Introduction

Shock is the condition in which the cells do not receive sufficient oxygen to meet all of the metabolic needs for energy. Shock States are the conditions that cause cell shock; the shock states are classified and diagrammed in Figure CVIII-1. There are two main classifications of the shock states: (1) Global; and (2) Focal. Global shock states affect the whole body. Focal shock states affect one organ or part of an organ. The goal of treatment of any of the shock states is to prevent as much irreversible cell damage as is possible.

Global Shock States

The global shock states result in shock involving the whole body. Regardless of the cause, global shock states activate the sympato-adrenal axis (sympathetic arm of the autonomic nervous system) and the adrenal glands that elaborate catecholamines (epinephrine, norepinephrine, dopamine). Symptoms and signs vary with the type and severity of the shock state, and may include: tachycardia (if possible), narrowing of the pulse pressure (if possible), hypotension, cold skin (moves progressively central), diaphoresis (clammy), cyanosis, prolonged capillary refill, and anxiety (feeling of impending doom).

The global shock states consist of two primary categories: (1) decreased cardiac output; and (2) normal or increased cardiac output. Low cardiac output shock states include: (1) hypovolemia; (2) cardiac (pump) failure; and (3) obstruction.

Decreased Cardiac Output

Hypovolemia

Hypovolemia is the condition of decreased volume in the respective circulatory compartments. Regardless of its cause, the principle physiological problem is that hypovolemia reduces the filling pressure of the ventricles (left and right); as a result, stroke volume and cardiac output fall with a decline in pressure in
the systemic arterial compartment. The severity of the symptoms and signs of existence of hypovolemia as well as the likelihood of survival relate to the proportion of the total blood volume lost: the intensity of the activation of the sympatho-adrenal compensatory mechanisms (see Table CVIII.1). Decreased intravascular volume may produce orthostatic decreases in blood pressure in the systemic arterial compartment due decreased venous return to the right ventricle due to the effects of gravity. Organ perfusion is determined primarily by the importance of the organ to the well-being of the whole person: generally, the organs with the longest anoxic survival time are deprived of flow first and then up the hierarchy of survival times. In Stage IV of a hypovolemic shock state, only the coronary, cerebral, and pulmonary circulations are protected: when the brain and coronary circulations are compromised, death ensues. Hypovolemia may result from hemorrhage, dehydration, vasodilatation, or capillary leak.

Treatment of hypovolemic shock states consists of stopping the fluid loss and filling the compartments (increasing filling pressure of both ventricles) with the most appropriate fluid. Always maintain cerebral and coronary perfusion even if the transient use of pressors is required. The mortality rate increases exponentially as the severity of the shock state increases. Thus, therapy must be directed to limiting the progression from one stage to another or to decrease the stage of the shock state.

<table>
<thead>
<tr>
<th></th>
<th>Stage 1</th>
<th>Stage II</th>
<th>Stage III</th>
<th>Stage IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loss (% total volume)</td>
<td>Up to 15</td>
<td>15-30</td>
<td>30-40</td>
<td>&gt;40</td>
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<tr>
<td>Volume loss (ml)</td>
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<td>750-1,500</td>
<td>1,500-2,000</td>
<td>&gt;2,000</td>
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<tr>
<td>Heart Rate/minute</td>
<td>&lt;100</td>
<td>100-120</td>
<td>120-140</td>
<td>&gt;140 or brady</td>
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<tr>
<td>Blood Pressure*</td>
<td>Normal</td>
<td>Normal</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Pulse Pressure</td>
<td>Normal or increased</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Ventilatory Rate/minute</td>
<td>14-20</td>
<td>20-30</td>
<td>30-40</td>
<td>&gt;35</td>
</tr>
<tr>
<td>Mental status</td>
<td>Slightly anxious</td>
<td>Mildly anxious</td>
<td>Anxious, confused</td>
<td>Confused, lethargic</td>
</tr>
</tbody>
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**Table CVIII-1—Stages of hypovolemic shock states**

