

Hypotension and Shock

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INTRODUCTION

Shock is an important life-threatening emergency and must be recognized early and intervention started to prevent progression, morbidity, and mortality. Unfortunately, the identification and treatment of shock in the out-of-hospital setting is fraught with many difficulties and potential pitfalls. For example, patient assessment is often limited by the challenging out-of-hospital environment and lack of diagnostic and therapeutic options. In addition, the early stages of compensated shock with subtle alterations in physical findings are easily overlooked or misinterpreted by out-of-hospital care providers. Ongoing treatment for medical conditions, such as beta-blockers for hypertension, may also mask the body's compensatory responses. As a result, the patient with severe shock may present with normal vital signs. The tools available for the diagnosis and treatment of shock in the field are limited.

PATHOPHYSIOLOGY

Shock is a complex physiologic process defined as the widespread reduction in tissue perfusion leading to cellular and organ dysfunction and death. In the early stages of shock, a series of complex compensatory mechanisms act to preserve critical organ perfusion.¹ In general, the following relationships drive this process:

$$\text{blood pressure} = \text{cardiac output} \times \text{peripheral vascular resistance}$$

$$\text{cardiac output} = \text{heart rate} \times \text{stroke volume}$$

Any condition that lowers cardiac output and/or peripheral vascular resistance may decrease blood pressure. Alterations of heart rate (very low or very high) can lower stroke volume and hence blood pressure. Also, decreasing stroke volume may also lower cardiac output with a possible reduction in perfusion as well. Stroke volume may be reduced by lower circulating blood volume (e.g., hemorrhage or dehydration), by damage to the heart (e.g., myocardial infarction or myocarditis), or by conditions obstructing blood flow through the thorax (e.g., tension pneumothorax, cardiac tamponade, or massive pulmonary embolism).

To aid in the evaluation and treatment of shock it is often useful for the physician and EMS personnel to categorize the etiology of the shock condition.² Most EMS providers are familiar with the pump-fluid-pipes model of the cardiovascular system, with the pump representing the heart; pipes representing the vascular system; and fluid representing the blood. Shock may therefore occur from increased resistance to flow of blood into the thorax, from diminished cardiac contractility, from diminished vascular resistance, and from decreased intravascular volume.³ Categorizing shock into four categories may help prehospital providers and EMS physicians organize their assessment and approach (Table 7.1). Accurate physical assessment is vital for the EMS provider to determine the etiology of the shock state (Table 7.2).

TABLE 7.1

Categories of Shock			
Type of Shock	Disorder	Example	Comments
Hypovolemic	Decreased fluids	A. External fluid loss 1 Hemorrhage 2 Gastrointestinal losses 3 Renal losses 4 Cutaneous loss B. Internal fluid loss 1 Fractures 2 Intestinal obstruction 3 Hemothorax 4 Hemoperitoneum	Hypovolemic shock states, especially hemorrhagic shock, produce flat neck veins, tachycardia, and pallor
Distributive	Increased “pipe” size	A. Drug or toxin induced B. Spinal cord injury C. Sepsis D. Anaphylaxis E. Anoxia	Distributive shock states usually show flat neck veins, tachycardia, and pallor. Neurogenic shock due to a cervical spinal cord injury tends to show flat neck veins, normal or low pulse rate, and pink skin
Obstruction	Pipe obstruction	A. Pulmonary embolism B. Tension pneumothorax C. Cardiac tamponade D. Severe aortic stenosis E. Venacaval obstruction	
Cardiogenic	“Pump” problems	A. Myocardial infarction B. Arrhythmias C. Cardiomyopathy D. Acute valvular incompetence E. Myocardial contusion F. Myocardial infarction	Cardiogenic and obstructive shock states tend to produce jugular venous distension, tachycardia, and cyanosis

TABLE 7.2

Signs and Symptoms of Shock
Cardiovascular
<ul style="list-style-type: none"> Tachycardia, arrhythmias, hypotension
Central Nervous System
<ul style="list-style-type: none"> Agitation, confusion Alterations in level of consciousness Coma
Respiratory
<ul style="list-style-type: none"> Tachypnea, dyspnea
Skin
<ul style="list-style-type: none"> Pallor, diaphoresis Cyanosis (in obstructive shock cases), mottling.

EVALUATION

The diagnosis of shock depends on a combination of key historical features and physical findings. Often historical features and clinical findings can provide clues as to the etiologies of the shock state. For example, tachycardia and hypotension in an elderly patient with fever, cough, and dyspnea may represent pneumonia with septic shock. Hemorrhagic shock may be suspected in a middle-aged man with epigastric pain, hematemesis, melena, and hypotension. Hypotension, tachycardia, and an urticarial rash in a victim of a recent bee sting strongly suggest distributive shock secondary to anaphylaxis. Obstructive shock precipitated by a tension pneumothorax should be suspected in a hypotensive trauma patient

with unilateral decreased breath sounds and tracheal deviation to the opposite side.

