Shock: The Silent Killer

As an EMT or paramedic, one of your most important roles is to recognize shock promptly and provide appropriate treatment. Unfortunately, for many prehospital personnel, the concept of shock is often misunderstood and prehospital textbooks and training courses vary significantly in their coverage and descriptions of this important topic. However, the concept of shock is really quite simple. To understand the effects of shock on the body, you must first understand its underlying pathophysiology.

Shock is best defined as inadequate tissue perfusion. It is caused by many conditions including trauma, fluid loss, heart attack, infection, spinal cord injury, anaphylaxis and many other causes. Whatever the cause, all forms of shock have the same underlying problem: inadequate tissue perfusion.

The various tissues of the body require a constant supply of oxygen and other essential nutrients. When tissue perfusion declines, the body reacts immediately to restore and maintain blood flow to the tissues through the activation of compensatory mechanisms. These physiological mechanisms often are effective in restoring tissue perfusion. However, if the underlying cause of the shock is not corrected, the compensatory mechanisms will ultimately fail. In such cases, shock becomes irreversible, and the patient dies.

Physiology of Perfusion

The circulatory system—the heart, blood and blood vessels—is responsible for the transport of oxygen and essential nutrients to the various body tissues. At the same time, it constantly removes waste products, such as carbon dioxide. The circulatory system works closely with the respiratory and gastrointestinal systems and is controlled by the nervous and endocrine systems.

Tissue perfusion depends on a functioning and intact circulatory system. Being a closed system, a problem with any one of its components—the heart, the blood or blood vessels—can affect the entire system and interrupt tissue perfusion.

The Heart

The heart is the pump of the circulatory system. It receives blood from the venous system and pumps it to the lungs, where it is oxygenated and carbon dioxide is removed. When the blood returns to the heart from the lungs, it is subsequently pumped to the peripheral tissues. The amount of blood pumped by the heart, called the stroke volume, depends on three factors: preload, contractile force and afterload.

Preload is the amount of blood delivered to the heart by the venous system during diastole. The venous system can be thought of as a storage system. It can be contracted or expanded as required to meet the physiological demands of the body. When additional blood is needed, the veins will constrict, effectively reducing the storage volume of the venous system. This subsequently increases the amount of blood delivered to the heart, which increases the stroke volume.
The actual strength of the cardiac contraction, the cardiac contractile force, can be increased through several mechanisms. One of these is by increasing the preload. The greater the amount of blood delivered to the heart by the venous system, the more the ventricles will be stretched. The greater the stretch, up to a certain point, the greater the force of the subsequent cardiac contraction.

Cardiac contractile strength is also affected by circulating hormones such as epinephrine and norepinephrine. During periods of stress, these hormones are released by the adrenal glands and carried to the heart, where they cause an increase in cardiac contractile force and heart rate. Stimulation of sympathetic nerves that innervate the heart also can increase the cardiac contractile force.

Stroke volume also is affected by afterload, which is the resistance against which the ventricle must pump. Afterload occurs in the arterial side of the circulatory system. Constriction of the arteries increases the pressure within this side, and the heart must generate enough force during the cardiac contraction to overcome the resistance and to pump blood. The less the resistance, the greater the stroke volume.

The amount of blood pumped by the heart in one minute is called the cardiac output. This is a function of the stroke volume and the heart rate. An increase in either stroke volume or heart rate will increase the cardiac output. A decrease in either stroke volume or heart rate will decrease the cardiac output.

Blood pressure, a frequently monitored vital sign, represents the pressure within the circulatory system at any given time. Blood pressure is a function of cardiac output and peripheral vascular resistance, which is the pressure against which the heart must pump. As discussed previously, the circulatory system is a closed system. Thus, increasing either cardiac output or peripheral vascular resistance will increase the blood pressure. Likewise, a decrease in cardiac output or peripheral vascular resistance will decrease the blood pressure.

The Blood

The second component of the circulatory system is blood, which is the system's fluid. To maintain tissue perfusion, an adequate amount of blood must fill the circulatory system; the typical adult has approximately 6 liters of blood. The blood consists of the plasma and the formed elements. Plasma is the liquid portion of the blood and the formed elements include red blood cells, white blood cells and platelets. Red blood cells contain hemoglobin and are responsible for transporting oxygen to the tissues. White blood cells aid the body in fighting infection. The platelets assist in blood clotting. Any significant decrease in the amount of blood within the circulatory system will adversely affect tissue perfusion.

