SHOCK: EARLY RECOGNITION AND MANAGEMENT

Author: Leigh Anne Wilmot, RN, CEN, CLN, Austin, TX

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Shock is a syndrome characterized by inadequate tissue perfusion. This physiological state arises from multiple causes: hypovolemia, neurogenic trauma, anaphylaxis, sepsis, and cardiac pump dysfunction.

Shock is a medical emergency that, if left untreated, leads to significant morbidity and mortality. While the data are limited regarding other causes of shock, septic shock is listed as the 10th leading cause of death in the United States. The monetary cost to the health care system is estimated to be $16.7 billion a year. The human cost is approximately 115,000 deaths a year.[1]

With these sobering statistics, the focus of providers should be on early recognition and intervention. Understanding the pathophysiology of the various shock states and implementing appropriate treatment modalities rapidly can significantly affect the outcomes of these critically ill patients.

Pathophysiology

The definition of shock is a syndrome in which the imbalance of oxygen supply and demand leads to decreased tissue perfusion and impaired cellular metabolism. Basically, when a cell does not receive an adequate supply of oxygen, it cannot effectively utilize the substrates such as carbohydrates and glucose necessary for energy and metabolism. This situation causes lactic acid and other waste products to build up in the cells. As these toxins accumulate, cellular injury, inflammation, and death occur. This condition is known as the systemic inflammatory response syndrome (SIRS). Providers should suspect SIRS in patients who present with two or more of the criteria listed in Figure 1.

Triggered by the body's immune response to infection or trauma, SIRS causes a variety of cellular processes. In the liver, the Kupffer cells are responsible for reharvesting the heme- or oxygen-carrying portion of the red blood cells. When the Kupffer cells become inflamed and die, the heme is unable to be reabsorbed and put back into circulation. Subsequently, a decrease in hemoglobin occurs, causing a further decrease of oxygen delivery to the tissues.

Most metabolic processes such as the growth and repair of tissue occur in the presence of oxygen (aerobic respiration). Through a process called glycolysis, substances such as carbohydrates and sugars are broken down into adenosine triphosphate (ATP). ATP is the "fuel bank" for the body. In aerobic respiration, up to 30 molecules of ATP are produced. Conversely, with anaerobic respiration or respiration without the presence of oxygen, only 8 molecules of ATP are produced.[2] Without the ATP necessary to carry out cellular processes such as regeneration of cells and processing of byproducts, cellular death starts to occur.

As cells die, lactic acid and other harmful byproducts are released into the circulation. Subsequent cellular death occurs as the byproducts wash across neighboring cells. Chemical processes detecting this cellular death then trigger the clotting cascade in an effort to halt the damage.

When there is significant cellular damage and significant over-production of clotting factors, small clots may become lodged in the blood vessels, causing interrupted blood flow to the organs. Microvascular thrombosis, hypoperfusion, ischemia and tissue injury can result, further triggering stimulation of inflammation and the clotting cascade. This hyperactivation eventually depletes the patient's ability to clot, resulting in disseminated intravascular coagulation.[3] Once the process of disseminated intravascular coagulation begins, multi-organ failure and death become almost inevitable.

Understanding Shock

Shock is divided into classifications based on mechanism: low circulating volume (hypovolemic shock), vasodilation (distributive shock—anaphylactic, neurogenic, and septic), or a primary decrease in cardiac output (cardiogenic).

Hypovolemic Shock

A 17-year-old male presents to the emergency department via EMS. He was riding his dirt bike on a cross-country trail when he struck a tree. He has bruising over his right upper quadrant and is complaining of severe pain with palpation. Vital signs are as follows: blood pressure (BP), 86/50; heart rate, 122; respiratory rate (RR), 24; temperature, 96.5°F;

Leigh Anne Wilmot is Staff RN IV, Team Leader, Seton Medical Center, Austin, TX.

For correspondence, write: Leigh Anne Wilmot, RN, CEN, CLNC, 10900 Research Blvd, Suite 160C, Austin, TX 78759; E-mail: lwilmot@seton.org.

