

# 18

# Bleeding and Shock

## Competency Areas

### Area 4: Assessment and Diagnostics

- 4.3.a Conduct primary patient assessment and interpret findings.
- 4.3.b Conduct secondary patient assessment and interpret findings.
- 4.3.c Conduct cardiovascular system assessment and interpret findings.
- 4.3.d Conduct neurological system assessment and interpret findings.
- 4.3.e Conduct respiratory system assessment and interpret findings.
- 4.3.f Conduct obstetrical assessment and interpret findings.
- 4.3.g Conduct gastrointestinal system assessment and interpret findings.
- 4.3.h Conduct genitourinary system assessment and interpret findings.
- 4.3.i Conduct integumentary system assessment and interpret findings.
- 4.3.j Conduct musculoskeletal assessment and interpret findings.
- 4.3.k Conduct assessment of the immune system and interpret findings.
- 4.3.l Conduct assessment of the endocrine system and interpret findings.
- 4.3.m Conduct assessment of the ears, eyes, nose, and throat and interpret findings.
- 4.3.n Conduct multisystem assessment and interpret findings.
- 4.4.a Assess pulse.
- 4.4.b Assess respiration.
- 4.4.c Conduct non-invasive temperature monitoring.
- 4.4.d Measure blood pressure by auscultation.
- 4.4.e Measure blood pressure by palpation.

- 4.4.f Measure blood pressure with non-invasive blood pressure monitor.
- 4.4.g Assess skin condition.
- 4.4.h Assess pupils.
- 4.4.i Assess level of mentation.
- 4.5.a Conduct oximetry testing and interpret findings.
- 4.5.b Conduct end-tidal carbon dioxide monitoring and interpret findings.
- 4.5.c Conduct glucometric testing and interpret findings.
- 4.5.d Conduct peripheral venipuncture.
- 4.5.e Obtain arterial blood samples via radial artery puncture.
- 4.5.f Obtain arterial blood samples via arterial line access.
- 4.5.g Conduct invasive core temperature monitoring and interpret findings.
- 4.5.h Conduct pulmonary artery catheter monitoring and interpret findings.
- 4.5.i Conduct central venous pressure monitoring and interpret findings.
- 4.5.j Conduct arterial line monitoring and interpret findings.
- 4.5.k Interpret laboratory and radiological data.
- 4.5.l Conduct 3-lead electrocardiogram (ECG) and interpret findings.
- 4.5.m Obtain 12-lead electrocardiogram and interpret findings.

### Appendix 4: Pathophysiology

- J. **Multisystem Diseases and Injuries**
  - Shock Syndromes: Anaphylactic
  - Shock Syndromes: Cardiogenic
  - Shock Syndromes: Hypovolemic
  - Shock Syndromes: Neurogenic
  - Shock Syndromes: Obstructive
  - Shock Syndromes: Septic

## Introduction

After managing the airway, recognizing bleeding and understanding how it affects the body are perhaps the most important skills you will learn as a paramedic. Any kind of bleeding is potentially dangerous because it may eventually lead to shock. Uncontrolled bleeding may lead to serious injury and, ultimately, death.

Bleeding is also the most common cause of shock. As used in this chapter, **shock** describes a state of collapse and failure of the cardiovascular system. Shock is actually a normal compensatory mechanism used by the body to maintain systolic blood pressure (BP) and brain perfusion during times of distress. This response can accompany a broad spectrum of events, ranging from trauma to heart attacks to allergic reactions. If not treated promptly, shock will injure the body's vital organs and ultimately lead to death. Your early and rapid actions can help significantly reduce the morbidity and mortality rates from bleeding and shock.

## Anatomy and Physiology of the Cardiovascular System

The cardiovascular system is designed to carry out one crucial job: keep blood flowing via the lungs to the peripheral tissues. In the lungs, blood dumps the gaseous waste products of

metabolism—chiefly carbon dioxide—and picks up life-sustaining oxygen. In the peripheral tissues, the process is reversed: Blood unloads oxygen and picks up wastes. If blood flow were to stop or slow significantly, the results would be catastrophic. Oxygen delivery to the heart, brain, and other vital organs would be disrupted. For a few minutes, the cells could switch to an emergency metabolic system—one that does not require oxygen (anaerobic metabolism), but that form of metabolism produces even more acids and toxic wastes. The cells of the organs of the body would have nowhere to eliminate their wastes and would rapidly be affected by the toxic by-products of their own metabolism.

To keep the blood moving continuously through the body, the circulatory system requires three intact components

### Figure 18-1 ▶ :

- A functioning pump: the heart
- Adequate fluid volume: the blood and body fluids
- An intact system of tubing capable of reflex adjustments (constriction and dilation) in response to changes in pump output and fluid volume: the blood vessels

All three components must interact effectively to maintain life. If any one becomes damaged or is deficient, the whole system is in jeopardy.

## Structures of the Heart

The heart is a muscular, cone-shaped organ whose function is to pump blood throughout the body. Located behind the sternum, the heart is about the size of a closed fist, weighing 280 to 350 g

# You are the Paramedic Part 1

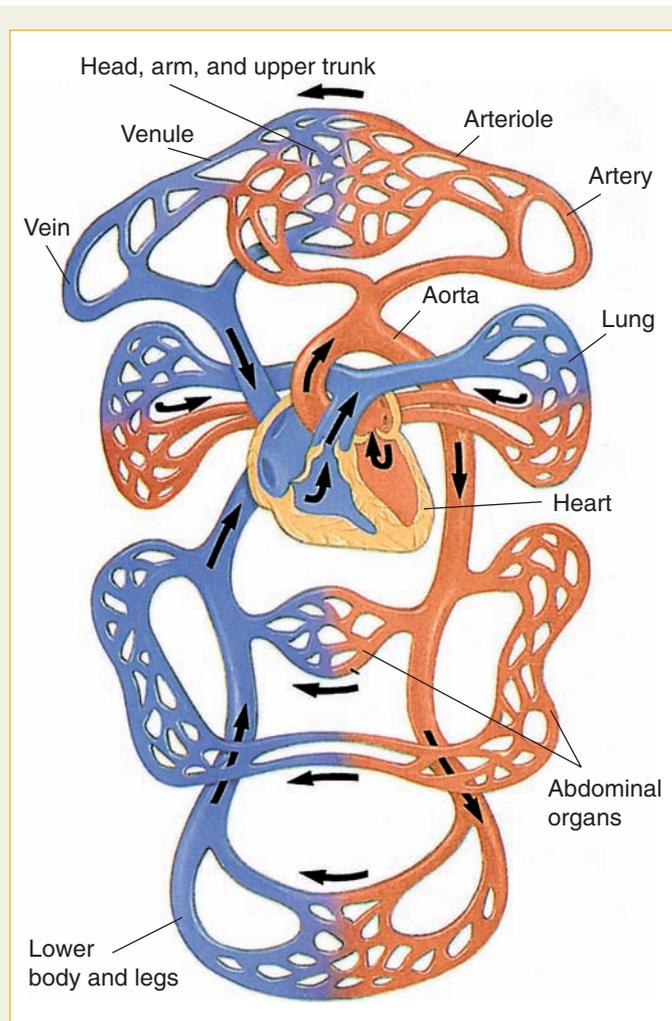
You and your partner have just finished dinner on what has been a quiet night shift. Suddenly, chatter erupts on the police scanner and you are called to a shooting at a local gas station/minimarket. The dispatcher alerts you that police on the scene are requesting “a rush on the bus.”

As you park in front of the store, you notice a crowd gathering on the sidewalk. Police officers are establishing a perimeter. You grab your initial assessment bag and head inside as your partner pulls out the stretcher with the help of a police officer.

As you enter the store, a detective informs you, “There was a robbery; the clerk was shot and the perpetrator has left the scene.” You observe a 22-year-old man sitting on the floor behind the counter and leaning against the wall. He is holding his left upper quadrant with his bloody hand. The patient appears to weigh about 80 kg. Although he is conscious, alert, and in obvious pain, he tells you that the shooting occurred just as the clock struck 23:00. It is now 23:10, and you hit the elapsed time counter on your digital watch as you don your personal protective equipment (PPE). As you begin to talk to the patient, you reach down to palpate his radial pulse but cannot feel it.

Initial Assessment	Recording Time: 0 Minutes
Appearance	Awake and anxious
Level of consciousness	A (Alert to person, place, and day)
Airway	Open and clear
Breathing	Rapid, shallow, and laboured
Circulation	Unpalpable radial pulse

1. Does the lack of significant visible bleeding and the fact that he is alert indicate that this patient is not bleeding seriously?
2. What is the significance of time in this type of incident?



**Figure 18-1** The circulatory system requires continuous operation of its three components: the heart, the blood and body fluids, and the blood vessels.

in men and 225 to 280 g in women. Roughly two thirds of the heart lies in the left part of the mediastinum, the area between the lungs that also contains the great vessels.

The human heart consists of four chambers: two atria (upper chambers) and two ventricles (lower chambers). Each atrium receives blood that is returned to the heart from other parts of the body; each ventricle pumps blood out of the heart. The upper and lower portions of the heart are separated by the atrioventricular valves, which prevent backward flow of blood. The semilunar valves, which serve a similar function, are located between the ventricles and the arteries into which they pump blood.

### Blood Flow Within the Heart

Two large veins, the superior vena cava and the inferior vena cava, return deoxygenated blood from the body to the right

atrium **Figure 18-2** . Blood from the upper part of the body returns to the heart through the superior vena cava; blood from the lower part of the body returns through the inferior vena cava (the larger of the two veins). From the right atrium, blood passes through the tricuspid valve into the right ventricle. The right ventricle then pumps the blood through the pulmonic valve into the pulmonary artery and then to the lungs.

In the lungs, oxygen is returned to the blood and carbon dioxide and other waste products are removed from it. The freshly oxygenated blood returns to the left atrium through the pulmonary veins. Blood then flows through the mitral valve into the left ventricle, which pumps the oxygenated blood through the aortic valve, into the aorta (the body's largest artery), and then to the entire body.

### The Cardiac Cycle

The cardiac cycle is the repetitive pumping process that begins with the onset of cardiac muscle contraction and ends with the beginning of the next contraction. Myocardial contraction results in pressure changes within the cardiac chambers, causing the blood to move from areas of high pressure to areas of low pressure. The valves ensure that blood is pumped in a forward direction.

The pressure in the aorta against which the left ventricle must pump blood is called the **afterload**. The greater the afterload, the harder it is for the ventricle to eject blood into the aorta. A higher afterload, therefore, reduces the **stroke volume**, or the amount of blood ejected per contraction.

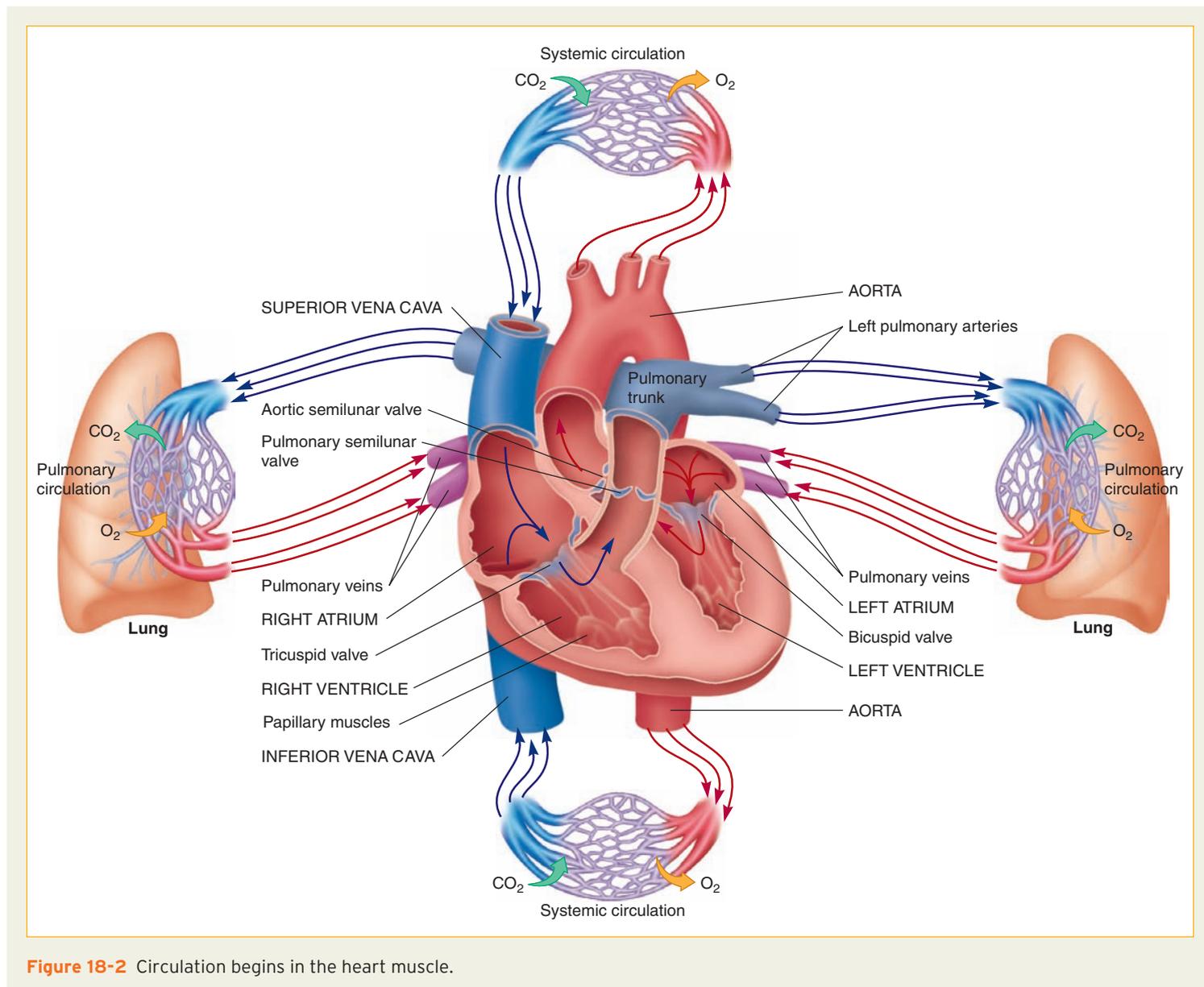
The amount of blood pumped through the circulatory system in 1 minute is referred to as the **cardiac output (CO)**. CO is expressed in litres per minute (l/min). The cardiac output equals the pulse rate multiplied by the stroke volume:

$$\text{Cardiac Output} = \text{Stroke Volume} \times \text{Pulse Rate}$$

Factors that influence the pulse rate, the stroke volume, or both will affect CO and, therefore, oxygen delivery (perfusion) to the tissues.

Increased venous return to the heart stretches the ventricles somewhat, resulting in increased cardiac contractility. This relationship, which was first described by the British physiologist Ernest Henry Starling, is known as the Starling law of the heart. Starling noted that if a muscle is stretched slightly before it is stimulated to contract, it would contract with greater force. Thus, if the heart is stretched, the muscle contracts more forcefully.

