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

C. Discuss the basic principles that apply to the emergency treatment of hemorrhagic shock and their application to the patient's clinical response to therapy.

D. Discuss the clinical significance of fluid management for problems unique to the trauma patient.

E. Demonstrate various techniques of central and peripheral intravenous line insertion, including cutdowns.

F. Discuss the indications, contraindications, and dangers of inpatient use of the pneumatic antishock garment.
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. Thus the definition of shock as an abnormality of the circulatory system that results in inadequate organ perfusion, 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. The majority of injured patients in shock are hypovolemic, but cardiogenic shock 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 will be 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. Septic shock is unusual but must be considered for patients whose arrival at the emergency facility has been greatly delayed.

The end result of inadequately treated hypoperfusion is organ failure leading to immediate or delayed patient demise. For that reason, the treatment of shock is directed toward restoring cellular and organ perfusion with adequately oxygenated blood, 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 will require surgical intervention, and many of these will require early intervention to relieve the shock. Therefore, the presence of shock in an injured patient demands the immediate involvement of qualified surgeons.

II. Hemorrhagic Shock in the Injured Patient

The trauma patient's response to blood loss is rendered more complex by changes in the body fluids (particularly in the extracellular fluid) that impact on the circulating blood volume and cellular function. The classic response to blood loss (described below) must be considered in the context of the fluid changes associated with soft tissue injury and the changes associated with severe, prolonged shock.

A. Pathophysiology

Early circulatory responses to blood loss are compensatory, ie, progressive vasoconstriction of cutaneous, visceral, and muscle circulation to preserve blood flow to the kidneys, heart, and brain. Tachycardia is the earliest measurable circulatory sign.

At the cellular level, inadequately perfused 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, 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 cyclic phenomenon with adequate oxygenation, ventilation, and appropriate fluid resuscitation.

B. Definition of Hemorrhage

Hemorrhage is defined as an acute loss of circulating blood. Normally, adult blood volume is approximately 7% of body weight. For example: a 70-kilogram male has approximately 5 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 to be 8% to 9% of the body weight (80 to 90 mL/kg). (See Chapter 10 - Pediatric Trauma.)

C. Direct Effect 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. Class I is exemplified by the condition of the blood donor. Class II is uncomplicated shock, but 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 will be dead within minutes. (See Estimated Fluid and Blood Requirements chart at conclusion of this chapter.)

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 occurrence 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 physiological 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, respiratory rate, or capillary refill test. The capillary refill test is performed by depressing the fingernail or hypothenar eminence with the finger. A normal response is for the color to return within the period it takes the examiner to say the phrase, "capillary return" (ie, two seconds). The capillary refill test cannot be interpreted in hypothermic patients. 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 will correct the circulatory state.

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

In a 70-kilogram male, this volume loss represents 800 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 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), and a positive capillary refill test. 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 initially stabilized 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 classical 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 will 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 (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 here. Loss of over 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 (755 mL) blood loss. Twice that amount (up to 1500 mL) is commonly associated with femur fractures.
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 acts as 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 would be represented by 1500 mL of interstitial fluid and 500 mL of plasma volume. In general, roughly 25% of such fluid translocation will be evidenced by a decrease in the plasma volume. The impact of these changes on circulating blood volume and the reason they compound fluid loss then becomes obvious.

III. 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. Remember, compensatory mechanisms may have precluded 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. A narrowed pulse pressure suggests significant blood loss and involvement of compensatory mechanisms. 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 normal 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; 100 in adult. The elderly patient may not exhibit tachycardia because of a limited cardiac response to catecholamine stimulation or certain medications such as propranolol.

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 near normal hematocrit does not rule out significant blood loss.

B. Clinical Differentiation of Etiology

Hemorrhage is the most common cause of shock after injury, and virtually all multiply injured patients have an element of hypovolemia. Therefore, once the shock state is identified, treatment is usually begun as if the patient were hypovolemic. As this 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 shock. The major differentiating factor in identifying the cause of shock in a trauma patient is whether the condition is hypovolemic or cardiogenic, especially for a patient with injuries above the diaphragm. A high index of suspicion and careful observation of the patient's response to 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.

