisal of the ATLS classification of hypovolaemic shock: does it really reflect clinical reality? *Resuscitation* 2013,84:309–313.

Class II Hemorrhage: 15% to 30% Blood Volume Loss

Clinical signs of class II hemorrhage include tachycardia, tachypnea, and decreased pulse pressure. The latter sign is related primarily to a rise in diastolic blood pressure due to an increase in circulating catecholamines, which produce an increase in peripheral vascular tone and resistance. Systolic pressure changes minimally in early hemorrhagic shock; therefore, it is important to evaluate pulse pressure rather than systolic pressure. Other pertinent clinical findings associated with this amount of blood loss include subtle central nervous system (CNS) changes, such as anxiety, fear, and hostility. Despite the significant blood loss and cardiovascular changes, urinary output is only mildly affected. The measured urine flow is usually 20 to 30 mL/hour in an adult with class II hemorrhage.

Accompanying fluid losses can exaggerate the clinical manifestations of class II hemorrhage. Some patients in this category may eventually require blood transfusion, but most are stabilized initially with crystalloid solutions.

Class III Hemorrhage: 31% to 40% Blood Volume Loss

Patients with class III hemorrhage typically present with the classic signs of inadequate perfusion, including marked tachycardia and tachypnea, significant changes in mental status, and a measurable fall in systolic blood pressure. In an uncomplicated case, this is the least amount of blood loss that consistently causes a drop in systolic blood pressure. The priority of initial management is to stop the hemorrhage, by emergency operation or embolization, if necessary. Most patients in this category will require packed red blood cells (pRBCs) and blood products to reverse the shock state.

Class IV Hemorrhage: >40% Blood Volume Loss

The degree of exsanguination with class IV hemorrhage is immediately life-threatening. Symptoms include marked tachycardia, a significant decrease in systolic blood pressure, and a very narrow pulse pressure or unmeasurable diastolic blood pressure. (Bradycardia may develop preterminally.) Urinary output is negligible, and mental status is markedly depressed. The skin is cold and pale. Patients with class IV hemorrhage frequently require rapid transfusion and immediate surgical intervention. These decisions are based on the patient's response to the initial management techniques described in this chapter.

PITFALL	PREVENTION
Diagnosis of shock can be missed when only a single parameter is used.	• Use all clinical information, including heart rate, blood pressure, skin perfusion, and mental status. • When available, obtain arterial blood gas measurements of pH, pO_2, PCO_2, oxygen saturation, and base deficit. • Measurement of end-tidal CO_2 and serum lactate can add useful diagnostic information.
Injury in elderly patients may be related to underlying infection.	• Always obtain screening urinalysis. • Look for subtle evidence of infection.

CONFOUNDING FACTORS

The physiologic classification system is helpful, but the following factors may confound and profoundly alter the classic hemodynamic response to the acute loss of circulating blood volume; all individuals involved in the initial assessment and resuscitation of injured patients must promptly recognize them:

- Patient age
- Severity of injury, particularly the type and anatomic location of injury
- Time lapse between injury and initiation of treatment
- Prehospital fluid therapy
- Medications used for chronic conditions

It is dangerous to wait until a trauma patient fits a precise physiologic classification of shock before initiating appropriate volume restoration. Initiate hemorrhage control and balanced fluid resuscitation when early signs and symptoms of blood loss are apparent or suspected—not when the blood pressure is falling or absent. Stop the bleeding.

FLUID CHANGES SECONDARY TO SOFT-TISSUE INJURY

Major soft-tissue injuries and fractures compromise the hemodynamic status of injured patients in two ways: First, blood is lost into the site of injury, particularly in major fractures. For example, a fractured tibia or humerus can result in the loss of up to 750 mL of blood. Twice that amount, 1500 mL, is commonly associated with femur fractures, and several liters of blood can accumulate in a retroperitoneal hematoma associated with a pelvic fracture. Obese patients are at risk for extensive blood loss into soft tissues, even in the absence of fractures. Elderly patients are also at risk because of fragile skin and subcutaneous tissues that injures more readily and tamponades less effectively, in addition to inelastic blood vessels that do not spasm and thrombose when injured or transected.

