Chapter 3: Shock

Objectives:

Upon completion of this topic, the physician will be able to identify and apply principles of management related to the initial diagnosis and treatment of shock in the injured patient. Specifically, the physician will be able to:

A. Define shock.

B. Discuss the similarities and differences in the various etiologies and presentations of shock.

C. Recognize the clinical shock syndrome and correlate the patient's acute clinical signs with the degree of volume deficit.

D. Discuss the basic principles of emergency treatment of hemorrhagic shock and their application based on the patient's clinical response to therapy.

E. Discuss fluid management problems unique to the trauma patient.

F. Demonstrate various techniques of central and peripheral vascular access, including cutdowns and intraosseous insertion.

I. Introduction

The initial step in managing shock in the injured patient is to recognize its presence. No laboratory tests immediately diagnose shock. The initial diagnosis is based on clinical appreciation of the presence of inadequate organ perfusion and tissue oxygenation. Thus, the definition of shock as an abnormality of the circulatory system that results in inadequate organ perfusion and tissue oxygenation also becomes an operative tool for diagnosis and treatment.

The second step in the initial management of shock is to identify the probable cause of the shock state. For the trauma patient this identification process is directly related to the mechanism of injury. All types of shock may be present in the trauma patient. Most injured patients in shock are hypovolemic, but cardiogenic shock or tension pneumothorax may be the cause and must be considered in patients with specific injuries above the diaphragm. The clinical situations that make this problem more likely are discussed later in this chapter. Neurogenic shock results from extensive injury to the central nervous system or the spinal cord. For all practical purposes, shock does not result from isolated head injuries. Spinal cord injury victims may initially present in shock from both vasodilatation and relative hypovolemia. Septic shock is unusual but must be considered for patients whose arrival at the emergency facility has been greatly delayed.

The physician's management responsibilities begin first with recognizing the presence of the shock state. Initiating treatment should be simultaneous with the identification of a probable cause of the shock state. The response to initial treatment, coupled with the findings
on initial and secondary patient survey, usually provides enough information to determine the cause of the shock state. Hemorrhage is the most common cause of the shock state in the injured patient.

Three components of cardiac physiology are classically defined in shock as: (1) preload, (2) pump, and (3) afterload. Preload is equated with venous capacitance and volume status. Myocardial contractility is the pump that drives the system. Afterload is systemic (peripheral) vascular resistance. The initial treatment of shock is directed toward restoring cellular and organ perfusion with adequately oxygenated blood. In hemorrhagic shock this means increasing preload or restoring adequate circulating blood volume rather than merely restoring the patient's blood pressure and pulse rate. Therefore, vasopressors are contraindicated for the treatment of hemorrhagic shock.

A significant percentage of injured patients who are in hypovolemic shock require surgical intervention, and many of these require early intervention to relieve the shock. Therefore, the presence of shock in an injured patient demands the immediate involvement of a qualified surgeon.

II. Initial Patient Assessment

A. Recognition of Shock

Full-blown circulatory shock, evidenced by inadequate perfusion of the skin, kidneys, and central nervous system, is easy to recognize. However, after the airway and breathing are evaluated, careful evaluation of the patient's circulatory status is important to identify earlier stages of shock. Sole reliance on systolic blood pressure as an indicator of shock results in a delayed recognition of the shock state. Remember, compensatory mechanisms may preclude a measurable fall in systolic pressure until the patient has lost up to 30% of his blood volume. Specific attention should be directed to pulse rate, respiratory rate, skin circulation, and pulse pressure (the difference between systolic and diastolic pressure). The earliest signs of shock are tachycardia and cutaneous vasoconstriction. Accordingly, any injured patient who is cool and tachycardic is in shock until proven otherwise. The norma heart rate varies with age. Tachycardia is present when the heart rate is greater than 160 in an infant, 140 in a preschool age child, 120 from school age to puberty, and 100 in an adult. The elderly patient may not exhibit tachycardia because of the limited cardiac response to catecholamine stimulation or certain medications such as propranolol. A narrowed pulse pressure suggests significant blood loss and involvement of compensatory mechanisms.

Use of the hematocrit (or hemoglobin concentration) is unreliable and inappropriate for estimating acute blood loss or diagnosing shock. Massive blood loss may produce a minimal acute decrease in hematocrit. Thus, a very low hematocrit suggests significant blood loss or pre-existing anemia, while a normal hematocrit does not rule out significant blood loss.
B. Clinical Differentiation of Etiology of Shock

1. Hemorrhagic shock

Hemorrhage is the most common cause of shock after injury, and virtually all multiply injured patients have an element of hypovolemia. In addition, most nonhemorrhagic shock states respond partially or briefly to volume resuscitation. Therefore, once the shock state is identified, treatment usually is begun as if the patient were hypovolemic. However, as treatment is instituted, it is important to identify the small number of patients whose shock has been caused by some other etiology, and the larger group of patients for whom a secondary factor complicates their hypovolemic/hemorrhagic shock. The specifics of treatment of hemorrhagic shock are covered in greater detail in the next section of this chapter. This section focuses on differentiating other potential etiologies of shock.

The major differentiating factor in identifying the cause of shock in a trauma patient is whether the condition is hemorrhagic or nonhemorrhagic. This is especially true for a patient with injuries above the diaphragm, when cardiogenic shock and tension pneumothorax may be a potential cause of the shock state. A high index of suspicion and careful observation of the patient's response to initial treatment should enable the physician to recognize and manage all forms of shock. The initial determination of the etiology depends on an appropriate history, a careful physical examination, and selected additional tests.

2. Nonhemorrhagic shock

a. Cardiogenic shock

Myocardial dysfunction may occur from myocardial contusion, cardiac tamponade, air embolus, or rarely a myocardial infarction associated with the patient's injury. Cardiac contusion is not uncommon in rapid deceleration blunt trauma to the thorax. All patients with blunt trauma need constant ECG monitoring to detect injury patterns and dysrhythmias. Blood CPK-isoenzymes and specific isotope studies of the myocardium rarely have any value in diagnosing or managing the patient in the emergency department. Ultrasound can be useful in the diagnosis of tamponade or valvular rupture, but it is often not practical or immediately available in the emergency department. Myocardial contusion may be an indication for early central venous pressure monitoring of fluid resuscitation in the emergency department.

Cardiac tamponade is most common in penetrating thoracic trauma. It can occur rarely in blunt trauma to the thorax. Tachycardia, muffled heart sounds, and dilated, engorged neck veins with hypotension resistant to fluid therapy suggest cardiac tamponade. Tension pneumothorax may mimic cardiac tamponade. Appropriate placement of a needle temporarily relieves these two life-threatening conditions.

b. Tension pneumothorax

Tension pneumothorax develops when a flap valve leak allows air to enter the pleural space but 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 fall in cardiac output. Tension pneumothorax is a true surgical emergency, requiring
immediate diagnosis and treatment. The presence of subcutaneous emphysema, absent breath sounds, hyperresonance to percussion, tracheal shift, and acute respiratory distress make the diagnosis and warrant thoracic decompression without waiting for roentgenographic confirmation.

c. Neurogenic shock

**Isolated head injuries do not cause shock.** The presence of shock in a patient with a head injury necessitates a search for another cause of shock. Spinal cord injury may produce hypotension due to loss of sympathetic tone. **Remember**, loss of sympathetic tone compounds the physiologic effects of hypovolemia, and hypovolemia compounds the physiologic effects of sympathetic denervation. The classic picture of neurogenic shock is hypotension without tachycardia or cutaneous vasoconstriction. A narrowed pulse pressure is not seen in neurogenic shock. Patients sustaining a spinal injury often have concurrent torso trauma. Therefore, patients with known or suspected neurogenic shock should be treated initially for hypovolemia. Failure to restore perfusion or blood pressure with fluid resuscitation may indicate either continuing hemorrhage or neurogenic shock. Central venous pressure monitoring may be helpful in managing this sometimes complex problem. (See Chapter 7, Spine and Spinal Cord Trauma.)

d. Septic shock

Shock due to infection immediately after injury is uncommon. However, if the patient's arrival at the emergency facility is delayed for several hours, this problem may occur. Septic shock is particularly likely to occur in patients with penetrating abdominal injuries and contamination of the peritoneal cavity with intestinal contents. The volume status of the patient in septic shock is clinically significant. Septic patients who are hypovolemic are difficult to distinguish clinically from those in hypovolemic shock (tachycardia, cutaneous vasoconstriction, impaired urinary output, decreased systolic pressure, narrow pulse pressure). Patients with sepsis and normal or nearly normal circulating volume may have a modest tachycardia, warm pink skin, a systolic pressure near normal, and wide pulse pressure.