Although the amount of blood returning to the right atrium varies somewhat from minute to minute, a normal heart continues to pump the same percentage of blood returned, a measure called the **ejection fraction**. If more blood returns to the heart, the stretched heart pumps harder rather than allowing the blood to back up into the veins. As a result, more blood



**Figure 18-2** Circulation begins in the heart muscle.

is pumped with each contraction, yet the ejection fraction remains unchanged: The amount of blood that is pumped increases, but so does the amount of blood returned. This relationship maintains normal cardiac function when a person changes positions, coughs, breathes, or moves.

### Blood and Its Components

**Blood** consists of plasma and formed elements or cells that are suspended in the plasma. These cells include red blood cells (RBCs), white blood cells (WBCs), and platelets. The purpose of blood is to carry oxygen and nutrients to the tissues and cellular waste products away from the tissues. In addition, the formed elements serve as the mainstay of numerous other body functions, such as fighting infections and controlling bleeding.

**Plasma** is a watery, straw-coloured fluid that accounts for more than half of the total blood volume. It consists of 92% water and 8% dissolved substances such as chemicals, minerals, and nutrients. Water enters the plasma from the digestive tract, from fluids between cells, and as a by-product of metabolism.

The disk-shaped RBCs (**erythrocytes**) are the most numerous of the formed elements. Erythrocytes are unable to move on their own; instead, the flowing plasma passively propels them to their destinations. RBCs contain **hemoglobin**, a protein that gives them their reddish colour. Hemoglobin binds oxygen that is absorbed in the lungs and transports it to the tissues where it is needed.

Several types of WBCs (**leukocytes**) exist, each of which has a different function. The primary function of all WBCs is to

fight infection. Antibodies to fight infection may be produced, or leukocytes may directly attack and kill bacterial invaders.

**Platelets** are small cells in the blood that are essential for clot formation. The blood clotting (coagulation) process is a complex series of events involving platelets, clotting proteins in the plasma (clotting factors), other proteins, and calcium. During coagulation, platelets aggregate in a clump and form much of the foundation of a blood clot. Clotting proteins produced by the liver solidify the remainder of the clot, which eventually includes red and white blood cells.

### Blood Circulation and Perfusion

Arteries are blood vessels that carry blood away from the heart. Veins are blood vessels that transport blood back to the heart. As arteries get farther from the heart, they become smaller. Eventually, they branch into many small arterioles, which themselves divide into even smaller capillaries (microscopic, thin-walled blood vessels). Oxygen and nutrients pass out of the capillaries and into the cells, and carbon dioxide and waste products pass from the cells and into the capillaries by a process called diffusion. To return deoxygenated blood to the heart, groups of capillaries gradually enlarge to form venules. Venules then merge together, forming larger veins that eventually empty into the heart, via the superior and inferior vena cavae.

**Perfusion** is the circulation of blood within an organ or tissue in adequate amounts to meet the cells' current needs for oxygen, nutrients, and waste removal. Blood must pass through the cardiovascular system at a speed that is fast enough to maintain adequate circulation throughout the body, yet slow enough to allow each cell time to exchange oxygen

and nutrients for carbon dioxide and other waste products. Although some tissues, such as the lungs and kidneys, never rest and require a constant blood supply, most tissues require circulating blood only intermittently, but especially when they are active. Muscles, for example, are at rest and require a minimal blood supply when you sleep. In contrast, during exercise, muscles need a large blood supply. As another example, the gastrointestinal (GI) tract requires a high flow of blood after a meal. After digestion is completed, it can do quite well with a small fraction of that flow.

The autonomic nervous system monitors the body's needs from moment to moment, adjusting the blood flow as required. During emergencies, it automatically redirects blood away from other organs and toward the heart, brain, lungs, and kidneys. Thus, the cardiovascular system is dynamic, constantly adapting to changing conditions. Sometimes, however, it fails to provide sufficient circulation for every body part to perform its function, resulting in hypoperfusion or shock.

The heart requires constant perfusion, or it will not function properly. The brain and spinal cord cannot go for more than 4 to 6 minutes without perfusion, or the nerve cells will be permanently damaged—recall that cells of the central nervous system do not have the capacity to regenerate. The kidneys will be permanently damaged after 45 minutes of inadequate perfusion. Skeletal muscles cannot tolerate more than 2 hours of inadequate perfusion. The GI tract can exist with limited (but not absent) perfusion for several hours. These times are based on a normal body temperature (37.0°C). An organ or tissue that is considerably colder is better able to resist damage from hypoperfusion because of the slowing of the body's metabolism.

## You are the Paramedic Part 2

Additional help arrives on the scene as you complete your initial assessment. Your partner has brought in the stretcher and is beginning to administer supplemental oxygen via a nonrebreathing mask at 15 l/min. Police inform you that the robber's weapon may have been a "sawed-off shotgun" that was fired at a fairly close range. The patient tells you a single shot was fired after he told the robber that he would not open the safe.

You give the patient some gauze and tell him to hold it firmly against the wound. When you complete your initial assessment of the patient, you decide to perform the rapid physical examination in the back of the ambulance and the SAMPLE history as you have time, given the higher priorities and need for rapid transport.

Initial Assessment	Recording Time: 3 Minutes
Breathing	Rapid, laboured, but no obstructions to breathing process (flail segment, punctures, or impaled objects). Oxygen has been started.
Circulation	A rapid, weak carotid pulse can be felt, the external bleeding is easily controlled with direct pressure and gauze. The skin is pale, cool, and moist.

- On the basis of the information you have so far, and remembering that the patient weighs approximately 80 kg, how much blood did he have before the incident? How much could he have lost so far?
- What phase or stage of shock is this patient in?
- Which BLS and ALS interventions would be most appropriate for this patient at this time? Should you insert an intravenous (IV) line at the scene?

## Pathophysiology of Hemorrhage

**Hemorrhage** simply means bleeding. Bleeding can range from a “nick” to a capillary while shaving, to a severely spurting artery from a deep slash with a knife, to a ruptured spleen from striking the steering column during a car collision. External bleeding (visible hemorrhage) can usually be easily controlled by using direct pressure or a pressure bandage. Internal bleeding (hemorrhage that is not visible) is usually not controlled until a surgeon locates the source and sutures it closed. Because internal bleeding is not as obvious, you must rely on signs and symptoms to determine the extent and severity of the hemorrhage.

### External Hemorrhage

External bleeding is usually due to a break in the skin. Its extent or severity is often a function of the type of wound and the types of blood vessels that have been injured. (Wound types are discussed in detail in Chapter 19.) Bleeding from a capillary usually oozes, bleeding from a vein flows, and bleeding from an artery spurts.

These descriptions are not infallible. For example, considerable oozing from capillaries is possible when a patient gets a very large abrasion (such as the road rash when a cyclist slides along the pavement without protective clothing). Likewise, varicose veins on the leg can produce copious bleeding.

Arteries may spurt initially, but as the patient's BP decreases, often the blood simply flows. In addition, an artery that is incised directly across or in a transverse manner will often recoil and attempt to slow its own bleeding. By contrast, if the artery is cut on a bias, it does not recoil and continues to bleed.

Some injuries that you might expect to be accompanied by considerable external bleeding do not always have serious hemorrhaging. For example, a person who falls off the platform at the train station and is run over by a train may have amputations of one or more extremities, yet experience little bleeding because the wound was cauterized by the heat of the train's wheels on the rail. Conversely, a person who pulled over on the shoulder of the road and was removing the jack from his car's trunk when another motorist slammed into the rear of the car, pinning him between the two vehicles, may have severely crushed legs. In such a case, bleeding may be severe, with the only effective means of bleeding control being two tourniquets.

### Internal Hemorrhage

Internal bleeding as a result of trauma may appear in any portion of the body. A fracture of a small bone (such as humerus, ankle, or tibia) produces a somewhat controlled environment in which a relatively small amount of bleeding can occur. By contrast, bleeding into the trunk (that is, thorax, abdomen, or pelvis), because of its much larger space, tends to be severe and uncontrolled. Nontraumatic internal hemorrhage usually occurs in cases of GI bleeding from the upper or lower GI tract, ruptured ectopic pregnancies, ruptured aneurysms, or other conditions.

Any internal bleeding must be treated promptly. The signs of internal hemorrhage (such as hematoma) do not always develop quickly, so you must rely on other signs and symptoms and an evaluation of the mechanism of injury (MOI) to make this diagnosis. Pay close attention to patient complaints of pain or tenderness, development of tachycardia, and pallor. In addition to evaluating the MOI, be alert for the development of shock when you suspect internal bleeding.

Management of a patient with internal hemorrhaging focuses on the treatment of shock, minimizing movement of the injured or bleeding part or region, and rapid transport. The patient will likely need a surgical procedure to stop the bleeding. In recent years, ultrasound has been used to locate bleeding in the emergency department (ED) before moving the patient to the surgical suite for the ultimate resolution of the problem.

### Controlled Versus Uncontrolled Hemorrhage

Bleeding that you can control (such as external bleeding that responds to a pressure bandage) and bleeding that you cannot control (such as a bleeding peptic ulcer) are serious emergencies. As a consequence, the initial assessment of the patient includes a search for life-threatening bleeding. If found, the hemorrhage must be controlled; if the hemorrhage cannot be controlled in the prehospital environment, all of your efforts should concentrate on attempting to control the bleeding as you rapidly transport the patient to the ED.

Most external bleeding can be managed with direct pressure, although arterial bleeding may take 5 or more minutes of direct pressure to form a clot. Military experience has shown that the use of pressure points is not as effective as previously thought and is difficult to manage while trying to rapidly evacuate a person from the battlefield. For this reason, most military medical training calls for use of a tourniquet for external bleeding to an extremity that cannot be controlled with direct pressure and a pressure bandage.

Because most cases of internal bleeding are rarely fully controlled in the prehospital setting, a patient with this type of injury needs rapid transport to the ED. Some strategies may be effective in the prehospital environment depending on the cause of the bleeding. For example, the external circumferential pressure of the pneumatic antishock garment/military antishock trousers (PASG/MAST) may help control the massive bleeding that accompanies a pelvic fracture. The paramedic should consult their local or regional protocols regarding use of binders or PASG/MAST.

### The Significance of Bleeding

When patients have serious external hemorrhage, it is often difficult to determine the amount of blood loss. Blood looks different on different surfaces, such as when it is absorbed in clothing versus when it has been diluted by being mixed in water. Although you should attempt to determine the amount of external blood loss, the patient's presentation and your assessment will ultimately direct your prehospital care and treatment plan.



### At the Scene

Not every person has the same amount of blood, so you must be able to estimate the patient's blood volume before the trauma occurred. Blood volume is relative to weight—it accounts for 6% to 8% of the total body weight. One litre of blood weighs approximately 1 kg.

Human adult male bodies contain approximately 70 ml of blood per kilogram of body weight, whereas adult female bodies contain approximately 65 ml/kg. The body cannot tolerate an acute blood loss of more than 20% of this total blood volume. If the typical adult loses more than 1 l of blood, significant changes in vital signs will occur, including increasing heart and respiratory rates and decreasing BP. An isolated femur fracture, for example, can easily result in the loss of 1 l or more of blood in the soft tissues of the thigh.

Because infants and children have less blood volume than their adult counterparts, they may experience the same effect with smaller amounts of blood loss. For example, a 1-year-old child has a total blood volume of about 800 ml, so significant symptoms of blood loss may occur after only 100 to 200 ml of blood loss. To put this in perspective, remember that a soft drink can holds roughly 345 ml of liquid **Figure 18-3** ▾.

How well people compensate for blood loss is related to how rapidly they bleed. A healthy adult can comfortably donate one unit (500 ml) of blood in a period of 15 to 20 minutes without having ill effects from this decrease in blood volume. If a similar blood loss occurs in a much shorter period, hypovolemic shock, a condition in which low blood volume results in inadequate perfusion and even death, may rapidly develop.



**Figure 18-3** A soft drink can holds roughly 345 ml of liquid.

Consider bleeding to be serious if any of the following conditions are present:

- A significant MOI, especially when the MOI suggests that severe forces affected the abdomen or chest
- Poor general appearance of the patient
- Signs and symptoms of shock
- Significant amount of blood loss
- Rapid blood loss
- Uncontrollable bleeding

### Physiologic Response to Hemorrhage

Typically, bleeding from an open artery is bright red (because of the high oxygen content) and spurts in time with the pulse. The pressure that causes the blood to spurt also makes this type of bleeding difficult to control. As the amount of blood circulating in the body drops, so does the patient's BP and, eventually, the arterial spurting diminishes.

Blood from an open vein is much darker (low oxygen content) and flows steadily. Because it is under less pressure, most venous blood does not spurt and is easier to manage. Bleeding from damaged capillary vessels is dark red and oozes from a wound steadily but slowly. Venous and capillary bleeding is more likely to clot spontaneously than arterial bleeding.

On its own, bleeding tends to stop rather quickly, within about 10 minutes, in response to internal clotting mechanisms and exposure to air. When vessels are lacerated, blood flows rapidly from the open vessel. The open ends of the vessel then begin to narrow (vasoconstrict), which reduces the amount of bleeding. Platelets aggregate at the site, plugging the hole and sealing the injured portions of the vessel, a process called **hemostasis**. Bleeding will not stop if a clot does not form. Direct contact with body tissues and fluids or the external environment commonly triggers the blood's clotting factors.

Despite the efficiency of this system, it may fail in certain situations. A number of medications, including anticoagulants such as aspirin and prescription blood thinners, interfere with normal clotting. With a severe injury, the damage to the vessel may be so extensive that a clot cannot completely block the hole. Sometimes, only part of the vessel wall is cut, preventing it from constricting. In these cases, bleeding will continue unless it is stopped by external means. In a case involving acute blood loss, the patient might die before the body's hemostatic defenses of vasoconstriction and of clotting can help.

### Assessment of a Bleeding Patient

The assessment of any patient begins with a good scene assessment and proceeds to your general impression and initial assessment. Once the scene is deemed safe to enter, you will need to don the appropriate level of personal protective equipment (PPE). Depending on the severity of bleeding and your initial diagnosis, this will entail gloves, mask, eyeshield, and, when the patient is very bloody or blood is spurting, a gown **Figure 18-4** ▶.



**Figure 18-4** Depending on the severity of bleeding and your initial diagnosis, PPE will entail gloves, mask, eyeshield, and, in some cases, a gown.

During the initial assessment, after determining the patient's mental status with AVPU, you must locate and manage immediate threats to life involving the airway, breathing, and circulation. Ensure that the patient has a patent airway. If you observe bleeding from the mouth or facial areas, keep the suction unit within reach.

If the patient has minor external bleeding, you can note it and move on with the initial assessment; management of this problem can wait until the patient

has been properly assessed and prioritized. Do not get sidetracked by applying dressings and bandages to a patient who has much more serious problems. If major external bleeding is present, you should deal with it during the initial assessment. If you suspect internal bleeding, begin management by keeping the patient warm and administering supplemental oxygen by a non-rebreathing mask at 15 l/min.



### At the Scene

When you are dealing with a bleeding patient, be sure to take necessary precautions to protect yourself from splashing or splattering. Wear appropriate PPE, including gloves, gown, mask, and eye protection. This is especially essential when arterial bleeding is present. Also remember that frequent, thorough handwashing between patients and after every call is a simple yet important protective measure.

Carefully assess the MOI in trauma patients because it may be your best indicator that the patient has sustained an internal injury and may be bleeding. **Table 18-1** lists some MOIs that can give clues about internal bleeding.