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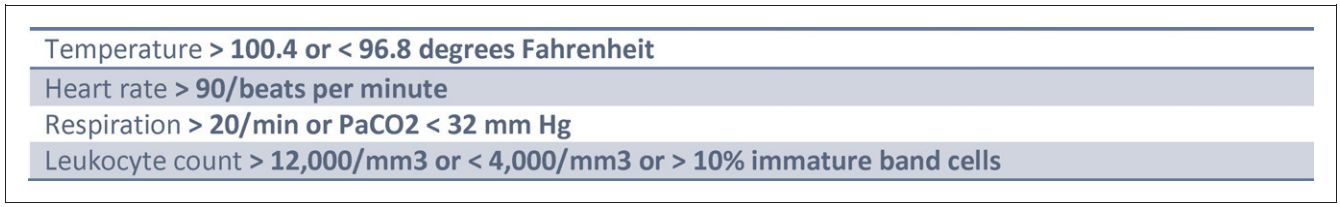

FIGURE 1

Criteria for systemic inflammatory response syndrome. $Paco_2$, Partial pressure of carbon dioxide in arterial blood. This figure can be viewed in color and as a full-page document at www.jenonline.org.

and oxygen saturation, 94% on room air. The patient is cool, sweaty, and appears confused. The most likely cause of this patient's distress is hypovolemic shock secondary to hemorrhage from a liver laceration.

Hypovolemic shock occurs from loss of blood, plasma or fluid. Trauma patients, patients with leaking abdominal aortic aneurysms, burn patients with large partial to full-thickness burns, alcoholic patients with esophageal varices, and patients with severe diarrhea and vomiting all are at risk for this condition.

This shock state is characterized by a critical decrease in intravascular circulating volume. When this volume is depleted, the amount of preload delivered to the heart is diminished. The lack of significant preload results in decreased cardiac output, which in turn causes decreased oxygen delivery to the tissues. As discussed previously, SIRS begins, and if left unchecked, it progresses to shock and death (Figure 2).

Therefore, treatment for hypovolemic shock includes rapid blood and/or fluid replacement with treatment to stop the cause of the fluid loss. Severe cases of hypovolemic shock may require vasopressive agents such as Levophed or dopamine depending on the cause. Cardiac monitoring, frequent vital signs, and monitoring of intake and output are essential. Patients should receive supplemental oxygen to assist with perfusion. These patients also are at high risk for falls secondary to dizziness and altered mental status. Close visual monitoring is recommended as well.

In cases of trauma, esophageal varices, or leaking abdominal aortic aneurysm, rapid surgical intervention may be required.

Distributive Shock: Anaphylactic, Neurogenic, Septic

The distributive shock state is characterized by changes in blood vessel tone. Often distributive shock mimics hypovolemic shock, but the mechanism is different. Distributive shock is analogous to turning on all of the faucets in a house at once. The circulating volume stays the same, but the systemic vascular resistance is decreased. Consequently, the vessels cannot deliver the circulating volume to the organs. Without the volume, oxygen delivery is impaired, beginning the cycle of cellular inflammation. To halt this cycle, the vessels need more volume to fill the void and the systemic vascular resistance needs to be increased.[4]

The cellular changes associated with distributive shock can be triggered by a number of factors including spinal cord injury, allergic reaction, and the body's response to infectious processes. Other factors to consider include cerebral edema, as in brain injury resulting from strokes and trauma, and depression of the medullary brain stem with general anesthetics or drug overdoses such as opiates, tranquilizers, and barbiturates. For these reasons, distributive shock is often subdivided into 3 other categories: anaphylactic, neurogenic and septic.

A listless 2 year old is rushed into the emergency department in his mother's arms. She relates that he was eating a peanut butter cookie when he began crying and rubbing his mouth. Within seconds his lips and eyes became swollen and a raised rash developed over his trunk and extremities. His breathing became labored, and audible wheezing could be heard. His mother states that he has never eaten nuts before. Vital signs are as follows: BP, 86/33; pulse, 185; RR, 52; temperature, 97.6°F axillary; and oxygen saturation, 88% on room air. The most likely cause of this patient's distress is distributive shock –anaphylactic.