Second, edema that occurs in injured soft tissues constitutes another source of fluid loss. The degree of this additional volume loss is related to the magnitude of the soft-tissue injury. Tissue injury results in activation of a systemic inflammatory response and production and release of multiple cytokines. Many of these locally active substances have profound effects on the vascular endothelium, resulting in increased permeability. Tissue edema is the result of shifts in fluid primarily from the plasma into the extravascular, or extracellular, space as a result of alterations in endothelial permeability. Such shifts produce an additional depletion in intravascular volume.

PITFALL	PREVENTION
Blood loss can be underestimated from soft-tissue injury, particularly in obese and elderly individuals.	• Evaluate and dress wounds early to control bleeding with direct pressure and temporary closure. • Reassess wounds and wash and close them definitively once the patient has stabilized.

INITIAL MANAGEMENT OF HEMORRHAGIC SHOCK

The diagnosis and treatment of shock must occur almost simultaneously. For most trauma patients, clinicians begin treatment as if the patient has hemorrhagic shock, unless a different cause of shock is clearly evident. The basic management principle is to stop the bleeding and replace the volume loss.

PHYSICAL EXAMINATION

The physical examination is focused on diagnosing immediately life-threatening injuries and assessing the ABCDEs. Baseline observations are important to assess the patient's response to therapy, and repeated measurements of vital signs, urinary output, and level of consciousness are essential. A more detailed examination of the patient follows as the situation permits.

Airway and Breathing

Establishing a patent airway with adequate ventilation and oxygenation is the first priority. Provide supplementary oxygen to maintain oxygen saturation at greater than 95%.

Circulation: Hemorrhage Control

Priorities for managing circulation include controlling obvious hemorrhage, obtaining adequate intravenous access, and assessing tissue perfusion. Bleeding from external wounds in the extremities usually can be controlled by direct pressure to the bleeding site, although massive blood loss from an extremity may require a tourniquet. A sheet or pelvic binder may be used to control bleeding from pelvic fractures. (See *Pelvic Binder video on MyATLS mobile app.*) Surgical or angioembolization may be required to control internal hemorrhage. The priority is to stop the bleeding, not to calculate the volume of fluid lost.

Disability: Neurological Examination

A brief neurological examination will determine the patient's level of consciousness, which is useful in assessing cerebral perfusion. Alterations in CNS function in patients who have hypovolemic shock do not necessarily imply direct intracranial injury and may reflect inadequate perfusion. Repeat neurological evaluation after restoring perfusion and oxygenation. (See *Chapter 6: Head Trauma*.)

Exposure: Complete Examination

After addressing lifesaving priorities, completely undress the patient and carefully examine him or her from head to toe to search for additional injuries. When exposing a patient, it is essential to prevent hypothermia, a condition that can exacerbate blood loss by contributing to coagulopathy and worsening acidosis. To prevent

hypothermia, always use fluid warmers and external passive and active warming techniques.

Gastric Dilation: Decompression

Gastric dilation often occurs in trauma patients, especially in children. This condition can cause unexplained hypotension or cardiac dysrhythmia, usually bradycardia from excessive vagal stimulation. In unconscious patients, gastric distention increases the risk of aspiration of gastric contents, a potentially fatal complication. Consider decompressing the stomach by inserting a nasal or oral tube and attaching it to suction. Be aware that proper positioning of the tube does not eliminate the risk of aspiration.

Urinary Catheterization

Bladder catheterization allows clinicians to assess the urine for hematuria, which can identify the genitourinary system as a source of blood loss. Monitoring urine output also allows for continuous evaluation of renal perfusion. Blood at the urethral meatus or perineal hematoma/bruising may indicate urethral injury and contraindicates the insertion of a transurethral catheter before radiographic confirmation of an intact urethra. (See *Chapter 5: Abdominal and Pelvic Trauma*.)