III. Hemorrhagic Shock in the Injured Patient

**Hemorrhage is the most common cause of shock in the trauma patient.** However, the trauma patient's response to blood loss is made more complex by changes in the body fluids (particularly in the extracellular fluid) that impacts on the circulating blood volume and cellular function. The classic response to blood loss must be considered in the context of the fluid changes associated with soft-tissue injury and the changes associated with severe, prolonged shock, and the pathophysiologic results of resuscitation and reperfusion.

A. Pathophysiology

Early circulatory responses to blood loss are compensatory, ie, **progressive vasoconstriction** of cutaneous, muscle, and visceral circulation to preserve blood flow to the kidneys, heart, and brain. **Tachycardia** is the earliest measurable circulatory sign.
At the cellular level, inadequately perfused and oxygenated cells initially compensate by shifting to anaerobic metabolism, which further results in the formation of lactic acid and the development of metabolic acidosis. If shock is prolonged, the cellular membrane loses its ability to maintain the normal electrical gradient, and cellular swelling occurs, leading to cellular damage and death, and tissue swelling. This process compounds the overall impact on blood loss and hypoperfusion. The administration of isotonic electrolyte solutions helps combat this process. Therefore, management is directed toward reversing this phenomenon with adequate oxygenation, ventilation, and appropriate fluid resuscitation. Resuscitation may be accompanied by a marked increase in interstitial edema, the result of the "reperfusion injury" to the capillary-interstitial membrane. This may result in larger volumes of resuscitation fluid than initially anticipated.

### B. Definition of Hemorrhage

Hemorrhage is defined as an **acute loss of circulating blood**. Although there is considerable variability, the normal adult blood volume is approximately 7% of body weight. For example: a 70-kilogram male has approximately five liters of circulating blood volume. 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. For children, the blood volume is calculated at 8% to 9% of the body weight (80 to 90 mL/kg). (See Chapter 10, Pediatric Trauma.)

### C. Direct Effects of Hemorrhage

Classes of hemorrhage, based on percentage of acute blood volume loss, are outlined individually in this chapter for the **purposes of teaching and comprehending** the physiologic and clinical manifestations of hemorrhagic shock. The distinction between classes is not always so apparent in any single trauma patient, and **treatment should be directed more by the response to initial therapy than relying solely on the initial classification**. Nevertheless, this classification system is useful in emphasizing the early signs and physiology of the shock state. **Class I** is exemplified by the condition of the blood donor. **Class II** is uncomplicated shock, but crystalloid fluid resuscitation is required. **Class III** is a complicated state in which at least crystalloid and perhaps blood replacement are required. **Class IV** can be considered as a preterminal event, and unless very aggressive measures are taken, the patient dies within minutes. (See Table 1, Estimated Fluid and Blood Losses.)

All medical personnel involved in the initial assessment and resuscitation of the patient in hemorrhagic shock must quickly recognize important factors that may accentuate or diminish the patient's physiologic response. Important factors that may profoundly alter the classic vascular dynamics include: (1) the patient's age; (2) severity of injury with special attention to type and anatomical location of injury; (3) time lapse between injury concurrence and initiation of treatment; and (4) prehospital fluid therapy and application of the pneumatic antishock garment (PASG).

**It is dangerous to wait until the trauma patient fits a precise physiologic classification of shock before initiating aggressive therapy.** Aggressive fluid resuscitation must be initiated when early signs and symptoms of blood loss are apparent or suspected, not when the blood pressure is falling or absent.
1. Loss of up to 15% - Class I Hemorrhage

The clinical symptoms of this volume loss 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. Transcapillary refill and other compensatory mechanisms restore blood volume within 24 hours. However, in the presence of other fluid changes, this amount of blood loss can produce clinical symptoms. Replacement of the primary fluid losses corrects the circulatory state.

2. 15% to 30% blood volume loss - Class II Hemorrhage

In a 70-kilogram male, this volume loss represents 750 to 1500 mL of blood. Clinical symptoms include tachycardia (heart rate above 100 in an adult), tachypnea, and a decrease in pulse pressure (the difference between the systolic and diastolic pressures). This decrease in pulse pressure is primarily related to a rise in the diastolic component. (The main reason for the rise in diastolic pressure is an elevation in catecholamines which produces an increase in peripheral resistance.) Because the systolic pressure changes minimally in early hemorrhagic shock, it is important to evaluate the pulse pressure rather than the systolic pressure. Other pertinent clinical findings with this degree of blood loss include subtle central nervous system changes (anxiety, which may be expressed as fright or hostility). Notably, despite the significant blood loss and cardiovascular changes, urinary output is only mildly affected (the measured flow is usually 20 to 30 mL per hour).

Again, accompanying fluid losses can compound the clinical expression of this amount of blood loss. The majority of such patients may eventually require blood transfusion, but can be stabilized initially with other replacement fluids.

3. 30% to 40% blood volume loss - Class III Hemorrhage

This amount of blood loss (approximately 2000 mL in an adult) can be devastating. Patients almost always present with the classic signs of inadequate perfusion, including marked tachycardia and tachypnea, significant changes in mental status, and a measurable fall in systolic pressure. Note that in an uncomplicated case, this is the smallest amount of blood loss that consistently causes a drop in systolic pressure. Although patients with this degree of blood loss almost always require transfusion, remember that these symptoms can result from lesser degrees of blood loss combined with other fluid losses. Thus, the decision to transfuse is based on the patient's response to initial fluid resuscitation and evidence of end-organ perfusion and oxygenation, as described later in this chapter.

4. More than 40% blood volume loss - Class IV Hemorrhage

This degree of exsanguination is immediately life-threatening. Symptoms include marked tachycardia, a significant depression in systolic blood pressure, and a very narrow pulse pressure (or an unobtainable diastolic pressure). Urinary output is negligible, and mental status is markedly depressed. The skin is cold and pale. Such patients 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. Loss of more than
50% of the patient's blood volume results in loss of consciousness, pulse, and blood pressure.

**D. Fluid Changes Secondary to Soft-Tissue Injury**

Major soft-tissue injuries and fractures compound the circulatory status of the injured patient in two ways. First, blood is frequently lost into the site of injury, particularly in cases of major fractures. For instance, a fractured tibia or humerus may be associated with as much as a unit and a half (750 mL) blood loss. Twice that amount (up to 1500 mL) is commonly associated with femur fractures, and several liters of blood may accumulate in a retroperitoneal hematoma associated with a pelvic fracture.

The second factor to be considered is the obligatory edema that occurs in injured soft tissues. This condition is related to the magnitude of soft-tissue injury and consists of extracellular fluid. Because the plasma is part of the extracellular fluid, these changes have a significant impact on circulating blood volume. For instance, the two liters of edema that may be associated with a massive femur fracture may consist of 1500 mL of interstitial fluid and only 500 mL of plasma volume. In general, roughly 25% of such fluid translocation is evidenced by a decrease in the plasma volume. The impact of these changes on circulating blood and the reason they compound fluid loss then becomes obvious.