_Hemorrhagic, hypovolemic shock state_—the cause of hemorrhagic hypovolemia is loss of blood from the vascular compartments. Therefore, in addition to a decline in the filling pressure of both ventricles, the oxygen content of the remaining blood decreases (decreased hemoglobin concentration of the systemic arterial blood; decreased oxygen carrying capacity even with saturation of the hemoglobin with O₂). Therefore, hypovolemia due to hemorrhage is not reflected in oxygen saturation measured by pulse.
oximeter. Treatment of hemorrhagic shock states is directed at maintaining the filling pressures of the ventricles by the administration of fluid or blood intravenously and controlling the hemorrhage. Resuscitation consists of the administration of fluids to restore/maintain the filling pressures of the ventricle and oxygen delivery to the tissues. If crystalloid solutions (lactated ringers, normal saline), two-thirds of the crystalloid solution administered diffuses out of the vascular system into the interstitial spaces and dilutes the hemoglobin remaining within the vascular system. When possible, volume restoration should consist of blood, blood products, and colloids that do not readily pass through the capillary pores,
Dehydration—dehydration is a condition in which the volume of fluids in the body is decreased. Dehydration involves not only the volume of blood in the vascular system, but decrease the volume of fluid of all of the body components. It can be caused by decreased intake of water or by the loss of water through vomiting or diarrhea. Decreases of intake result in thirst and the development of gradual loss of...
organ function. No intake of water is accelerated by the insensible loss of fluids by breathing and evaporation. The onset of manifestation of dehydration from lack of adequate intake take several days in the adult, but may be more rapid in infants. Vomiting or diarrhea can occur at any age, but dehydration from vomiting and diarrhea may be rapid in infants and small children. A major effect of ketoacidosis associated with diabetes is dehydration as well as manifestations of disturbances in the concentrations of electrolytes and ketoacidosis. Cholera is an example of severe dehydration caused by an infection and has been a major cause of death during the recent epidemic in Haiti. Dehydration is manifested by dry skin and mucous membranes, and changes in brain function. Dehydration results in progressive decreases in preloads to both ventricles and decreased cardiac output. Thus, manifestations of activation of the sympatho-adrenal access appear. The skin remains tented when gently pinched. Treatment consists of rehydration using intravenous or oral administration of electrolyte-containing solutions or use of rehydrating solutions administered orally, and correction of the underlying cause.

Capillary leak syndromes—loss of fluid from the vascular compartments into the interstitial spaces may be related to injury of the capillaries from circulating toxins or other harmful substances such as circulating products of cell injury or death. These substances circulating in the vascular system result in increased capillary permeability (increased diameter of the capillary pores) so that more fluid is lost at the arteriolar end of the capillary than can be reabsorbed at the venous end. This results in the development of interstitial edema. Interestingly, this process also occurs with hemorrhagic hypovolemia—2/3rds of the crystalloid solutions infused winds up outside of the vascular system. Therefore, three times the anticipated blood loss must be infused when using crystalloid solutions (lactated ringers or normal saline) to increase the filling pressures of the ventricles. Treatment must be aimed at correcting the underlying cause and intravenous administration of fluids containing substances that are substances too large to diffuse through the dilated capillary pores (e.g., albumen, red blood cells).

Vasodilatation—vasodilation may involve loosening of the screw claps, dilatation of the systemic venous compartment, or the opening of precapillary sphincters. The precapillary sphincters open channels that connect the arterioles with the venules without the blood passing through the capillaries. Precapillary shunts open during sepsis, anaphylaxis, or circulating toxins that are likely to harm the tissues if they perfuse through the capillaries. Many chemicals (drugs) may result in vasodilatation as a primary or secondary effect of the administration of the agent. For example, nitroglycerin, lasix, and morphine are powerful dilators of the systemic venous compartment. Dilatation of the systemic venous compartment decreases the preload of the right ventricle and results in decreased stroke volume of the right ventricle. Their administration is noted below as a treatment for patients in left ventricular caused pulmonary
edema—decrease right ventricular output to allow the output of the left ventricle to catch up. In addition, some drugs are administered to decrease arteriolar tone (arteriolar dilators, i.e., nitroglycerine, antihypertensive agents, alpha blockers). Many other drugs taken by patients may have vasodilating capabilities.

**Cardiac dysfunction**

The two hearts (right and left ventricles) must have normal pacemakers and a conduction system to be able to deliver normal or increased levels of cardiac output. Many conditions may cause decreased outputs of one or both of the pumps. Electrical disturbances (dysrhythmias or arrhythmias) affect both ventricles while impairment of the mechanics of the hearts may affect either or both ventricles.

**Mechanical**

Impaired performance of ventricular contraction (negative inotrope) may result from myocardial ischemia or infarction, persistent or inadequately treated hypertension, infection of the myocardial muscle, degeneration of the muscle, or insufficiency of the cardiac valves.

**Myocardial injury/infarction**—oxygen deprived myocardium associated with the coronary syndromes may affect the contractility of the myocardium. Decreased stroke volume and pulmonary congestion may result from ischemia—patients with an acute myocardial ischemia injury may present with shortness of breath due to dysfunction of the ischemic portion of the left ventricle. As a result, patients experiencing an acute myocardial injury often manifest with the symptoms and signs of decreased output of the left ventricle, pulmonary edema, and activation of the sympatho-adrenal axis.

