EMS “Live” In-Station Continuing Education

CE Provider:	University of Texas Southwestern Medical Center at Dallas Department of Emergency Medicine Division of Emergency Medical Services
Course Title:	Shock/Hypoperfusion
Course Approval Number:	UTSW – 04M2017 TDH – 101606
Date:	April 2017
Paramedic-Level National Registry Content Area and Hours:	Mandatory: Airway Breathing and Circulation – 1.0 hour Mandatory: Obstetrics and Pediatrics – 1.0 hour Flexible: Airway Breathing and Circulation – 2.0 hours
Paramedic-Level National Continued Competency Program (NCCP) Content Area and Hours:	Airway, Respiration, and Ventilation • Ventilation – 0.5 hour • Capnography – 0.5 hour • Advanced Airway Management in the Perfusing Patient – 1.0 hour Cardiovascular • Congestive Heart Failure – 0.5 hour • Acute Coronary Syndrome – 0.5 hour Trauma • Fluid Resuscitation – 0.5 hour Local or Individual Component – 0.5 hour
Paramedic-Level TDH Content Area and Hours:	Airway Management and Ventilation – 1.0 hour Medical – 3.0 hours
Skills Proficiency Verification:	
Class Location:	_____
Instructor Name:	_____
Student Name:	_____

In order to accrue the CE hours required for recertification, the student must attend and participate in the live CE component represented by this module and complete any required skill demonstrations.

STUDENT VERSION

This form shall serve as a written record of the participant's successful completion of the EMS educational activity as outlined in the Texas Administrative Code, Title 25, Part 1, Chapter 157, Subchapter C and as outlined in CECBEMS Standards and Requirements for Organization Accreditation.

Shock / Hypoperfusion

EMT-Level National Registry Content Area and Hours:	Preparatory – 1.0 hours Airway - 0.5 hours Medical – 1.0 hours Trauma – 0.5 hours Patient Assessment – 1.0 hours
EMT-Level National Continued Competency Program (NCCP) Content Area and Hours:	Ventilation - 0.5 hours Oxygenation – 0.5 hours Cardiac Rate Disturbances – 0.5 hours Local or Individual Component – 2.5 hours
EMT-Level TDH Content Area and Hours:	Preparatory – 1.0 hours Airway - 0.5 hours Medical – 1.0 hours Trauma – 0.5 hours Patient Assessment – 1.0 hours

Cognitive Objectives: Upon successful completion of this course, the student will be able to:

- Provide ventilatory support for a patient
- Provide care to a patient who is experiencing cardiovascular compromise
- Assess and provide care to an infant or child who has shock/hypoperfusion
- Assess and provide care for an adult patient who has respiratory distress
- Use oxygen delivery system components
- Perform techniques to ensure a patent airway
- Assess and provide care to a patient who is experiencing non-traumatic chest pain/discomfort

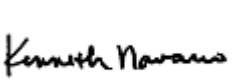
Psychomotor Objectives:

- No psychomotor objectives listed for this CE module

Affective Objectives:

- No affective objectives listed for this CE module

This continuing education activity is approved by the University of Texas Southwestern Medical Center at Dallas, an organization accredited by the Texas Department of State Health Services.



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For ECG practice or review, visit **www.ecglibrary.com**. To access the latest version of the protocols, for patient care alerts or updates on new medications, visit **www.biotel.ws**. References and bibliographies for all CE modules are on file and available upon request. To comment on this module, good or bad, e-mail **kenneth.navarro@utsouthwestern.edu**

Shock / Hypoperfusion

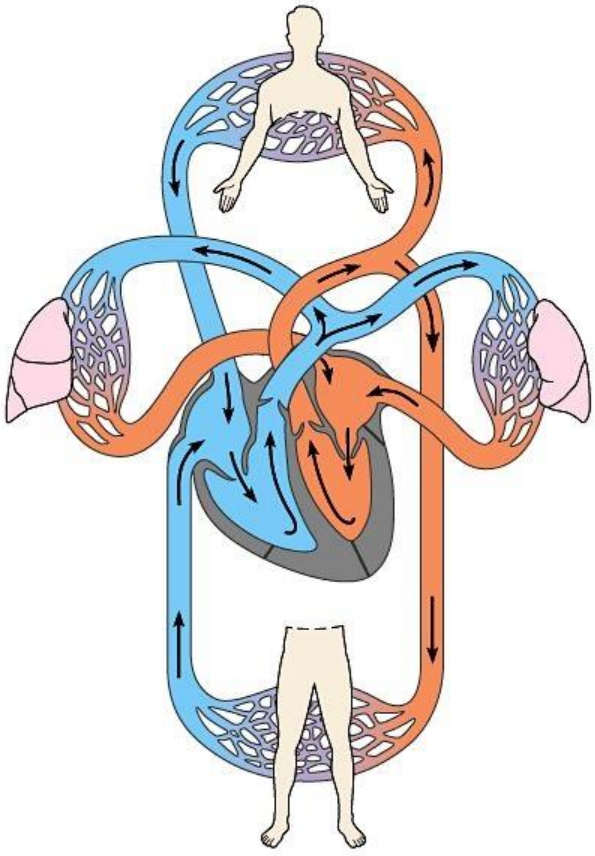
In a very broad sense, healthcare providers can define shock as inadequate capillary perfusion. However, shock is not a singular event with a specific cause and treatment. It is a complex group of physiological abnormalities caused by a variety of disease states and injuries. This lesson will review some of the more common causes of shock and outline a management strategy for each.

Introduction

Because shock is such a complex process, one cannot adequately define the condition in terms of pulse rate, blood pressure, or cardiac function alone. Shock may affect the entire body or it may occur at a tissue or cellular level, even in the presence of normal hemodynamic measurements. A review of cellular physiology will help us recognize very subtle aspects of shock and to assess its severity with some degree of accuracy.

Anatomy and Physiology Review

Most medics learn the physiology of the circulatory system using a simplified pump/pipes/tank model. In this model, the pump represents the beating heart. The pipes represent the various blood vessels while the tank represents the volume of blood itself. In order for the tissue cells to receive an adequate supply of oxygen, all three components of this system must function properly. Impairment in any singular component can result in a decrease in cellular oxygenation.



A beating heart produces pressure changes that move blood throughout the body. The volume of blood leaving the left ventricle, also known as the left ventricular stroke volume (SV), is very important in determining the oxygenation status of the tissues.

The term cardiac output reflects the amount of blood pumped out of the heart every minute. One can see the relationship between cardiac output and stroke volume by examining the following formula:

Cardiac output (CO) = Stroke Volume (SV) x Heart Rate (HR)

Referencing this formula, it is easy to see how the body must respond to keep cardiac output constant in the face of a variety of problems.