VASCULAR ACCESS

Obtain access to the vascular system promptly. This measure is best accomplished by inserting two large-caliber (minimum of 18-gauge in an adult) peripheral intravenous catheters. The rate of flow is proportional to the fourth power of the radius of the cannula and inversely related to its length, as described in Poiseuille's law. Hence, short, large-caliber peripheral intravenous lines are preferred for the rapid infusion of fluid, rather than longer, thinner catheters. Use fluid warmers and rapid infusion pumps in the presence of massive hemorrhage and severe hypotension.

The most desirable sites for peripheral, percutaneous intravenous lines in adults are the forearms and antecubital veins. This can be challenging in the young, very old, obese patients, and intravenous drug users. If peripheral access cannot be obtained, consider placement of an intraosseous needle for temporary access. If circumstances prevent the use of peripheral veins, clinicians may initiate large-caliber, central venous (i.e., femoral, jugular, or subclavian vein) access. (See *Appendix G: Circulation Skills* and *Intraosseous Puncture video on MyATLS mobile app.*) The clinician's experience and skill are critical determinants in selecting the most appropriate procedure or route for establishing vascular access. Intraosseous access with specially designed equipment is possible in all *age groups*. This access may be used in the hospital until intravenous access is obtained and is discontinued when it is no longer necessary.

As intravenous lines are started, draw blood samples for type and crossmatch, appropriate laboratory analyses, toxicology studies, and pregnancy testing for all females of childbearing age. Blood gas analysis also may be performed at this time. A chest x-ray must be obtained after attempts at inserting a subclavian or internal jugular line to document the position of the line and evaluate for a pneumothorax or hemothorax. In emergency situations, central venous access is frequently not accomplished under tightly controlled or completely sterile conditions. Therefore, these lines should be changed in a more controlled environment as soon as the patient's condition permits.

INITIAL FLUID THERAPY

The amount of fluid and blood required for resuscitation is difficult to predict on initial evaluation of a patient. Administer an initial, warmed fluid bolus of isotonic fluid. The usual dose is 1 liter for adults and 20 mL/kg for pediatric patients weighing less than 40 kilograms. Absolute volumes of resuscitation fluid should be based on patient response to fluid administration, keeping in mind that this initial fluid amount includes any fluid given in the prehospital setting. Assess the patient's response to fluid resuscitation and identify evidence of adequate end-organ perfusion and tissue oxygenation. Observe the patient's response during this initial fluid administration and base further therapeutic and diagnostic decisions on this response. Persistent infusion of large volumes of fluid and blood in an attempt to achieve a normal blood pressure is not a substitute for definitive control of bleeding.

■ **TABLE 3-2** outlines general guidelines for establishing the amount of fluid and blood likely required during resuscitation. If the amount of fluid required to restore or maintain adequate organ perfusion and tissue oxygenation greatly exceeds these estimates, carefully reassess the situation and search for unrecognized injuries and other causes of shock.

The goal of resuscitation is to restore organ perfusion and tissue oxygenation, which is accomplished with administering crystalloid solution and blood products to replace lost intravascular volume. However, if the patient's blood pressure increases rapidly before the hemorrhage has been definitively

TABLE 3-2 RESPONSES TO INITIAL FLUID RESUSCITATION[a]

	RAPID RESPONSE	TRANSIENT RESPONSE	MINIMAL OR NO RESPONSE
Vital signs	Return to normal	Transient improvement, recurrence of decreased blood pressure and increased heart rate	Remain abnormal
Estimated blood loss	Minimal (<15 %)	Moderate and ongoing (15%–40%)	Severe (>40%)
Need for blood	Low	Moderate to high	Immediate
Blood preparation	Type and crossmatch	Type-specific	Emergency blood release
Need for operative intervention	Possibly	Likely	Highly likely
Early presence of surgeon	Yes	Yes	Yes

[a] Isotonic crystalloid solution, up to 1000 mL in adults; 20 mL/kg in children

PITFALL	PREVENTION
Shock does not respond to initial crystalloid fluid bolus.	• Look for a source of ongoing blood loss: "floor and four more (abdomen/ pelvis, retroperitoneum, thorax, and extremities). • Consider a non-hemorrhagic source of shock. • Begin blood and blood component replacement. • Obtain surgical consultation for definitive hemorrhage control.

controlled, more bleeding can occur. For this reason, administering excessive crystalloid solution can be harmful.