Patients who have infarcted myocardium may develop “cardiogenic shock”—dead muscle does not contract. Therefore, stroke volume of the affected ventricle decreases. The degree of impairment is related to the size and location of the infarct. The larger the amount of dead muscle, the greater will be the impairment of function. Small infarcts may be compensated by hypertrophy of the healthy myocardium. Larger infarcts may destine the patient to repeated episodes of CHF or shock related to impaired stroke volume. Currently, there is not treatment that replaces infarcted muscle. Treatments are directed at enhancing the function of the remaining myocardium (digoxin) and decreasing the afterload on the ventricle (decreasing arteriolar resistance). These patients repeated present in pulmonary edema or have a ventricular assist device installed as a temporizing measure while awaiting a heart transplant.

**Hypertension**—persistent elevation of systemic hypertension has been discussed above. Eventually, if untreated, left ventricular failure results and pulmonary edema and/or shock ensue/
Myocarditis—myocarditis is inflammation of the heart muscle most often caused by viruses or bacteria. Myocarditis also may result from circulating toxins or other chemicals. The muscle is sick and cannot perform well. The inflammatory process generally involves both ventricles. Patients may have a history of a recent infection. If the infectious organisms or the toxins they elaborate make their way into the circulation, they may invade the myocardium—the hearts see all of the blood volume each minute and thus, have a huge exposure to the offending organism or substances both intra-ventricularly and through the coronaries. A typical case occurs with a viremia from a respiratory infection—the time when a person feels crappy—malaise, chills, fever, muscle aches, etc. Exercise increases cardiac output and coronary perfusion that exposes the myocardium to higher levels of viruses and may increase the likelihood for developing viral myocarditis. Thou shalt not exercise when thou hast a viremia! Sick muscle does not perform well and cardiac output may deteriorate relatively quickly with the patient presenting with decreased cardiac output and tissue shock or develop a dysrhythmias or arrhythmia due to changes in the irritability of sick muscle. The sick muscle may recover completely, partially, or may heal by scarring and permanent dysfunction. Treatment consists of support of vital functions, rest, and treatment of rhythm abnormalities. Myocarditis is a cause of cardiomyopathy.

Cardiomyopathy—cardiomyopathy is a functional change in the myocardium that impairs myocardial function. Myocarditis or poisoning with chemicals (i.e., alcohol) may cause the development of cardiomyopathy. Sudden increases in afterload may cause irreversible dilatation and failure of the myocardium (pulmonary embolism). Many cases of cardiomyopathy currently have no specific cause that can be identified. The myocardium develops severe failure with the manifestation of sympatho-adrenal activation. Most patients with severe cardiomyopathy require a heart transplant and many will have a cardiac assist device inserted while they are awaiting a heart transplant.

Valve insufficiency, Congenital Heart Disease—disease may involve the cardiac valves. Insufficient valves and abnormal pathways that are genetically determined facilitate backward (!!!),(wrong directional flow.) Backward flow increases the amount of work the ventricle must provide as part of the ejection goes the wrong way. Insufficiency of a valve results in ventricular hypertrophy and ultimately ventricular failure. Thus, ventricular work must increase (if it can) to provide normal levels of cardiac output. These conditions may be severe but will not be diagnosed in the field. Treatments are directed at keeping them alive until they can be transferred to the hospital staff. Patients or their families generally know of the pathology.
**Electrical**

Electrical disturbances are abnormal or absent cardiac rhythms. The rhythms that cause shock may be too slow or too fast. The arrhythmias of asystole and ventricular fibrillation are called cardiac arrest (no cardiac output without chest compressions). The treatment for ventricular rates that are going too fast to allow adequate ventricular filling are treated by slowing the rates of ventricular contractions, and rhythms that are going too slow must be sped up using drugs or electrical pacing. Electrical disturbances affect the output of both ventricles.

*Too Fast*—cardiac rhythms that are too fast generally are associated with decreased time for ventricular filling from the venous compartment. This results in less ventricular filling not related to decreased preload. Decreased filling results in decreased stroke volume and impaired cardiac output. Rhythms that may be too fast include ventricular tachycardia, supraventricular tachycardia (sinus tachycardia, nodal tachycardia), and atrial fibrillation or atrial flutter. Slowing the ventricular rates is accomplished by carotid sinus massage, the administration of negative chronotropic agents (adenosine, lidocaine, amiodarone, beta blockers (metoprolol, atenolol)), or/and synchronized cardioversion.

*Too Slow*—cardiac rhythms that are too slow impair cardiac output but are not related to changes in filling pressures. Rhythms that are too slow may include agonal, ideoventricular, nodal bradycardia, supraventricular bradycardia (including sinus and nodal), and atroventricular blocks. The treatment consists of speeding up the ventricular rate using drugs such as atropine, sympathomimetic agents, e.g., isuprel, epinephrine, dopamine), or/and electrical pacing.