During your focused history, elaborate on the patient's chief complaint using the OPQRST mnemonic, and obtain a history of the present illness using SAMPLE. Are there any



### At the Scene

Consider any patient exhibiting signs and symptoms of shock without obvious injury to have probable internal bleeding, usually in the abdominal cavity.

**Table 18-1** The MOI: Indicators of Internal Bleeding

Mechanism of Injury	Potential Internal Bleeding Sources
Fall from a ladder striking head	Head injury or hematoma
Fall from a ladder striking extremities	Possible fractures; consider chest injury
Child struck by car (Waddell triad)	Head trauma, chest and abdomen injuries, leg fractures
Fall on outstretched arm	Possible broken bone or joint injury
Child thrown or falls from height	Children usually have a head-first impact, causing head injury
Unrestrained driver in head-on collision (up-and-over route)	Head and neck, chest, abdomen injuries
Unrestrained driver in head-on collision (down-and-under route)	Knees, femur, hip, and pelvis injuries
Unrestrained front-seat passenger, side impact collision with intrusion into vehicle	Humerus broken exposing the chest wall (possible flail chest); pelvis and acetabulum injuries
Unrestrained driver crushed against steering column	Chest and abdomen injuries, ruptured spleen, neck trauma
Road bike or mountain bike (over the handlebars)	Fractured clavicle, road rash, head trauma if no helmet
Abrupt motorcycle stop, causing rider to catapult over the handlebars	Fractured femurs, head and neck injuries
Diving into the shallow end of a swimming pool	Head and neck injuries
Assault or fight	Punching or kicking injury to chest and abdomen and the face
Blast or explosion	Injury from direct strike with debris; indirect and pressure wave in enclosed space

signs and symptoms of hypovolemic shock? Ask the patient if they have pain or if he or she experiences any dizziness. Ask the patient about current medications that may thin the blood and about any history of clotting insufficiency. Assess for tenderness, bruising, guarding, or swelling. These signs and symptoms may indicate internal bleeding.

During the physical examination (rapid or focused, depending on the MOI), note the colour of bleeding and try to determine its source. Bright red blood from a wound or the mouth, rectum, or other orifice indicates fresh arterial bleeding. Coffee-ground emesis is a sign of upper GI bleeding; this kind of blood is old and looks like used coffee grounds.

**Melena**, the passage of dark, tarry stools, indicates lower GI bleeding. **Hematochezia**, by contrast, is the passage of stools containing bright red blood and may indicate bleeding near the external opening of the colon. Hemorrhoids in the lower colon



### Special Considerations

In older patients, dizziness, syncope, or weakness may be the first sign of nontraumatic internal hemorrhage.

tend to cause hematochezia. Hematuria (blood in the urine) may suggest serious renal injury or illness. Nonmenstrual vaginal bleeding is always significant as well.

## Management of a Bleeding Patient

Always wear appropriate PPE when treating bleeding patients. As with all prehospital care, ensure that the patient has an open airway and is breathing adequately. Provide high-flow supplemental oxygen, and assist ventilation if needed, paying special attention to cervical spine control in trauma patients.

### Managing External Hemorrhage

To control external hemorrhaging, follow these steps:

1. Apply direct pressure over the wound.
2. Elevate the injury above the level of the heart if no fracture is suspected.
3. Apply a pressure dressing.

A tourniquet is generally used only as a last resort, when it may be necessary to sacrifice the limb to save the life.

### Bleeding From the Nose, Ears, and Mouth

Bleeding from the nose (*epistaxis*) or bleeding from the ears following a head injury may indicate a skull fracture. In such a case, you should not attempt to stop the blood flow. If you suspect a skull fracture, cover the bleeding site loosely with a sterile gauze pad to collect the blood and help keep contaminants away from the site—there is always a risk of infection to the brain with a skull fracture. Apply light compression by wrapping the dressing loosely around the head. If blood or drainage contains cerebrospinal fluid, there will be a characteristic staining of the dressing that resembles a bull's-eye target.

### Bleeding From Other Areas

With bleeding from other areas of the body, control bleeding through use of direct pressure and elevation, if appropriate. Apply pressure dressings, especially at pressure points for the upper and lower extremities. In addition, use splints as necessary, always following your local protocols. Pack large, gaping wounds with sterile dressings. Consider applying the PASG/MAST, if your local protocols permit, but reserve the tourniquet for use as a last resort.

Once bleeding is controlled and a sterile dressing and pressure bandage have been applied, keep the patient warm and in the appropriate position. Allow the patient's condition to dictate the mode of transport.

### Special Management Techniques for External Hemorrhage

Much of the bleeding associated with broken bones occurs because the sharp ends of the bones lacerate vessels, muscles, and other tissues. As long as a fracture remains unstable, the bone ends will move and continue to damage tissues and vessels. They may also break up clots that have partially formed, resulting in ongoing bleeding. For these reasons, immobilizing a fracture is a priority in the prompt control of bleeding. Often, simple splints will quickly control the bleeding associated with a fracture. If not, you may need to use another splinting device.

#### Air Splints

Air splints can control the bleeding associated with severe soft-tissue injuries, such as massive or complex lacerations, or with fractures. They also stabilize the fracture itself. An air splint acts like a pressure dressing applied to an entire extremity rather than to a small, local area.

Once you have applied an air splint, monitor circulation in the distal extremity. Because an air splint is typically inflated to approximately 50 mm Hg (so you can still dent the splint with your fingertips), it would not be appropriate to use on arterial bleeding because the splint would not actually control the bleeding until the patient's systolic BP dropped to the pressure of the splint.

#### Hemostats

Although hemostats may be helpful when a vessel has been severed, especially if it has retracted into the surrounding tissue, they often cause significant damage to the vessel and surrounding tissue. Be sure to check your local protocols about the use of hemostats in your area.

#### Tourniquets

The tourniquet is useful if a patient is bleeding severely from a partial or complete amputation and other methods of bleeding control have proved ineffective. The paramedic should realize that its application can cause permanent damage to nerves, muscles, and blood vessels, resulting in the loss of an extremity. The procedure for tourniquet application is shown in Skill Drill 19-2 in Chapter 19. Whenever applying a tourniquet, make sure you observe the following precautions:

- Do not apply a tourniquet directly over any joint. Keep it as close to the injury as possible.
- Use the widest bandage possible. Make sure that it is tightened securely.
- Never use wire, rope, a belt, or any other narrow material as the tourniquet; it could cut into the skin.
- Use wide padding under the tourniquet, if possible, to protect the tissues and help with arterial compression.

- Never cover a tourniquet with a bandage. Leave it open and in full view.
- Do not loosen the tourniquet after you have applied it. Hospital personnel will loosen it once they are prepared to manage the bleeding.

### Pneumatic Antishock Garment/Military Antishock Trousers

MAST, also known as PASG, is an inflatable garment that surrounds the legs and abdomen of a patient to provide circumferential pressure. It is by far one of the most controversial pieces of equipment used in the prehospital setting. This device is primarily used for controlling blood loss and is not designed for resuscitation, except with authorization of direct medical control in a few situations of extreme hypotension **Figure 18-5** ▾.

In the 1980s, researchers began to question whether this device, and IV fluid infusion, were really effective in the treatment of shock. The use of this device remains highly controversial, be sure to check with and adhere to your local protocols **Figure 18-6** ▶.

By applying uniform pressure to sources of bleeding, the PASG/MAST, when pumped up to the point where the Velcro crackles (60 to 80 mm Hg), seems to control bleeding and promote hemostasis. The circumferential pressure also compresses the tissue and vessels and, ultimately, results in a decrease in the vascular container size under the suit. With this increase in the systemic vascular resistance (SVR), it has been theorized that a small amount of blood (approximately 200 ml) is autotransfused back to the torso and the mean arterial pressure (MAP) increases. These effects increase the patient's CO.

The PASG/MAST raises the BP of a patient in shock. Whether elevating the BP is beneficial has not been proved. Some researchers believe that raising the BP before bleeding has been controlled may have harmful effects, for example by

promoting further bleeding. However, raising the patient's BP improves perfusion to vital organs and can be useful to *paramedics*, because veins that were collapsed and invisible may "pop up" after the device has been inflated, making the job of inserting IV lines easier.

The inflated device provides a good splint for a fractured pelvis, but only does a marginal job splinting fractures of the lower extremities. Ideally, fractures of the femur should be traction-splinted in conjunction with the application of the PASG/MAST.

As yet, we do not know whether the PASG/MAST improves the overall outcome for seriously injured patients. Recent research at Baylor College of Medicine suggests that, at least in certain types of injuries, the device does not improve chances of survival and may adversely affect the outcome. Medical directors of local EMS systems should stay abreast of the research in this area and make their decisions regarding deployment of the device accordingly.

In EMS systems that continue to use the PASG/MAST, use is appropriate in patients with shock from blood loss (hemorrhagic shock) in the following circumstances:

- To stabilize suspected pelvic fractures with hypotension
- To begin to control severe hypotension (systolic BP < 50 to 60 mm Hg)
- To begin to control suspected intraperitoneal bleeding with hypotension (solid organs such as the liver and spleen, mesenteric vessels)
- To begin to control retroperitoneal bleeding with hypotension (such as in kidneys, aorta, and vena cavae)

Current contraindications to use of the PASG/MAST include the following:

- Penetrating thoracic trauma
- Splinting of the lower extremities in the absence of hypotension. The PASG/MAST is not a good splint and has been known to cause compartment syndrome of the calf when fractures were present.

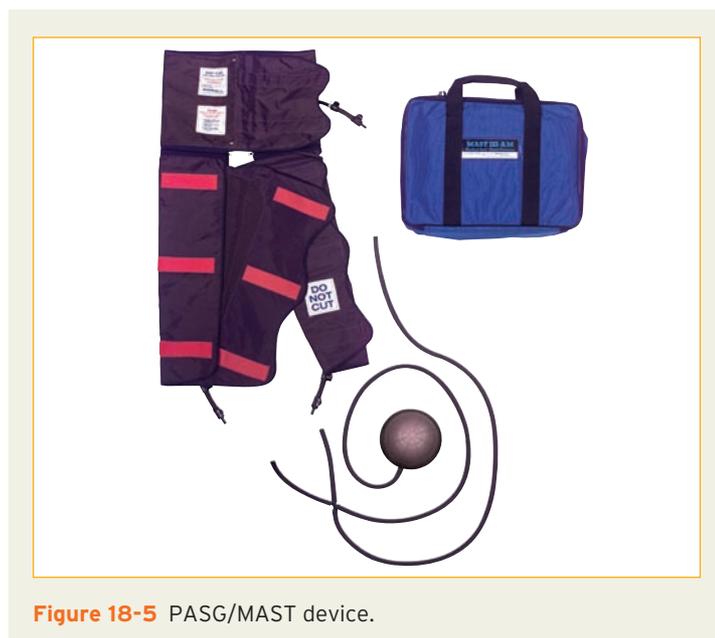


Figure 18-5 PASG/MAST device.



Figure 18-6

- Evisceration of abdominal organs
- Impaled objects of the abdomen
- Pregnancy
- Acute pulmonary edema
- Traumatic cardiac arrest
- Major head injuries

The steps in applying the device are described here and shown in **Skill Drill 18-1** :

1. Rapidly expose and examine the areas to be covered by the PASG/MAST. Pad any exposed bone ends to prevent puncture of the garment as it is inflated.
2. Apply the garment. If you will immobilize or move the patient on a backboard, lay the device out on the board before rolling the patient onto it. Position the top of the abdominal section of the PASG/MAST below the lowest rib to ensure that it does not compromise chest expansion **Step 1**.
3. Close and fasten both leg compartments and the abdominal compartment **Step 2**.
4. Open the stopcocks (valves) to the compartments you are preparing to inflate, ensuring that the other compartments are closed off.
5. Auscultate breath sounds for pulmonary edema before inflation **Step 3**.
6. Inflate the compartments with the foot pump to 60 to 80 mm Hg (until the Velcro crackles). Turn off compartment valves after inflation to maintain pressure in the garment. When using the device to stabilize a pelvic fracture, apply pressure only until the garment is firm to the touch. Overinflation may cause the bones to shift, creating further injury and bleeding. Higher inflation pressures may cause local tissue damage and/or compartment syndrome **Step 4**.
7. Check the patient's BP. Continue to monitor serial vital signs at least every 5 minutes because a patient who is subjected to this intervention is considered unstable. Remember that the pressure gauges on the device

## Skill Drill 18-1: Applying PASG/MAST



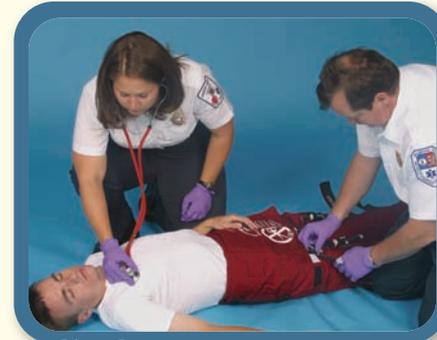
Step 1

Rapidly expose and examine the areas to be covered by the device. Pad any exposed bone ends. Apply the garment so that the top is below the lowest rib.



Step 2

Close and fasten both leg compartments and the abdominal compartment.



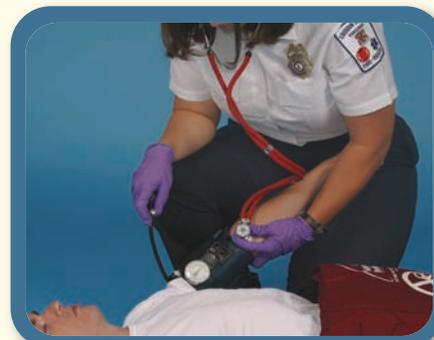
Step 3

Open the three stopcocks (valves). Auscultate breath sounds for pulmonary edema before inflation.



Step 4

Inflate with the foot pump until the Velcro crackles (approximately 60 to 80 mm Hg).



Step 5

Check the patient's BP again, and begin transport if not already in the transport mode.

measure the air pressure in the device—not the patient's BP. Be aware of temperature extremes or external pressure changes that might significantly affect the pressure exerted by the PASG/MAST, thus requiring frequent monitoring and adjustment (**Step 5**).

The simplest rule to remember regarding deflation of the PASG/MAST is this: Do not deflate the device in the prehospital environment. To the extent that the device supports the BP and provides hemostasis, the effects will be reversed when the PASG/MAST is deflated. An extreme case in which it might be necessary to deflate the device in the prehospital setting—albeit with direct medical control's permission—would be for a suspected ruptured diaphragm (causing abdominal contents to herniate into the chest cavity immediately after inflation of the device). It is desirable, therefore, to have restored at least some of the patient's circulating blood volume before releasing the pressure provided by the PASG/MAST. Remember that the patient's container size will have been decreased and deflation will increase the container, potentially leading to dramatic declines in the SVR, preload (venous return), and CO.

Before the PASG/MAST is deflated in the hospital setting, the patient should have at least two large-bore IV infusions running, with typed and cross-matched blood on standby. If the patient's serial vital signs are relatively stable and the ED physician so instructs, cautious deflation of the PASG/MAST may proceed as follows:

1. Record the patient's pulse and BP.
2. Slowly deflate the abdominal section *only*.
3. Recheck the patient's serial vital signs for 5 to 10 minutes. If the BP drops by 5 mm Hg or more, infuse 100 to 200 ml of fluid until the BP restabilizes.
4. When the patient's vital signs are again stable, slowly deflate one leg section.
5. Recheck the vital signs for 5 to 10 minutes. If there is another BP drop, again infuse volume until the BP comes back up.
6. If vital signs are stable, deflate the other leg section—again slowly, with careful monitoring of BP every few minutes.