Exercise 1: Without the assistance of any compensatory mechanisms, what would happen to stroke volume if the patient began to have uncontrollable bleeding or if the left ventricle suddenly became weaker? What would be the effect on cardiac output?
Answer:

Exercise 2: What compensatory mechanism will ensure adequate cardiac output in the face of a slight decrease in stroke volume.
Answer:

Another useful formula worthy of remembering is the formula for the relationship between cardiac output and blood pressure.

Exercise 3: Complete the formula: Blood pressure (BP) = Cardiac Output (CO) x _____
Answer:

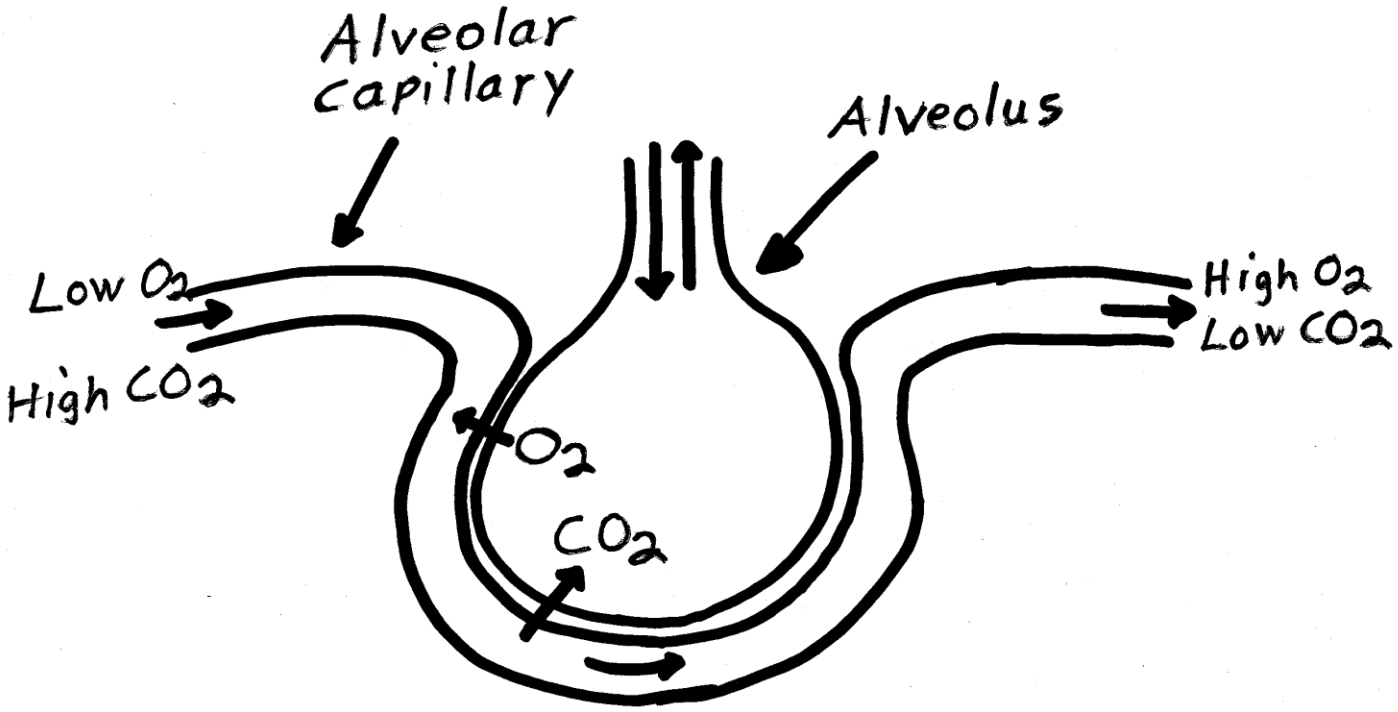
Blood leaves the heart through a single vessel known as the aorta. Almost immediately, smaller vessels begin branching off the aorta to take blood to various parts of the body. As blood continues to move forward, blood vessels get smaller and smaller. Because blood is a viscous fluid, it rubs against the inside of the vessel as it passes. This creates friction or resistance to forward blood flow. The effects of the friction accumulate throughout the length of a blood vessel thus creating a pressure gradient from one end of a vessel to the other. The pressure gradient (difference) between the two ends of the tube determines blood flow.

Exercise 4: Where in the closed system is the highest pressure? Where is the lowest pressure?
Answer:

In general, various muscle fibers surround the vascular tubes and control the vessel diameter. If the muscle fibers relax, the vessel enlarges and the pressure within that area falls. If the muscle fibers contract, the vessel shrinks, thereby increasing the local pressure. Vasoconstriction or vasodilation of the vasculature can profoundly affect blood flow and oxygen delivery to the tissues.

Fluctuations in the performance of heart muscle and the vasculature attempt to regulate the supply of blood flow to the tissues. However, the cells in the tissues need more than just the blood; they need the oxygen the blood carries.

In order for red blood cells to pick up oxygen, they must periodically have access to a fresh supply of the gas. One function of the lungs is to allow oxygen to flow into the body and encounter the blood supply. Once the red blood cells have picked up a fresh supply of oxygen, the heart can pump the cells though the vasculature to the tissues where the oxygen can diffuse out to the cells.



Shock begins when the cardiovascular system cannot meet tissue oxygen demands. This may occur because the heart is not able to pump effectively, the vasculature (pipes) enlarges causing pressure within a specific area (or the entire system) to drop, or when there is not enough fluid (or red blood cells) to carry to carry an adequate amount of oxygen to the tissues.

Hypotension (as we now know it) is NOT Synonymous to Shock

Often, medics associate shock with a systolic B/P less than 90 mm Hg. However, patients who are chronically hypertensive may have inadequate tissue perfusion with systolic B/P in the

normal range (Gamper et al., 2016). Similarly, it is possible for individuals who appear hypotensive to have normal tissue perfusion (Gamper et al., 2016).

It is also worth noting that in patients truly suffering from the effects of shock, hypotension is a late sign. In a large sample (> 115,000) of patients from the National Trauma Data Bank of the American College of Surgeons, systolic B/P did not fall below 90 mm Hg until the degree of shock was profound, which was defined as a base deficit (a marker for anaerobic metabolism) of less than -20 (Parks, Elliott, Gentilello, & Shafi, 2006). For the group that developed hypotension, mortality was high (about 60%). This study suggests the life threatening complications of inadequate tissue perfusion (shock) are present well before the patient's blood pressure falls below a commonly recognized shock threshold.