Fluid resuscitation and avoidance of hypotension are important principles in the initial management of patients with blunt trauma, particularly those with traumatic brain injury. In penetrating trauma with hemorrhage, delaying aggressive fluid resuscitation until definitive control of hemorrhage is achieved may prevent additional bleeding; a careful, balanced approach with frequent reevaluation is required. Balancing the goal of organ perfusion and tissue oxygenation with the avoidance of rebleeding by accepting a lower-than-normal blood pressure has been termed "controlled resuscitation," "balanced resuscitation," "hypotensive resuscitation," and "permissive hypotension." Such a resuscitation strategy may be a bridge to, but is not a substitute for, definitive surgical control of bleeding.

Early resuscitation with blood and blood products must be considered in patients with evidence of class III and IV hemorrhage. Early administration of blood products at a low ratio of packed red blood cells to plasma and platelets can prevent the development of coagulopathy and thrombocytopenia.

Measuring Patient Response to Fluid Therapy

The same signs and symptoms of inadequate perfusion that are used to diagnose shock help determine the patient's response to therapy. The return of normal blood pressure, pulse pressure, and pulse rate are signs that perfusion is returning to normal, however, these observations do not provide information regarding organ perfusion and tissue oxygenation. Improvement in the intravascular volume status is important evidence of enhanced perfusion, but it is difficult to quantitate. The volume of urinary output is a reasonably sensitive indicator of renal perfusion; normal urine volumes generally imply adequate renal blood flow, if not modified by underlying kidney injury, marked hyperglycemia or the administration of diuretic agents. For this reason, urinary output is one of the prime indicators of resuscitation and patient response.

Within certain limits, urinary output is used to monitor renal blood flow. Adequate volume

replacement during resuscitation should produce a urinary output of approximately 0.5 mL/kg/hr in adults, whereas 1 mL/kg/hr is adequate urinary output for pediatric patients. For children under 1 year of age, 2 mL/kg/hr should be maintained. The inability to obtain urinary output at these levels or a decreasing urinary output with an increasing specific gravity suggests inadequate resuscitation. This situation should stimulate further volume replacement and continued diagnostic investigation for the cause.

Patients in early hypovolemic shock have respiratory alkalosis from tachypnea, which is frequently followed by mild metabolic acidosis and does not require treatment. However, severe metabolic acidosis can develop from long-standing or severe shock. Metabolic acidosis is caused by anaerobic metabolism, as a result of inadequate tissue perfusion and the production of lactic acid. Persistent acidosis is usually caused by inadequate resuscitation or ongoing blood loss. In patients in shock, treat metabolic acidosis with fluids, blood, and interventions to control hemorrhage. Base deficit and/or lactate values can be useful in determining the presence and severity of shock, and then serial measurement of these parameters can be used to monitor the response to therapy. Do not use sodium bicarbonate to treat metabolic acidosis from hypovolemic shock.

Patterns of Patient Response

The patient's response to initial fluid resuscitation is the key to determining subsequent therapy. Having established a preliminary diagnosis and treatment plan based on the initial assessment, the clinician modifies the plan based on the patient's response. Observing the response to the initial resuscitation can identify patients whose blood loss was greater than estimated and those with ongoing bleeding who require operative control of internal hemorrhage.

The potential patterns of response to initial fluid administration can be divided into three groups: rapid response, transient response, and minimal or no response. Vital signs and management guidelines for patients in each of these categories were outlined earlier (see Table 3-2).

Rapid Response

Patients in this group, referred to as "rapid responders," quickly respond to the initial fluid bolus and become hemodynamically normal, without signs of inadequate tissue perfusion and oxygenation. Once this occurs, clinicians can slow the fluids to maintenance rates. These patients typically have lost less than 15% of their blood volume (class I hemorrhage), and no further fluid bolus or immediate blood administration is indicated. However, typed and crossmatched blood should be kept available. Surgical consultation and evaluation are necessary during initial assessment and treatment of rapid responders, as operative intervention could still be necessary.