*Nothing*—any rhythm is better than no rhythm. Arrhythmias (without rhythm) result in no cardiac output (note: atrial fibrillation is an arrhythmia that does produce cardiac output). The arrhythmias of concern are asystole and ventricular fibrillation. The treatment of patients with either consists of trying to get something from nothing. The most important of these arrhythmias is ventricular fibrillation. Ventricular fibrillation often occurs when a premature ventricular complex (PVC), originating from a region in the myocardium that is oxygen deprived, occurs on the T-wave of the preceding QRS (regional hypoxia). VF is a sudden-onset event, usually has some oxygen reserve, and very amenable to treatment with defibrillation—the earlier after onset, the greater the likelihood of successful conversion. Failure of defibrillation usually is due to inadequate oxygenation of the
myocardium by the coronary circulation. Coronary circulation may be enhanced by the performance of CPR and the use of pressor agents (epi, norepi) followed by repeat defibrillation.

Asystole is a terminal state. Whereas, ventricular fibrillation results from regional hypoxia of a portion of the myocardium, asystole is the result of global hypoxia/anoxia of the myocardium that results in a gradual drop in pacemaker from sinus tachy to sinus brady to nodal brady to idioventricular to agonal; asystole does not occur suddenly. Hopefully, all of the bodies in the morgue are in asystole. Successful restarting of a heart from asystole is rare (may be better in kids). Treatment consists of CPR and the administration of pressors to optimize coronary blood flow during CPR and positive chronotropic drugs (atropine, isuprel, epi, norepi). Capture may be obtained (though unlikely) using external pacing.

**Obstructive Shock States**

Obstructive shock states are caused by something being in the way of the circulation such as a blood clot or compression of the systemic arterial compartment. Treatment consists of removal of the obstruction and maintenance of coronary and cerebral perfusion.

*Tension pneumothorax*—as described in the pulmonary pathophysiology section of this course, a tension pneumothorax is associated with air getting in between the two pleural surfaces and not being able to escape from the interpleural space. A tension pneumothorax can occur from a leak from the lung parenchyma, a disrupted major airway, or through a sucking chest wound. As more air gets into the interpleural space, the intrapleural pressure gradually increases and eventually will be greater than the pressure in the systemic venous compartment, and the pulmonary and systemic veins are compressed interfering with filling of the right and left ventricles, and hence, compromised cardiac output due to poor ventricular filling pressures. Tension pneumothorax can be detected by asymmetry of the two sides of the chest with the hyperinflated side being larger than the unaffected side; the affected side moves less during breathing than does the unaffected side of the thorax. Breath sounds during spontaneous breathing will be decreased or absent, but may be present during positive pressure inspiration. Tracheal deviation away from the side of the pneumothorax may be present. Often, cyanosis of the skin and mucous membranes above the nipple line is present. Subcutaneous emphysema may appear in the area of injury or in the neck and may dissect down to the abdomen and into the genitalia. Treatment consists of venting the affected hemithorax by insertion of a catheter over a needle into the interpleural space in the second intercostals space in the mid-clavicular line, bouncing off of the third rib (so as not to impinge a intercostal vessels along the bottom of the second rib). Relief of the pressure usually allows return of the filling pressures of the ventricles and restores cardiac output. After relief of the intrathoracic pressure, sucking chest wounds
should be covered by Vaseline gauze dressing taped only on three sides—the open side will allow venting of pressure if there is further accumulation of air in the hemithorax. Following trauma, a tension pneumothorax may become apparent or more apparent on initiation of positive pressure ventilation.

**Pulmonary embolism**—a pulmonary embolism is a part of a blood clot that forms in the systemic venous compartment (usually legs or pelvis) and migrates through the systemic venous compartment including the right atrium, through the right ventricle, and passes through the pulmonic valve into the pulmonary arterial compartment where it continues until it lodges in the smallest artery it can get into, where it obstructs blood flow. A pulmonary embolism may cause an infarction of the lung distal to the obstruction. Generally, only a part of the whole clot breaks away from the body of the clot. Depending on the size of the embolism determines the problems created. The clot can be relatively small and occlude flow only through a small artery, or it may be so large as to occlude the main pulmonary artery and occlude flow to both lungs (saddle embolism). Sudden obstruction of part (or all) of the pulmonary arterial compartment increases the resistance of blood flow through the lungs, suddenly raising the afterload on the right ventricle, which may dilate and fail. An acute pulmonary embolism may cause chest pain in the area of the lung involved, may caused sudden onset of shortness of breath, cyanosis above the nipple line, venous distension of the neck veins, and/or sudden cardiac arrest. Wheezing may be present over the affected area of the lung and the chest pain may be present only with or accentuated by breathing. Immediate treatment consists of efforts to maintain coronary and cerebral blood flow. Jabbing chest compressions may push the clot further out into the pulmonary arterial compartment.