**IV. Initial Management of Hemorrhagic Shock**

As in many emergency situations, diagnosis and treatment must be performed in rapid succession. For most trauma patients, treatment is instituted as if the patient had hypovolemic shock, unless evidence to the contrary is clear.

**A. Physical Examination**

The physical examination is directed at the immediate diagnosis of life-threatening injuries and includes assessment of the ABCs. Baseline recordings are important to the subsequent monitoring of the patient. Vital signs, urinary output, and level of consciousness are important. A more detailed examination of the patient follows as the situation permits.

1. **Airway and breathing**

   Establishing a patent airway with adequate ventilatory exchange and oxygenation is the first priority. Supplementary oxygen via a bag-valve-mask reservoir system is delivered to maintain arterial oxygen tension between 80 and 100 mm Hg.

2. **Circulation - Hemorrhage control**

   Priorities include: Control obvious hemorrhage; obtain adequate intravenous access; and access tissue perfusion. Bleeding from external wounds usually can be controlled by direct pressure to the bleeding site, eg, scalp, neck, and upper and lower extremities. The pneumatic antishock trousers may be used to control bleeding from pelvic or lower extremity fractures, but the device should not interfere with rapid re-establishment of intravascular volume by the intravenous route. The adequacy of tissue perfusion dictates the amount of fluid resuscitation required.
3. Disability - Neurologic examination

A brief neurologic examination determining the level of consciousness, eye motion and pupillary response, motor function, and degree of sensation is performed. These data are useful in assessing cerebral perfusion, following the evolution of neurologic disability, and prognosticating future recovery. (See Chapter 6, Head Trauma.)

4. Exposure - Complete examination

After addressing the life-saving priorities, the patient must be completely undressed and carefully examined from "head to toe" as part of the search for associated injuries. The performance of other diagnostic and therapeutic maneuvers is part of this secondary survey. Remember, when undressing the patient, preventing iatrogenic hypothermia is essential.

5. Gastric dilatation - Decompression

Gastric dilatation often occurs in the trauma patient and may cause unexplained hypotension. This condition makes shock difficult to treat, and in the unconscious patient, it represents a significant risk of aspiration - a potentially fatal complication. The physician's responsibility does not end with the passage of the gastric tube. The tube must be properly positioned, attached to appropriate suction, and be functioning.

6. Urinary catheter insertion

Bladder decompression allows for the assessment of urine for hematuria and constant monitoring of renal perfusion via the urinary output. Blood at the urethral meatus or a nonpalpable prostate in the male is a contraindication to inserting a transurethral catheter. (See Chapter 5, Abdominal Trauma.)

B. Vascular Access Lines

Access to the vascular system must be obtained promptly. This is best done by establishing two large-caliber (minimum of 16-gauge) peripheral intravenous catheters before any consideration is given to a central line. Poiseuille's Law states that flow is proportional to the fourth power of the radius of the cannula, and inversely related to its length. Hence, short, large-caliber peripheral IVs are preferred to infuse large volumes of fluid rapidly. The most desirable sites for peripheral intravenous lines in adults are: (1) percutaneous peripheral access via the forearm or antecubital veins, and (2) cutdown on the saphenous or arm veins. If circumstances prevent the use of peripheral veins, large-caliber, central venous access is indicated, using the Seldinger technique. In children younger than six years, intraosseous needle access should be attempted before central line insertion. The important determinant for selecting a procedure and route for establishing vascular access is the level of the physician's skill and experience.

As intravenous lines are started, blood samples are drawn for appropriate laboratory analyses, including type and crossmatch, toxicology studies, and pregnancy testing of all females of childbearing age. It also may be useful to obtain arterial blood gases at this time. A chest roentgenogram should be obtained after attempts at inserting a subclavian or internal
jugular central venous pressure monitoring line to document its position and evaluate for pneumothorax.

C. Initial Fluid Therapy

Isotonic electrolyte solutions are used for initial resuscitation. This type of fluid provides transient intravascular expansion and further stabilizes the vascular volume by replacing accompanying fluid losses into the interstitial and intracellular space. Ringer’s lactate solution is the initial fluid of choice. Normal saline is the second choice. Although normal saline is satisfactory replacement fluid in the volumes administered to injured patients, it has the potential to cause hyperchloremic acidosis. This potential is enhanced if renal function is impaired.

An initial fluid bolus is given as rapidly as possible. The usual dose one to two liters for an adult and 20 mL/kilogram for a pediatric patient. The patient's response is observed during this initial fluid administration, and further therapeutic and diagnosis decisions are based on this response.

The amount of fluid and blood required for resuscitation is difficult to predict on initial evaluation of the patient. Table 1, Estimated Fluid and Blood Losses, outlines general guidelines for establishing the amount and type of fluid and blood the patient probably requires. A rough guideline for the total amount of crystalloid volume acutely required is to replace each one milliliter of blood loss with three milliliters of crystalloid fluid, thus allowing for restitution of plasma volume lose into the interstitial and intracellular spaces. However, it is more important to assess the patient's response to fluid resuscitation and evidence of adequate end-organ perfusion and oxygenation. If, during resuscitation, the amount of fluid administered deviates widely from these estimates, a careful reassessment of the situation and a search for unrecognized injuries or other causes of shock are necessary.

V. Evaluation of Fluid Resuscitation and Organ Perfusion

A. General

The same signs and symptoms of inadequate perfusion that are used to diagnose shock are useful determinants of patient response. The return of normal blood pressure, pulse pressure, and pulse rate are positive signs and indicate that circulation is normalizing. However, these observations give no information regarding organ perfusion. Improvements in the central nervous system status and skin circulation are important evidence of enhanced perfusion, but are difficult to quantitate. The urinary output can be quantitated and the renal response to restoration of perfusion is reasonably sensitive (if not modified by diuretics). For this reason urinary output is one of the prime monitors of resuscitation and patient response. Changes in central venous pressure can provide useful information, and the risk of a central venous pressure line is justified for complex cases. Measurements of left heart function (obtained with a Swan-Ganz catheter) are rarely indicated for the management of the injured patient in the emergency department.
B. Urinary Output

Within certain limits, urinary output can be used as a monitor of renal blood flow. Adequate volume replacement should produce a urinary output of approximately **50 mL/hour in the adult.** One mL/kg/hour is an adequate urinary output for the pediatric patient. For children under one year of age, two mL/kg/hour should be maintained. Inability to obtain urinary output at these levels (or decreasing urinary output with an increasing specific gravity) suggests inadequate resuscitation. This situation should stimulate further volume replacement and diagnostic endeavors.

C. Acid/Base Balance

Patients in early hypovolemic shock have respiratory alkalosis due to tachypnea. Respiratory alkalosis gives way to mild metabolic acidosis in the early phases of shock and does not require treatment. Severe metabolic acidosis may develop from long-standing or severe shock. Metabolic acidosis is due to anaerobic metabolism resulting from inadequate tissue perfusion, and persistence is usually due to inadequate fluid resuscitation. Persistent acidosis in the normothermic shock patient should be treated with increased fluids and not intravenous sodium bicarbonate, unless the pH is less than 7.2.