Anaphylactic shock is typically triggered by an acute allergic reaction to bee stings, drugs, or food-based or iodine-based substances such as seafood or contrast dye. However, the immune system is dynamic and can react to anything in the environment.

Patients typically present complaining of headache and lightheadedness. They may become flushed with raised whelps that itch profusely. These patients are extremely anxious and may complain of an impending sense of doom. If left unchecked, severe allergic reactions can progress to respiratory and cardiovascular collapse.

Interventions should include establishing and maintaining an airway and providing supplemental administration of

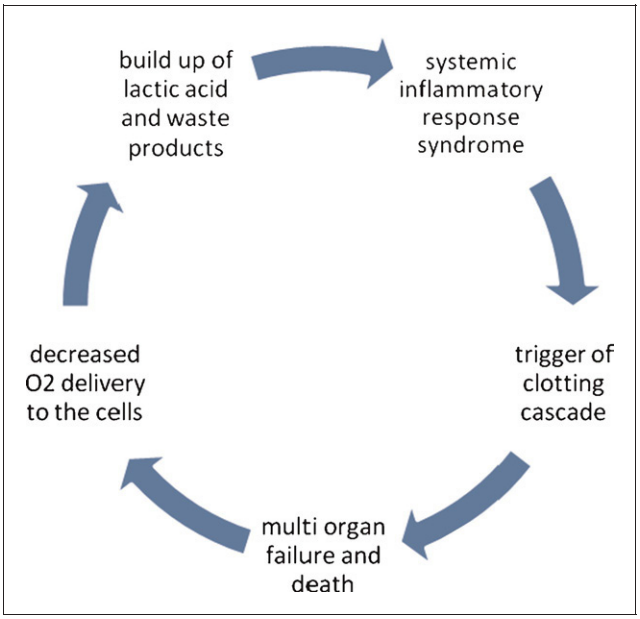

FIGURE 2
Shock cycle.

oxygen. Some patients may require bronchodilators or racemic epinephrine if wheezing or stridor is present. In severe cases, intubation may be required with inline nebulizer treatments. Atrovent inhalers should be avoided in patients with soya lecithin allergies (soybean and peanut) because of a preservative the inhaler contains. Patients have been known to adversely react to this medication when in this form. However, albuterol and Atrovent nebulizers are safe in this patient population as they do not contain the preservative.

Intravenous fluid boluses with crystalloids (lactated Ringers or normal saline solution) are recommended. The provider should consider administration of subcutaneous epinephrine to increase systemic vascular resistance. Other treatment modalities include administration of histamine blockers such as Benadryl and Pepcid. Steroids such as Solu-Medrol or dexamethasone are utilized to decrease the body's inflammatory response to the allergen. These are long-acting medications, however, so a result is not immediately achieved.

The patient will need to be observed for several hours to ensure that he or she does not rebound once the antihistamines and epinephrine have worn off. Upon discharge, the patient may be prescribed an Epi-Pen (self-administered epinephrine) to carry with him or her at all times in the event of another episode.

A 72-year-old man is brought to the emergency department via EMS. He sustained a 10-foot fall from a ladder onto his back. He is awake and alert. His vital signs are as follows: BP, 80/50; pulse, 55; RR, 26; temperature, 96.6; and oxygen saturation, 91% on room air The patient complains of mid low-back pain and decreased ability to move his legs. His legs are pink, warm, and dry, but you notice that above his waistline he is pale, cool, and clammy. The most likely cause of this patient's distress is distributive shock—neurogenic.

The most common cause of neurogenic (spinal) shock is traumatic injury to the spinal cord. These injuries can be sustained from motor vehicle accidents, falls, violence, or sports injuries. Other causes of neurogenic shock can be from tumors, infectious conditions, vertebral fractures that occur with osteoporosis, and iatrogenic injuries such as postepidural catheter placement or spinal injections.