**Cardiac Tamponade**—the pericardium is a tough membrane that surrounds the heart. The epicardial surface of the hearts and the inner surface of the pericardium consist of a shiny, slippery material that allows the heart to move within the pericardial sac with very little resistance. The pericardium is not very elastic and does not stretch easily. Cardiac tamponade occurs when fluid collects between the epicardium (outside of the heart) and the pericardium. Relatively large accumulations (400-600+ ml) of fluid between these surfaces impair ventricular filling for the same preload. As the fluid accumulates, the heart cannot expand sufficiently to allow adequate ventricular filling, and thus, stroke volume declines progressively.

Cardiac tamponade can occur with fluid, pus, or blood. Blood can leak into the pericardial sac following a penetrating wound to the heart, heart rupture from compression or through an area of infarction, pericarditis, decreased proteins in the blood, or cancer.

Symptoms relate to the activation of the sympathoadrenal axis. Heart tomes may be decreased or may not be heard. Changes may occur in the ECG—the amplitude of the QRS-complexes may be low.
The treatment for life-threatening tamponade is pericardiocentesis (removal of the fluid). Pericardiocentesis may be life-saving.

**Pericarditis** (constrictive)—pericarditis is inflammation of the pericardium. The most common causes include infections, trauma, myocardial infarction, and cancer. Pericarditis involves both the epicardium and the pericardium. Pericarditis may result in accumulation of fluid in the pericardial sac. Most importantly, pericarditis may heal with the formation of scar tissue that welds the pericardium to the epicardium. Scar tissue is even less elastic than is the pericardium. The scar tissue constricts the heart much the same way as does tamponade. Constrictive pericarditis may follow an acute/chronic infection of the pericardium.

**Aortic and/or Mitral Valve stenosis**—stenosis (narrowing/scarring) of the aortic valve increases the resistance to ejection of blood from the left ventricle—the left ventricle must work harder to eject blood into the aorta. This increase in the work of the ventricle results in hypertrophy of the left ventricle and the problems previously discussed. Aortic stenosis may occur with advancing age, with formation of atheromas, or in association with rheumatic fever. Aortic stenosis may be relieved by dilation of the valve with a balloon inserted on a catheter or with surgery. Importantly, recall that the orifices of the coronary arteries are incorporated into the aortic valve. This may complicate the process and/or the treatment. When the aortic valve is open, coronary flow decreases (during systole).

Mitral stenosis (narrowing of the mitral valve is a common complication of rheumatic fever. Mitral stenosis impedes the rate of flow from the left atrium into the left ventricle and thus, decreases filling of the left ventricle. Stenosis of the aortic and mitral valve will not occur suddenly, and patients generally know of their condition as they have been evaluated multiple times. Treatment includes incising the mitral valve leaflets (scarred together) or replacement of the mitral valve with an artificial mechanical valve or a valve harvested from a pig. Patients with uncomplicated valvular disease generally will not present to EMS unless the processes are complicated resulting in the development of CHF or of acute myocardial injury. EMS personnel will not treat the valve, but will be called for severe complications such as ventricular fibrillation or sudden onset/worsening of pulmonary edema.

**Rupture of papillary muscle**—a papillary muscle may be involved in a myocardial infarction. The muscle may rupture several days following an acute myocardial infarction. An infarct of a papillary muscle results in the sudden onset of mitral insufficiency and the patient may present with pulmonary edema.

**Normal or Increased Cardiac Output**
Global cellular shock also may result without compromise in in cardiac output and may occur with elevated levels of cardiac output. The perfusing blood may have inadequate oxygen content to meet the metabolic demands of the tissue or the blood with adequate or increased oxygen content may not reach the capillaries (distributive).

**Decreased Oxygen Content**

The oxygen content of the blood perfusing the tissues may be decreased because of decreased hemoglobin concentration of the blood (“anemia”), blood loss, inability of the pulmonary system to oxygenate the blood as it passes through the pulmonary capillaries, or abnormal hemoglobin that cannot transport oxygen (hemoglobin binding sites cannot grab oxygen on all of its oxygen binding sites).