In severely injured patients, this deflation procedure, which can take between 20 and 60 minutes, will usually not be feasible. Instead, the patient must be taken straight to the operating room with the PASG/MAST still on and inflated.

### Managing Internal Hemorrhage

The definitive management of a patient with internal hemorrhage occurs in the hospital. Prehospital management of suspected internal bleeding involves treating for shock and splinting injured extremities:

1. Keep the patient supine, open the airway, and check breathing and pulse.
2. Administer high-flow supplemental oxygen and assist ventilation if needed.
3. Splint broken bones or joint injuries. If a pelvic fracture is suspected, you may consider use of the PASG/MAST if your local protocols permit.

4. Place blankets under and over the patient to maintain body heat.
5. If no fractures are suspected, elevate the legs 30 cm. While en route to the ED, insert a large-bore (14- or 16-gauge) IV cannula, and administer a fluid challenge of 250 ml (provided the lungs are clear). Insert an IV line at the scene only if transport is delayed (such as if the patient is entrapped). Whenever possible, use warm IV fluids to prevent the patient from becoming chilled.
6. Consider giving pain medication if the vital signs are stable and after consultation with direct medical control.
7. Monitor the serial vital signs, and watch diligently for developing shock.

If the patient shows any signs of shock (hypoperfusion), transport rapidly while providing aggressive management en route. Because a patient in shock is usually emotionally upset, you should provide psychological support as well.

## Transportation of Patients With Hemorrhage

In case of hemorrhage, the issue is not whether the patient will be transported, but rather how fast the transport decision should be made and where the patient should be taken for definitive care. There are a few exceptions to this rule—for example, in the case of a minor wound, the decision to transport a patient should take into consideration factors such as the need for stitches, whether the patient has had a tetanus shot in the past 10 years, and whether the patient or his or her companion is reliable and will follow up properly. (Wounds are discussed in more detail in Chapter 19.)

Most patients with internal or external hemorrhage will need to be transported to a hospital for further care. Consideration for the priority of the patient and the availability of a regional trauma centre should be your concerns when making a transport decision in such cases. Patients who have severe internal or external bleeding, especially if uncontrolled, will usually be candidates for surgical interventions and should be transported to a facility with those capabilities. Patients with specific causes of bleeding such as major trauma or specific devastating wounds (such as leg amputation, glove avulsion) should be taken to a facility that is fully prepared to care for the patient. In EMS systems with helicopters available, it may be appropriate to consider this method of transportation for a patient with suspected severe internal or uncontrollable external bleeding.

## Pathophysiology of Shock

**Hypoperfusion** occurs when the level of tissue perfusion decreases below normal. Early decreased tissue perfusion may result in subtle changes, such as altered mental status, before a

patient's vital signs (blood pressure, pulse, respiratory rate) appear abnormal. Shock refers to a state of collapse and failure of the cardiovascular system that leads to inadequate circulation, creating inadequate tissue perfusion. Like internal bleeding, shock cannot be seen. It is not a specific disease or injury, but rather a dangerous condition that results in inadequate flow of

### Notes from Nancy

Don't wait until the BP falls before you suspect shock and begin treatment!



blood to the body's cells and failure to rid the body of metabolic wastes.

When the body senses tissue hypoperfusion, compensatory mechanisms are

set into action. In some cases, this is enough to stabilize the patient's condition. In other cases, the severity of disease or injury overwhelms the normal compensatory mechanisms, leading to progressive deterioration in the patient's condition. Evaluation of a patient's level of organ perfusion is important in diagnosing shock. If the conditions causing shock are not promptly addressed, the patient will rapidly deteriorate. Perfusion depends on CO, SVR, and transport of oxygen:

$$\text{Cardiac Output} = \text{Pulse Rate} \times \text{Stroke Volume}$$

$$\text{Blood Pressure} = \text{Cardiac Output} \times \text{Systemic Vascular Resistance}$$

Because the heart cannot pump out what is not in its holding chambers, BP varies directly with CO, SVR, and blood volume. Hypoperfusion, therefore, can result from inadequate CO, decreased SVR, or the inability of RBCs to deliver oxygen to tissues.



### At the Scene

Remember that BP may be the last measurable factor to change in shock. The body has several automatic mechanisms to compensate for initial blood loss and to help maintain BP. Thus, by the time you detect a drop in BP, shock is well developed. This is particularly true in infants and children, who can maintain their BP until they have lost close to half their blood volume.

## Mechanisms of Shock

Recall that normal tissue perfusion requires three intact mechanisms: a pump (heart), fluid volume (blood and body fluids), and tubing capable of reflex adjustments (constriction and dilation) in response to changes in pump output and fluid volume (blood vessels). If any one of those mechanisms is damaged, tissue perfusion may be disrupted, and shock will ensue.

When shock arises because of failure of the heart's ability to pump effectively due to muscle dysfunction, it is called **cardiogenic shock** (*cardio* = heart + *genic* = causing). Cardiac arrest is the most drastic form of cardiogenic shock, but not the only form. Cardiogenic shock may occur secondary to

myocardial infarction, cardiac arrhythmias, pulmonary embolism, severe acidosis, or a variety of other conditions. All of these conditions have one thing in common: They interfere with the heart's ability to pump normally.

Shock may also occur because of a loss of fluid volume; perfusion cannot take place if there isn't enough fluid to propel through the system. When shock comes about because of inadequate volume, it is termed hypovolemic shock (*hypo* = deficient + *vol* = volume + *emia* = in the blood). Volume can be lost as blood (hemorrhagic shock), plasma (as in burns), or electrolyte solution (as in vomiting, diarrhea, sweating). Suspect a hypovolemic component of shock in any patient with unexplained shock, and treat the patient for hypovolemia first.

### Notes from Nancy

Suspect a hypovolemic component of shock in any patient with unexplained shock, and treat for hypovolemia first.



Failure of vasoconstriction (that is, a decrease in the peripheral vascular resistance [PVR]) may lead to neurogenic shock, so called because the sympathetic nervous system ordinarily controls the dilation and constriction of blood vessels. In a healthy person, the calibre of the blood vessels constantly changes in response to signals from the nervous system, allowing the body to adapt to changes in position, fluid volume, and so forth. When you stand up, for example, blood vessels in your legs reflexively constrict to divert the circulation toward more vital areas, like the brain. Similarly, when you donate a pint of blood or sweat a litre of fluid, your blood vessels constrict to accommodate a smaller fluid volume. In certain situations, nervous system control over the calibre of blood vessels becomes deranged—for example, after spinal cord injury—and the blood vessels lose their tone and dilate. A given blood volume then has to be accommodated quite suddenly in a much larger container. The net effect is a relative hypovolemia (the volume in the container is now inadequate relative to the increased size of the container), which the body experiences as shock.

More than one component of the circulatory system may be affected in case of shock. For example, a patient in shock after a myocardial infarction is likely to have an element of cardiogenic shock, because the damaged heart can no longer pump efficiently, and an element of hypovolemic shock, if the patient has been vomiting, sweating, or too nauseated to take in fluids. Some types of shock always result from combined deficits from both fluid leakage into the interstitial space as well as vasodilatation.

Certain categories of patients are at high risk to develop shock. They include patients known to have had trauma or bleeding, elderly people, patients with massive myocardial infarction, pregnant women, and patients with a possible source for septic shock (such as burned patients and people with diabetes or cancer).

## Compensation for Decreased Perfusion

Central among the homeostatic mechanisms that regulate cardiovascular dynamics are those that maintain BP. When any event results in decreased perfusion (such as in blood loss, myocardial infarction, loss of vasomotor tone, or tension pneumothorax), the body must respond immediately to preserve the vital organs. Baroreceptors located in the aortic arch and carotid sinuses sense the decreased blood flow and activate the vasomotor center, which oversees changes in the diameter of blood vessels, to begin constriction of the vessels.

Stimulation typically occurs when the systolic pressure is between 60 and 80 mm Hg in adults or even lower in children. A decrease in systolic pressure to less than 80 mm Hg stimulates the vasomotor centre to increase arterial pressure by constricting vessels. The drop in arterial pressure decreases the stretching of the arterial walls, thereby decreasing baroreceptor firing, stimulating the vasoconstrictor centre of the medulla. The sympathetic nervous system is also stimulated as the body recognizes a potential catastrophic event.

In response to hypoperfusion, the renin-angiotensin-aldosterone system is activated and antidiuretic hormone is released from the pituitary gland. Together, these mechanisms trigger salt and water retention and peripheral vasoconstriction. The result is an increase in the patient's BP and CO. Depending on the severity of the insult, variable amounts of fluid will shift from the interstitial tissues into the vascular compartment. The spleen also releases some RBCs that are normally sequestered there to augment the blood's oxygen-carrying capacity. The overall response of the initial compensatory mechanisms is to increase the preload, stroke volume, and pulse rate, which usually results in an increase in CO.

As hypoperfusion persists, the myocardial oxygen demand continues to increase. Eventually, the accelerated compensatory mechanisms are no longer able to keep up with the body's demand. Myocardial function then worsens, with decreased CO and ejection fraction. Tissue perfusion decreases, leading to impaired cell metabolism. Often, the systolic BP decreases, especially in progressive hypoperfusion or "decompensated" shock. Fluid may leak from the blood vessels, causing systemic and pulmonary edema. Other signs of hypoperfusion may also be present, such as dusky skin colour, oliguria, and impaired mentation.

The body produces its own "medicines," epinephrine and norepinephrine, in the adrenal glands in response to hypoperfusion. These substances are released by the body as part of the global compensatory state. Epinephrine is also administered by caregivers in cases of anaphylaxis, severe airway disease, and cardiac arrest.

Release of epinephrine improves CO by increasing the pulse rate and strength. The alpha-1 response to its release includes vasoconstriction, increased peripheral vascular resistance, and increased afterload from the arteriolar constriction. Alpha-2 effects ensure a regulated release of alpha-1. Beta responses from the release of epinephrine primarily affect the heart and lungs. Increases in pulse rate, contractility, conductivity, and automaticity occur in tandem with bronchodilation.

Effects of norepinephrine are primarily alpha-1 and alpha-2 in nature and centre on vasoconstriction and increasing PVR. This vasoconstriction allows the body to shunt blood from areas of lesser need to areas of greater need; that is, it serves to keep the brain and other vital organs perfused in the early phases of shock. In an effort to maintain circulation to the brain, the body

## You are the Paramedic Part 3

You decide that the patient does not have spinal involvement. He reports that he was not blown to the ground, but rather felt dizzy and sat down on his own. You and your partner decide to quickly pick the patient up and load him onto the stretcher, rather than spending the time for spinal immobilization to a backboard. You also decide to insert the IV line en route to the regional trauma centre.

The patient is starting to become confused as you place him into Trendelenburg position and head out the door. He states that he is nauseated and thirsty and asks your partner, "Am I going to die?" When closing the back of the ambulance, you note on your watch that 7 minutes have elapsed on the scene. Your plan for the next few minutes is to redo the initial assessment, get IV fluids running, notify the ED, do the rapid trauma assessment and SAMPLE history, and consider PASG/MAST.

Vital Signs	Recording Time: 5 Minutes
Mental status	V (Responsive to verbal stimuli), confused about place and day
Respirations	26 breaths/min, shallow and laboured
Pulse	120 beats/min, thready (core, only not peripheral)
Blood pressure	106 mm Hg by palpation
Skin	Pale, cool, and moist
Sa <sub>o</sub> <sub>2</sub>	95% on nonrebreathing mask at 15 l/min of supplemental oxygen
ECG	Sinus tachycardia with no ectopy

- For this patient, is the Sa<sub>o</sub><sub>2</sub> a helpful indicator?
- Why weren't the baseline vital signs taken on the scene?

will shunt blood away from the following tissues, in this order: placenta, skin, muscles, gut, kidneys, liver, heart, lungs. The skin and muscles can survive with minimal blood flow from vasoconstriction for a much longer period than can major organs such as the kidneys, liver, heart, and lungs. If the blood supply is inadequate to the major organs for more than 60 minutes, they often develop complications that will lead to death, such as renal failure and shock lung. This concept has been traditionally referred to as the “golden hour of trauma,” and it explains why it is so important to address the cause of the shock in as timely a manner as possible.

Failure of compensatory mechanisms to preserve perfusion leads to decreases in preload and CO. Myocardial blood supply and oxygenation decrease, reducing myocardial perfusion. As CO further decreases, coronary artery perfusion also decreases, leading to myocardial ischemia.

### Types of Shock

The inadequate oxygen and nutrient delivery to the metabolic apparatus of the cell experienced in shock results in impaired cellular metabolism. Once a certain level of tissue hypoperfusion is reached, cell damage proceeds in a similar manner, regardless of the type of initial insult. Impairment of cellular metabolism results in the inability to properly use oxygen and glucose at the cellular level. The cell converts to anaerobic metabolism, which causes increased lactic acid production and metabolic acidosis, decreased oxygen affinity for hemoglobin, decreased adenosine triphosphate (ATP) production, changes in cellular electrolytes, cellular edema, and release of lysosomal enzymes. The blood glucose level may be elevated due to release of catecholamines and cortisol. In addition, fat breakdown (lipolysis) with ketone formation may occur.

The Weil-Shubin classification considers shock from a mechanistic point of view. From this perspective, two types of shock are distinguished: **central shock**, which consists of cardiogenic shock and obstructive shock, and **peripheral shock**, which includes hypovolemic shock and distributive shock.

Regardless of type, shock is characterized by reduced CO, circulatory insufficiency, and tachycardia. Most types of shock also include pallor, except for spinal shock and sepsis. The patient's mental status may be altered. Low BP, although classically associated with shock, is a late sign, especially in children.

### Cardiogenic Shock

Cardiogenic shock occurs when the heart is unable to circulate sufficient blood to maintain adequate peripheral oxygen delivery. Circulation of blood throughout the vascular system requires the constant pumping action of a normal and vigorous heart muscle. Many diseases can cause destruction or inflammation of this muscle. Within certain limits, the heart can adapt to these problems. If too much muscular damage occurs, however, the heart no longer functions effectively. Filling is impaired because of a lack of pressure to return blood to the heart (preload), or outflow is reduced by a lack of pumping function.

In either case, direct pump failure is the cause of shock. In the case of ischemic heart disease, pump failure is generally due to a loss of 40% or more of the functioning myocardium.

The most common cause of cardiogenic shock is extensive infarction of the left ventricle, diffuse ischemia, or decompensated congestive heart failure resulting in primary pump failure. The heart damage may be due to a single massive event or result from cumulative damage. Other forms of cardiac dysfunction may result in cardiogenic shock as well—for example, large ventricular septal defect, cardiomyopathy, or hemodynamically significant arrhythmias.

Patients have a poor prognosis when more than 40% of the left ventricle is destroyed. Historically, in about 7.5% of patients with acute myocardial infarction, cardiogenic shock develops, and mortality rates range as high as 80%, even with appropriate therapy.