**Table 1. Estimated Fluid and Blood Losses**\(^1\) Based on Patient's Initial Presentation

<table>
<thead>
<tr>
<th></th>
<th>Class I</th>
<th>Class II</th>
<th>Class III</th>
<th>Class IV</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Blood Loss (mL)</strong></td>
<td>Up to 750</td>
<td>750-150</td>
<td>1500-200</td>
<td>&gt; 2000</td>
</tr>
<tr>
<td><strong>Blood Loss (%BV)</strong></td>
<td>Up to 15%</td>
<td>15-30%</td>
<td>30-40%</td>
<td>&gt; 40%</td>
</tr>
<tr>
<td><strong>Pulse Rate</strong></td>
<td>&lt; 100</td>
<td>&gt; 100</td>
<td>&gt; 120</td>
<td>&gt; 140</td>
</tr>
<tr>
<td><strong>Blood Pressure (mm Hg)</strong></td>
<td>Normal</td>
<td>Normal</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td><strong>Pulse Pressure</strong></td>
<td>Normal or increased</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td><strong>Respiratory Rate</strong></td>
<td>14-20</td>
<td>20-30</td>
<td>30-40</td>
<td>&gt; 35</td>
</tr>
<tr>
<td><strong>Urinary Output (mL/hr)</strong></td>
<td>&gt; 30</td>
<td>20-30</td>
<td>5-15</td>
<td>Negligible</td>
</tr>
<tr>
<td><strong>CNS/Mental Status</strong></td>
<td>Slightly anxious</td>
<td>Mildly anxious</td>
<td>Anxious and confused</td>
<td>Confused and lethargic</td>
</tr>
<tr>
<td><strong>Fluid Replacement (3:1 Rule)</strong></td>
<td>Crystalloid</td>
<td>Crystalloid</td>
<td>Crystalloid and blood</td>
<td>Crystalloid and blood</td>
</tr>
</tbody>
</table>

\(^1\) For a 70-kg male.

The guidelines in Table 1 are based on the "three-for-one" rule. This rule derives from the empiric observation that most patients in hemorrhagic shock require as much as 300 mL of electrolyte solution for each 100 mL of blood loss. Applied blindly, these guidelines can result in excessive or inadequate fluid administration. For example, a patient with a crush injury to the extremity may have hypotension out of proportion to his blood loss and require fluids in excess of the 3:1 guideline. In contrast, a patient whose ongoing blood loss is being replaced requires less than 3:1. The use of bolus therapy with careful monitoring of the patient's response can moderate these extremes.
VI. Therapeutic Decisions Based on Response to Initial Fluid Resuscitation

The patient's response to initial fluid resuscitation is the key to determining subsequent therapy. (See Table 2, Responses to Initial Fluid Resuscitation.) Having established a preliminary diagnosis and plan based on the initial evaluation of the patient, the physician can now modify management based on the patient's response to the initial fluid resuscitation. Observing the response to the initial resuscitation identifies those patients whose blood loss was greater than estimated and those with ongoing bleeding. In addition, it limits the probability of overtransfusion or unneeded transfusion of blood in those whose initial status was disproportionate to the amount of blood loss. It is particularly important to distinguish the patient who is "hemodynamically stable" from one who is "hemodynamically normal." A hemodynamically stable patient may be persistently tachycardic, tachypneic, and oliguric, clearly remaining underperfused and underresuscitated. In contrast, the hemodynamically normal patient is one who exhibits no signs of inadequate tissue perfusion. The potential response patterns can be discussed in three groups.

A. Rapid Response to Initial Fluid Administration

A small group of patients respond rapidly to the initial fluid bolus and remain stable and hemodynamically normal when the initial fluid bolus has been completed and the fluids are slowed to maintenance rates. Such patients usually have lost minimal (less than 20%) blood volume. No further fluid bolus or immediate blood administration is indicated for this small group of patients. Type and crossmatched blood should be kept available. Surgical consultation and evaluation are necessary during initial assessment and treatment.

B. Transient Response to Initial Fluid Administration

The largest group of patients responds to the initial fluid bolus. However, in some patients, as the initial fluids are slowed, the circulatory perfusion indices may begin to show deterioration, indicating either an ongoing blood loss or inadequate resuscitation. Most of these patients initially have lost an estimated 20% to 40% of their blood volume. Continued fluid administration and initiation of blood administration are indicated. The response to blood administration should identify patients who are still bleeding and require rapid surgical intervention.

C. Minimal or No Response to Initial Fluid Administration

This response is seen in a small but significant percentage of injured patients. For most of these patients, failure to respond to adequate crystalloid and blood administration in the emergency department dictates the need for immediate surgical intervention to control exsanguinating hemorrhage. On very rare occasions, failure to respond may be due to pump failure as a result of myocardial contusion or cardiac tamponade. The possible diagnosis of nonhemorrhagic shock always should be entertained in this group of patients. Central venous pressure monitoring helps differentiate between the various shock etiologies.
VII. Blood Replacement

The decision to begin transfusion is based on the patient's response, as described in the previous section.

A. Packed Red Blood Cells versus Whole Blood Therapy

Either whole blood or packed red blood cells can be used to resuscitate the trauma patient. However, in an effort to maximize blood product availability, most blood centers currently provide component therapy (packed cells, platelets, fresh frozen plasma, etc). The main purpose in transfusing blood is to restore the oxygen-carrying capacity of the intravascular volume. Volume resuscitation itself can be accomplished with crystalloids, with the added advantage of contributing to interstitial and intracellular volume restitution.

Table 2. Responses to Initial Fluid Resuscitation*

<table>
<thead>
<tr>
<th>Vital Signs</th>
<th>Rapid Response</th>
<th>Transient Response</th>
<th>No Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Return to normal</td>
<td>Transient improvement; recurrence of low BP and high HR</td>
<td>Remain abnormal</td>
<td></td>
</tr>
<tr>
<td>Estimated Blood Loss</td>
<td>Minimal (10-20%)</td>
<td>Moderate and ongoing (20-40%)</td>
<td>Severe (&gt; 40%)</td>
</tr>
<tr>
<td>Need for More Crystalloid</td>
<td>Low</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>Need for Blood Preparation</td>
<td>Low</td>
<td>Moderate - high Type-specific</td>
<td>Immediate Emergency blood release</td>
</tr>
<tr>
<td>Type and crossmatch</td>
<td></td>
<td>Type-specific</td>
<td></td>
</tr>
<tr>
<td>Need for Operative Intervention</td>
<td>Possibly</td>
<td>Likely</td>
<td>Highly likely</td>
</tr>
<tr>
<td>Surgical Consultation</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

* 2000 mL Ringer's lactate in adults, 20 mL/kg Ringer's lactate in children, over 10 to 15 minutes.

B. Crossmatched, Type-specific and Type O Blood

1. Fully crossmatched blood is preferable. However, the complete crossmatching procedure requires approximately one hour in most blood banks. For patients who stabilize rapidly, crossmatched blood should be obtained and should be available for transfusion when indicated.

2. Type-specific or "saline crossmatched" blood can be provided by most blood banks within ten minutes. Such blood is compatible with ABO and Rh blood types. Incompatibilities of minor antibodies may exist. Such blood is of first choice for patients with life-threatening shock situations, such as transient responders described in previous section.
3. If type-specific blood is unavailable, type O packed cells are indicated for patients with exsanguinating hemorrhage. For life-threatening blood loss, the use of unmatched, type-specific blood is preferred over type O blood, unless multiple unidentified casualties are being treated simultaneously. To avoid sensitization and future complications, Rh-negative cells are preferable, particularly for females of childbearing age.

C. Blood Filters

Macropore (160 microns) intravenous filtering devices are used when whole blood transfusions are given. These filters remove microscopic clots and debris. The use of micropore blood filters has not been demonstrated to be of significant value.

D. Warming Fluids - Plasma and Crystalloid

Iatrogenic hypothermia in the resuscitation phase of trauma patients can and must be prevented. The use of blood warmers is cumbersome yet most desirable in the emergency department. The most efficient and easiest way to prevent hypothermia in any patient receiving massive volumes of crystalloid is to heat the fluid to 39 degrees centigrade before using it. Blood, plasma, and glucose-containing solutions cannot be warmed in the microwave oven.