With neurogenic shock, the sympathetic nervous system is interrupted. When this interruption occurs, catecholamines are not released into the blood stream, and without this release, the classic signs of shock such as tachycardia and diaphoresis are inhibited. Instead, patients in neurogenic shock will have a triad—slow, weak pulses, hypotension, and hypothermia.[5] Unless there is a cervical spine injury that inhibits the phrenic nerve, patients also will have rapid respiratory rates to compensate for the increased demand of oxygen to the tissues.

Neurogenic shock is further complicated by the patient's general appearance. Vasodilatation below the level of the injury causes these patients to remain pink, warm, and dry. However, proximal to the injury, skin is cool, clammy, and pale. This mismatch occurs because the sympathetic nerves above the injury are not interrupted and can still respond as they normally would. For these reasons, a detailed physical assessment and history is imperative along with close monitoring of neurologic status.

Interventions should be focused on rapid treatment. Establishing and maintaining an airway is always a priority, and supplemental oxygen should be provided to assist with oxygen delivery to the tissues. At least 2 large-bore intravenous lines should be placed and rapid fluid administration with crystalloids begun.

In some cases of severe hypotension, vasopressors such as dopamine or Levophed may need to be started. The provider should be cautious in administration of Levophed, however, because of the vasoconstrictive effects on the kidneys.

If the patient is severely bradycardic, he or she may require atropine administration.

Patients should be kept warm to prevent shivering, which can increase the body's oxygen demand and decrease oxygen delivery to the tissues.

The provider should recognize that neurogenic shock can be worsened as swelling around the spinal cord increases. High doses of steroids such as Solu-Medrol and dexamethasone can be effective in cord swelling if administered within

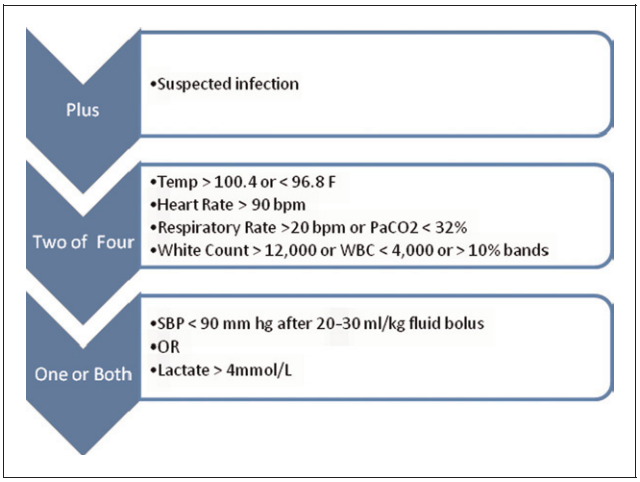

FIGURE 3

Early recognition of sepsis. *bpm*, Beats per minute; *Pa_{CO_2}*, partial pressure of carbon dioxide in arterial blood; *SBP*, systolic blood pressure; *WBC*, white blood cell count.

8 hours of the injury and should be considered as a treatment. However, use of steroids may not be appropriate in all spinal trauma, as surgical intervention to relieve pressure around the spinal cord may be the most effective treatment.

A 55-year-old diabetic woman presents to the emergency department complaining of bilateral flank pain, foul-smelling urine, vomiting, and chills for 3 days. She is lethargic and her skin is pale and cool. Vital signs are as follows: BP, 90/60; pulse, 112; temperature, 96.6°F; RR, 22; and oxygen saturation, 93% on room air. The most likely cause of this patient's distress is distributive shock—sepsis.

Any patient with an infectious process has the potential to become septic. Infection triggers the release of cytokines and other inflammatory processes (SIRS). If left unchecked, SIRS then triggers the clotting cascade (Figure 2), as discussed previously. Through a complex series of events, a simple infection can turn into fulminate septic shock. Providers should maintain a high index of suspicion for sepsis if the following is present: Vulnerable populations such as the elderly, persons with diabetes, and those who are immunocompromised (ie, oncology, HIV and hepatitis positive, status, postoperative splenectomy) are at increased risk for sepsis and should be treated with high priority when infection is suspected.