### Obstructive Shock

**Obstructive shock** occurs when blood flow in the heart or great vessels becomes blocked. In pericardial tamponade, diastolic filling of the right ventricle is impaired, leading to a decrease in CO. Obstruction of the superior or inferior vena cava (such as vena cava syndrome as in third-trimester pregnancy) decreases CO by decreasing venous return. A large pulmonary embolus or tension pneumothorax may prevent adequate blood flow to the lungs, resulting in inadequate venous return to the left side of the heart.

### Hypovolemic Shock

**Hypovolemic shock** occurs when the circulating blood volume does not deliver adequate oxygen and nutrients to the body. It is subdivided into two types, exogenous and endogenous, depending on where the fluid loss occurs.

The most common cause of exogenous hypovolemic shock is external bleeding. Hemorrhage is most prevalent in trauma patients due to blunt or penetrating injuries to vessels or organs, long bone or pelvic fractures, major vascular injuries (as in traumatic amputation), and multisystem injury. The organs and organ systems with a high incidence of exsanguination from penetrating injuries include the heart, liver, spleen thoracic vascular system, abdominal vascular system (such as abdominal aorta, superior mesenteric artery), and venous system (such as inferior vena cava or portal vein).

Endogenous hypovolemic shock occurs when the fluid loss is contained within the body, as in dehydration, burn injury, crush injury, and anaphylaxis. With severe thermal burns, for example, intravascular plasma leaks from the circulatory system into the burned tissues that lie adjacent to the injury. By comparison, crushing injuries may result in the loss of blood and plasma from damaged vessels into injured tissues.

Abnormal losses of fluids and electrolytes (that is, dehydration) may occur through a variety of mechanisms:

- GI losses, especially through vomiting and diarrhea
- Increased loss as a consequence of fever, hyperventilation, or high environmental temperatures (through the lungs)

- Increased sweating
- Internal losses (“third-space” losses), as in peritonitis, pancreatitis, and ileus
- Plasma losses from burns

Other causes of body fluid deficits include ascites, diabetes insipidus, acute renal failure, and osmotic diuresis secondary to hyperosmolar states (such as diabetic ketoacidosis).

In each case, the fluid lost has a unique electrolyte composition, and long-term therapy aims to restore the deficient body chemicals. For treatment in the prehospital environment, however, all excessive fluid losses can be considered to lead to dehydration.

Symptoms of dehydration include loss of appetite (anorexia), nausea, vomiting, and sometimes fainting when standing up (postural syncope). Physical examination of a dehydrated patient reveals poor skin turgor (the skin over the forehead or sternum will “tent” when pinched); a shrunken, furrowed tongue; and sunken eyes. The pulse will be weak and rapid, rising more than 15 beats/min when the patient is raised from a recumbent to a sitting position (a maneuver that may cause the patient to feel faint). When fluid and electrolyte depletion are severe, shock and coma may be present.

A dehydrated patient needs replacement of fluid and electrolytes and should be given an IV infusion of normal saline or lactated Ringer’s solution at a rate of 100 to 200 ml/h for an adult, depending on the circumstances. Keep the patient flat to optimize circulation to the brain.

### Distributive Shock

**Distributive shock** occurs when there is widespread dilation of the **resistance vessels** (small arterioles), the **capacitance vessels** (small venules), or both. As a result, the circulating blood volume pools in the expanded vascular beds and tissue perfusion decreases. The three most common types of distributive shock are septic shock, neurogenic shock, and anaphylactic shock.

### Septic Shock

Sepsis comes from the Greek word meaning “to putrefy.” **Septic shock** is defined as the presence of sepsis syndrome plus a systolic BP of less than 90 mm Hg or a decrease from the baseline BP of more than 40 mm Hg.

Sepsis occurs as a result of widespread infection, usually due to gram-negative bacterial organisms; gram-positive bacteria, fungi, viruses, and rickettsia can also be causative agents. Complex interactions occur between the pathogen and the body’s defence systems. Initially, the body’s defence mechanisms may keep the infection at bay. The infection activates the inflammatory-immune response, which invokes humoral, cellular, and biochemical pathways. This response results in increased microvascular permeability (leaky capillaries), vasodilation, third-space fluid shifts, and microthrombi formation. In some patients, an uncontrolled and unregulated inflammatory-immune response occurs, resulting in hypoperfusion to the cells, tissue destruction, and organ death. Left untreated, the result is multiple-organ dysfunction syndrome and, often, death.

Septic shock is a complex problem. First, there is an insufficient volume of fluid in the container, because much of the blood has leaked out of the vascular system (hypovolemia). Second, the fluid that leaks out often collects in the respiratory system, interfering with ventilation. Third, a larger-than-normal vascular bed is asked to contain the smaller-than-normal volume of intravascular fluid.

### Neurogenic Shock

**Neurogenic shock** usually results from spinal cord injury, resulting in loss of normal sympathetic nervous system tone and vasodilation.

In neurogenic shock, the muscles in the walls of the blood vessels are cut off from the nerve impulses that cause them to contract. As a consequence, all vessels below the level of the spinal injury dilate widely, increasing the size and capacity of the vascular system and causing blood to pool. The available 5 to 6 l of blood in the body can no longer fill this enlarged vascular system. Perfusion of organs and tissues becomes inadequate, even though no blood or fluid has been lost, and shock occurs. The patient experiences relative hypovolemia, which leads to hypotension (systolic BP usually between 80 and 100 mm Hg). In addition, relative bradycardia occurs because the sympathetic nervous system is not stimulated to release catecholamines. The skin is pink, warm, and dry because of cutaneous vasodilation. There is no release of epinephrine and norepinephrine, which would otherwise produce the classic sign of pale, cool, diaphoretic skin. Instead, a characteristic sign of neurogenic shock is the absence of sweating below the level of injury.

Neurogenic shock is not to be confused with *spinal shock*, which refers to the local neurologic condition that occurs immediately after a spinal injury produces motor and sensory losses (which may not be permanent). Spinal shock is characterized by flaccid paralysis, flaccid sphincters, and absent reflexes. There is an absence of all pain, temperature, touch, proprioception, and pressure below the level of the lesion; absent or impaired thermoregulation; absent somatic and visceral sensations below the lesion; bowel distension; and loss of peristalsis.

### Anaphylactic Shock

**Anaphylaxis** occurs when a person reacts severely to a substance to which he or she has been sensitized. **Sensitization** means developing a heightened reaction (becoming allergic) to a substance. An allergic reaction typically does not occur, or occurs in a milder form, during sensitization. Do not be misled by a patient who reports no history of allergic reaction to a substance following a first or second exposure: Each subsequent exposure after sensitization tends to produce a more severe reaction.

In anaphylactic shock, there is no loss of blood, no vascular damage, and only a slight possibility of direct cardiac muscular injury. Instead, the patient experiences widespread vascular dilation, resulting in relative hypovolemia.

In anaphylaxis, immune system chemicals, such as histamine and other vasodilator proteins, are released on exposure

to an allergen. Their release causes the severe bronchoconstriction that accounts for wheezing if the patient is actually moving enough air. Anaphylaxis is also accompanied by urticaria (hives). The results are widespread vasodilation, which causes distributive shock, and blood vessels that continue to leak. Fluid leaks out of the blood vessels and into the interstitial spaces, resulting in hypovolemia and potentially causing significant swelling. In some cases, this swelling may occlude the upper airway, resulting in a life-threatening condition

**Figure 18-7** ▶

Recurrent large areas of subcutaneous edema of sudden onset, usually disappearing within 24 hours and mainly seen in young women (frequently as a result of allergy to food or drugs), is called **angioedema**.

### Shock-Related Events at the Capillary and Microcirculatory Levels

As perfusion decreases, cellular ischemia occurs. Minimal blood flow passes through the capillaries, causing the cells to switch from **aerobic metabolism** to **anaerobic metabolism**, which can quickly lead to metabolic acidosis. With less circulation, the blood stagnates in the capillaries. The precapillary sphincter relaxes in response to the buildup of lactic acid, vasomotor centre failure, and increased amounts of carbon dioxide. The postcapillary sphincters remain constricted, causing the capillaries to become engorged with fluid.

The capillary sphincters—circular muscular walls that constrict and dilate—regulate blood flow through the capillary beds. These sphincters are under the control of the autonomic nervous system, which regulates involuntary functions such as sweating and digestion. Capillary sphincters also respond to other stimuli at the local level such as heat, cold, increased demand for oxygen, and the need for waste removal. Thus, regulation of blood flow is determined by cellular need and is accomplished by vessel constriction or dilation, working in tandem with sphincter constriction or dilation.

The body can tolerate anaerobic metabolism for only a limited time. Anaerobic metabolism is much less efficient than aerobic metabolism and leads to systemic acidosis and depletion of the body's normally high energy reserves (ATP).

During anaerobic metabolism, incomplete glucose breakdown leads to an accumulation of pyruvic acid. Pyruvic acid cannot be converted to acetyl coenzyme A without oxygen, however, so it is transformed in greater amounts to lactate and other acid by-products. Acidosis develops because ATP is hydrolyzed to adenosine diphosphate and phosphate with the release of a proton. Hydrogen ion accumulates, decreasing the pool of bicarbonate buffer. Lactate also buffers protons, and lactic acid accumulates in the body.

At the same time, ischemia stimulates increased carbon dioxide production by the tissues. The higher the body's metabolic rate, the higher the carbon dioxide level in hypoperfused states. The excess carbon dioxide combines with intracellular water to produce carbonic acid. Increased tissue acids will, in



**Figure 18-7** In anaphylaxis, interstitial fluid may cause significant swelling. In some cases, this swelling may occlude the upper airway, resulting in a life-threatening condition.

turn, react with other buffers to form more intracellular acidic substances. Thus, acidosis serves as an indirect measure of tissue perfusion. The acidic condition of the blood inhibits hemoglobin in the RBCs from binding with and carrying oxygen. This adds to the cellular oxygen debt, shifting the oxyhemoglobin dissociation curve to the right.

Meanwhile, sodium, which is usually more abundant outside the cells than inside them, is naturally inclined to diffuse into the cells. Normally the sodium-potassium pump acts like a “bouncer” at the cell membrane, sending the sodium back out against the concentration gradient. This mechanism involves active transport and requires an ample supply of ATP. Reduced levels of ATP, however, result in a dysfunctional sodium-potassium pump and alter the cell membrane function. Excessive sodium begins to diffuse into the cells, along with water, which ultimately depletes the interstitial compartment.

The intracellular enzymes that usually help digest and neutralize bacteria introduced into a cell are bound in a relatively impermeable membrane. Cellular flooding damages that membrane and releases these lysosomal enzymes, which then autodigest the cell. If enough cells are destroyed in this way, organ failure will become evident. The release of the lysosomes opens the floodgates for the onset of the last phase of shock, called irreversible shock.

To compound these problems, accumulating acids and waste products act as potent vasodilators, further decreasing venous return and diminishing blood flow to the vital organs and tissues. The arterial pressure falls to the point at which even the “protected organs” such as the brain and heart are no longer being perfused. When aortic pressures fall below a MAP of 60 mm Hg, the coronary arteries no longer fill, the heart is weakened, and the CO falls. Myocardial depressant factor is released from an ischemic pancreas, further decreasing the pumping action of the heart and reducing the CO.

Eventually, the reduced blood supply to the vasomotor centre in the brain results in slowing and then stopping of sympathetic nervous system activity. The metabolic wastes are released into the slower-flowing blood. The blood's sluggish flow coupled with its acidity leads to platelet agglutination and formation of microthrombi. Because the capillary walls are stretched, they lose their ability to retain large molecules, allowing them to leak into the surrounding interstitial spaces. Hydrostatic pressure forces plasma into the interstitial spaces, further increasing the distance from the capillaries to the cells. In turn, oxygen transport decreases, increasing cellular hypoxia.

The continuing buildup of lactic acid and carbon dioxide acts as a potent vasodilator, leading to relaxation of the post-capillary sphincters. The accumulated hydrogen, potassium, carbon dioxide, and thrombosed (clotted) RBCs wash out into the venous circulation, increasing the metabolic acidosis. This has been referred to as the capillary "washout phase." The result is an even greater drop in CO. Ischemia and necrosis ultimately lead to multiple-organ dysfunction syndrome, in which the various organ systems fail.

### Systemic Inflammatory Response Syndrome

**Systemic inflammatory response syndrome (SIRS)** is a systemic inflammatory response to a variety of severe clinical insults. It does not confirm a diagnosis of infection or sepsis because the features of SIRS can be seen in many other conditions, including trauma, burns, and pancreatitis. Simply put, SIRS + infection = sepsis. SIRS is not a diagnosis itself, nor is it a good indicator of outcome.

The definition was proposed in 1991 by the American College of Chest Physicians and the Society of Critical Care to improve the ability of clinicians to make early bedside detection of sepsis, thus allowing early intervention. Also, standardizing the definition allows for improved analysis in research.

The inflammatory response is manifested by two or more of the following conditions: (1) temperature greater than 38°C or less than 36°C; (2) heart rate greater than 90 beats/min; (3) respiratory rate greater than 20 breaths/min or PCO<sub>2</sub> less than 32 mm Hg; and (4) a white blood cell count greater than 12,000/μl or less than 4,000/μl. The SIRS criteria may be considered a crude stratification for patients with systemic inflammation. In a prospective study of SIRS in medical and surgical patients, mortality rates were 3% in patients without SIRS, 6% in those with two criteria, 10% in those with three positive criteria, and 17% in those meeting all four criteria.

The release of cytokines in severe sepsis and injury is central to the development of SIRS. Initially, a central mediator is activated and released, which results in a cascaded secretion of various secondary mediators and the activation of neutrophils, the complement system, and vascular endothelial cells. The outcome of this complex cascade produces the physiologic changes recognized as SIRS.

SIRS affects specific organs and organ systems in the following ways:

- **Cardiovascular.** Manifests as tachycardia and hypotension. Myocardial depression and ischemia lead to pump failure.

Peripheral pulses may be weak or absent, and extremities may be cool and cyanosed.

- **Respiratory.** Manifest as tachypnea and increased minute ventilation. Reduced pulmonary capillary blood flow results in impaired gas exchange. Alveolar cells become ischemic and slow their production of surfactant, resulting in massive atelectasis and a reduction in pulmonary compliance. At the same time, pulmonary capillaries become permeable to water, resulting in edema. The net result is adult respiratory distress syndrome (ARDS) with respiratory failure, severe hypoxemia, and respiratory acidosis.
- **Central nervous system.** Decreased cerebral perfusion results in confusion, progressing to reduced level of consciousness and eventually unresponsiveness.
- **Renal system.** Reduced renal blood flow results in acute tubular necrosis, which in turn leads to oliguria (urine output < 20 ml/h). Toxic waste products cannot be excreted, so they are retained in the blood, worsening metabolic acidosis.
- **Liver and gastrointestinal tract.** Hypoperfusion results in ischemic gut. Impaired liver function and alterations in clotting factors produce coagulopathies, such as disseminated intravascular coagulation, in which clotting and bleeding occur at the same time. Liver cell death is evidenced by an increase in liver enzyme levels.
- **Metabolic.** Respiratory alkalosis is the first acid-base abnormality. Shock progresses with metabolic acidosis.