Transient Response

Patients in the second group, "transient responders," respond to the initial fluid bolus. However, they begin to show deterioration of perfusion indices as the initial fluids are slowed to maintenance levels, indicating either an ongoing blood loss or inadequate resuscitation. Most of these patients initially have lost an estimated 15% to 40% of their blood volume (class II and III hemorrhage). Transfusion of blood and blood products is indicated, but even more important is recognizing that such patients require operative or angiographic control of hemorrhage. A transient response to blood administration identifies patients who are still bleeding and require rapid surgical intervention. Also consider initiating a massive transfusion protocol (MTP).

Minimal or No Response

Failure to respond to crystalloid and blood administration in the ED dictates the need for immediate, definitive intervention (i.e., operation or angioembolization) to control exsanguinating hemorrhage. On very rare occasions, failure to respond to fluid resuscitation is due to pump failure as a result of blunt cardiac injury, cardiac tamponade, or tension pneumothorax. Non-hemorrhagic shock always should be considered as a diagnosis in this group of patients (class IV hemorrhage). Advanced monitoring techniques such as cardiac ultrasonography are useful to identify the cause of shock. MTP should be initiated in these patients (■ FIGURE 3-4).

BLOOD REPLACEMENT

The decision to initiate blood transfusion is based on the patient's response, as described in the previous section. Patients who are transient responders or nonresponders require pRBCs, plasma and platelets as an early part of their resuscitation.

■ **FIGURE 3-4** Massive transfusion of blood products in a trauma patient.

CROSSMATCHED, TYPE-SPECIFIC, AND TYPE O BLOOD

The main purpose of blood transfusion is to restore the oxygen-carrying capacity of the intravascular volume. Fully crossmatched pRBCs are preferable for this purpose, but the complete crossmatching process requires approximately 1 hour in most blood banks. For patients who stabilize rapidly, crossmatched pRBCs should be obtained and made available for transfusion when indicated.

If crossmatched blood is unavailable, type O pRBCs are indicated for patients with exsanguinating hemorrhage. AB plasma is given when uncrossmatched plasma is needed. To avoid sensitization and future complications, Rh-negative pRBCs are preferred for females of childbearing age. As soon as it is available, the use of unmatched, type-specific pRBCs is preferred over type O pRBCs. An exception to this rule is when multiple, unidentified casualties are being treated simultaneously, and the risk of inadvertently administering the wrong unit of blood to a patient is increased.

PREVENT HYPOTHERMIA

Hypothermia must be prevented and reversed if a patient is hypothermic on arrival to the hospital. The use of blood warmers in the ED is critical, even if cumbersome. The most efficient way to prevent hypothermia in any patient receiving massive resuscitation of crystalloid and blood is to heat the fluid to 39°C (102.2°F) before infusing it. This can be accomplished by storing crystalloids in a warmer or infusing them through intravenous fluid warmers. Blood products cannot be stored in a warmer, but they can be heated by passage through intravenous fluid warmers.

AUTOTRANSFUSION

Adaptations of standard tube thoracostomy collection devices are commercially available, allowing for sterile collection, anticoagulation (generally with sodium citrate solutions rather than heparin), and transfusion of shed blood. Consider collection of shed blood for autotransfusion in patients with massive hemothorax. This blood generally has only low levels of coagulation factors, so plasma and platelets may still be needed.

MASSIVE TRANSFUSION

A small subset of patients with shock will require massive transfusion, most often defined as > 10 units of pRBCs within the first 24 hours of admission or more than 4 units in 1 hour. Early administration of pRBCs, plasma, and platelets in a balanced ratio to minimize excessive crystalloid administration may improve patient survival. This approach has been termed "balanced," "hemostatic," or "damage-control" resuscitation. Simultaneous efforts to rapidly control bleeding and reduce the detrimental effects of coagulopathy, hypothermia, and acidosis in these patients are extremely important. A MTP that includes the immediate availability of all blood components should be in place to provide optimal resuscitation for these patients, because extensive resources are required to provide these large quantities of blood. Appropriate administration of blood products has been shown to improve outcome in this patient population. Identification of the small subset of patients that benefit from this can be a challenge and several scores have been developed to assist the clinician in making the decision to initiate the MTP. None have been shown to be completely accurate. (See *Trauma Scores: Revised and Pediatric* and *ACS TQIP Massive Transfusion in Trauma Guidelines*.)