1. Cardiogenic shock

Myocardial dysfunction may occur from tension pneumothorax, 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 thoracic trauma need constant ECG monitoring to detect injury patterns and dysrhythmias. Blood CPK and isoenzymes should be drawn on admission, but rarely have any value in diagnosing or managing the patient in the emergency room. Ultrasound and specific isotope studies of the myocardium are not practical tests for the emergency room. Myocardial contusion may be an indication for early central venous pressure monitoring of fluid resuscitation in the emergency department.

Cardiogenic tamponade is most common in penetrating thoracic trauma. It can occur rarely in blunt trauma to the thorax or in a patient who had myocardial infarction with tamponade as a cause or result of the accident. Tachycardia; muffled heart sounds; and dilated, engorged neck veins with hypotension resistant to fluid therapy suggest cardiac tamponade. The only condition that mimics cardiac tamponade is tension pneumothorax. Appropriate placement of a needle temporarily relieves these two life-threatening conditions.

2. Neurogenic shock

Isolated head injuries do not cause shock. The presence of shock in a patient with a head injury indicates 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. Patients with known or suspected neurogenic shock should be treated initially for hypovolemia. Vasoactive drugs should not be administered until volume is restored. Venous pressure monitoring is extremely helpful in managing this sometimes complex problem.

3. 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 a wide pulse pressure.
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 - Breathing

The adequacy of ventilation is assessed. Establishing a patent airway with adequate ventilatory exchange is the first priority. After establishing an adequate airway, oxygen is administered. Supplementary oxygen via a bag-valve-mask reservoir system is delivered to maintain arterial oxygen tension between 80 and 100 mm Hg.

2. Bleeding - Hemorrhage control

Bleeding from external wounds usually can be controlled by direct pressure to the bleeding site, eg, scalp, neck, and upper and lower extremity. Pneumatic antishock trousers may be used to control bleeding from pelvic and open lower extremity fractures. However, this device should not interfere with rapid re-establishment of intravascular volume by the intravenous route.

3. Gastric dilatation - Decompression

Gastric dilatation often occurs in the trauma victim in spite of a nasogastric tube. This condition makes shock difficult to treat, and the unconscious patient is always in danger of aspiration, a potentially fatal complication. The physician's responsibility does not end with the passage of the tube. The tube must be properly positioned, attached to appropriate suction, and be functioning.

B. Vascular Access Lines

Access to the vascular system must be obtained promptly. This is best done by establishing two large-bore (#16-gauge or larger) catheters before any consideration is given to a central line. The most desirable sites for peripheral intravenous lines (in order of priority) are: 1) percutaneous peripheral access via forearm or antecubital veins, and 2) cutdown on the saphenous or arm veins. If circumstances prevent the use of peripheral veins, central venous access is indicated, using the Seldinger technique.

As the intravenous lines are started, blood samples are drawn for appropriate laboratory analyses, type and crossmatch, and toxicology. It also may be useful to obtain arterial blood gases at this time.
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. Ringer's lactate solution is the initial fluid of choice. Normal saline is the second choice. Although normal saline is a 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 is one to two litres 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 diagnostic 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. However, general guidelines are available for establishing the amount and type of fluid and blood the patient will probably require. If, during resuscitation, the amounts administered deviate widely from these estimates, a careful reassessment of the situation and a search for unrecognized injuries or other causes of shock are necessary. (See Table 1 - Estimated Fluid and Blood Requirements, at conclusion of this chapter.)

V. Evaluation of Response / Continued Therapy

A. General

The same signs and symptoms of inadequate perfusion that were used to diagnose shock are useful determinators of patient response. The return of normal blood pressure, pulse pressure, and pulse rate are positive signs, and indicate that circulation is stabilizing. 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 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 emergency room management of the injured patient.

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 a 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.