Uncontrolled SIRS leads to profound hypotension, inadequate perfusion, and death. Both SIRS and sepsis can progress to **multiple-organ dysfunction syndrome (MODS)**, defined as the presence of altered organ function in acutely ill patients such that hemostasis cannot be maintained without intervention. MODS is well established as the final stage in a continuum and carries a high mortality rate of 60% to 90%.

### Phases of Shock

Shock occurs in three successive phases (compensated, decompensated, and irreversible). Your goal is to recognize the clinical signs and symptoms of shock in its earliest phase and begin immediate treatment before permanent damage occurs. To do so, you must be aware of the subtle signs exhibited while the body is compensating effectively **Table 18-2 ▶** and treat the patient aggressively. Anticipate the potential for shock from the scene assessment and evaluation of the MOI. Recognize the signs of poor perfusion that precede hypotension, and do not rely on any one sign or symptom to determine the phase of shock the patient is going through. Always err on the side of caution when treating a potential shock patient. Rapid assessment and immediate transportation are essential to preserve any chance of survival.

#### Compensated Phase of Shock

The earliest stage of shock, in which the body can still compensate for blood loss, is called **compensated shock**. In this phase, the patient's level of responsiveness is a better indicator of tissue perfusion than most other vital signs. Release of chemical mediators by the autonomic nervous system as it recognizes a potential

**Table 18-2** Compensated Versus Decompensated Hypoperfusion

Compensated Hypoperfusion	Decompensated Hypoperfusion
<ul style="list-style-type: none"> <li>■ Agitation, anxiety, restlessness</li> <li>■ Sense of impending doom</li> <li>■ Weak, rapid (thready) pulse</li> <li>■ Clammy (cool, moist) skin</li> <li>■ Pallor with cyanotic lips</li> <li>■ Short of breath</li> <li>■ Nausea, vomiting</li> <li>■ Delayed capillary refill in infants and children</li> <li>■ Thirst</li> <li>■ Normal BP</li> </ul>	<ul style="list-style-type: none"> <li>■ Altered mental status (verbal to unresponsive)</li> <li>■ Hypotension</li> <li>■ Laboured or irregular breathing</li> <li>■ Thready or absent peripheral pulses</li> <li>■ Ashen, mottled, or cyanotic skin</li> <li>■ Dilated pupils</li> <li>■ Diminished urine output (oliguria)</li> <li>■ Impending cardiac arrest</li> </ul>

**At the Scene**

Regardless of the type of bleeding—whether hidden in the body cavities or visible on the kitchen floor—all patients will proceed through the phases of shock if their bleeding is not controlled. In hemorrhagic shock, the specific phase of shock is a function of the percentage of blood volume lost. Because internal bleeding is more likely to be uncontrolled, stay alert for the subtle signs of shock and be aggressive in your management of its early phase to avoid the slippery downward slope of shock's later phases.

catastrophic event causes the arterial BP to remain normal or slightly elevated. There is an increase in the rate and depth of respirations as the body attempts to bring in more oxygen and remove more carbon dioxide. This effort helps to maintain the acid-base balance by creating respiratory alkalosis to offset the metabolic acidosis.

During the compensated phase of shock, BP is maintained. Blood loss in hemorrhagic shock can be estimated at 15% to 30% at this point. A narrowing of the **pulse pressure** (the difference between the systolic and diastolic pressures) also occurs. The pulse pressure reflects the tone of the arterial system and is more sensitive to changes in perfusion than the systolic or diastolic BP alone. Patients in the compensated phase will also have a positive orthostatic tilt test result.

Treatment at this stage of shock will typically result in recovery.

**Decompensated Phase of Shock**

The next stage of shock, when BP is falling, is **decompensated shock** (also called uncompensated shock or progressive shock). It occurs when blood volume drops by more than 30%. The compensatory mechanisms begin to fail, and signs and symptoms become much more obvious. The CO falls dramatically, leading to further reductions in BP and cardiac function. The signs and symptoms become more obvious as blood is shunted to the

**At the Scene**

The term orthostatic has to do with positioning. **Orthostatic hypotension**, for example, is a drop in systolic BP when moving from a sitting to a standing position. An orthostatic tilt test is used to determine dehydration or hypovolemia. Blood pressure and pulse are measured as patients are lying, seated, and standing. A positive tilt test result occurs if the patient becomes dizzy, has a pulse increase of at least 20 beats/min, or has a systolic BP decrease of at least 20 mm Hg.

brain, heart, and kidneys. At this point, vasoconstriction can have a disastrous effect if allowed to continue. Cells in the nonperfused tissues become hypoxic, leading to anaerobic metabolism. Treatment at this stage will sometimes result in recovery.

Blood pressure may be the last measurable factor to change in shock. The body has several automatic mechanisms to compensate for initial blood loss and to help maintain BP. Thus, by the time you detect a drop in BP, shock is well developed. This is particularly true in infants and children, whose BP may be maintained until they have lost more than half of their blood volume.

**Irreversible (Terminal) Phase of Shock**

The last phase of shock, when this condition has progressed to a terminal stage, is **irreversible shock**. Arterial BP is abnormally low (typically in hemorrhagic shock there is a 40% or greater blood volume loss). A rapid deterioration of the cardiovascular system occurs that cannot be reversed by compensatory mechanisms or medical interventions. Life-threatening reductions in CO, BP, and tissue perfusion are observed. Blood is shunted away from the liver, kidneys, and lungs to keep the heart and brain perfused. Cells begin to die. Even if the cause of shock is treated and reversed, vital organ damage cannot be repaired, and the patient will eventually die. Even aggressive treatment at this stage does not usually result in recovery.

**The Clinical Picture of Hypovolemic Shock**

Most of the typical symptoms and signs of shock result from inadequate tissue oxygenation and the body's attempts to compensate for volume loss. Probably the earliest signs of shock are restlessness and anxiety: The patient looks scared! The decline in tissue perfusion is setting off alarms all over the body, to which the patient responds with a feeling of apprehension. If conscious, the patient may complain of thirst, reflecting the deficit of fluids in the body; at the same time, the patient may feel nauseated and even vomit. The diversion of blood flow away from low-priority peripheral tissues causes the skin to become pale, cold, and clammy; sometimes it has a mottled appearance. Meanwhile, the heart speeds up to circulate the remaining RBCs more rapidly, producing a rapid, weak pulse.

While the arteries are constricting and the heart is speeding up, the brain discovers that it is not getting enough oxygen. It assumes that the lungs must be malfunctioning, so the respiratory centre in the brain stem signals the respiratory muscles

to speed up their activity. The result is rapid, shallow breathing (tachypnea).

As bleeding continues, the BP finally falls in the shock patient. Do not wait until the BP falls before you suspect shock and begin treatment! Falling BP is a *late* sign in shock, signaling the collapse of all compensatory mechanisms. Furthermore, the BP measured at the arm gives little information about perfusion of vital organs.

The goal in treating shock is to save the brain and kidneys; these organs must remain perfused if the patient is to survive and return to a healthy life. The best indication of brain perfusion is the patient's state of consciousness. If the patient is conscious and alert, the brain is being perfused adequately no matter what the sphygmomanometer says. If the patient is confused, disoriented, or unconscious, perfusion of the brain is likely inadequate. Kidney perfusion can be gauged by urine output in a catheterized patient. Adequately perfused kidneys put out at least 30 to 50 ml of urine per hour; poorly perfused kidneys shut down and stop putting out any urine.

In the prehospital setting—where patients will not ordinarily have urinary catheters—you can estimate the patient's peripheral perfusion by testing for capillary refill, although this is not the most reliable indicator. To do so, press on one of the patient's fingernails until it blanches, then release the pressure. If the skin under the nail doesn't "pink up" within 2 seconds (about as long as it takes to say "good capillary refill"), peripheral perfusion is compromised. To determine how well the *vital* organs are being perfused, you must rely on the patient's state of consciousness.

## General Assessment of a Patient With Suspected Shock

The general assessment of a patient who is suspected of having hypoperfusion or shock follows the plan reinforced throughout this book. After sizing up the scene for hazards, taking BSI precautions, and addressing the need for additional help, begin the initial assessment.

Include a quick assessment of the MOI. For a patient with suspected shock, this information can give you clues about the causes and the extent of any bleeding (whether internal or external) or the causes of nonhemorrhagic shock.

Start the assessment by forming an initial diagnosis. Next, assess the patient's mental status (using AVPU) and manage any life threats to airway, breathing, and circulation. In conscious patients, you will usually assess the pulse at the radius; in unconscious patients, you will typically take the carotid pulse in the neck. The radial pulse can give you clues about the phase of shock and the patient's ability to compensate for shock. Ask yourself, "Is the radial pulse strong and regular, or weak and thready, or irregular?" If the radial pulse is barely palpable, yet the patient is sitting up and talking to you with a bullet hole in the abdomen, remember that the purpose of the shock syndrome is to keep the brain perfused—but the reduction in the radial pulse is an indicator that the systolic BP is dropping fast. In such a case, you may even decide that you

simply cannot take the time to measure the patient's BP, because you already know the patient is hypotensive (indicating decompensated shock) and immediate transport to the ED is the best course of action.

Patients with shock will usually be prioritized as "high." If the shock originates from a medical problem, the patient should be fast-tracked through the assessment of the chief complaint (OPQRST). In the more likely case—that the patient has had some sort of trauma—the MOI will guide your rapid trauma assessment of the major body cavities and regions.



### Documentation and Communication

Recording frequent serial vital signs—and observing perfusion indicators such as skin condition and mental status—will give you a window into the progression of shock. Use your documentation to remind you to suspect shock early and treat it aggressively.

The SAMPLE history and the baseline vital signs come next; they can be done en route to the ED along with your ongoing assessment. Time is of the essence in shock cases, so focus on moving toward the ED and keep the on-scene care to the essential items that must be done before moving the patient (that is, ABCs and spinal immobilization). Unless the patient is pinned and there may be a delay in extrication, delay inserting IV lines until en route to the ED.

Shock is considered hypovolemic or hemorrhagic until proven otherwise. **Table 18-3** summarizes the hemodynamic parameters in the differentiation of shock. The phase of shock in hypovolemic or hemorrhagic shock (compensated, decompensated, or terminal/irreversible) relates to the percentage of blood loss. The percentage blood loss is easily remembered by thinking of the score in a tennis game: 15% to 30% compensated, 30% to 40% decompensated, greater than 40% irreversible.

## Management of a Patient With Suspected Shock

As with any patient, airway and ventilatory support take top priority when treating a patient with suspected shock. Maintain an open airway, and suction as needed. Give high-flow supplemental oxygen via nonrebreathing mask or assist ventilation with a bag-valve-mask device. Consider early definitive management in patients who are unable to maintain their own airway. Control any external hemorrhage, and try to estimate the amount of blood lost. Look for signs of internal hemorrhage, and consider the potential for loss in the area of suspected hemorrhage. For example, a patient may lose as much as 1 l of blood in the tissues of the thigh in a closed, uncomplicated femur fracture. Consider the MOI, and maintain a high index

## 18.22 Section 4 Trauma

of suspicion for occult injuries, especially when the patient has signs of shock with no obvious cause.

Without delaying time at the scene, establish IV access with two large-bore cannulas (14 or 16 gauge) and administer IV fluid to replace blood loss. Isotonic crystalloids, such as normal saline or lactated Ringer's, should be used (synthetic solutions may also be used). Solutions of dextrose in water are not effective for resuscitation of trauma patients. The goal of volume replacement is to maintain perfusion without increasing internal or uncontrollable external hemorrhage. For this reason, most protocols advise administration of IV fluid in boluses of 20 ml/kg until radial pulses return. The presence of radial pulses equates to a systolic BP of 80 to 90 mm Hg, which is generally sufficient to perfuse the brain and other vital organs. In certain cases of shock, especially those caused by penetrating trauma, fluid therapy to maintain the systolic BP at approximately 80 mm Hg may be safer for the patient than to attempt restoration of normotension, which may aggravate ongoing bleeding.

Hypoperfusion with an unstable pelvis is the primary indication for use of the PASG/MAST. Conditions of decreased SVR not corrected by other means, such as increasing fluid volume in cases of neurogenic shock, may also benefit from use of the PASG/MAST. Effects resulting from inflation of the PASG/MAST include an increased arterial BP above the garment, increased SVR, immobilization of the pelvis and possibly the lower extremities, and increased intra-abdominal pressure. Because

use of the device is highly controversial, it is imperative that you follow local protocols.

If the patient exhibits signs of a tension pneumothorax, perform needle chest decompression to improve CO. In cases of suspected cardiac tamponade, you must recognize the need for expeditious transport for pericardiocentesis at the ED. Both of these conditions further impair circulation by compressing the heart and decreasing CO.

Nonpharmacologic interventions for shock include proper positioning of the patient, prevention of hypothermia, and rapid transport. Apply the cardiac monitor, and be alert for possible arrhythmias. When making your transport decision, consider the need for a regional trauma centre. If travel time is lengthy, air medical transportation may be the best option. Provide psychological support en route; speak calmly and reassuringly to the patient throughout assessment, prehospital care, and transport.

**Skill Drill 18-2** provides a review of shock management:

1. Use PPE. Make sure the patient has an open airway, and check breathing and pulse. In general, keep the patient in a supine position. Patients who have experienced a severe heart attack or who have lung disease may find it easier to breathe in the Fowler's or semi-Fowler's position. Always provide supplemental oxygen, assist with ventilation as needed, and continue to monitor the patient's breathing

**Step 1**

**Table 18-3** Differentiation of Shock

Origin	Etiology	Characteristics				
		BP	Pulse	Skin	Lungs	EMS Treatment
↓ Pump performance	Cardiogenic	↓	↓ → ↑	Pale, cool, moist	Crackles	Low-dose dopamine
↓ Fluid volume	Hypovolemic, hemorrhagic	↓	↑	Pale, cool, moist	Clear	IV fluids
Vessels or container dilates: maldistribution of blood; low peripheral resistance	Neurogenic	↓	↓	Flushed, dry, warm	Clear	IV fluids, atropine, high-dose dopamine, norepinephrine
	Septic	↓	↑	Flushed or pale, hot or cool, moist	Crackles if pulmonary origin	IV fluids, high-dose dopamine, norepinephrine
	Anaphylactic	↓	↑	Flushed, warm, moist	May have wheezes; may be ↓ with no sounds	Epinephrine, diphenhydramine, salbutamol, ipratropium, corticosteroids
Parameter	Hemodynamic Parameters					
	Hypovolemic	Cardiac	Neurogenic	Septic		
Mean arterial pressure	↓	↓	↓	↓		
HR	↑	↑ or ↓	↓	↑		
Central venous pressure	↓	variable	↓	↑		
CO	↓	↓	↓	↑ then ↓		
Peripheral vascular resistance	↑	↑	↓	↑		
pH	↓	↓	↓	↓		
PaO <sub>2</sub>	↓	↓	↓	↓		
Paco <sub>2</sub>	↓	↓	Increase and decrease the rate	↓		

2. Control all obvious external bleeding. Place dry, sterile dressings over the bleeding sites, and secure them with pressure bandages (Step 2).
3. Splint bone or joint injuries to minimize pain and bleeding, which can aggravate shock. Splinting also prevents the ends of the broken bone from further damaging adjacent soft tissue and, in general, makes it easier to move the patient. To minimize time spent on the scene, you may use a backboard as a temporary splint until you are headed to the ED. Handle the patient gently and no more than is necessary (Step 3). Use PASG/MAST only with the approval of direct medical control or established local protocols.
4. To prevent the loss of body heat, place blankets under and over the patient. Do not overload the patient with covers or attempt to warm the body too much, however; the goal is to maintain a normal body temperature. Do not use external heat sources, such as hot water bottles or heating pads because they may cause vasodilation and decrease BP even more (Step 4).
5. Once you have positioned the patient on a backboard or a stretcher, place him or her in the Trendelenburg position: Raise the foot of the backboard or stretcher about 30 cm. If the patient is not on a backboard and no lower extremity or back fractures are suspected, place the patient in the shock position: Elevate the patient's legs 30 cm by propping them up on several blankets or other stable objects. These positions help blood return from the extremities to the core of the body, where it is needed most. Patients with respiratory distress do not generally benefit from a Trendelenburg position; it may aggravate breathing because the abdominal organs push against the diaphragm (Step 5).