COAGULOPATHY

Severe injury and hemorrhage result in the consumption of coagulation factors and early coagulopathy. Such coagulopathy is present in up to 30% of severely injured patients on admission, in the absence of preexisting anticoagulant use. Massive fluid resuscitation with the resultant dilution of platelets and clotting factors, as well as the adverse effect of hypothermia on platelet

aggregation and the clotting cascade, contributes to coagulopathy in injured patients.

Prothrombin time, partial thromboplastin time, and platelet count are valuable baseline studies to obtain in the first hour, especially in patients with a history of coagulation disorders or who take medications that alter coagulation (also see Anticoagulation Reversal table in ***Chapter 6: Head Trauma***). These studies may also be useful in caring for patients whose bleeding history is unavailable. Point-of-care testing is available in many EDs. Thromboelastography (TEG) and rotational thromboelastometry (ROTEM) can be helpful in determining the clotting deficiency and appropriate blood components to correct the deficiency.

Some jurisdictions administer tranexamic acid in the prehospital setting to severely injured patients in response to recent studies that demonstrated improved survival when this drug is administered within 3 hours of injury. The first dose is usually given over 10 minutes and is administered in the field; the follow-up dose of 1 gram is given over 8 hours. (See ***Guidance Document Regarding the Pre-Hospital Use of Tranexamic Acid for Injured Patients.***)

In patients who do not require massive transfusion, the use of platelets, cryoprecipitate, and fresh-frozen plasma should be guided by coagulation studies, along with fibrinogen levels and balanced resuscitation principles. Of note, many newer anticoagulant and antiplatelet agents cannot be detected by conventional testing of PT, PTT, INR, and platelet count. Some of the oral anticoagulants have no reversal agents.

Patients with major brain injury are particularly prone to coagulation abnormalities. Coagulation parameters need to be closely monitored in these patients; early administration of plasma or clotting factors and/or platelets improves survival if they are on known anticoagulants or antiplatelet agents.

CALCIUM ADMINISTRATION

Most patients receiving blood transfusions do not need calcium supplements. When necessary, calcium administration should be guided by measurement of ionized calcium. Excessive, supplemental calcium can be harmful.

SPECIAL CONSIDERATIONS

Special considerations in diagnosing and treating shock include the mistaken use of blood pressure as a direct measure of cardiac output. The response of elderly patients, athletes, pregnant patients, patients on medications, hypothermic patients, and patients with pacemakers or implantable cardioverter-defibrillators (ICDs) may differ from the expected.

PITFALL	PREVENTION
Uncontrolled blood loss can occur in patients taking antiplatelet or anticoagulant medications.	• Obtain medication list as soon as possible. • Administer reversal agents as soon as possible. • Where available, monitor coagulation with thromboelastography (TEG) or rotational thromboelastometry (ROTEM). • Consider administering platelet transfusion, even with normal platelet count.
Thromboembolic complications can occur from agents given to reverse anticoagulant and antiplatelet medications.	• Weigh the risk of bleeding with the risk of thromboembolic complications. • Where available, monitor coagulation with TEG or ROTEM.

EQUATING BLOOD PRESSURE TO CARDIAC OUTPUT

Treatment of hemorrhagic shock requires correction of inadequate organ perfusion by increasing organ blood flow and tissue oxygenation. Increasing blood flow requires an increase in cardiac output. Ohm's law ($V = I \times R$) applied to cardiovascular physiology states that blood pressure (V) is proportional to cardiac output (I) and systemic vascular resistance (R; afterload). An increase in blood pressure should not be equated with a concomitant increase in cardiac output or recovery from shock. For example, an increase in peripheral resistance with vasopressor therapy, with no change in cardiac output, results in increased blood pressure but no improvement in tissue perfusion or oxygenation.

ADVANCED AGE

In the cardiovascular system, the aging process produces a relative decrease in sympathetic activity.