Through examination of the aforementioned case scenario, the provider easily could be misled into believing that the patient was simply volume depleted from the vomiting or experiencing a diabetic crisis. The ominous vital sign, however, is actually the temperature.

When a person's immune system is overwhelmed, he or she may lose the ability to generate a fever or white blood cell response to fight the infection. Septic patients often present as hypothermic with normal or low white blood cell counts. Recognizing the potential for infection in the absence of fever and elevated white blood cell count and intervening appropriately are imperative in the treatment and prevention of sepsis.

The focus of care should be on rapid identification of potentially septic patients and prioritization of treatment modalities (Figure 3). Supporting breathing and ventilation with supplemental oxygen is a priority. Providers should establish 2 different intravenous sites with the forethought that the patient potentially will need incompatible medications. Establishing a central line takes time and can delay treatment modalities, but peripheral intravenous lines usually are established rapidly.

Blood cultures should be obtained with each intravenous line start. A urine sample and sputum culture should be obtained, as well as cultures from indwelling vascular access devices. As soon as a potential source of infection is identified, appropriate antibiotics should be administered. Ideally, the potential sources of infection are collected prior to the administration of antibiotics to ensure proper treatment of the infection, but the key to combating sepsis is rapid administration of antibiotics. Studies have shown that for every hour that antibiotics are delayed, survival decreases by 7.6%.[6] Providers play a vital role in this regard and should be advocating for antibiotic therapy when sepsis is suspected.

Other treatment modalities are similar to those used for the other shock states, with one of the first-line treatments in septic shock being fluid boluses of crystalloids to compensate for the vasodilatation. Blood transfusions are given to replenish the hemoglobin that is lost when the Kupffer cells are affected.

Oftentimes these patients require intubation to increase oxygen delivery to the tissues. One way of effectively monitoring the body's oxygen consumption rate is through central venous oxygen saturation ($SCVO_2$) monitoring. This monitoring is accomplished with a special central venous catheter that has an attachment for $SCVO_2$ monitoring. In knowing this measurement, the effectiveness of treatment on tissue perfusion can be established. As discussed previously, improved oxygen delivery to the tissues is the goal in halting SIRS. The $SCVO_2$ monitor indicates whether the patient is responding to interventions.

Central venous lines become imperative so that the central venous pressure can be monitored accurately as well. In knowing the central venous pressure, fluids can be titrated accordingly. Septic patients can appear hydrated when in fact they have a fluid volume deficit that is only picked up with central venous monitoring. For this reason,

monitoring of the mean arterial pressure (MAP) is helpful. For a MAP of less than 60 mmHg or a drop of 40 mmHg from baseline, the patient is considered to be in shock.

Vasopressors may be utilized if the patient becomes hypotensive, with Levophed being the preferred vasopressor over dopamine. With dopamine, the adrenal glands are stimulated, putting more catecholamines into the circulatory system. This catecholamine release is potentially detrimental because catecholamines are instrumental in SIRS, leading to worsening of the shock cycle (Figure 2). However, if the MAP falls below 60 mmHg, dopamine is utilized concurrently to help with renal perfusion. (Recall that Levophed has vasoconstrictive properties.)

A newer modality in the treatment of sepsis is the use of recombinant human activated protein C. With activation of SIRS, the clotting cascade can be stimulated. Recombinant human activated protein C is thought to inhibit thrombosis and inflammation as well as regulate coagulation.[4] Due to the mechanism of action of this endogenous protein, however, the patient is at risk for bleeding and must be monitored closely. This treatment also can be cost-intensive and is not readily available in most facilities.