VI. Therapeutic Decisions

 Having established a preliminary plan based on the initial evaluation of the patient, the physician can now modify management, depending on the patient's response to initial resuscitative fluids. This approach 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. The potential response patterns can be discussed in three groups.

A. Rapid Response to Initial Fluid Administration

 A small group of patients will respond rapidly to the initial fluid bolus, and will remain stable when the initial fluid has been completed and the fluids are slowed. Such patients will usually have lost less than 20% of their 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 will responds to the initial fluid bolus. However, as the initial fluids are slowed, the circulatory perfusion indices will begin to show deterioration in these patients (most of whom will have lost 20% to 40% of their blood volume or who are still bleeding). Continued fluid administration and initiation of blood administration are indicated. The response to blood administration should identify patients who are still bleedsing 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. Central venous pressure monitoring helps differentiate between these two groups of patients.
VII. Blood Replacement

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

A. Crossmatched, Type-Specific, and Type O Blood

1. Fully crossmatched blood is preferable. However, the complete crossmatching procedure requires approximately an hour in most blood banks. For patients who stabilize rapidly, crossmatched blood should be obtained and 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 the previous section.

3. If type-specific blood is unavailable, type O packed cells are indicated for patients with exsanguinating hemorrhage. To avoid sensitization and future complications, Rh-negative cells are preferable, particularly for women of child-bearing age.

B. Blood Filters

Macropore (160 micron) 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.

C. Warming Fluids - Plasma and Crystalloid

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

D. Coagulopathy

Coagulopathy is a rare problem in the first hour of treatment of the multiply 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. Routine use of fresh frozen plasma and platelets in patients with dilutional coagulopathy after massive fluid and blood replacement is costly, dangerous, and unwarranted.

E. Calcium Administration

The majority of the patients receiving blood transfusions do not need calcium supplements in the first hour of treatment.
VIII. Pneumatic Antishock Garment (PASG)

The application of the PASG can raise systolic pressure by increasing peripheral vascular resistance and myocardial afterload. Use of the PASG is controversial. If the need for application has been recognized during the delivery of prehospital care, most patients will arrive in the emergency department with the PASG applied.

A. Indications

Current indications for inhospital use of the PASG are:

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

2. Intra-abdominal trauma with severe hypovolemia in patients who are en route to the operating room or another facility. A number of investigative studies suggest that the garment may reduce intra-abdominal hemorrhage.

B. Contraindications

1. Uncontrolled hemorrhage outside the confines of the garment is a relative contraindication.

2. Pulmonary edema, known rupture of the diaphragm, and left ventricular dysfunction are absolute contraindications.

C. Dangers

The use of the PASG must not delay volume replacement or rapid transport for penetrating trauma. Precise time that the leg compartments have been inflated must be documented on the patient's records. Any patient with extremity trauma and associated history of shock should not have the leg segments inflated for prolonged periods of time.

If inflation of the abdominal component of the PASG causes an increase in the patient's respiratory 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 PASG

Several specific points bear emphasis in deflation procedures. The trousers can be removed after the shock state is adequately managed, and vital signs are within acceptable limits. Individual segments may be carefully deflated for examination of extremities, angiography, etc. In general, if the patient requires transfer to another facility, the garment is left inflated. For patients transferred by air, effective inflation pressures may increase due to changes in atmospheric pressure. A similar change 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. Deflation of the garment begins with the waist or abdominal segment. A small amount of air is allowed to escape from the valve, while the patient's blood pressure is carefully monitored. Deflation is continued until the patient exhibits a blood pressure drop of 5 mm Hg. At this point, deflation is stopped, and intravenous fluid replacement is increased until the blood pressure returns to normal limits. Deflation is resumed. When the abdominal segment has been deflated, the legs are sequentially deflated, carefully monitoring the patient's blood pressure.

IX. Pitfalls in the Diagnosis and Treatment of Shock

A. Age

The older patient has difficulty tolerating hypotension from hemorrhage due to trauma. 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.