This is thought to result from a deficit in the receptor response to catecholamines, rather than reduced production of catecholamines. Cardiac compliance decreases with age, and unlike younger patients, older patients are unable to increase their heart rate or the efficiency of myocardial contraction when stressed by blood volume loss.

Atherosclerotic vascular occlusive disease makes many vital organs extremely sensitive to even the slightest reduction in blood flow. In addition, many elderly patients have preexisting volume depletion resulting from long-term diuretic use or subtle malnutrition. For these reasons, elderly trauma patients exhibit poor tolerance to hypotension secondary to blood loss. For example, a systolic blood pressure of 100 mm Hg may represent shock in an elderly patient. ß-adrenergic blockade can mask tachycardia as an early indicator of shock, and other medications can adversely affect the stress response to injury or block it completely. Because the therapeutic range for volume resuscitation is relatively narrow in elderly patients, consider using early advanced monitoring to avoid excessive or inadequate volume restoration.

Reduced pulmonary compliance, decreased diffusion capacity, and general weakness of the muscles of respiration limit elderly patients' ability to meet increased demands for gas exchange imposed by injury. This compounds the cellular hypoxia already produced by a reduction in local oxygen delivery. Glomerular and tubular senescence in the kidney reduces elderly patients' ability to preserve volume in response to the release of stress hormones such as aldosterone, catecholamines, vasopressin, and cortisol. The kidney is also more susceptible to the effects of reduced blood flow, and nephrotoxic agents such as drugs, contrast agents, and the toxic products of cellular destruction can further decrease renal function.

For all of these reasons, the mortality and morbidity rates increase directly with age. Despite adverse effects of the aging process, comorbidities from preexisting disease, and general reduction in the "physiologic reserve" of geriatric patients, most of these patients may recover and return to their preinjury status. Treatment begins with prompt, aggressive resuscitation and careful monitoring. (See *Chapter 11: Geriatric Trauma*.)

ATHLETES

Rigorous athletic training routines change the cardiovascular dynamics of this group of patients. Blood volume may increase 15% to 20%, cardiac output can increase 6-fold, stroke volume can increase 50%, and the resting pulse can average 50 BPM. Highly trained athletes' bodies have a remarkable ability to compensate for blood loss, and they may not manifest the usual responses to hypovolemia, even with significant blood loss.

PREGNANCY

The normal hypervolemia that occurs with pregnancy means that it takes a greater amount of blood loss to manifest perfusion abnormalities in the mother, which also may be reflected in decreased fetal perfusion. (See *Chapter 12: Trauma in Pregnancy and Intimate Partner Violence*.)

MEDICATIONS

Specific medications can affect a patient's response to shock. For example, ß-adrenergic receptor blockers and calcium channel blockers can significantly alter a patient's hemodynamic response to hemorrhage. Insulin overdosing may be responsible for hypoglycemia and may have contributed to the injury-producing event. Long-term diuretic therapy may explain unexpected hypokalemia, and nonsteroidal anti-inflammatory drugs (NSAIDs) may adversely affect platelet function and increase bleeding.

HYPOTHERMIA

Patients suffering from hypothermia and hemorrhagic shock do not respond as expected to the administration of blood products and fluid resuscitation. In hypothermia, coagulopathy may develop or worsen. Body temperature is an important vital sign to monitor during the initial assessment phase. Esophageal or bladder temperature is an accurate clinical measurement of the core temperature. A trauma victim under the influence of alcohol and exposed to cold temperatures is more likely to have hypothermia as a result of vasodilation. Rapid rewarming in an environment with appropriate external warming devices, heat lamps, thermal caps, heated respiratory gases, and warmed intravenous fluids and blood will generally correct hypotension and mild to moderate hypothermia. Core rewarming techniques includes irrigation of the peritoneal or thoracic cavity with crystalloid solutions warmed to 39°C (102.2°F); for severe hypothermia, extracorporeal bypass is indicated. Hypothermia is best treated by prevention. (See *Appendix B: Hypothermia and Heat Injuries*.)