## Skill Drill 18-2: Treating Shock



Step 1

Keep the patient supine, open the airway, and check breathing and pulse. Give high-flow supplemental oxygen, and assist ventilations if needed.



Step 2

Control obvious external bleeding.



Step 3

Splint broken bones or joint injuries.



Step 4

Place blankets under and over the patient.



Step 5

If no fractures are suspected, elevate the legs 30 cm. Insert an IV line, and administer warm fluid en route to the ED. Insert an IV line at the scene only if transport of the patient is delayed (such as if the patient is pinned).

## IV Therapy

Intravenous lines are inserted for one of two general purposes: to provide a route for immediate replacement of fluid in patients who have already lost significant volumes or to provide a route for potential fluid replacement in patients who are at risk of losing significant volumes of fluid or blood. The IV fluid of choice will be normal saline or lactated Ringer's.

Specifically, all patients in hypovolemic shock need IV fluid replacement. In addition, IV access should be obtained in patients who are likely to develop hypovolemic shock because they have one or more of the following conditions: profuse external bleeding, internal bleeding, vaginal bleeding, blunt trauma to the abdomen, fracture of the pelvis or femur, severe or widespread burns, heat exhaustion, intractable vomiting or diarrhea, and neurogenic shock or septic shock. As always, consult your local or regional protocols regarding access and fluid administration in the setting of shock.

In case of need for emergency administration of drugs, IV lines should also be inserted to keep a vein open. When a patient has poor CO (as in shock), blood is shunted away from the skin and skeletal muscles. Thus, drugs administered subcutaneously or intramuscularly are absorbed at a low and unpredictable rate. Giving a drug directly into the vein ensures that the desired dose of the drug reaches the circulation. Patients who need a vein kept open include those at risk of cardiac arrest (it's easier to start the IV before the arrest) and patients who may need parenteral medication (such as patients with seizures, diabetes, congestive heart failure, or coma).

The IV flow rate is typically determined by local protocol. The decision on flow rates usually reflects the patient's presumptive diagnosis and the condition of his or her lungs (wet or dry), and takes into account whether the IV line was inserted for fluid administration to keep the vein open for future medication administration. **Table 18-4** shows the BP indicators that are often referenced when determining IV flow rates for patients with dry lungs (not pulmonary edema).

<b>Table 18-4 IV Fluid Therapy for Adult Patients in Suspected Shock</b>	
<b>Adult Systolic BP (mm Hg)</b>	<b>Fluid Volume (presumes dry lungs)*</b>
Normotensive (100 to 130; higher end depends on age)	Fluid challenge of 250 ml normal saline, and reevaluate patient
Hypotensive (80 to 90)	Fluid challenge of 500 ml normal saline, and reevaluate patient
Severe hypotension (50 to 80)	Fluid challenge of 1 l normal saline, then titrate additional fluid to achieve low end or normotensive state

\*Fluid therapy for burn patients should follow the Parkland formula (see Chapter 20).

## Volume Expanders and Plasma Substitutes

Hypovolemic shock should be treated with volume expanders to replace what has been lost or to "fill the container" in relative hypovolemia. For cardiogenic shock, cautious use of volume expanders may increase preload and, subsequently, CO. Positive cardiac inotropic drugs may be administered to increase the strength of contractions, along with rate-altering medications to further enhance perfusion. An example is epinephrine, which serves both purposes with its beta-1 effects.

The vasodilation that accompanies distributive shock creates relative hypovolemia. Treatment involves volume expanders and positive cardiac inotropic drugs. Volume expanders are also indicated for obstructive shock and spinal shock.

A variety of macromolecular solutions have colloidal and osmotic properties similar to those of plasma and are used to maintain circulatory volume in the emergency treatment of shock. Although such solutions cannot replace the RBCs, platelets, or plasma proteins lost in hemorrhage, they are more readily available than whole blood or plasma in an emergency because they do not require typing and can be carried in the ambulance. Furthermore, during multiple-casualty incidents, the supply of blood and blood products may not be adequate, and substitutes must be used. Available plasma substitutes and volume expanders include dextran, plasma protein fractions, and polygeline.

Given that prehospital research has yet to show dextran as superior to crystalloid solution administration, medical directors are not likely to approve its use in the prehospital setting at this time.

## Crystalloids

Crystalloids are solutions that do not contain proteins or other large molecules; that is, they are noncolloids. Their effects in restoring volume in shock are usually quite transitory because the fluid rapidly equilibrates across the capillary walls into the tissues. For example, approximately 60% of infused normal saline, when given as a bolus, diffuses out of the intravascular space within 20 minutes of administration. Thus, when noncolloid solutions are used in the treatment of hemorrhagic shock, you need to give two to three times the volume of blood lost.

Crystalloids are clearly the fluids of choice when only salt and water have been lost, such as in dehydration. Where debate continues, however, is about the role of crystalloids versus colloids in the treatment of shock. Despite a great deal of research on the subject, no overwhelming evidence supports one therapeutic approach over the other. Until such evidence is forthcoming, practical considerations will continue to favour the use of crystalloids for initial fluid resuscitation in the prehospital setting.

The crystalloids most commonly used for that purpose are normal saline and lactated Ringer's solution. Normal saline is simply sodium chloride (0.9% NaCl) in water at a concentration isotonic with the extracellular fluid. Lactated Ringer's solution is similarly constituted but includes small amounts of potassium and calcium. Lactated Ringer's solution contains 28 mEq of lactate as well, which is added as a buffer (the liver breaks lactate

down into bicarbonate). None of these solutions is superior to the others for acute resuscitation, so the choice remains a matter of the physician's preference.

Recently, there has been considerable interest in the use of hypertonic saline solution (7.5% NaCl solution) for emergency treatment of blood loss. Infusing a hypertonic solution should, in theory, attract interstitial fluid into the vascular space, so such solutions should, at least temporarily, improve intravascular volume. This solution seems to be an effective plasma expander, with infusion of 250 ml producing the same effect as infusion of 2 to 3 l of isotonic crystalloid solution. The effectiveness of this regimen in humans and its ability to improve survival rates over those seen with use of isotonic solution remain to be demonstrated.

### Management of Specific Types of Shock

The following sections provide guidance for treatment of specific types of shock. In all cases, be aggressive during the compensated phase of shock to avoid the need to make up for fluid loss if the patient moves into the decompensated phase of shock. If the patient may be in cardiogenic shock, fluids are still indicated, but you must be diligent about monitoring the lung sounds so as to not overload the patient.

#### Hypovolemic and Hemorrhagic Shock

The priorities in treating a patient in hemorrhagic or hypovolemic shock are the same as in treating any other patient—namely, the ABCs. Establish and maintain an open airway. Keep suction at hand to clear the mouth and pharynx if the patient should vomit. Administer supplemental oxygen, and assist ventilation as needed. Control bleeding, if present, by using direct pressure

over the site of external bleeding. Apply the PASG/MAST, if it is part of your local protocol, and begin transport.

Insert at least one, and preferably two, large-bore peripheral IV lines (14 to 16 gauge). Give normal saline or lactated Ringer's solution. For guidance, refer to the IV fluid flow rates in Table 18-4 and to your local protocol. Run in the first 500-ml "fluid challenge" as fast as it will flow, and then reassess the patient to see the impact of the intervention. If warmed fluids are available, consider their use as well. There is growing evidence that aggressive use of crystalloid to resuscitate a patient with hypovolemic shock due to blunt trauma may actually be harmful. While not definitive, recent research suggests that allowing the systolic blood pressure to remain low, in the range of 70 to 80, may actually improve survival compared to resuscitating the patient until their blood pressure is in the normal range. While this evidence is new, local and regional EMS protocols and paramedic practice may change to conservative use of crystalloid resuscitation in patients with hypovolemic shock due to blunt trauma. Paramedics should consult their protocols to determine fluid resuscitation guidelines in their service.

Do not give the patient anything by mouth because he or she is very likely to vomit. Keep the patient at normal temperature, which usually means covering the patient with a blanket—patients in hypovolemic shock are often unable to conserve body heat effectively and are easily chilled. Place the patient in a position with the head elevated 15° to 30° and the legs propped up 30° on pillows (injuries permitting).

Monitor the ECG rhythm because any critically ill or injured patient is apt to have arrhythmias. Also monitor the state of consciousness, pulse, and BP. In a patient with substantial vasoconstriction, the BP sounds may be difficult to hear, especially in the

## You are the Paramedic Part 4

Luckily, a paramedic from another unit was able to drive your ambulance, so you and your paramedic partner can focus on the patient for the entire 15-minute trip to the hospital. You insert a large-bore IV cannula and give normal saline, with a pressure infuser attached to the bag. You have just enough time to repeat the initial assessment before you call the ED to report the patient's condition. The physician advises you to hold off on inflation of the PASG/MAST and insert a second IV line. Meanwhile, your partner prepares to begin ventilatory assistance with a bag-valve-mask device. The patient's mental status has diminished, signaling he is truly fighting for his life.

Reassessment	Recording Time: 15 Minutes
Skin	Pale, cool, clammy
Pulse	120 beats/min; thready and getting difficult to palpate
ECG	Sinus tachycardia with occasional premature ventricular contractions
Blood pressure	Unobtainable
Respirations	28 breaths/min, shallow
Sa <sub>o</sub> <sub>2</sub>	86% with bag-valve-mask ventilation and supplemental oxygen
Pupils	Midsized and sluggish

When you return to the ED after another call, about 4 hours later, you check on the young man. According to the ED physician, the patient is still holding on after a few hours of intensive surgery. Apparently, he had major internal bleeding and damage to his stomach and spleen and major vessel damage from a bullet fragment. The ED physician states that in this case, the patient's best chance is a dose of the "bright lights and cold steel" of the operating suite. Because of your quick judgment about how to save time on the scene and your willingness to call ahead so the ED was ready, the patient made it to the operating suite about 35 minutes after the shooting.

- Without interventions in the prehospital setting, how long would this patient have lived?
- What is the benefit of calling ahead and focusing on the time factor?

prehospital setting. If you can feel a pulse over the femoral artery but not over the radial artery, for example, the systolic BP is probably somewhere between 70 and 80 mm Hg.

### Cardiogenic Shock

Prolonged efforts to stabilize the condition of a patient in cardiogenic shock in the prehospital setting are not recommended. Because this is a time-sensitive patient, you should expedite transport as quickly as possible. Place the patient in a supine position, secure the airway, monitor the SpO<sub>2</sub>, and administer supplemental oxygen via a nonrebreathing mask at 12 to 15 l/min. Apply ECG electrodes, and document the initial rhythm. Your IV access should be with a crystalloid solution. Auscultate the lungs; if they are clear and protocols allow, try a fluid challenge of 200 ml to increase the preload and evaluate the effects on the BP and lung sounds.

Some EMS systems advocate the use of dopamine (Intropin) at low doses in the beta range (5 µg/kg/min) if the patient has a MAP of less than 60 mm Hg. Elevating the BP by using high-dose dopamine in the alpha range may be temporarily ordered by direct medical control at the expense of other target organs. In such a case, anticipate very rapid tachycardia that could adversely impact ventricular filling. Combination drug therapy is often needed at the hospital (eg, dopamine plus dobutamine, or norepinephrine) while awaiting cardiac catheterization, hemodynamic monitoring catheters, and insertion of an intra-aortic balloon pump.

### Neurogenic Shock

The prehospital care of a patient with suspected neurogenic shock is similar to the general management approach for any patient with shock. In addition, the patient should be immobilized to minimize further movement and injury to the spine. Specific concerns relate to keeping the patient warm because a spinal injury can disrupt the thermoregulatory mechanisms and leave the patient vulnerable to hypothermia.

Another specific concern relates to the issue of fluid therapy. Determine the necessity for IV fluids based on the patient's hemodynamic status. Maintain adequate hydration and volume status to keep the systolic BP at 90 mm Hg or higher. General hemodynamic resuscitation includes volume loading with normal saline IV fluid boluses in 200-ml increments up to 2 l through a large-bore IV cannula. If possible, use warm fluid to prevent hypothermia.

In pure neurogenic shock not associated with hypovolemic shock, vagal blockers—such as atropine, 0.5 mg, by rapid IV push (up to a maximum of 3 mg) if the pulse remains bradycardic—and pressor agents—such as a dopamine drip beginning at 10 µg/kg/min and titrating to 20 µg/kg/min—may be used to better advantage than overhydrating the patient. Monitor the patient's response to vasopressors because it may be less than expected owing to the compromise of the sympathetic nervous system.

### Anaphylactic Shock

In a case involving shock due to a severe allergic reaction, you need to act fast. Remove the inciting cause if possible. Resolve

any immediate life threats to the ABCs, which may require aggressive airway management and supplemental oxygen administration. Evaluate the patient's ventilatory status and the need for bag-valve-mask device assistance.

Provide cardiovascular support with IV fluid challenges of crystalloid solution. Reverse the target-organ effect by administering epinephrine or a vasopressor such as dopamine (Intropin) in high doses. Consider the need for a bronchodilator such as salbutamol or ipratropium (Atrovent). Impede further mediator release with an antihistamine such as diphenhydramine (Benadryl). If the patient has an epinephrine injector, such as an Epi-Pen, consider using it and taking a spare along to the hospital because its effect will wear off quickly.

## Transportation of Patients With Suspected Shock

If a patient is suspected to be in shock, transport is inevitable; the questions to be asked are simply when and where. Consideration for the priority of the patient and the availability of a regional trauma centre should be your concerns, and local transport protocols may specifically deal with these issues. Patients who have suspected shock, whether compensated or decompensated, will benefit from early surgical intervention and should be transported to a facility with those capabilities. Patients with cardiogenic shock may need to go to a hospital with comprehensive cardiac care capabilities (that is, a catheterization lab and heart surgery program). If a facility of this type is not readily available, direct medical control should help you make the transport decision. In some communities, this will involve transport to a local facility and transfer (often aeromedical) to a tertiary care facility with the appropriate facilities and staff to handle the patient's complex needs.

## Prevention Strategies

Of course the best prevention of shock would involve not having the incident that led to the shock in the first place! Probably the number one strategy that would prevent many lost lives is simply wearing seatbelts whenever driving or riding in a motor vehicle!