Some have argued that medical providers should raise the systolic B/P threshold for determining the presence of shock. Data from over 800,000 patients from the National Trauma Data Bank suggests the diagnostic shock threshold should probably be around 100 mm Hg for systolic B/P readings (Eastridge et al., 2007). Even that threshold may be too low for elderly patients (Oyetunji et al., 2011). After reviewing over 24,000 moderate to severely injured patients without major head injury cases from the Los Angeles County Trauma Database, researchers determined the optimal definition of hypotension was systolic B/P of 100 mm Hg for patients 20 to 49 years, 120 mm Hg for patients 50 to 69 years, and 140 mm Hg for patients 70 years and older (Edwards et al., 2010)

Stages of Shock

Healthcare providers can categorize shock into stages depending upon how the body responds. The three stages are (1) compensated shock, (2) uncompensated or decompensated shock, and (3) irreversible shock.

Compensated Shock

During compensated shock, some mechanism reduces tissue perfusion; however, a complex series of compensatory responses help to preserve critical organ perfusion. During this stage, the body increases catecholamine production, which maintains cardiac output and a normal systolic B/P.

However, in some of the tissues of the body, individual cells are not getting all the oxygen they need and switch from aerobic to anaerobic metabolism with a resultant increase in lactic acid production. The body responds by increasing the rate and depth of ventilation in order to exhale more carbon dioxide and compensate for the acidosis. Thus, although compensatory mechanisms help to maintain adequate perfusion status of critical organs, they do so at the expense of other organs and systems (Graham & Parke, 2005).

Exercise 5: If cardiac output remains fairly normal during this stage of shock, what might be a better indicator of early shock instead of monitoring systolic B/P?

Answer:

With time, the compensated stage gets more complicated. The same stimulation that is increasing the heart rate to maintain cardiac output also causes bronchodilation and vasoconstriction. The vasoconstriction diverts capillary flow in some capillary beds (the skin, for example) to the vital organs. This selective reduction in capillary flow may manifest as delayed capillary refill and cool skin.

Even though the patient is able to maintain a normal B/P during this stage, the decreased capillary flow may cause a decrease in central nervous system perfusion. The patient may exhibit confusion or combativeness. At this stage, if medics discover and properly treat the underlying cause of shock, the patient has a good chance of recovery. Left untreated, the compensatory mechanisms begin to collapse.

Uncompensated Shock

Uncompensated shock occurs when the compensatory mechanisms begin to fail and the B/P drops. Generally, systolic pressure is dependent on blood volume and during decompensation, systolic B/P drops before the diastolic pressure. Diastolic pressure is more dependent upon the size of the vessels and may rise initially because of vasoconstriction.

Exercise 6: What is the term for the difference between the systolic and the diastolic blood pressures?

Answer:

As B/P drops, blood flow to the brain decreases. Confusion and combativeness develop as the compensated stage worsens. The heart beats even faster in an attempt to maintain cardiac output. Respirations increase as the body tries to compensate for the acid accumulation in the

tissues. Continued shunting of blood away from the surface and tissue hypoxia can cause the patient to have cold extremities and cyanosis.

For patients who present to EMS in decompensated shock, about 33 to 52% of them will die in the hospital (Holler et al., 2015). However, as those numbers show, some of these patients can still survive if medics correct the causes early.

Irreversible Shock

As most of the compensatory mechanisms fail, there is a progression of cellular ischemia, necrosis, and subsequent organ death. The patient has now slipped into the final stage, irreversible shock. Even if medics restore adequate oxygenation and perfusion, patients with irreversible shock do not usually survive.

Near the end, the patient will become bradycardic, serious dysrhythmia will develop, and the patient will be profoundly hypotensive. At that point, cardiopulmonary collapse usually is imminent.

Compensated Shock	Uncompensated Shock	Irreversible Shock
• Pulse rate increases • Respirations increase • Pulse weakens • Skin becomes cool and clammy • Anxious, restless and combative • Thirsty and weak	• Very weak or absent pulses • Severe drop in blood pressure • Altered mental status or unconsciousness • Slow breathing to apnea	• Cell death • Cellular washout • Organ system failure • Hemorrhaging all over • Patient dies
Stage I and II Hemorrhages • 500-1250 mL blood loss • 5-25% blood volume lost	**Stage II and IV Hemorrhages** • 1250-1750+ mL blood loss • 25-35%+ mL blood volume lost	**Stage IV Hemorrhage** • 1750+ mL blood loss • 35%+ blood volume lost
• Stop the bleeding • Oxygenation • Give fluids • Keep the patient warm. • Get them to definitive care	• Work very fast	• Work even faster but recognize that most of these patients will die

Initial Assessment of the Shock Patient

A standardized initial assessment of all patients will help identify the degree of perfusion in the cells of the body.

Airway

Always begin the assessment with the airway. If assessment suggests obstruction, take whatever steps are necessary to ensure airway patency. Use basic techniques first, such as head-tilt, chin-lift method or a modified jaw thrust. If the patient requires long-term airway maintenance, insert a supraglottic airway or an endotracheal tube after restoring adequate oxygenation and ventilation.

Breathing

Insure that breathing efforts are adequate. In many shock patients, the rate and depth of ventilation increase in an attempt to reduce carbon dioxide content of the blood and compensate for the metabolic acidosis. If breathing is inadequate or absent, provide assisted ventilation using a bag-valve mask connected to an oxygen source. If the breathing is adequate, administer supplemental oxygen using a non-rebreather mask.

Circulation

Next, assess the patient's circulatory status. Feel for a radial pulse. If it is absent, feel for the carotid pulse. If the carotid pulse is absent, begin CPR immediately.

If you feel a carotid pulse but no radial or brachial pulse, a depressed circulatory status is likely. As mentioned earlier, pulse rates slightly increase early in shock to help maintain an adequate cardiac output. As the shock progresses, the tachycardia increases. The pulse may be strong or weak, which allows for only a rough estimation of the systolic B/P. It is clear when the patient is lying down and a carotid pulse is the only pulse you can feel, the B/P is not high enough to perfuse the peripheral tissues.

Historically, EMS educators (as well as some trauma courses) have taught that the presence or absence of pulses at certain location can be used to estimate the patient's systolic B/P. For example, many medics believe that if a carotid pulse is present, the systolic B/P must be at least 60-70 mm Hg. If both the carotid and femoral pulses are present, the systolic B/P must be at least 70-80 mm Hg. Finally, the systolic B/P must be at least 80 mm Hg if radial pulses are also present.