The Blood Vessels

The third component of the circulatory system is it's container or the blood vessels. The blood vessels can be thought of as a continuous, closed and pressurized pipeline that moves the blood throughout the body.

Like the heart, the blood vessels are under the control of the nervous and endocrine systems. The nervous system can modify the size of the container by constricting or dilating the blood vessels. For example, during exercise, blood supply is shunted to areas of the body that are under increased demand, such as the heart and the muscles. The body is very adept at directing blood flow to tissues that require additional amounts.

Hormones, such as norepinephrine and epinephrine, also affect the blood vessels. As mentioned previously, these hormones are released into the circulatory system.

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from the adrenal glands during periods of stress. They act on receptors within the
vascular system and cause vasoconstriction. This diverts blood to essential tis-
sues, such as the heart and brain.

In summary, tissue perfusion depends on an intact and functioning circulatory
system. It also depends on adequate air exchange in the lungs to get oxygen into
the blood. Asphyxia, or respiratory failure, can lead to inadequate oxygen delivery to
the blood, ultimately leading to diminished tissue perfusion and, eventually, shock.
Thus, a problem with either the heart, the blood, the blood vessels or the respiratory
system can adversely affect tissue perfusion and result in shock.

The ultimate targets of decreased tissue perfusion are the cells. When oxygen supply
to a cell is interrupted, the cell will change the way in which it produces energy.
Instead of producing energy with the aid of oxygen (aerobic metabolism), it
switches to energy production without the aid of oxygen (anaerobic metabolism).
Anaerobic metabolism is very inefficient. It also results in the production of acids,
such as lactic acid, which are harmful to the cells. If this situation persists for a
prolonged period of time, cellular death will eventually occur, ultimately leading
to tissue death. Tissue death will ultimately lead to organ death and organ death
ultimately will lead to death of the individual.

**The Body’s Response to Shock**

Following the onset of inadequate tissue perfusion, the body’s various compensatory
mechanisms kick in to restore tissue perfusion. First, the heart rate and the
strength of the cardiac contraction will increase. In addition, the peripheral blood
vessels will constrict to increase systemic vascular resistance. Both of these actions
serve to maintain adequate blood pressure.

As shock progresses, these compensatory mechanisms may fail. In these cases,
the body is unable to maintain adequate blood pressure, and thus, blood supply to
essential organs, such as the kidneys and brain, begins to decline. As the peripheral
tissues become more hypoxic, the small capillaries eventually will relax and dilate
causing blood flow to slow. As inadequate tissue perfusion continues, cellular death
will occur. When enough cells die, tissue and organ death occurs. At some point,
when enough cells in the vital organs are disrupted, shock becomes irreversible, and
death is inevitable.

**Stages of Shock**

Shock, regardless of its cause, can be broken down into three distinct stages. These stages are:
- compensated
- decompenstated
- irreversible

**Compensated Shock**
The earliest stage of shock, compensated shock, is the most difficult to detect. During
compensated shock, the body’s various compensatory mechanisms are able to
maintain an adequate blood pressure. However, with peripheral vasoconstriction,
there may be some evidence of decreased end-organ perfusion.

The signs and symptoms of compensated shock are often subtle and related to
the underlying physiological events. They include:
- a slight increase in heart rate
- moist, cool skin
- normal blood pressure
- normal mental status

**Decompensated Shock**
The second stage of shock is easier to detect. In decompensated shock, the com-
ponsatory mechanisms fail. Most notably,
blood pressure begins to fall and end-organ perfusion diminishes further. Signs and symptoms of decompensated shock include:

- pronounced increase in heart rate
- markedly prolonged capillary refill time
- pale, cool, diaphoretic skin
- decreased blood pressure
- altered mental status (ranging from slight agitation to coma)

Irreversible Shock

The final stage of shock, irreversible shock, is a retrogressive diagnosis. If inadequate tissue perfusion is prolonged, various body cells and tissues eventually will die. When enough cells and tissues in vital organs die, the brain will die, and further shock will be irreversible. The liver, kidneys, heart, lungs, and brain are all very susceptible to hypoxia and death of these organs can occur even if effective blood pressure has been restored. Irreversible shock may not become evident for several days following the original event.