E. Autotransfusion

Adaptation of standard tube thoracostomy collection devices are commercially available to allow for the sterile collection, anticoagulation (generally with sodium-citrate solutions, not heparin), and retransfusion of shed blood. Collection of shed blood for autotransfusion should be considered for any major hemothorax. Equipment to collect, wash, and retransfuse blood lost during operative procedures also is commercially available. Bacterial contamination may limit the usefulness of such devices during many operations.

F. Coagulopathy

Coagulopathy is a rare problem in the first hour of treatment of the multiply injured patient. Hypothermia and massive transfusion with resultant dilution of platelets and clotting factors are the usual causes of coagulopathies in the injured patient. Prothrombin time, partial thromboplastin time, and platelet count are valuable baseline studies to obtain in the first hour, especially if the patient has a history of coagulation disorders or takes medications that alter coagulation. Transfusion of platelets, cryoprecipitate, and fresh frozen plasma should be guided by these coagulation parameters, including fibrinogen. Routine use of such products is generally not warranted.

G. Calcium Administration

Most patients receiving blood transfusions do not need calcium supplements. Excessive, supplemental calcium may be harmful.
VIII. Pneumatic Antishock Garment (PASG)

The application of the PASG can raise systolic pressure by increasing peripheral vascular resistance and myocardial afterload. The efficacy of PASG in hospital or in the rural setting remains unproven, and in the urban prehospital setting, controversial.

A. Indications

Current indications for use of the PASG are:

1. Splinting and control of pelvic fractures with continuing hemorrhage and hypotension

2. Intra-abdominal trauma with severe hypovolemia in patients who are en route to the operating room or another facility.

B. Contraindications

1. Pulmonary edema

2. Known diaphragmatic rupture

3. Uncontrolled hemorrhage outside the confines of the garment (ie, thoracic, upper extremity, scalp, face, or neck injury).

C. Dangers

The use of the PASG must not delay volume replacement or rapid transport. Prolonged inflation of the leg components of the PASG can result in compartment syndrome, particularly in the patient in shock with extremity trauma. Precise time of garment inflation must be recorded. If inflation of the abdominal component of the PASG causes an increase in the patient's respiratory rate or respiratory distress, it must be deflated immediately, regardless of the patient's blood pressure. Diaphragmatic rupture is assumed until proven otherwise.

D. Deflation and Removal of the PASG

Several specific points bear emphasis in deflation procedures. The PASG should not be an impediment to IV access and volume resuscitation. Individual segments may be carefully deflated for IV access, examination of extremities, angiography, etc. In general, if the patient requires transfer to another facility, the garment is left in situ, inflated as indicated. For patients transferred by air, effective inflation pressures may increase due to changes in atmospheric pressure. A similar change may occur when the PASG is applied in a cold environment, and the patient is then brought into a warm emergency department. The deflation process is gradual, beginning with the abdominal segment. Air is allowed to escape slowly, while blood pressure is closely monitored. A fall in systolic blood pressure of more than 5 mm Hg is an indication for more fluid resuscitation prior to continued deflation.
IX. Pitfalls in the Diagnosis and Treatment of Shock

A. Equating Blood Pressure with Cardiac Output

Treatment of hypovolemic (hemorrhagic) shock requires correcting inadequate organ perfusion, which means increasing organ blood flow and tissue oxygenation. Increasing blood flow means increasing 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). It is a mistake to necessarily equate an increase in blood pressure with a concomitant increase in cardiac output. An increase in peripheral resistance (ie, vasopressor therapy) with no change in cardiac output results in increased blood pressure, but no improvement in tissue perfusion or oxygenation.

B. Age

Hypotension from hemorrhage due to trauma is poorly tolerated by the older patient. Aggressive therapy with fluids and early surgery are often warranted to save the patient and prevent serious complications, such as myocardial infarction or a cerebrovascular accident.

C. Athletes

Rigorous training routines change the cardiovascular dynamics of this group of patients. Blood volume can increase 15% to 20%, cardiac output can increase six-fold, stroke volume can increase 50%, and resting pulse is generally at 50. This group's ability to compensate for blood loss is truly remarkable. The usual responses to hypovolemia may not be manifested in athletes, even though significant blood loss may have occurred.

D. Medications

Beta-adrenergic receptor blockers and calcium antagonists can significantly alter the patient's hemodynamic response to hemorrhage.

E. Hypothermia

Patients suffering from hypothermia and hemorrhagic shock are resistant to appropriate blood and fluid resuscitative measures and often develop a coagulopathy. Therefore, body temperature is an important vital sign to record during the initial assessment phase. Theoretically, esophageal or bladder recording is an accurate clinical measurement of the core temperature. Although not ideal, a rectal temperature alerts the treating physician to the problem. A trauma victim under the influence of alcohol and exposed to cold temperature extremes may become hypothermic. Rapid rewarming in a warmed environment with appropriate external warming devices, heated respiratory gases, and warmed intravenous fluids generally correct the patient's hypotension and hypothermia. Core rewarming (peritoneal or thoracic cavity irrigation with warmed - 39 degrees centigrade - crystalloid solutions, or extracorporeal bypass) may occasionally be indicated. (See Chapter 9, Injury due to Burn and Cold.) Hypothermia is best treated by prevention.
F. Pacemaker

Patients with pacemakers are unable to respond to blood loss in the expected fashion. Considering the significant number of patients with myocardial conduction defects who have such devices in place, central venous pressure monitoring is invaluable in these patients to guide fluid therapy.

X. Reassessing Patient Response and Avoiding Complications

Inadequate volume replacement with subsequent organ failure is the most common complication of hemorrhagic shock. Immediate, appropriate, and aggressive therapy that restores organ perfusion minimizes these untoward events. Three specific concerns are outlined in the succeeding paragraphs.

A. Continued Hemorrhage

Obscure hemorrhage is the most common cause of poor patient response to fluid therapy. These patients are generally included in the Transient Response category as defined previously. Under this circumstance, consider immediate surgical intervention.

B. Fluid Overload and CVP Monitoring

After the patient's initial assessment and management have been completed, the risk of fluid overload is minimized by monitoring the patient carefully. Remember, the goal of therapy is restoration of organ perfusion and adequate tissue oxygenation, signified by appropriate urinary output, central nervous system function, skin color, and return of pulse and blood pressure toward normal.

Central venous pressure (CVP) monitoring is a relatively simple procedure and is used as a standard guide for assessing the ability of the right side of the heart to accept a fluid load. Properly interpreted, the response of the CVP to fluid administration helps evaluate volume replacement. Several points to remember are:

1. The precise measure of cardiac function is the relationship between ventricular-end diastolic volume and stroke volume. It is apparent that comparison of right atrial pressure (CVP) to cardiac output (as reflected by evidence of perfusion or blood pressure, or even by direct measurement) is an indirect and, at best, an insensitive estimate of this relationship. Remembering these facts is important to avoid over dependence on CVP monitoring.

2. The initial CVP level and the actual blood volume are not necessarily related. The initial CVP is sometimes high even with a significant volume deficit, especially in patients with chronic obstructive pulmonary disease, generalized vasoconstriction, and rapid fluid replacement. The initial venous pressure also may be high secondary to the application of the PASG or the inappropriate use of exogenous vasopressors.

3. A minimal rise in the initial, low CVP with fluid therapy suggests the need for further volume expansion (minimal or no response to fluid resuscitation category).
4. A declining CVP suggests ongoing fluid loss and the need for additional fluid or blood replacement (transient response to fluid resuscitation category).