Despite this time-honored belief, the accuracy of these recommendations is questionable. In an observational study of 20 patients with a systolic B/P less than 90 mm Hg following a traumatic injury, these pulse/pressure estimate guidelines correctly predicted systolic B/P only one-fourth of the time (Poulton, 1988). In half of the patients, the guidelines falsely overestimated the B/P by an average of 34 mm Hg. The greatest overestimation occurred in patients with the lowest pressure. Similarly, an observational study using invasive arterial monitoring found that 83% of the seriously injured patients predicted by the guidelines to have a systolic pressure of at least 80 mm Hg actually had pressures below that threshold (Deakin & Low, 2000).

Assess for and control any major bleeding. Evaluate the patient's skin color and temperature. Pale, cyanotic, or blue-grey skin is an indication of a circulatory system in trouble. Likewise, if the skin is cool and clammy, it may be an indication of an impending cardiovascular disaster. IF THE SKIN IS WET, THE PATIENT IS IN TROUBLE!

Disability: Level of Consciousness

Next, evaluate the patient's level of consciousness. You can use the A-V-P-U scale to gauge their response. If the patient immediately responds to your presence, consider the patient alert.

If the patient does not acknowledge your presence when you approach, but responds to your voice, the patient is responsive to verbal stimuli.

For patients who do not respond to the sound of your voice, try some type of painful stimulus. Rub the patient's sternum with your knuckle or press on one of the patient's nail beds with the side of your pen. If the patient attempts to move away from the pain or push you away, they are responsive to painful stimuli.

Finally, if there is no attempt to get away from the pain, the patient is unresponsive. Any alteration in the patient's mental status is an indicator of a critical perfusion problem.

Expose

Following the airway, breathing, circulation, and disability assessments, expose the body surfaces to check for life-threatening injuries. Remove as much clothing as necessary to accomplish this task.

Detailed Physical Examination

After the initial assessment and management of any life threatening conditions, perform a detailed physical examination. This assessment begins with the baseline vital sign measurements and evaluation of the patient's EKG.

The pulse rate usually increases above normal limits after a fluid loss of 10% to 15%. However, consider the patient's pulse rate as only one factor in an evaluation of perfusion because some patients continue to have normal pulse rates even though a volume deficit of this magnitude exists.

There are several potential causes for bradycardia, including hypoxemia, neurological injury, or preexisting illnesses. Some medications, such as beta-blockers prevent the body's compensatory mechanism from producing tachycardia. Severe myocardial ischemia, a primary cause of cardiogenic shock, will also produce bradycardia. Immediately after discovering a bradycardic rhythm, administer 100% oxygen by non-rebreather mask and assist ventilations if necessary.

Although obtaining a B/P measurement is a necessary component of a thorough prehospital patient assessment, B/P used in isolation is not a reliable indicator of significant blood loss (Bulger et al., 2008) or hospital length of stay (Newgard et al., 2010). Initially, the systolic B/P may remain normal during shock. The diastolic B/P may rise slightly as shock begins. Later, when the heart can no longer pump enough to fill the blood vessels, the systolic pressure will drop. As the fluid deficit approaches 25%, both the systolic and diastolic pressures will drop.

What type of shock are we dealing with?

There are a variety of classification schemes for shock. One common scheme used for decades classifies shock by the mechanism that produces inadequate tissue perfusion (Hinshaw & Cox, 1972).

Hypovolemic Shock

A decrease in circulating blood volume causes hypovolemic shock. Always assume hypovolemia as the underlying cause for shock until you can prove otherwise. Paramedics normally associate hypovolemic shock with trauma and hemorrhage; however, there are other potential causes. Severe diarrhea, prolonged vomiting and endocrine disorders can cause a significant loss in circulating volume. Hypovolemic shock may also result from internal fluid shifts caused by burns and massive abdominal infections.

The mechanism of injury, such as steering wheel damage or extensive passenger compartment intrusion in a motor vehicle accident can raise the suspicion of additional injuries in the trauma patient. If you suspect internal bleeding, rapid transportation to an appropriate trauma center is the highest priority after securing an airway and ensuring adequate ventilation. Suspect internal bleeding in any trauma patient with signs of shock, especially those without evidence of external blood loss.

Chest, abdominal, or back pain in non-traumatic shock patients may indicate a vascular disorder. The classic sign of a thoracic aneurysm is a tearing pain radiating to the back. Abdominal aortic aneurysms usually result in tearing pain in the abdomen. Ask about blood clotting disorders, vomiting of blood, bloody stools, alcohol history, and excessive use of medications that cause GI bleeding, such as nonsteroidal anti-inflammatory drugs.

If you suspect a gynecologic cause, inquire about the last menstrual period, vaginal bleeding (including amount and duration), and the vaginal passage of products of conception.

Signs of hypovolemic shock include tachycardia, tachypnea, pulse pressure decrease, cool clammy skin, delayed capillary refill, and slight anxiety. As the hypovolemia progresses, the patient will develop marked tachypnea and tachycardia, the systolic B/P will fall, the patient will complain of being thirsty, and significant changes in mental status will occur.

Cardiogenic Shock

Cardiogenic shock is simply a decrease in the pumping function of the heart that causes inadequate perfusion to the tissues. It is most commonly associated with the damage caused by a myocardial infarction. Dead heart muscle is unable to contract. Once damage to more than 40% of the heart occurs, it is unable to supply the tissues with an adequate blood supply (O'Connor et al., 2010).

Cardiogenic shock occurs in about 5 to 15% of patients with an acute myocardial infarction (Kalavrouziotis, Rodés-Cabau, & Mohammadi, 2017). Mortality rates are very high at about 40-50% (Thiele, Ohman, Desch, Eitel, & de Waha, 2015). Outcomes improve only when physicians can restore the blood supply to the heart in enough time to keep the damaged heart from dying.

Most patients with cardiogenic shock present with signs and symptoms of acute cardiac ischemia, including chest pain, shortness of breath, profound diaphoresis, nausea and vomiting. Patients experiencing cardiogenic shock also may present with pulmonary edema and presyncopal or syncopal symptoms.

Clinical assessment begins with attention to airway, breathing, and circulation. The patient may eventually require endotracheal intubation but the airway is usually patent initially. Breathing may be labored, with audible coarse crackles or wheezing.

As in any shock like state, circulation is noticeably impaired. Tachycardia is present, the extremities are cool and clammy, and the peripheral pulses are very weak. The patient will also have varying degrees of end-organ dysfunction, such as decreased mental function.