Prevention of shock and its deadly effects begins with your immediate assessment of the MOI, initial assessment findings, and the patient's clinical picture. Be alert, and search for early signs of shock. Don't rationalize irregularities away because they will soon become much more obvious if the patient truly is in shock—but by then it may be too late to stop the patient from sliding down the slippery slope. For example, at the scene of a motor vehicle collision where the patient's brand-new car has a dented driver's door, don't say to yourself, "The patient has tachycardia because he is upset!" Instead, consider the MOI and decide to manage the suspected shock aggressively now.

## You are the Paramedic Summary

### 1. Does the lack of significant visible bleeding and the fact that he is alert indicate that this patient is not bleeding seriously?

No—don't get caught in that trap! The purpose of the compensatory mechanism in the early phase of shock is to ensure the brain is well perfused. If this mechanism is working, as one would expect early in blood loss, the patient would be alert. Don't confuse nervousness or anxiousness with an altered mental state, which implies diminished brain perfusion. As for the lack of visible bleeding, be aware that a patient can lose most of his or her blood volume into large cavities (such as the thigh, chest, or abdomen) without a drop of external blood.

### 2. What is the significance of time in this type of incident?

Set the timer on your digital watch because this patient does not have a lot of time to spare. In the "golden hour of trauma," a lot of things need to occur, so paramedics in the prehospital setting do not have any more than a "platinum 10 minutes" for the initial assessment and prehospital management of the patient.

### 3. On the basis of the information you have so far, and remembering that the patient weighs approximately 80 kg, how much blood did he have before the incident? How much could he have lost so far?

Based on 6% to 8% of the total body weight, this patient would have about 4.8 to 6.4 l of blood before the incident. Given that he has no palpable radial (peripheral) pulse but is still alert (and, thus, has a carotid pulse), one could estimate that his systolic BP is between 60 and 90 mm Hg. Rather than take the time at this point to ponder the exact reading, simply note that the patient is hypotensive—that is, he is in decompensated shock. In decompensated shock, this patient may have lost 30% of his blood volume, in this case some 4 units of blood.

### 4. What phase of shock is this patient in?

Because the patient has no radial pulse, he is hypotensive. He is therefore in the decompensated phase of shock.

### 5. Which BLS and ALS interventions would be most appropriate for this patient at this time? Should you insert an IV line at the scene?

The BLS treatment for this patient would be to administer high-concentration supplemental oxygen and consider the PASG/MAST if permitted by local protocols. If you suspect a spinal injury, rapidly immobilize the patient on a backboard; if no spinal injury is suspected, place the patient on the stretcher and take him to the ED. Perform the rapid trauma assessment and detailed physical examination en route to the hospital. The ALS interventions would involve two large-bore IV lines with normal saline wide open, ECG monitoring, and calling ahead to the ED so staff are prepared.

A paramedic could certainly provide another set of trained hands to help care for this patient and get him away from the scene quickly. The insertion of an advanced airway can help in the management of the patient should his level of consciousness decrease en route to the hospital. Critical trauma patients

often have severe hypoxia and vomiting. A well-managed airway can help control these problems.

Intravenous access for fluid resuscitation is helpful but should not delay transport of a trauma patient in critical condition. Insert the large-bore IV lines en route to the ED; alternatively, you may insert them on the scene, but only while the paramedics are simultaneously packaging the patient for transport. Two arguments are put forth to explain why you should not wait at the scene to insert the IV line in a trauma patient.

First, paramedics usually infuse lactated Ringer's or normal saline, but what the patient in this late phase of hemorrhagic shock really needs is a colloid fluid expander followed by whole blood and likely a trip to the operating room to control bleeding. Although the fluid infused will expand the patient's circulating volume temporarily until it leaks out of the vascular space, it does not contain hemoglobin and, therefore, cannot carry oxygen. Most experts recommend switching to a colloid solution when 2 to 3 l of crystalloid solution is not enough to restore blood volume and whole blood is unavailable.

Second, if the patient was found in decompensated shock, you cannot catch up with the ongoing blood loss by inserting one or two large-bore lines at the scene. The mere time it takes to prep the site and equipment and make a successful venipuncture can add dramatically to the scene time as the patient continues to bleed. For the patient in this case, the paramedic unit should be called, and initiation of one or two IV lines is appropriate en route to the hospital. If the patient becomes unconscious, you should intubate the patient to ensure an adequate airway.

### 6. For this patient, is the $SaO_2$ a helpful indicator?

This measurement has limited usefulness in this case. The  $SaO_2$  is only one of many indicators but is sometimes not accurate in low perfusion states. Go with the clinical signs and symptoms, and move fast!

### 7. Why weren't the baseline vital signs taken on the scene?

In the initial assessment, you discovered that the patient had a pulse and you "guesstimated" that he was hypotensive based on the location of the pulse. You also have a handle on his respirations, so the formal baseline vital signs can be delayed until you get going with this patient.

### 8. Without interventions in the prehospital environment, how long would this patient have lived?

If it took about 10 minutes for the patient to lose 30% of his blood volume into his belly, he has very little time. At 40% blood loss, his pulse is apt to be lost. He would be dying without your rapid interventions.

### 9. What is the benefit of calling ahead and focusing on the time factor?

With this patient bleeding so severely, time is precious and any intervention at the scene must be absolutely justified as essential and lifesaving. Otherwise, do it en route to the ED. Let the ED personnel know you are on the way so they can be ready to move the patient through the ED and to the "bright lights and cold steel" of the operating room and expertise of the surgeons.

# Prep Kit

## Section 4 Trauma

### Ready for Review

- The cardiovascular system is designed to carry out one crucial job: keep blood flowing between the lungs and the peripheral tissues.
- Hemorrhage simply means bleeding.
  - Bleeding can range from a “nick” to a capillary while shaving, to a severely spurting artery from a deep slash with a knife, to a ruptured spleen from striking the steering column during a car collision.
  - External bleeding can usually be easily controlled by using direct pressure or a pressure bandage.
  - Internal bleeding is usually not controlled until a surgeon locates the source and sutures it closed.
- The assessment of any patient begins with a good scene assessment and proceeds to your general impression and initial assessment.
  - Once the scene is deemed safe to enter, you will need to wear the appropriate level of PPE.
  - Depending on the severity of bleeding and your initial diagnosis, this will entail gloves, mask, eyeshield, and, when the patient is very bloody or blood is spurting, a gown.
- In case of hemorrhage, the issue is not whether the patient will be transported, but rather how fast the transport decision should be made and where the patient should be taken for definitive care.
- Hypoperfusion occurs when the level of tissue perfusion decreases below normal.
  - Early decreased tissue perfusion may result in subtle changes, such as altered mental status, long before a patient’s vital signs (that is, BP, pulse, respiratory rate) appear abnormal.
  - Shock refers to a state of collapse and failure of the cardiovascular system that leads to inadequate circulation, creating inadequate tissue perfusion.
- As with any patient, airway and ventilatory support take top priority when treating a patient with suspected shock.
- If a patient is suspected to be in shock, transport is inevitable; the questions to be asked are simply when and where.
  - Consideration for the priority of the patient and the availability of a regional trauma centre should be your concerns, and local transport protocols may specifically deal with these issues.
  - Patients who have suspected shock, whether compensated or decompensated, will benefit from early surgical intervention and should be transported to a facility with those capabilities.

- Prevention of shock and its deadly effects begins with your immediate assessment of the MOI, initial assessment findings, and the patient’s clinical picture.
  - Be alert, and search for early signs of shock.

### Vital Vocabulary

**aerobic metabolism** Metabolism that can proceed only in the presence of oxygen.

**afterload** The pressure in the aorta against which the left ventricle must pump blood.

**anaerobic metabolism** Metabolism that takes place in the absence of oxygen.

**anaphylaxis** A severe life-threatening allergic reaction to foreign protein or other substances.

**angioedema** Recurrent large areas of subcutaneous edema of sudden onset, usually disappearing within 24 hours, which is seen mainly in young women, frequently as a result of allergy to food or drugs.

**blood** The fluid that is pumped by the heart through the arteries, veins, and capillaries and consists of plasma and formed elements or cells, such as red blood cells, white blood cells, and platelets.

**capacitance vessels** The smallest venules.

**cardiac output (CO)** The amount of blood pumped through the circulatory system in 1 minute.

**cardiogenic shock** A condition caused by loss of 40% or more of the functioning myocardium; the heart is no longer able to circulate sufficient blood to maintain adequate oxygen delivery.

**central shock** A condition that consists of cardiogenic shock and obstructive shock.

**compensated shock** The early stage of shock, in which the body can still compensate for blood loss. The systolic blood pressure and brain perfusion are maintained.

**decompensated shock** The late stage of shock, when blood pressure is falling.

**distributive shock** A condition that occurs when there is widespread dilation of the resistance vessels, the capacitance vessels, or both.

**ejection fraction** The portion of the blood ejected from the ventricle during systole.

**epistaxis** A nosebleed.

**erythrocytes** Red blood cells.

**hematochezia** Passage of stools containing bright red blood.

**hemoglobin** The oxygen-carrying pigment in red blood cells.

**hemorrhage** Profuse bleeding.

**hemostasis** Stopping hemorrhage.

**hypoperfusion** A condition that occurs when the level of tissue perfusion decreases below that needed to maintain normal cellular functions.

**hypovolemic shock** A condition that occurs when the circulating blood volume is inadequate to deliver adequate oxygen and nutrients to the body.

**irreversible shock** The final stage of shock, resulting in death.

**leukocytes** White blood cells.

**melena** Passage of dark, tarry stools.

**multiple-organ dysfunction syndrome (MODS)** A progressive condition usually characterized by combined failure of several organs, such as the lungs, liver, and kidney, along with some clotting mechanisms, which occurs after severe illness or injury.

**neurogenic shock** Circulatory failure caused by paralysis of the nerves that control the size of the blood vessels, leading to widespread dilation; seen in spinal cord injuries.

**obstructive shock** Shock that occurs when there is a block to blood flow in the heart or great vessels, causing an insufficient blood supply to the body's tissues.

**orthostatic hypotension** A drop in systolic blood pressure when moving from a sitting to a standing position.

**perfusion** The delivery of oxygen and nutrients to the cells, organs, and tissues of the body.

**peripheral shock** A condition that consists of hypovolemic shock and distributive shock.

**plasma** The fluid portion of the blood from which the cells have been removed.

**platelets** Small cells in the blood that are essential for clot formation.

**pulse pressure** The difference between the systolic and diastolic pressures.

**resistance vessels** The smallest arterioles.

**sensitization** Developing sensitivity to a substance that initially caused no allergic reaction.

**septic shock** Shock caused by severe infection, usually a bacterial infection.

**shock** An abnormal state associated with inadequate oxygen and nutrient delivery to the metabolic apparatus of the cell.

**stroke volume** The amount of blood that the left ventricle ejects into the aorta per contraction.

**systemic inflammatory response syndrome (SIRS)** The systemic inflammatory response to a variety of severe clinical insults.



## Assessment in Action

You are called to a shopping centre by security on a Friday night for a person who was assaulted. When you arrive, you see a man in his late 20s who is sitting on a bench holding a towel to his face. There is a trail of blood from the men's restroom, and the towel is dripping with blood. According to the security officer, there was a fight in the restroom and they found this man, Joey, stumbling and drenched in blood. Apparently, a couple of gang members beat him up and slashed his face and neck with razors.

You quickly don PPE and ensure that the scene is safe. The police are just arriving behind you. The initial assessment reveals an alert and oriented but anxious patient who has an open and clear airway, has 26 shallow breaths/min and has a very weak and thready radial pulse. He has external bleeding from the face and neck, which your partner is attempting to control with direct pressure. His carotid pulse is 120 beats/min, and his skin is pale, cool, and clammy. You begin to administer supplemental oxygen with a nonrebreathing mask at 15 l/min and lay the patient down with his feet raised so you can do a rapid trauma assessment.

Meanwhile, your supervisor arrives with the stretcher so the patient can be rapidly removed from the scene. The assessment reveals that there may also be potential for internal bleeding because the patient was kicked in the ribs and abdomen when he was down on the floor. You quickly load the patient and decide to insert two IV lines en route to the regional trauma centre. Your supervisor locks his vehicle so you can have plenty of personnel working up your patient en route to the ED because there is much to do to save his life.

- This patient has one very obvious injury involving:**
  - a flail chest.
  - the facial and neck lacerations.
  - a ruptured spleen.
  - an injured left kidney.
- With a patient who has so much obvious external bleeding, you should don which PPE?**
  - Disposable gloves
  - An eye shield
  - A disposable mask
  - All of the above
- An example of an injury that is potentially life threatening yet difficult to see in this patient would be:**
  - internal bleeding.
  - the facial laceration.
  - the neck laceration.
  - head trauma.
- What is the significance of the weak radial pulse in this patient?**
  - It demonstrates he is generally physically fit.
  - It demonstrates that his bleeding is actually minimal.
  - It indicates that his systolic blood pressure is already dropping.
  - It indicates that his body is compensating well for the injuries.
- What is the significance of the pale, cool, and clammy skin in this patient?**
  - It demonstrates that he was on a cold floor.
  - It shows that the vessels in the skin have been constricting.
  - It shows that the vessels in the skin have been dilating.
  - It shows that he has an adequate supply of blood to the brain.
- With the patient alert and oriented at this point, how serious is his condition?**
  - Not very serious at all once a bandage is applied to the face and neck.
  - The lacerations are serious and will need to be sutured in the ED.
  - Very serious owing to the combination of external and internal bleeding.
  - Very serious owing to the symptoms of a head injury.
- With deep lacerations to the face and neck, how should the bleeding be controlled?**
  - Pressure point
  - Tourniquet
  - Cold application
  - Direct pressure
- What phase of shock is this patient in at this point?**
  - Terminal
  - Decompensated
  - Compensated
  - Guarded
- What type of shock does this patient potentially have?**
  - Septic
  - Neurogenic
  - Hemorrhagic
  - Anaphylactic
- Aside from controlling the external bleeding, giving supplemental oxygen, and using the Trendelenburg position, what other treatment would be appropriate for this patient en route to the regional trauma centre?**
  - Two large-bore IV lines for normal saline
  - The PASG/MAST inflated until the Velcro crackles
  - Cooling the patient with chilled IV fluid
  - A dopamine drip in the alpha dose range

### Challenging Question

- If the patient weighed 90 kg and you obtained a BP of 86 on palpation en route to the ED, what phase of shock is he in, how much blood did he have (roughly) before the assault, and how much blood has he already lost?**

### Points to Ponder

You respond to the scene of a single motor vehicle collision in which a 45-year-old woman is walking around the scene. It is obvious from the spiderlike crack in the windshield and her forehead laceration that she struck the glass with her head. She is nervous that she will get in trouble and states she was wearing a seatbelt and did not hit the windshield. You also note the smell of alcohol on her breath, and she denies any medical history. She allowed you to feel her weak rapid radial pulse but now is refusing to let you do any further assessment. Her head lacerations are no longer bleeding, although she has plenty of blood on her white blouse. She is refusing to go to

the hospital, stating there is nothing wrong with her. The police officer, who has been dealing with the traffic congestion, states he is going to administer a breathalyzer test.

How should you deal with this patient's refusal to go to the hospital?

**Issues:** MOI for Internal Injury, Recognition of Shock, Estimating Phases of Shock, Compensation Versus Decompensation During Shock, Treatment Plan for a Patient With Suspected Shock, and Patient Refusal.