5. An abrupt or persistent elevation in the CVP suggests volume replacement is adequate, is too rapid, or that cardiac function is compromised.

6. Remember, the CVP line is not a primary intravenous fluid resuscitation route. It should be inserted on an elective rather than emergent basis.

7. Pronounced elevations of the CVP may be caused by hypervolemia as a result of overtransfusion, cardiac dysfunction, cardiac tamponade, or increased intrathoracic pressure from a pneumothorax. Catheter malposition may produce an erroneously measured high CVP.

Access for a central venous pressure line can be obtained via a variety of routes. Appropriate antiseptic techniques are used when central lines are placed. Ideal placement of an intravenous catheter is in the superior vena cava, just proximal to the right atrium. Techniques are discussed in detail in Skill Station IV, Vascular Access and Monitoring.

Central venous lines are not without complications. Infections, vascular injury, embolization, thrombosis, and pneumothorax are encountered. Central venous pressure monitoring reflects right heart function. It may not be representative of the left heart function in patients with primary myocardial dysfunction or abnormal pulmonary circulation.

C. Recognition of Other Problems

When the patient fails to respond to therapy, consider ventilatory problems, unrecognized fluid loss, acute gastric distention, cardiac tamponade, myocardial infarction, diabetic acidosis, hypoadrenalism, and neurogenic shock. Constant re-evaluation, especially when patients deviate from expected patterns, is the key to recognizing such problems as early as possible.

XI. Summary

Shock therapy, based on sound physiologic principles, is usually successful. Hypovolemia is the cause of shock in most trauma patients. Management of these patients requires immediate hemorrhage control and fluid replacement. Other possible causes of the shock state must be considered. The patient's response to initial fluid therapy determines further therapeutic and diagnostic procedures. The goal of therapy is restoration of organ perfusion. In hypovolemic shock, vasopressors are rarely, if ever, needed. Central venous pressure measurement is a valuable tool for confirming the volume status and monitoring the rate of fluid administration.
Skill Station IV: Vascular Access and Monitoring

Resources and Equipment

This list is the recommended equipment to conduct this skill session in accordance with the stated objectives for and intent of the procedures outlined. Additional equipment may be used providing it does not detract from the stated objectives and intent of this skill, or from performing the procedures in a safe method as described and recommended by the ACS Committee on Trauma.

1. Live patient model.
2. Jugular and subclavian IV manikin (optional).
3. Fresh chicken or turkey legs.
4. Subclavian intravenous set-up.
5. Central venous pressure set-up.
   a. Assorted intravenous over-the-needle catheters (#14-, #16-, and #18-gauge, 15-20 cm or 6-8 inches in length)
   b. #18-gauge needle (3.75 cm or 1.5 inches in length)
   c. #19-gauge needle (3.75 or 1.5 inches in length)
   d. #16- or #18-gauge bone aspiration/transfusion needle (1.25 cm or 0.5 inch in length).
7. 7.0-8.5 Fr rapid infuser catheter kit with guidewire.
10. 12- and 20-mL syringes.
11. Lidocaine 1% (demonstration purposes only).
12. 1000 mL Ringer's lactate with macrodrip.
13. Large-caliber intravenous and extension tubings.
15. 3x3 gauze sponges.
16. Vial of sterile saline.
17. Pulse oximetry monitoring device, power cord, sensors, and operator's manual.

Objectives

1. Performance at this station will allow the participant to practice and demonstrate the techniques of subclavian, internal jugular, and femoral intravenous, as well as intraosseous line insertions.

2. Upon completion of this station, the participant will be able to describe the surface markings for percutaneous access into a:
   a. Subclavian vein.
   b. Internal jugular vein.
   c. Femoral vein.
   d. Proximal tibia or distal femur.
3. Upon completion of this station, the participant will be able to identify the intravenous fluid used for the following types of shock and associated trauma:
   a. Hemorrhagic (hypovolemic) shock.
   b. Nonhemorrhagic shock.

4. Upon completion of this station, the participant will be able to:
   a. Discuss the purpose of pulse oximetry monitoring.
   b. Demonstrate the proper method of activating the device, setting alarm limits, and applying the optical sensors to appropriate body parts.
   c. Discuss the indications for using a pulse oximetry monitoring device, its functional limits of accuracy, and reasons for malfunction or inaccuracy.
   d. Accurately interpret the pulse oximeter monitor readings and relate their significance to the care of the trauma patient.

**Procedures**

2. Internal jugular venipuncture.
3. Femoral venipuncture.
4. Intraosseous puncture/infusion.
5. Pulse oximetry monitoring.

**Note:** The Broselow Pediatric Resuscitation Measuring Tape is included on the equipment list for this station. A specific skill is not outlined for its use; however, participants need to be aware of its availability and its use when managing pediatric trauma. By measuring the height of the child, the child's estimated weight can be determined readily. One side of the tape provides drugs and their recommended doses for the pediatric patient based on weight. The other side provides equipment needs for the pediatric patient based on size. Therefore, participation at this station should include an orientation to the tape and its use.
Skills Procedures: Vascular Access and Monitoring

Note: Universal precautions are required whenever caring for the trauma patient.

I. Subclavian Venipuncture: Infraclavicular Approach (See Figure 1).

A. Place the patient in a supine position, at least 15 degrees head-down to distend the neck veins and prevent an air embolism. Only if the cervical spine has been cleared radiographically can the patient's head be turned away from the venipuncture site.

B. Cleanse the skin well around the venipuncture site and drape the area. Sterile gloves should be worn when performing this procedure.

C. If the patient is awake, use a local anesthetic at the venipuncture site.

D. Introduce a large-caliber needle, attached to a 12-mL syringe with 0.5 to 1 mL saline, 1 cm below the junction of the middle and medial thirds of the clavicle.

E. After the skin has been punctured, with the bevel of the needle upward, expel the skin plug that may occlude the needle.

F. The needle and syringe are held parallel to the frontal plane.

G. Direct the needle medially, slightly cephalad, and posteriorly behind the clavicle toward the posterior, superior angle to the sternal end of the clavicle (toward finger placed in the suprasternal notch).

H. Slowly advance the needle while gently withdrawing the plunger of the syringe.

I. When a free flow of blood appears in the syringe, rotate the bevel of the needle caudally, remove the syringe, and occlude the needle with a finger to prevent an air embolism.

J. Quickly insert the catheter to a predetermined depth (tip of catheter should be above the right atrium for fluid administration).

K. Remove the needle and connect the catheter to the intravenous tubing.

L. Affix the catheter (eg, with suture), apply antibiotic ointment, and dress the area.

M. Tape the intravenous tubing in place.

N. Attach the central venous pressure set-up to the intravenous tubing and adjust the manometer (level at zero) with the level of the patient's right atrium.

O. Obtain a chest film to identify the position of the intravenous line and a possible pneumothorax.
P. Optional Method: Seldinger technique as outlined in III. Femoral Venipuncture: Seldinger Technique.
II. Internal Jugular Venipuncture: Middle or Central Route (See Figure 2).

A. Place the patient in a supine position, at least 15 degrees head-down to distend the neck veins and to prevent air embolism. Only if the cervical spine has been cleared radiographically, can the patient's head be turned away from the venipuncture site.

B. Cleanse the skin well around the venipuncture site and drape the area. Sterile gloves should be worn when performing this procedure.

C. If the patient is awake, use a local anesthetic at the venipuncture site.

D. Introduce a large-caliber needle, attached to a 12-mL syringe with 0.5 to 1 mL of saline, into the center of the triangle formed by the two lower heads of the sternomastoid and the clavicle.

E. After the skin has been punctured, with the bevel of the needle upward, expel the skin plug that may occlude the needle.