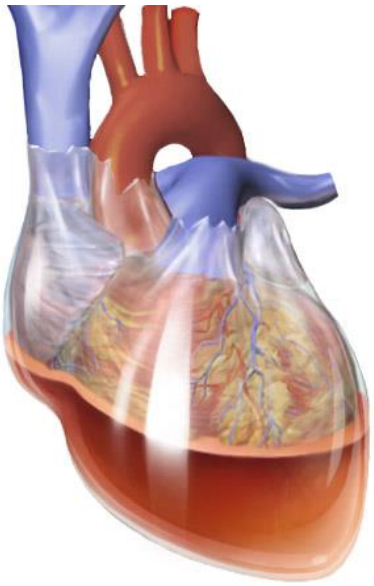
Neck examination may reveal jugular venous distention, which may be prominent. This finding is evidence of right ventricular failure. With increasing ventricular dysfunction, pulmonary edema and severe hypotension may develop.

Obstructive Shock

Obstructive shock occurs when there is a physical obstruction to blood flow through the great vessels or inhibition of adequate contraction of heart muscle. This obstruction could be in the form of a blood clot or other material that lodges in one of the large blood vessels that serve the lungs (pulmonary embolism). Alternatively, excess pressure in the thorax (tension pneumothorax) or in the pericardial sac (cardiac tamponade) may interfere with the ability of the heart to fill or empty.

The classic presentation of a massive **pulmonary embolism** is tachycardia, tachypnea, and hypoxemia (Wacker & Winters, 2014). However, these signs are not specific to pulmonary embolism and are present in many other medical emergencies. T-wave inversions in leads V1-V4 are also suggestive of pulmonary embolism (Vanni et al., 2009).

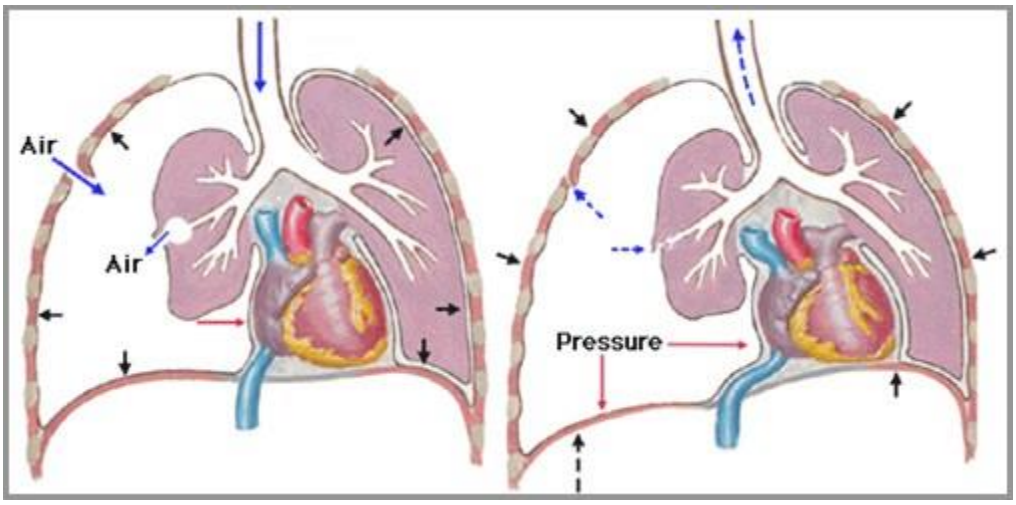
Cardiac tamponade occurs when blood or other fluid collects in the fibrous sac that surrounds the heart. This fluid begins to compress the heart and reduce the heart's ability to pump blood effectively. As a result, cardiac output begins to fall.



The physical examination findings with the highest sensitivity for cardiac tamponade are pulsus paradoxus (82%), tachycardia (77%), and elevated jugular venous pressure (76%) (Roy, Minor, Brookhart, & Choudhry, 2007). Often medics associate Beck's triad with cardiac tamponade. Beck's triad is the coexistence of hypotension, muffled heart sounds, and elevated jugular venous pressure in the same patient (Wacker & Winters, 2014). However, this triad generally only appears with hemorrhage into the pericardial space due to trauma or to myocardial or aortic rupture (Roy, Minor, Brookhart, & Choudhry, 2007). It does not generally occur when the intrapericardial fluid accumulates slowly, as is often the case with pericardial effusions (Guberman, Fowler, Engel, Gueron, & Allen, 1981).

Tension pneumothorax occurs when air collects in the pleural space and increases intrathoracic pressure. Rising intrathoracic pressure inhibits venous return which significantly lowers cardiac output. Patients with tension pneumothorax often present with tachypnea, decreased breath sounds, and hypertympany on the affected side (Wacker & Winters, 2014), although ambient noise levels in the field can impair a medic's ability to appreciate thoracic sounds.

Obstructive shock is rare and many paramedics will go their entire career without ever seeing a single case of obstructive shock.



Distributive Shock

Distributive shock is a type of shock where excessive dilation of the blood vessels reduces B/P in the system and impairs the ability of the cardiovascular system to deliver adequate amounts of blood and oxygen to the tissues.

The most common form of distributive shock among patients admitted to the intensive care unit is septic shock (Gamper et al., 2016). In one multi-center study involving over 1600 patients treated with a vasopressor for undifferentiated shock, septic shock was diagnosed in 62% of the cases followed by cardiogenic shock (16%), hypovolemic shock (16%), other forms of distributive shock (4%) and obstructive shock (2%) (De Backer et al., 2010).

Another form of distributive shock results from sudden increases in the diameter of the blood vessels. This is called **neurogenic or vasogenic shock**. Usually, this type of shock occurs when trauma to the central nervous system causes this loss of vasomotor tone in the muscle layers of blood vessels. This produces significant systemic vasodilation as the muscles relax. Since the space formerly occupied by 5 liters of blood now changes to a 7-liter space, the B/P suddenly drops. Because the system has lost pressure, the tissues can no longer get the blood or oxygen supply they need.

You can generally differentiate neurogenic shock from other types of shock by the patient's history or scene assessment. Shock in the presence of trauma to the central nervous system especially when there is no evidence of blood loss suggests neurogenic shock. The same mechanisms that cause the vessels to dilate suddenly will also keep the heart from receiving signals from the brain to increase the rate. Therefore, patients in neurogenic shock will frequently have a slow heart rate instead of the tachycardia associated with other forms of shock.

Exposure to substances to which the body is allergic can bring about a third type of distributive shock called **anaphylactic shock**. This type of shock is similar to hypovolemic and neurogenic shock combined. Common substances that cause anaphylaxis are antibiotic medications, venoms, and insect stings.