**Decreased hemoglobin concentration**—the concentration of hemoglobin may be decreased due to loss of hemoglobin through bleeding (often slow oozing rather than hemorrhage); for EMS this is the major cause), or may be due to inadequate production of hemoglobin or rapid destruction of the hemoglobin. Hemoglobin is produced primarily in the bone marrow, and in diseases that affect the bone marrow, the production of hemoglobin may be impaired (i.e., leukemia or other cancers, fibrosis). These are chronic conditions and do not occur quickly. Hemoglobin production may be impaired by lack of adequate stores of iron which is a key component of hemoglobin.

Hemolytic diseases are associated with the destruction of red blood cells. The by-products of hemolysis cause jaundice. There are many such diseases, and they are too numerous to describe here. Some hemoglobins cannot carry oxygen. This condition may be caused by exposure to carbon monoxide or poisoning of the cells by chemicals (i.e., cyanide, some drugs). It has been discussed in the Respiratory portions of this course.

**Carboxyhemoglobin**—human hemoglobin has 235 times greater affinity for carbon monoxide than for oxygen. CO occupies the oxygen binding sites on the hemoglobin molecule. Each hemoglobin molecule has four binding sites for oxygen. If all of the oxygen binding sites are filled by oxygen, the hemoglobin is 100% saturated with oxygen. If two of the four binding sites are filled with CO, the hemoglobin saturation with CO is 50% and if the remaining oxygen binding sites are filled with oxygen, the oxygen saturation of the hemoglobin is 50%, and so on. Unfortunately the carotid and aortic bodies cannot separate the carboxyhemoglobin from oxygenated hemoglobin and the differences are not picked up by pulse oximeters. Manifestations of CO include altered CNS function with gradual decreasing levels of consciousness. Treatments include getting the victim out of the CO-rich environment to render the pCO in the alveolar air less than in the blood—CO then diffuses out of the blood, the provision of high
concentrations of oxygen in the inspiratory air, life support as needed, and potentially exposure to hyperbaric oxygen that will supplement the free oxygen dissolved in the plasma until the CO leaves the hemoglobin. CO-Hgb detectors are available that detect the Hgb saturation with CO.

**Pulmonary failure**—inability of the lungs to transfer oxygen from the ambient environment to the blood perfusing the pulmonary capillaries results in decreased oxygen content of the pulmonary venous and systemic arterial blood. The causes and presentations of hypoxemia have been discussed elsewhere.

**Abnormal hemoglobins**—there are several hemoglobins that do not carry oxygen as well as does normal hemoglobin. Generally, the presence of these abnormal hemoglobins is well known to the patients and do not influence prehospital care.

**Abnormal Distribution of Perfusion**

Tissue hypoxia may result from conditions in which well-oxygenated blood seemingly is being delivered to the tissue, but little of the oxygenated blood actually perfuses the capillaries—oxygenated blood actually bypasses the tissue capillaries by shunting past the tissues by passing directly through low resistance pathways that link the arterioles directly with the venules (precapillary shunts). Such reactions seemingly are responses to circulating toxins or allergic reaction. Since these precapillary shunts offer little resistance and bypass the screw clamps, the flow from the arterioles into the venous compartment is very rapid, and hence, the pressure in the systemic arterial compartment falls rapidly following systole. The fall in blood pressure of the systemic arterial compartment is detected by the baroreceptors in the carotid and aortic sinuses. The baroreceptors notify the brain and activate the sympathetic nervous system responds by increasing heart rate—cardiac output increases and the system becomes hyperdynamic. However, the increase in cardiac output does not result in increasing tissue oxygenation, and tissue shock continues. In such circumstances, the brain and heart are in danger due to the lack of perfusion and administration of high doses of pressors may be required to sustain coronary and cerebral perfusion.

**Anaphylaxis**—anaphylaxis is a systemic, whole body reaction to an allergen. The rapid fall in blood pressure in the systemic arterial compartment due to precapillary shunting may be life threatening. Pulse pressure widens with a prompt fall in diastolic pressure (rapid run-off from the systemic arterial compartment into the systemic venous compartment through the pre-capillary bypass shunts). Activation of the SA axis results in tachycardia, but the alpha receptors in the arterioles are ineffective in restoring perfusion through the capillaries. The fall in diastolic pressure is threatening to coronary blood flow: coronary flow occurs primarily during diastole, and thus, diastolic pressure in the aorta is the primary
determinant of coronary perfusion. Blood pressure support with pressors and rapid infusion of intravenous fluids is essential. These reactions may be minimized by the administration of epinephrine and corticosteroids, i.e., methylprednisolone, and elimination or control of the precipitating materials. Special attention must be directed towards maintenance of cerebral and coronary perfusion.