F. Direct the needle caudally, parallel to the sagittal plane, at a 30-degree posterior angle with the frontal plane.

G. Slowly advance the needle while gently withdrawing the plunger of the syringe.

H. When a free flow of blood appears in the syringe, remove the syringe and occlude the needle with a finger to prevent an air embolism. If the vein is not entered, withdraw the needle and redirect it 5 to 10 degrees laterally.

I. Quickly insert the catheter to a predetermined depth (tip of catheter should be above the right atrium for fluid administration).

J. Remove the needle and connect the catheter to the intravenous tubing.

K. Affix the catheter in place (eg, with suture), apply antibiotic ointment, and dress the area.

L. Tape the intravenous tubing in place.

M. Attach the central venous pressure set-up to the intravenous tubing and adjust the manometer (level at zero) with the level of the patient's right atrium.

N. Obtain a chest film to identify the position of the intravenous line and possible pneumothorax.

O. Optional Method: Seldinger technique as outlined in III. Femoral Venipuncture: Seldinger Technique.
Complications of Central Intravenous Venipuncture

1. Hematoma formation.
2. Cellulitis.
3. Thrombosis.
4. Phlebitis.
6. Arterial puncture.
7. Pneumothorax.
8. Hemopneumothorax (eg, with subclavian venipuncture).
10. Chylothorax (eg, with internal jugular venipuncture).
11. Arteriovenous fistula.
12. Peripheral neuropathy.
13. Lost catheters.
15. Improperly placed catheters.
III. Femoral Venipuncture: Seldinger Technique (see Figure 3).

A. Place the patient in supine position.

B. Cleanse the skin well around the venipuncture site and drape the area. Sterile gloves should be worn when performing this procedure.

C. Locate the femoral vein by palpating the femoral artery. The vein lies directly medial to the femoral artery (nerve, artery, vein, empty space). A finger should remain on the artery to facilitate anatomical location and to avoid insertion of the catheter into the artery.

D. If the patient is awake, use a local anesthetic at the venipuncture site.

E. Introduce a large-caliber needle attached to a 12-mL syringe with 0.5 to 1 mL of saline. The needle, directed toward the patient's head, should enter the skin directly over the femoral vein.

F. The needle and syringe are held parallel to the frontal plane.

G. Directing the needle cephalad and posteriorly, slowly advance the needle while gently withdrawing the plunger of the syringe.

H. When a free flow of blood appears in the syringe, remove the syringe and occlude the needle with a finger to prevent an air embolism.

I. Insert the guidewire and remove the needle. Then insert the catheter over the guidewire.

J. Remove the guidewire and connect the catheter to the intravenous tubing.

K. Affix the catheter in place (ie, with suture), apply antibiotic ointment, and dress the area.

L. Tape the intravenous tubing in place.

M. Attach the central venous pressure set-up to the intravenous tubing and adjust the manometer (level at zero) with the level of the patient's right atrium.

N. Obtain chest and abdominal roentgenograms to identify the position and placement of the intravenous catheter.
Complications of Femoral Venipuncture

1. Hematoma formation.
2. Cellulitis.
3. Thrombosis.
4. Phlebitis.
5. Nerve transection and/or puncture.
6. Arterial puncture.
7. Arteriovenous fistula.
8. Peripheral neuropathy.
9. Lost catheters.
10. Inaccurate monitoring techniques.
11. Improperly placed catheter.
IV. Intraosseous Puncture/Infusion: Proximal Tibial Route (See Figure 4).

Note: This procedure is limited to children six years of age or younger, for whom venous access is impossible due to circulatory collapse or for whom percutaneous peripheral venous cannulation has failed on two attempts. Intraosseous infusions should be limited to emergency resuscitation of the child and discontinued as soon as other venous access has been obtained. (Methylene-blue dye may be mixed with the sterile saline for demonstration purposes only. Providing the needle has been properly placed within the medullary canal, the methylene-blue dye/saline solution seeps from the upper end of the chicken or turkey bone when the solution is injected. See item H.)

A. Place the patient in a supine position. Selecting an uninjured lower extremity place sufficient padding under the knee to effect an approximate 30-degree angle and allow the patient's heel to rest comfortably on the gurney.

B. Identify the puncture site - anteromedial surface of the proximal tibia, approximately one finger-breadth (1 to 3 cm) below the tubercle.

C. Cleanse the skin well around the puncture site and drape the area. Sterile gloves should be worn when performing this procedure.

D. If the patient is awake, use a local anesthetic at the puncture site.

E. At a 90-degree angle, introduce a short (threaded or smooth), large-caliber bone-marrow aspiration needle (or a short, #18-gauge spinal needle with stylet) into the skin with needle bevel direct toward the foot and away from the epiphyseal plate.

F. Using a gentle twisting or boring motion, advance the needle through the bone cortex and into the bone marrow.

G. Remove the stylet and attach to the needle a 12-mL syringe filled with approximately 6 mL of sterile saline. Gently withdraw on the plunger of the syringe. Aspiration of bone marrow into the syringe signifies entrance into the medullary cavity.

H. Inject the saline into the needle to expel any clot that may occlude the needle. If the saline flushes through the needle easily and there is no evidence of swelling, the needle should be in the appropriate place. If bone marrow was not aspirated as outlined in G, but the needle flushes easily when injecting the saline without evidence of swelling, the needle should be in the appropriate place. Additionally, proper placement of the needle is indicated if the needle remains upright without support and intravenous solution flows freely without evidence of subcutaneous infiltration.

I. Connect the needle to the large-caliber intravenous tubing and begin fluid infusion. The needle is then carefully screwed further into the medullary cavity until the needle hub rests on the patient's skin. If a smooth needle is used, it should be stabilized at a 45 to 60 degree angle to the anteromedial surface of the child's leg.

J. Apply antibiotic ointment and a 3x3 sterile dressing. Secure the needle and tubing
in place.

K. Routinely re-evaluate the placement of the intraosseous needle, assuring that it remains through the bone cortex and in the medullary canal. Remember, intraosseous infusion should be limited to emergency resuscitation of the child and discontinued as soon as other venous access has been obtained.

**Complications of Intraosseous Puncture**

1. Local abscess and cellulitis.
2. Osteomyelitis.
3. Sepsis.
4. Through and through penetration of the bone.
5. Subcutaneous or subperiosteal infiltration.
6. Pressure necrosis of the skin.
7. Transient bone marrow hypocellularity.
V. Pulse Oximetry Monitoring

The pulse oximeter provides continuous, noninvasive measurements of oxygen saturation of functional hemoglobin and pulse rate that are updated with each heart beat. The device combines the principles of spectrophotometric oximetry and plethysmography. It consists of an electro-optical sensor that is applied to the patient, and a monitor that processes and displays the measurements. The sensor contains low-voltage, low-intensity light-emitting diodes (LEDs) as light sources and photodiode as a light receiver. (An oximeter is a photoelectric instrument that measures the oxygen saturation of blood. A spectrophotometer is a tool that estimates the quantity of colored material in a light path by determining the amount of light absorbed. A plethysmograph is a device that measures variations in the volume of an organ, part, or limb caused by pulsatile variations in the amount of blood present or passing through it.)

When the light from the LEDs is transmitted through blood and other body tissues, a portion of the light is absorbed by the blood and by each tissue component. The photodiode in the sensor measures the light that passes through without being absorbed, and this measurement is used to determine how much light is absorbed. The results of these measurements are conveyed both audibly and visually into pulse rate and percent arterial hemoglobin oxygen saturation displays on the device.

With each heart beat, a pulse of oxygenated arterial blood flows to the sensor site. The relative amount of light absorbed by oxygenated hemoglobin differs from deoxygenated hemoglobin. The pulse oximeter uses these measurements to determine the percentage of hemoglobin that is saturated with oxygen.