In response to exposure to one of these allergens, the body mounts a chemical defense, which quickly spirals out of control. Some of these chemicals cause blood vessels to dilate suddenly, similar to the vasodilation seen in neurogenic shock (De Bisschop & Bellou, 2012). Some of the blood vessels begin to leak, which causes fluid to shift from the blood stream into the spaces between individual tissue cells. This fluid shift can be substantial, producing a loss of circulating blood volume as in hypovolemic shock.

In addition, some of the cellular mediators (chemicals) released in response to the allergen exposure cause constriction of the upper and lower airways, which affects the ability of the lungs to receive oxygen. Patients often present with hoarseness, stridor, bronchospasm, wheezing and decreased breath sounds. Complete airway obstruction is also possible.

The patient may experience nausea, vomiting, abdominal cramps, and diarrhea. The patient's tissues become swollen and gain a reddish appearance called urticaria, or hives. About 80% to 90%

of all anaphylactic reactions will produce some form of cutaneous sign that medics should be able to visualize (Stoloff, 2010).

General Management Principles

Regardless of the etiology of shock, the initial management goals are similar. The medic must ensure a patent airway, provide adequate oxygenation and ventilation, restore effective perfusion.

Simultaneously with airway, breathing and circulatory assessment and interventions, medics must attempt to control any external bleeding. Examine area of controlled hemorrhage frequently to determine that hemorrhage remains controlled.

EMS personnel must establish and maintain an open airway. Give the patient high-concentrations of supplemental oxygen at 10-15 lpm (100% NRBM). Assist ventilation, as needed. Oxygen saturation values in the hypovolemic patient may be misleading because bleeding reduces the total amount of hemoglobin in the body. When a pulse oximeter reads 100% saturation, it is accurately reporting that 100% of the hemoglobin passing the sensor is saturated. However, there may not be enough hemoglobin left in the body to carry enough oxygen to meet the demands of the tissues.

In a healthy individual, each breath eliminates between 35 and 45 mm Hg of carbon dioxide. Since capnography is an indirect measure of cardiac output, carbon dioxide monitoring is useful in shock. As cardiac output drops, perfusion drops. This reduces blood flow to the lungs and as a result, there is less carbon dioxide available at the alveolar surface to make the diffusion into the airway. End-tidal carbon dioxide measurements will drop as the shock progresses.

In addition to oxygen saturation and waveform capnography, medics should also monitor the patient's ECG. If arrhythmias develop, treat according to specific arrhythmia treatment guidelines.

Finally, take steps to maintain the patient's normal body temperature. Shock often interferes with the body's ability to conserve body heat and patients easily become hypothermic.

Management of Specific Forms of Shock

In addition to the general management appropriate to all victims of shock, it is appropriate to obtain IV or IO access and begin fluid replacement regardless of the etiology of shock. Fluid replacement guidelines, as well as other therapies are specific to each specific form of shock.

For some forms of shock, administration of IV fluids in the prehospital setting is controversial and should never delay patient transportation. The controversy exists because prehospital IV solutions cannot restore the oxygen carrying capacity of blood. In most situations, the patient is best

served by rapid assessment, airway stabilization, protection from further injury, and rapid transportation to an appropriate medical facility.

Hypovolemic Shock

Fluid administration is generally the field therapy of choice for patients suffering from hypovolemic shock.

For hypovolemic shock, BioTel can order additional fluid boluses in order to keep the systolic B/P above 90 mmHg. Eventually, replacement of the lost blood cells must occur. You should not consider the management of hypovolemic shock complete until the patient receives definitive surgery to repair the leaking system.

If uncontrolled internal or external bleeding is present, administer fluid in an attempt to maintain a radial pulse. It may be dangerous for the patient to receive fluid doses in excess of that amount. Administer 20 mL/kg boluses, as needed, to maintain radial pulses (do not exceed systolic B/P of 90 mmHg).

For the pediatric patient in hypovolemic shock, administer 20 milliliters per kilogram of saline. You may repeat this dose twice if the systolic B/P remains below 70 mm Hg and there are no signs of volume overload. Contact BioTel if additional boluses are needed, especially for hypovolemic shock secondary to diabetic ketoacidosis (DKA). If the patient is unconscious, consider the use of an intraosseous infusion early.

Cardiogenic Shock

The management focus of cardiogenic shock is on improving the pumping action of the heart and managing any cardiac arrhythmias. During an acute myocardial infarction, injured cardiac muscle affects the conduction system. Bradycardia often begins at the onset of AMI symptoms and can lead to cardiogenic shock. If the heart rate is less than 60 and the patient is symptomatic with signs and symptoms of decreased cardiac output and poor perfusion, proceed immediately to application of a transcutaneous pacemaker.

When applying the transcutaneous pacer, set the rate at 60 beats per minute. Increase the current until you achieve electrical capture. Once you do, check for mechanical capture by assessing for a pulse. If the pulse is present, increase the energy setting by 5 milliamps.

In the conscious patient, pacing can be an uncomfortable experience. To reduce the discomfort associated with transcutaneous pacing, administer 2.5 mg - 5 mg midazolam slow IV/IO/IM/IN prior to pacing. Standing orders allow for one repeat dose, if needed.

After assuring heart rate and rhythm are adequate, if the patient remains hypotensive, infuse a single 20 mL/kg IV/IO bolus, unless pulmonary edema is present. If the patient remains hypotensive, consider one repeat bolus while preparing norepinephrine drip, as long as there is no evidence of

pulmonary edema. The standing order dose of norepinephrine is 8-12 mcg/minute administered as an IV drip. Evaluate the lungs frequently for evidence of pulmonary edema.

For the pediatric patient in cardiogenic shock, infuse the saline at a TKO rate and contact BioTel for authorization to administer a vasopressor or a fluid bolus.

Obstructive Shock

When pulmonary embolism is large enough to produce obstructive shock, mortality is very high (Goldhaber, Visani, & De Rosa, 1999). These patients need either fibrinolytic therapy or some form of invasive procedure to remove the obstruction (Jaff et al., 2011; Kearon et al., 2012). In the meantime, medics should provide supportive therapy as needed. This includes infusing or titrating a fluid bolus of 20 mL/kg fluid to achieve a systolic B/P of 90 mmHg. Do not exceed 1 liter of fluid under standing orders.

Patient suffering from obstructive shock secondary to cardiac tamponade must receive pericardiocentesis as early as possible. Emergently, there are no contraindications to this procedure (L'Italien, 2013). However, since paramedics in the BioTel system are not credentialed to perform this skill, the patient must get to an emergency department as quickly as possible. In the meantime, medics should provide supportive therapy as needed. This includes infusing or titrating a fluid bolus of 20 mL/kg fluid to achieve a systolic B/P of 90 mmHg. Do not exceed 1 liter of fluid under standing orders.