B. Athletes

Rigorous training routines change the cardiovascular dynamics of this group of patients. Blood volume can increase 15% to 20%, cardiac outputs 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. Any athlete with suspected hemorrhage due to trauma must be watched carefully.

C. Medications

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

D. Hypothermia

Body temperature is one of the most important vital signs recorded in the initial assessment phase. Theoretically, esophageal recording is the most accurate measurement of the core temperature. Although not ideal, a rectal temperature will alert the treating physician to the problem. A trauma victim under the influence of alcohol and exposed to cold temperature extremes may become hypothermic. Patients suffering from hypothermia and hemorrhagic shock are resistant to appropriate blood and fluid resuscitative measures and surgical treatment. The only true indication for vasopressors in hypovolemic shock is the hypothermic trauma patient who sustains a cardiac arrest. Rapid rewarming with an appropriate warming blanket, warm fluids, and blood generally corrects the patient's hypotension and hypothermia.

E. Pacemaker

About 150,000 such devices are placed in patients with myocardial conduction defects every year in North America. These patients are unable to respond to blood loss in the expected fashion. For this group, central venous pressure monitoring is invaluable to guide fluid therapy.
X. 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 will minimize 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. Under this circumstance, consider immediate surgical intervention.

B. Fluid Overload and CVP Monitoring

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

Central venous pressure monitoring is a relatively simple procedure and is used as a standard guide for assessing the right heart's ability to accept a fluid load. Properly interpreted, the response of the central venous pressure 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. The CVP is valid for gross evaluation of appropriate clinical situations. Remembering these facts is important to avoid overdependence on CVP monitoring.

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

3. A minimal rise in the initial, low central venous pressure with fluid therapy suggests the need for further volume expansion.
4. A declining central venous pressure suggests ongoing fluid loss and the need for additional fluid or blood replacement.

5. An abrupt or persistent elevation in the central venous pressure suggests volume replacement has been adequate, is too rapid, or that cardiac function has been compromised.

6. **Remember**, the central venous pressure 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 central venous pressure may be caused by catheter malposition, hypervolemia as a result of overtransfusion, cardiac dysfunction, or cardiac tamponade. Increased intrathoracic pressure from a pneumothorax may also cause an elevation in the central venous pressure.

Access for a central venous pressure line is through the antecubital, internal jugular, or subclavian veins. 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 will be discussed in detail in Skill Station IV - Percutaneous Venous Access.

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 infection, 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 physiological 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.
**Table 1. Estimated Fluid and Blood Requirements**
*(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>750-1500</td>
<td>1500-2000</td>
<td>2000 or more</td>
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<tr>
<td><strong>Blood Loss (%BV)</strong></td>
<td>up to 15%</td>
<td>15-30%</td>
<td>30-40%</td>
<td>40% or more</td>
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<td><strong>Pulse Rate</strong></td>
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<td>&gt; 100</td>
<td>&gt; 120</td>
<td>&gt; 140</td>
</tr>
<tr>
<td><strong>Blood Pressure</strong></td>
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<td>Normal</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td><strong>Pulse Pressure (mm Hg)</strong></td>
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<td>Decreased</td>
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<td>30-40</td>
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<td><strong>Urine Output (mL/hr)</strong></td>
<td>30 or more</td>
<td>20-30</td>
<td>5-15</td>
<td>Negligible</td>
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<td><strong>CNS - Mental Status</strong></td>
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<td>Mildly anxious</td>
<td>Anxious - confused</td>
<td>Confused - lethargic</td>
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<td><strong>Fluid Replacement (3:1 Rule)</strong></td>
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<td></td>
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<td>+ blood</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* 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 will 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 will have hypotension out of proportion to his blood loss and will require fluids in excess of the 3:1 guidelines. In contrast, a patient whose ongoing blood loss is being replaced will require less than 3:1. The use of bolus therapy with careful monitoring of the patient's response can moderate these extremes. (See Chapter 3 - Shock, "Therapeutic Decisions".)