Tight glycemic control recently has been recognized in the care of critically ill patients as a potential benefit for improving patient outcomes. Some studies are recommending stringent glycemic control in the treatment of sepsis as well. The Surviving Sepsis Campaign focuses on maintaining blood glucose levels of less than 150 mg/dL, but other studies have shown an increased survival rate with blood glucose levels of 80-110 mg/dL.[7]

Cardiogenic Shock

A 68-year-old man presents to the emergency department complaining of severe midsternal chest pain that radiates to his left arm and jaw. He reports shortness of breath, nausea, and dizziness. He is lethargic, pale, and diaphoretic with mottled extremities. Rales are heard bilaterally upon auscultation of his lung sounds. Vital signs are as follows: BP, 72/50; pulse, 118; temperature, 96.8°F; RR, 22; and oxygen saturation, 89% on room air. The most likely cause of this patient's distress is cardiogenic shock from an acute myocardial infarction.

Cardiogenic shock is characterized by the decreased ability of the heart to pump effectively, causing decreased tissue perfusion. The most common cause of cardiogenic shock is acute myocardial infarction.[8] Nonviable myocardium does not contract, leading to a reduced ejection fraction and decreased cardiac output. This in turn causes decreased tissue perfusion and the initiation of SIRS, as discussed previously.

Other causes of cardiogenic shock include thoracic aortic dissection, aortic stenosis, cardiomyopathy, mitral valve regurgitation, pericarditis, and in rare cases, systemic lupus erythematosus.[9]

Providers also need to be cognizant of medications that can induce cardiogenic shock. These medications include β-blockers such as Metoprolol and calcium channel blockers such as Diltiazem. These medications directly affect the heart's ability to contract and should be suspect in cases of unexplained cardiogenic shock.

Treatment for cardiogenic shock includes rapid identification of the underlying cause. Until the patient is transferred to a higher level of care, interventions should focus on supporting the patient. Establishing and maintaining an airway is of vital importance. Providers also should be prepared to administer vasopressors, vasodilators, diuretics, and analgesics to improve cardiac output and decrease myocardial oxygen demand.

For discussion, administering vasodilators, analgesics, and diuretics to hypotensive patients is in conflict with basic medical knowledge. In the case of cardiogenic shock, however, the benefit may outweigh the risk.

Too much preload in the heart causes blood to back flow into the lungs. This situation in turn can cause pulmonary edema, which is characterized by rales and hypoxia. As the body responds to pain and the anxiety from oxygen deprivation, myocardial oxygen demand increases and tissue perfusion decreases. For this reason, cardiogenic shock must be managed through multiple treatment approaches that are often at odds.

Once the patient is stabilized, surgical intervention may be required to correct the underlying cause of the cardiogenic failure. For example, percutaneous transluminal coronary angioplasty is utilized for the treatment of acute myocardial infarction to improve blood flow.[10] Repair of mitral valve prolapse may be necessary as well, if there is significant mitral regurgitation causing decreased cardiac output.

As mentioned previously, other possible surgical causes of cardiogenic shock include thoracic aortic dissection, cardiac tamponade, and pericarditis. Not all aortic dissections are operable, however, but some may be managed with medication and lifestyle changes. As to cardiac tamponade, pericardiocentesis or needle aspiration of fluid around the heart is the treatment for this condition. In the matter of pericarditis, the cause of the inflammation dictates the treatment modality. Surgical intervention is utilized in severe cases of constrictive pericarditis, but other forms of pericarditis are inoperable.

Lastly, in cases of severe cardiomyopathy, patients may require the use of an left ventricular assist device until such time as the heart can heal or the patient becomes eligible for heart transplantation.

Conclusion

Shock is a medical emergency that, if left untreated, results in significant morbidity and mortality in all patient populations. Understanding the pathophysiology and clinical presentation of the various shock states is imperative in improving the outcomes of these critically ill patients. If providers are to administer effective care, their focus should be on early recognition and prevention. Detailed physical assessments, thorough history taking, and repeated examinations are imperative in comprehensive patient care. Providers should maintain a high index of suspicion for shock and adjust treatment modalities accordingly.

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