Types of Shock

Basically, there are only three major types of shock.

- cardiogenic shock (pump failure)
- hypovolemic shock (fluid or blood loss)
- distributive shock (neurogenic shock)

Each represents a problem in one or more components of the circulatory system. The following discussion will detail each type of shock and present a brief overview of prehospital treatment strategies.

Cardiogenic Shock

An inability of the heart to pump enough blood to all parts of the body is referred to as cardiogenic shock. This can result from any one or more of the following conditions:

- inadequate preload
- inadequate cardiac contractile strength
- excessive afterload
- inadequate heart rate

Cardiogenic shock usually is the result of left ventricular failure, the most common cause of which is acute myocardial infarction. As cardiogenic shock progresses, hypotension soon develops and blood supply to the heart is reduced. This decreases coronary perfusion, which further damages the heart muscle, complicating the situation.

In addition, the activation of the body’s compensatory mechanisms actually can worsen cardiogenic shock. An increase in the heart rate and systemic vascular resistance increases the amount of work the heart must perform. This increases myocardial oxygen demand. Since coronary perfusion is already diminished, increased myocardial work can worsen cardiac ischemia.

Finally, in cardiogenic shock, the heart often fails to function as an effective forward pump. This often results in accumulation of fluid within the lungs (pulmonary edema). Pulmonary edema can adversely affect oxygen exchange in the lungs, worsening both hypoxia and tissue perfusion.

Prehospital Treatment

Treatment of cardiogenic shock involves several modalities. First, supplemental, high-flow oxygen must be administered to increase oxygen saturation in the blood and help maintain the myocardial oxygen supply. The patient also should be positioned supine unless there is associated pulmonary edema. In addition, IV access should be obtained. If there is no evidence of pulmonary edema, a fluid bolus of 250 ml to 500 ml should be administered. Fluids should not be administered if there is evidence of pulmonary edema (rales, tachypnea, orthopnea).

Drug therapy in cardiogenic shock is aimed at increasing cardiac output, treating life-threatening dysrhythmias, correcting problems with heart rate and decreasing myocardial work. Commonly used medications include:

- Dopamine—Dopamine increases both cardiac output and peripheral vascular resistance in patients who are hypotensive (systolic <100 mmHg). It achieves this by increasing cardiac contractile force, slightly increasing heart rate, and by causing peripheral vasoconstriction. Unlike some other drugs, dopamine maintains blood flow to the kidneys and intestines when used in therapeutic dosages.

Norepinephrine—Norepinephrine works by increasing systemic vascular resistance. It has much less effect on cardiac contractile force than does dopamine and is only used in severe hypotension (systolic <70 mmHg) or when dopamine fails. Dopamine, however, is usually the preferred vasoopressor drug.

Furosemide—Furosemide is a potent diuretic used to treat pulmonary edema associated with cardiogenic shock. It functions in two ways. First, it causes venous vasodilation, which causes a decrease in preload and in cardiac work. Second, it causes the kidneys to eliminate water, thus decreasing the intravascular fluid volume. This also decreases cardiac work and helps eliminate pulmonary edema. The average dose is 20-80 milligrams intravenously.

Morphine—Morphine is an important drug in the treatment of cardiogenic shock with associated pulmonary edema. It, too, causes venous vasodilation, which decreases preload. It also acts on the central nervous system to decrease pain and anxiety. This ultimately causes a decrease in sympathetic stimulation and in cardiac work.

Despite the use of medications and other therapy, the mortality rate associated with cardiogenic shock is high (approximately 90 percent). Thus, prehospital personnel should concentrate on early recognition and treatment. Prompt administration of oxygen, as well as drugs, such as dopamine, to maintain blood pressure, is essential.