Sepsis—sepsis is the presence of bacteria, viruses, or fungi and/or their byproducts in the blood. Sepsis causes the similar reactions to those in anaphylaxis; precapillary shunting from the arterioles into the venules without passing through the capillaries. The treatment is similar to those modalities used in the support of patients with anaphylaxis with the addition of appropriate antibiotics/anti-viral agents. Sepsis often is complicated by increases in capillary permeability and leaking of fluid into the interstitial space (edema formation). The use of corticosteroids is controversial at present.

Neurogenic—neurogenic shock states are associated with interruption of sympathetic nerves to the caudal division of the sympathetic nervous system, generally secondary to damage to the spinal cord. This results in interruption of the nerve fibers that regulate the state of arteriolar constriction distal to the level of injury, arteriolar dilatation, and a rapid fall in blood pressure as the run-off between the arterial and venous compartments is more rapid. The fall in pressures in the systemic arterial compartment stimulates the baroreceptors in the carotid and aortic sinuses, which in turn, causes a tachycardia. Filling pressures remain normal or slightly elevated. Treatment may involve the administration of pressor agents to restore vasomotor tone (arteriolar constriction), and possibly administration of fluids if filling pressures fall.

Pharmacologic—certain pharmacologic agents are potent vasodilators. Some cause dilatation of the systemic venous compartment while other may decrease systemic arteriolar tone. Some do both, e.g., when nitroglycerin is administered sublingually or by spray, it is a potent dilator of the systemic venous compartment as well as a systemic arteriolar dilator. Thus, filling pressure (preload) of the right ventricle decreases and right ventricular stroke volume decreases, and the runoff from the systemic arteriolar to the systemic venous compartment is more rapid, thus decreasing pressure in the systemic arterial compartment and dropping the afterload on the left ventricle. Similarly, the administration of furosemide intravenously results in dilatation of the systemic venous compartment and also is a loop diuretic. Thus, its administration results in decreasing the preload on the right ventricle and diuresis resulting in decreases in the volume of all of the vascular compartments. Thus, it is important to understand the actions of pharmacologic agents in order to anticipate what will happen to the patient when you administer the agent. Often, the effects can be used to the benefit of the patient. Just, be ready!
Sickle cell—sickle cell disease is a genetically transmitted abnormality of the red cells. The normally compliant red blood cells assume a sickle shape and become rigid. This results in the sickle-shaped red blood cells getting stuck in capillaries and preventing blood flow through the capillaries distal to the obstruction. This can occur in any or all of the organs and manifestations depend on the organs involved as well as the amount of obstruction. Generally, the presenting symptom is pain generated by the hypoxic organs; abdominal pain, chest pain, etc. Treatments consists of maintaining hydration and the administration of high flow oxygen until the obstruct clears.

FOCAL SHOCK STATES
The focal shock states occur in a single organ or part of an organ. They do not involve the whole body. If persistent, the may result in failure of the organ involved. Focal shock states may be the result of external compression or from a blockade of an artery leading to or within an organ. Regardless of cause, blood flow to the target organ and consequently, oxygen delivery to the organ is interrupted and the cells suffer from oxygen lack and go through the dying process as outlined above. Also, as noted, different organs or parts of organs have different anoxic survival times. Treatment consists of decompression or restring blood flow though an artery that is obstructed.

Compression
Compression refers to compression of the arterial and venous supply to an organ and can result from accumulation of fluid inside of a rigid structure (pressure increases as compartment size either becomes smaller or fluid accumulates within the compartment m arteries within the area of compression may collapse causing impaired perfusion distal to the compression.

Compartment syndrome—in compartment syndromes, edema fluid accumulates within a relatively rigid structure. This can occur in extremities that have been burned (thermal injury) circumferentially. The escar that forms is rigid and will not expand as edema fluid is formed due to increases in capillary permeability related to the products of injury to the capillary bed. As the volume of fluid trapped in this area increases the pressure within the rigid compartment rises. This pressure may increase to levels at or above the systemic arterial pressure—this process results in collapse of arteries and impairment of arterial blood flow distal to the area compression. Similar problems may result in the tissue (muscle) compartments in areas surrounding fractures of extremities. Treatment consists of prompt relief of the pressure within compartment. This may require incisions in the compartments to release the pressure. In burns, this is called “escarotomy”.