Initially, light absorption is determined when the pulsatile blood is not present, serving as the "reference" or "baseline" absorption of tissue and nonpulsatile blood. Absorption is then measured after the next heart beat when pulsatile blood enters the tissue. Subsequently, the light absorption is changed by the presence of the pulse of arterial blood.

The microprocessor in the monitor then corrects the measurements during the pulsatile flow for the amount of light absorbed during the initial (no pulsatile flow) measurements. The ratio of the corrected absorption is then used to determine functional oxygen saturation (oxygenated hemoglobin conveyed as a percentage of only the hemoglobin capable of transporting oxygen - oxyhemoglobin and reduced hemoglobin; dysfunctional hemoglobin is excluded from this calculation).

The pulse oximeter is calibrated to read oxyhemoglobin saturation (%SaO_2) of functional hemoglobin as compared to a CO-oximeter. Significant levels of dysfunctional hemoglobin (hemoglobin incapable of reversibly binding oxygen, eg, carboxyhemoglobin, methemoglobin), may affect the accuracy of the instrument. Indocyanine green, methylene-blue, and other intravascular dyes, depending on their concentrations, may interfere with the accuracy of the instrument. Instrument performance also may be affected by excessive patient movement, electrocautery interference, intense surrounding light, severe anemia, severe vasoconstriction, and hypothermia. (See Chapter 2, Airway and Ventilatory Management.)
A. Assemble the necessary equipment and set up the device.

1. Plug one end of the power cord into the device's AC power inlet and the other end into a properly grounded AC outlet.

2. Connect the patient sensor module to the device.

3. If ECG synchronization is to be used, provide an ECG signal to the device.

B. Select an appropriate sensor and apply it to the patient's ear lobe, finger tip, toe tip, nose bridge, or foot band (neonates). Remember, the sensor should not be applied to an ischemic or injured body part.

C. Turn the device to "On", review the "Start Up" displays (pulse search, oxygen saturation, pulse rate, ECG in use), and confirm effective operation of the device.

D. Identify the "Oxygen Saturation" and "Pulse Rate" readings, test the alarms, and reset the alarm limits as necessary.

E. Evaluate the "Oxygen Saturation" and "Pulse Rate" findings.

1. Are they appropriate?

2. What do they indicate?

3. What might affect these results?

4. What is the estimated arterial oxygen partial pressure?

F. Turn the device to "Off" and disconnect the sensors from the patient.

G. Discuss the following potential problems and causes:

1. No indication of pulse, saturation, and/or pulse rate were displayed.

2. The perfusion indicator tracks the pulse, but there is no oxygen saturation or pulse rate display.

3. Saturation and/or pulse rate displays are changing rapidly, and the perfusion indicator is erratic.

4. The pulse rate does not correlate with other monitors or monitoring methods.

5. The oxygen saturation measurement does not correlate with blood gas determinations.
Hemoglobin Oxygen Saturation versus Arterial Oxygen Pressure

The relationship between partial pressure of oxygen in arterial blood (PaO₂) and percent arterial hemoglobin oxygen saturation (%SaO₂) is displayed in Figure 5. The sigmoid shape of this curve indicates that the relationship between saturation and arterial oxygen partial pressure is nonlinear. This is particularly important in the middle range of this curve, where small changes in PaO₂ will effect large changes in saturation. Remember, the pulse oximeter measures arterial oxygen saturation, not arterial oxygen partial pressure. (See Table 1, PaO₂ versus O₂ Saturation Levels, Chapter 2, Airway and Ventilatory Management.)

Standard blood gas measurements report both arterial oxygen pressure (PaO₂) and a calculated hemoglobin saturation (%SaO₂). When oxygen saturation is calculated from blood gas PaO₂, the calculated value may differ from the oxygen saturation measured by the pulse oximeter. This is because an oxygen saturation value that has been calculated from the blood gas PaO₂ has not necessarily been correctly adjusted for the effects of variables that shift the relationship between PaO₂ and saturation. These variables include temperature, pH, PaCO₂, 2,3-DPG, and the concentration of fetal hemoglobin.
Skill Station V: Venous Cutdown

This surgical procedure, if performed on a live-anesthetized animal, must be conducted in a USDA-Registered Animal Laboratory. (See ATLS Instructor Manual, Section II, Chapter 9 - Policies, Procedures, and Protocols for Surgical Skills Practicum.)

Resources and Equipment

This list is the recommended equipment to conduct this skill session in accordance with the stated objectives for and intent of the procedures outlined. Additional equipment may be used providing it does not detract from the stated objectives and intent of this skill, or from performing the procedures in a safe method as described and recommended by the ACS Committee on Trauma

1. Live, anesthetized animals.
2. Licensed veterinarian (see guidelines referenced above).
3. Animal troughs, ropes (sandbags optional).
4. Animal intubation equipment:
   a. Endotracheal tubes
   b. Laryngoscope blade and handle
   c. Respiratory with 15-mm adapter.
5. Electric shears with #40 blade.
6. Local anesthetic set, including antiseptic.
7. Tables or instrument stand.
8. #14- to #20-gauge cutdown catheters.
9. Suture:
   a. 3-0 ties
   b. 4-0 suture with swaged needle.
10. Surgical instruments:
    a. Scalpel handles with #10 and #11 blades
    b. Small hemostats
    c. Needle holders
    d. Single-toothed spring retractors.
11. 4x4 gauze sponges.
12. Surgical drapes.
13. 500-mL Ringer's lactate with macrodrip tubing.
15. Surgical garb (gloves, shoe covers, and scrub suits or cover gowns).

Objectives

1. Performance at this station will allow the participant to practice and demonstrate on a live anesthetized animal the technique of peripheral venous cutdown.

2. Upon completion of this station, the participant will be able to identify the surface markings and structures to be noted in performing a peripheral venous cutdown.

3. Upon completion of this station, the participant will be able to discuss the
indications and contraindications for a peripheral venous cutdown.

**Anatomic Considerations for Venous Cutdown**

1. The primary site for a peripheral venous cutdown is the greater saphenous vein at the ankle, which is located at a point approximately 2 cm anterior and superior to the medial malleolus. (See Figure 1, Saphenous Venous Cutdown.)

2. A secondary site is the antecubital medial basilic vein, located 2.5 cm lateral to the medial epicondyle of the humerus at the flexion crease of the elbow.
Skills Procedure: Venous Cutdown

Note: Universal precautions are required whenever caring for the trauma patient.

I. Venous Cutdown

A. Prepare the skin of the ankle with antiseptic solution and drape the area.

B. Infiltrate the skin over the vein with 0.5% lidocaine.

C. A full-thickness transverse skin incision is made through the area of anesthesia to a length of 2.5 cm.

D. By blunt dissection, using a curved hemostat, the vein is identified and dissected free from any accompanying structures.

E. Elevate and dissect the vein for a distance of approximately 2 cm, to free it from its bed.

F. Ligate the distal, mobilized vein, leaving the suture in place for traction.

G. Pass a tie about the vein, cephalad.

H. Make a small transverse venotomy and gently dilate the venotomy with the tip of a closed hemostat.

I. Introduce a plastic cannula through the venotomy and secure it in place by tying the upper ligature about the vein and cannula. The cannula should be inserted an adequate distance to prevent dislodging.

J. Attach the intravenous tubing to the cannula and close the incision with interrupted sutures.

K. Apply a sterile dressing with a topical antibiotic ointment.

Complications of Peripheral Venous Cutdown

1. Cellulitis.
2. Hematoma.
3. Phlebitis.
4. Perforation of the posterior wall of the vein.
5. Venous thrombosis.
7. Arterial transection.