Patients suffering from obstructive shock secondary to tension pneumothorax must have needle decompression performed right away.

- Insert a 14-gauge or 16-gauge IV catheter into the affected pleural space at the second intercostal space in the midclavicular line. The needle and catheter must be at least 2½" long.
- Failure to identify the midclavicular line increases the risk of puncturing structures in the mediastinum.
- Insert the catheter just above the third rib to avoid the nerve, artery and vein that lie just beneath each rib.
- Advance the catheter its entire length, followed by needle removal, thus leaving the plastic catheter as a conduit into the pleural space.
- This allows the air trapped in the space to be released or aspirated.
- Treatment guidelines for the BioTel system DO NOT recommend attaching a one-way valve or making one from a finger of a sterile glove.

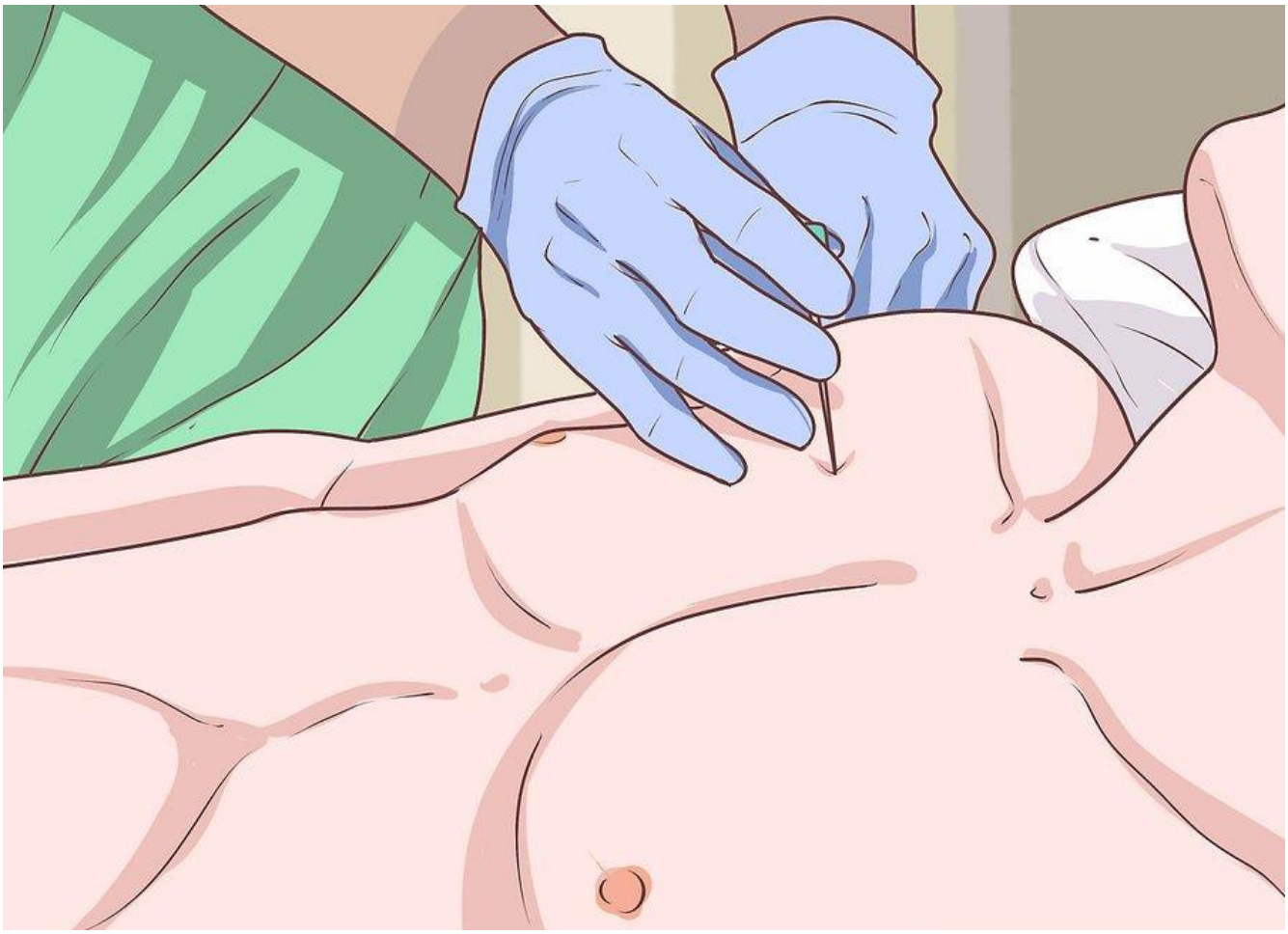
- The maneuver effectively relieves the pressure and converts a tension pneumothorax into a simple pneumothorax.

Complications and Precautions of Needle Thoracostomy

- Commonly available 14–16 gauge IV catheters have an associated failure rate ranging from 50–65% with anterior insertion because they are too short (Ball et al., 2010; Stevens et al., 2009)
- Longer catheters may prevent under-penetration, but also increases over-penetration, which may result in injury to the lung, subclavian vessels, heart and great vessels (Zengerink et al., 2008)
- A tension pneumothorax that was initially relieved may also re-accumulate undetected as a result of catheter kinking or plugging.

Pearls

- Needle thoracostomy is a relatively simple procedure but diagnosis of a tension pneumothorax is not easy, especially in the field.
- Conscious patients will not want to co-operate with examination because they are hypoxic
- Hyperresonant percussion is hard to detect and the affected side is not silent as sounds are transmitted across the chest
- Diagnosis is even more difficult if there is significant thoracic adipose tissue
- If the person is unconscious respiratory rate can be depressed due to a head injury, not elevated.
- Even if the patient produces a hiss of air during insertion, you probably won't hear it because of prehospital background noise.
- Don't expect the patient to rapidly improve
    - Try blowing through an IV catheter!
    - It takes minutes for the pressure to dissipate, not seconds.



Distributive Shock

For all other forms of shock, infuse 20 mL/kg boluses and reassess, titrating to achieve a systolic B/P of 90 mmHg. Do not exceed 1 liter of fluid under standing orders.

For the pediatric patient, administer 20 mL/kg of saline. You may repeat this dose once if the systolic pressure does not rise above 70 mm Hg. As with hypovolemic shock, you may repeat the dose twice if systolic B/P remains below 70 mm Hg and there are no signs of volume overload. Contact BioTel if additional boluses are required, especially for DKA, cardiac history, or signs of pulmonary edema (e.g. rales). If the patient is unconscious and peripheral IV access is unavailable, consider early use of IO infusion.