PRESENCE OF PACEMAKER OR IMPLANTABLE CARDIOVERTER-DEFIBRILLATOR

Patients with pacemakers or ICDs with pacemakers are unable to respond to blood loss as expected, because cardiac output is directly related to heart rate. Heart rate may remain at the device's set rate regardless of volume status in these patients. In a significant number of patients with myocardial conduction defects who have such devices in place, additional monitoring may be required to guide fluid therapy. Many devices can be adjusted to increase heart rate if clinically indicated.

REASSESSING PATIENT RESPONSE AND AVOIDING COMPLICATIONS

Inadequate volume replacement is the most common complication of hemorrhagic shock. Patients in shock need immediate, appropriate, and aggressive therapy that restores organ perfusion.

CONTINUED HEMORRHAGE

An undiagnosed source of bleeding is the most common cause of poor response to fluid therapy. These patients, also classed as transient responders, require persistent investigation to identify the source of blood loss. Immediate surgical intervention may be necessary.

MONITORING

The goal of resuscitation is to restore organ perfusion and tissue oxygenation. This state is identified by appropriate urinary output, CNS function, skin color, and return of pulse and blood pressure toward normal. Monitoring the response to resuscitation is best accomplished for some patients in an environment where sophisticated techniques are used. For elderly patients and patients with non-hemorrhagic causes of shock, consider early transfer to an intensive care unit or trauma center.

RECOGNITION OF OTHER PROBLEMS

When a patient fails to respond to therapy, causes may include one or more of the following: undiagnosed bleeding, cardiac tamponade, tension pneumothorax, ventilatory problems, unrecognized fluid loss, acute gastric distention, myocardial infarction, diabetic acidosis, hypoadrenalism, or neurogenic shock. Constant reevaluation, especially when a patient's condition deviates from expected patterns, is the key to recognizing and treating such problems as early as possible.

TEAMWORK

One of the most challenging situations a trauma team faces is managing a trauma victim who arrives in profound shock. The team leader must direct the team decisively and calmly, using ATLS principles.

Identifying and controlling the site of hemorrhage with simultaneous resuscitation involves coordinating multiple efforts. The team leader must ensure that rapid intravenous access is obtained even in challenging patients. The decision to activate the massive transfusion protocol should be made early to avoid the lethal triad of coagulopathy, hypothermia, and acidosis. The team must be aware of the amount of fluid and blood products administered, as well as the patient's physiological response, and make necessary adjustments.

The team leader ensures that the areas of external hemorrhage are controlled and determines when to perform adjuncts such as chest x-ray, pelvic x-ray, FAST, and/or diagnostic peritoneal lavage (DPL). Decisions regarding surgery or angioembolization should be made as quickly as possible and the necessary consultants involved. When required services are unavailable, the trauma team arranges for rapid, safe transfer to definitive care.

CHAPTER SUMMARY

1. Shock is an abnormality of the circulatory system that results in inadequate organ perfusion and tissue oxygenation.

2. Hemorrhage is the cause of shock in most trauma patients. Treatment of these patients requires immediate hemorrhage control and fluid or blood replacement. Stop the bleeding.

3. Diagnosis and treatment of shock must occur almost simultaneously.

4. Initial assessment of a patient in shock requires careful physical examination, looking for signs of

tension pneumothorax, cardiac tamponade, and other causes of shock.

5. Management of hemorrhagic shock includes rapid hemostasis and balanced resuscitation with crystalloids and blood.

6. The classes of hemorrhage and response to interventions serve as a guide to resuscitation.

7. Special considerations in diagnosis and treatment of shock include differences in the response to shock in extremes of age, athleticism, pregnancy, hypothermia, and presence of some medications and pacemakers/ICDs. Avoid the pitfall of equating blood pressure with cardiac output.

ADDITIONAL RESOURCES

The STOP the Bleeding Campaign
Rossaint et al. *Critical Care* 2013;17(2):136
http://ccforum.com/content/17/2/136

ACS TQIP Massive Transfusion in Trauma Guidelines
https://www.facs.org/~/media/files/quality%20programs/trauma/tqip/massive%20transfusion%20in%20trauma%20guildelines.ashx

Management of Bleeding and Coagulopathy Following Major Trauma: An Updated European Guideline
Spahn et al. *Critical Care* 2013;17(2):R76
http://ccforum.com/content/17/2/R76

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