Increased Intracranial Pressure (ICP)—the brain is encased by a rigid box (skull). This is a variance of the compartment syndrome. Any accumulation of fluid (blood, edema) within the skull results in increases in the pressure within the skull. If the pressure becomes greater than the pressure within the venous sinuses, the venous sinuses collapse. If the accumulation of fluid (blood, extracellular fluid, cerebrospinal fluid) results in increased intracranial pressure that can become greater than the pressure within the systemic arterial compartment, blood flow to the brain ceases and the brain experiences a period of shock. Similarly, masses growing within the brain increase the volume within the skull and may result in increased intracranial pressure ultimately impairing perfusion to the brain. Treatment consists of fluid removal from inside of the skull or by the administration of pharmacological agents that draw fluid out of the brain that can be removed from the body by the kidney by osmotic diuresis. Victims of closed head injuries may have pressure monitoring devices placed inside the skull and/or may have catheters placed into the ventricles of the brain from which fluid can be withdrawn. Some congenital defects may cause obstruction of outflow of cerebrospinal fluid from the skull into the spinal canal. Such conditions result in increased fluid within the skull (hydrocephalus) and this, increases in intracranial pressure. Often, “shunts are placed that drain the CSF from inside the skull into the GI tract. These shunts may become plugged/kinked and may result in increases in ICP; if such occurs, the shunts must repaired/replaced by the neurosurgeons.

Crush syndrome—pressure by external masses on extremities (i.e., heavy weights) may compress the arteries under the area of contact. The results are the same as occur in the compartment syndromes, namely that those areas distal to the area that is being compressed are deprived of perfusion, and hence suffer from deprivation of oxygen. Further, the compression may physically disrupt cell membranes in the tissues underlying the compressing object. The obvious treatment is to relieve the compression as quickly as is possible. However, this must be done with caution as cell membranes may have been disrupted and cell contents that can damage other cells may have been released into the intercellular spaces (interstitium) and may make their way into the circulation, especially via the lymphatics. Thus, cells distant from the area of compression may be damaged (e.g., lungs, kidney). Renal failure is a relatively common complication of crush.

Traumatic asphyxia—traumatic asphyxia results from a heavy weight compressing the thorax and/or the abdomen. The compression compromises ventilation and the increased intrathoracic pressure impairs venous return to the right heart. Blood accumulates in the pulmonary venous compartment, especially in the area drained through the superior vena cava (few and incompetent valves). Intracapillary pressure
increases and many capillaries rupture. This process creates cyanosis of the skin above the nipple line due to deoxygenated hemoglobin leaking into the interstitial space. Victims appear dead. Many patients with traumatic asphyxia are not dead and resuscitation should be attempted. It should be noted, that the compression must be released, as such victims cannot breathe with the chest being compressed. Abdominal compression forces the diaphragm high into the thorax. This was a major problem during the crush in Camp Randall in 1993. All 10 victims who were pulseless and non-breathing survived!

*PASG*—the pneumatic antishock trousers (PASG) are pants with air bags that can be wrapped circumferentially around the lower extremities and across the abdomen. Inflation of the bladders compresses the extremities and abdomen. The pressures under the inflated trousers are sufficient to collapse the circulation in the extremities and abdomen and may be used to tamponade hemorrhage or to centralize perfusion as only the chest, lungs, arms, coronaries and brain are perfused. They have been used in treatment of hypovolemia and for tamponade of a leaking/ruptured abdominal aortic aneurysm.

**Intravascular Obstruction**

Complete or partial obstruction of systemic arteries leading to an organ or part of an organ can occur due to the formation of a blood clot with the vessel lumen or the formation of subintimal plaques in the arteries. The result is shock related to ischemia of the end organs. The intravascular obstruction may be complete or incomplete. Incomplete obstruction compromises flow to the organ and the tissues may become ischemic (cell shock) especially when the metabolic activity of the organ increases. This is characteristic of the coronary syndromes, in which increased energy requirement created by the augmented myocardial work cannot be met by the oxygen delivery often resulting in chest discomfort and other symptoms and signs associated with the acute coronary syndromes.

**Atherosclerosis**—the role of atherosclerosis has been discussed elsewhere. Arterial obstruction is common in patients with diabetes and often results in losses of parts of extremities. Recall that blood squirting through a narrowed lumen of an artery tends to break blood cells and platelets apart. The contents of these cells are thromboplastic and may result in the formation of a clot beyond an area of partial occlusion created by a plaque. This is a common mechanism for the development of myocardial or brain ischemic injuries associated with myocardial infarction and strokes. Hemorrhage may occur under an atheromatous plaque and push it further in to the lumen creating arterial obstruction. Treatment is to dissolve the clots using thrombolytic agents and/or dilate the offending vessel using an inflatable balloon that breaks up the plaque (angioplasty) and is followed by placing an intravascular stent into the area to help keep the vessel open.
Blood clots—as noted above, blood clots may form in any part of the systemic arterial compartment. The formation of clots results in ischemic injuries to the tissue beyond the clot, and eventually failure of that organ and infarction. Focal shock states must be reversed before infarction occurs.

Shock states cause much damage to the tissues. The causes must be diagnosed by history, physical examination, laboratory studies, and sometimes by imaging.