Neurogenic Shock

The management of neurogenic shock mirrors the management for hypovolemia. One major difference is that you must take care during fluid resuscitation to avoid a circulatory overload. Closely monitor the patient's lung sounds for signs of pulmonary congestion.

Airway assessment is critical because upper airway obstruction is responsible for most deaths from anaphylactic shock. Evaluate the conscious patient for voice changes, stridor, or a barking cough. Complaints of tightness in the neck and dyspnea should alert you of impending airway obstruction. Treat all allergic symptoms with the same degree of urgency because the reaction can quickly progress from annoying hives to life-threatening bronchospasm of shock within 5 to 30 minutes (Pumphrey, 2000). If the patient begins to show signs or symptoms of impending airway obstruction, consider early endotracheal intubation.

If the patient is in shock as the result of anaphylaxis, administer fluids in 250 ml increments to maintain a systolic B/P of 90 mm Hg. Do not exceed 1 liter under standing orders although effective fluid resuscitation may require up to 4 liters (Sinz, Navarro, & Soderberg, 2013)..

The definitive treatment for anaphylactic shock is epinephrine administration (Vande Hoek et al., 2010). Epinephrine is a powerful vasoconstrictor and increases the blood pressure. Do not delay epinephrine administration. In fact, studies have established a link between medication administration delay and poor outcome (Sampson, Mendelson, & Rosen, 1992; Yunginger et al., 1988).

The BioTel Medical Direction Team has authorized the use of epinephrine autoinjectors by EMTs within the BioTel system as long as the autoinjector belongs to the patient and paramedics are not yet on the scene. There is no standing order authorization for EMTs to use an autoinjector carried by the EMS agency.

For profound anaphylactic shock, administer intramuscular (IM) epinephrine in a 1:1,000 concentration while attempting to establish an IV. As a standing order, give the adult patient 0.3 - 0.5 milligrams IM. For the pediatric patient, standing orders allow you to give epinephrine in a 1:1,000 concentration at 0.01 milligrams per kilogram up to a maximum dose of 0.3 milligrams IM. Standing orders allow the administration of up to three doses of 1:1,000 epinephrine. If the patient received any epinephrine from an autoinjector before development of anaphylaxis, that dose counts as one of the three doses.

Although diphenhydramine is the drug of choice for the treatment of mild allergic reactions, it still has a place in the treatment of anaphylaxis. Histamine release is ultimately responsible for the symptoms. Epinephrine can overcome those symptoms, but diphenhydramine can ensure that the mast cells stop releasing histamine. For anaphylaxis, give diphenhydramine just as you would for a mild allergic reaction. Treatment guidelines also allow for 25 - 50 milligrams of diphenhydramine as a standing order medication for the adult patient either as an intramuscular or intravenous injection.

For the pediatric patient, the standing order dose of diphenhydramine is 1 - 2 milligrams per kilogram of body weight either as an intramuscular or intravenous injection.

Summary

One of the simplest definitions of shock is inadequate capillary perfusion. However, shock is a very complex group of physiological abnormalities caused by a variety of disease states and injuries.

The goals of prehospital care for the patient in shock include rapid recognition of the cause, initiation of treatment, and protection from additional injury. In many shock situations, the very treatment required by the patient is not available in the field. In those situations, rapid transport to an appropriate medical facility affords the patient the best possibility of survival.

SHOCK

Inclusion Criteria: Any patient experiencing signs and symptoms consistent with shock and hypoperfusion. Refer also to **TRAUMA,** specific arrhythmia, and **ALLERGIC REACTION** Guidelines, and to the **NEEDLE THORACOSTOMY** Special Procedure.

Basic Level

1. Assess and support ABCs.
2. Initiate **SPINAL MOTION RESTRICTION**, if indicated. Place the patient supine and elevate the legs, unless contraindicated.
3. Control any obvious external hemorrhage.
4. Cover the patient to avoid heat loss, but do not over-bundle.
5. Administer oxygen via non-rebreather mask at 10-15 lpm (100% NRBM) or assist ventilations via BVM, if indicated.
6. Begin transport as soon as possible.

Advanced Level

7. Continuously monitor ECG, SpO_2 and $ETCO_2$.
8. Establish one large bore IV and infuse Normal Saline according to the following guidelines:

Hypovolemic/Hemorrhagic shock:	
Adult • Administer 20 mL/kg boluses, as needed, to maintain radial pulses (do not exceed systolic BP of 90 mmHg). • Examine area of controlled hemorrhage frequently to determine that hemorrhage remains controlled.	**Pediatric** • Infuse Normal Saline bolus IV/IO: 20 mL/kg. • Repeat twice if systolic BP remains below 70 mm Hg *and* there are no signs of volume overload. • Contact BioTel if additional boluses are needed, especially for DKA. • If the patient is unconscious and peripheral IV access is unavailable, consider early use of IO infusion.

Cardiogenic Shock - Assure heart rate and rhythm are adequate, then;	
Adult • Infuse a single 20 mL/kg IV/IO bolus, unless pulmonary edema is present. • Consider one repeat bolus while preparing norepinephrine drip, *IF* there is no evidence of pulmonary edema. • Norepinephrine bitartrate drip 8 to12 mcg/min, if there is no response to fluid bolus.	**Pediatric** • Run fluid at TKO rate. • Contact BioTel for vasopressor dosing and possible IV/IO fluid bolus (5 to 10 mL/kg).

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All Other Forms of Shock (Except Tension Pneumothorax):	
Adult	**Pediatric**
• Infuse 20 mL/kg boluses and reassess, titrating to achieve a systolic BP of 90 mmHg. • Do not exceed 1 L under standing orders.	• Infuse Normal Saline bolus IV/IO: 20 mL/kg. • Repeat twice if systolic BP remains below 70 mm Hg *and* there are no signs of volume overload. • Contact BioTel if additional boluses are required, especially for DKA, cardiac history, or signs of pulmonary edema (e.g. rales). • If the patient is unconscious and peripheral IV access is unavailable, consider early use of IO infusion.

Obstructive Shock due to Tension Pneumothorax (Pneumothorax, with Hypotension and Severe Hemodynamic Compromise):	
Adult	**Pediatric**
• Perform needle thoracostomy. • Refer to the **NEEDLE THORACOSTOMY** Special Procedure.	• Perform needle thoracostomy. • Contact BioTel as soon as possible. • Refer to the **NEEDLE THORACOSTOMY** Special Procedure.

9. Continuously monitor vital signs, ECG, SpO_2, $ETCO_2$ and neurological status during transport.

10. For additional patient care considerations not covered under standing orders, consult BioTel.