Hypovolemic Shock

Loss of fluid from within the circulatory system can cause hypovolemic shock. This fluid loss can result from several conditions including:

- internal or external hemorrhage
- trauma
- long-bone or open fractures
- severe dehydration from prolonged vomiting and diarrhea
- plasma loss from burns
- diabetic ketoacidosis with resultant osmotic fluid loss
- third space losses from bowel obstruction, peritonitis and pancreatitis.
Treatment

Prehospital treatment of hypovolemia is directed at early recognition and management. The first step is to complete the primary assessment. Next, high-flow supplemental oxygen should be administered, and the patient should be placed in a supine position with the feet elevated (unless there is an associated head injury). Body temperature should be maintained by use of a blanket.

ALS personnel should obtain IV access and begin fluid replacement using lactated Ringer’s or normal saline. Adults in hypovolemic shock should initially receive a fluid bolus with the IV wide open, and children should receive fluid boluses at a rate of 20 ml/kg.

In severe hypovolemic shock, the pneumatic antishock garment (PASG) can buy critical time. This device increases lower body peripheral vascular resistance, helping maintain blood pressure. It is also helpful in stabilizing lower extremity and pelvic fractures. The PASG should not be used with penetrating trauma to the chest.

Definitive IV fluid administration usually occurs in the hospital setting. Blood loss through hemorrhage is usually replaced with blood or blood products. Other fluid losses are corrected based on the underlying cause and the results of laboratory studies.

Distributive Shock

Distributive shock results from inadequate peripheral vascular resistance due to widespread vasodilation. Common causes include spinal cord injury, central nervous system injury, overwhelming infection, and anaphylactic reactions. Whatever the cause of the shock, the ultimate effect is peripheral vasodilation that results in decreased peripheral vascular resistance and hypotension.

The peripheral blood vessels are under the control of the nervous system. A spinal cord or central nervous system (CNS) injury can interrupt nervous control, causing immediate peripheral vasodilation. This condition, often called neurogenic shock, results in pooling of the blood in the dilated peripheral vessels.

Prehospital treatment includes completion of the primary assessment with attention to stabilization of the spine if trauma is suspected. Peripheral vascular resistance can be increased by the use of the PASG. In addition, norepinephrine can be administered. As mentioned, norepinephrine causes peripheral vasoconstriction and increased peripheral vascular resistance, helping to maintain blood pressure.

Overwhelming bacterial infections, especially those caused by certain Gram negative bacteria, also can cause distributive shock. Some bacteria will release specialized toxins, called endotoxins, when they die. Many of these endotoxins act directly on the peripheral blood vessels, causing vasodilation. This process, called septic shock, can cause hypotension and inadequate tissue perfusion. Treatment is directed at correcting the underlying cause of the shock.

Another cause of distributive shock is anaphylaxis. Anaphylaxis is a severe allergic reaction that is far more pronounced than would be expected in an individual. During an anaphylactic reaction, the body releases several chemicals including histamine. When released by specialized cells of the immune system, histamine has several effects. First, it causes bronchoconstriction. Second, it causes the capillaries to dilate and become “leaky.” This allows fluid to migrate from the circulatory system into surrounding tissues. In addition, in severe reactions, histamine and other chemicals can cause peripheral vasodilation, leading to decreased peripheral vascular resistance and decreased tissue perfusion.

Treatment of anaphylaxis involves several things. First, the primary assessment should be completed, high-flow oxygen should be administered and respirations supported. Then, if the patient is hypotensive, the legs should be elevated.

ALS personnel should initiate IV therapy with a crystalloid solution. A fluid bolus is often indicated by the associated hypovolemia due to increased permeability of the capillaries. The drug of choice is epinephrine 1:10,000 administered intravenously at doses of 0.5 mg to 1.0 mg. In severe cases, an epinephrine drip may be required. Antihistamines, such as diphenhydramine, should be administered to counter the effects of histamine. Potent corticosteroids, such as methylprednisolone, are also indicated, especially with a long transport time.

Summary

The key to successful management of shock in the prehospital setting is early recognition and prompt intervention. The key to prompt recognition is a fundamental understanding of the underlying pathophysiology of shock. As an EMT or paramedic, you should look for the early, subtle indicators of shock, such as tachycardia and decreased skin perfusion. Remember, a fall in blood pressure is a late sign of shock. Anybody can recognize shock when the patient is hypotensive. The key is to recognize it early, provide appropriate care and prevent deterioration into the later, more severe stages of shock.

References


Graphics


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