An important problem in the prehospital diagnosis of shock is the frequent inaccuracy of field assessment. For example, Cayten et al. found an error rate of more than 20% for vital signs obtained by EMTs in a nonemergency setting.⁴ The researchers suggest that when critical medical decisions will be based on the data gathered in the field, multiple assessment measures should be performed.

Out-of-hospital care providers should look for the signs and symptoms of system-wide reduction in tissue perfusion, such as tachycardia, tachypnea, mental status changes, and cool, clammy skin (see Table 7.2). Overall, the clinical presentation of shock depends on the patient's degree of compensation, the etiology of the shock state, the existence of other clinical conditions, and other concurrent treatments.

Vital signs that fall outside of expected ranges must be correlated with the overall clinical presentation. A petite 45-kg, 16-year-old female with lower abdominal pain and a reported blood pressure of 88 mm Hg systolic by palpation may have a ruptured ectopic pregnancy, or she may normally have a systolic blood pressure of 88 mm Hg. An elderly patient with significant epistaxis may be hypertensive due to catecholamine release and vasoconstriction despite being relatively volume depleted.

In the noisy field environment, providers often measure blood pressure by palpation rather than auscultation. Blood pressure by palpation provides only an estimate of systolic pressure.⁵ Without an auscultated diastolic pressure, the pulse pressure (difference between systolic and diastolic pressure) cannot be calculated. Because a decrease in the pulse pressure may provide an early clue to the presence of hypovolemic shock, the field provider measuring only palpated systolic blood pressure may miss this important clue.¹

Previously healthy victims of acute hypovolemic shock may maintain relatively normal vital signs with up to 25% blood volume loss.¹ Sympathetic nervous system stimulation with vasoconstriction and increased cardiac contractility may result in normal blood pressure in the face of decreasing vascular volume. In some patients with intra-abdominal bleeding (e.g., ruptured abdominal aneurysm, ectopic pregnancy) the pulse may be relatively bradycardic despite significant blood loss.⁶

EMS personnel may equate "normal" vital signs with normal cardiovascular status.³ The field team may be lulled into a false sense of security initially

if the early signs of shock are overlooked, only to be caught off guard when the patient's condition dramatically worsens during transport. Early recognition and aggressive treatment of shock may prevent progression to the later stages of shock that can result in the death of potentially salvageable patients.⁷

Prehospital hypotension may predict in-hospital morbidity and mortality in both trauma and medical patients.⁸⁻¹⁰ Jones et al. noted a 30% higher mortality rate for medical patients with prehospital hypotension.⁸ Other studies have shown similar findings in trauma patients with prehospital hypotension, even with subsequent normotension in the emergency department.^{9,10} Therefore, hospital providers should consider any episode of prehospital hypotension as evidence of significant shock and illness.

Despite their variable value, orthostatic vital signs are often evaluated in the emergency department, and occasionally in the field. The most sensitive use of orthostatic vital signs is in moving the patient from the lying to the standing positions. A positive orthostatic vital sign test for pulse rate would show a pulse increase of 30 beats per minute after 1 minute of standing.¹¹ Symptoms of lightheadedness or dizziness would also be considered a positive test. Orthostatic blood pressure checks are sporadically performed in the field. Occasionally orthostatic vital signs are performed serendipitously by the patient who refuses treatment while lying down, then stands up to leave the scene, and suffers a syncopal episode. This demonstration of orthostatic hypotension is often helpful in convincing the patient to allow treatment and transport. However, rescuers should not equate absence of orthostatic response with normovolemia.

Capillary refill testing as a clinical test for shock has variable support in the literature. In a study of patients with evidence of hypovolemia, Schriger and Baraff found that capillary refill was not a useful test for mild to moderate hypovolemia.¹² Moreover, environmental considerations, such as cold temperatures and adverse lighting conditions, also affect the accuracy of this technique of shock assessment.

Out-of-hospital personnel often provide estimates of on-scene blood loss for trauma victims. These estimates may influence therapeutic interventions, including fluid administration. However, a recent study suggests that these blood loss estimates are not accurate at estimating spilled blood volumes.¹³

Hypoxia is a common theme for many shock states. However, a study by Brown et al. suggests that the detection of hypoxia in the prehospital setting without a

pulse oximeter may prove difficult.¹⁴ Patients in various stages of exsanguination may not have sufficient blood volume to adequately perfuse the body with oxygen. Pulse oximetry alone cannot detect the adequacy of oxygen delivery. Pulse oximetry may fail to detect a pulse when blood flow is reduced.^{15,16} In the prehospital setting in nonintubated patients, one study showed that pulse oximetry falsely alerted three or four times per patient transport, whereas the capnography alert rate was 0.3 per patient transport.¹⁵

Like pulse oximetry, capnography may also serve as an important tool in the evaluation and treatment of shock in the prehospital setting.^{17–20} Capnography reflects the expiration of carbon dioxide from the lungs. Exhaled end-tidal carbon dioxide (ETCO₂) levels vary inversely with minute ventilation, providing feedback regarding the effect of changes in ventilatory parameters.^{21,22} In addition, changes in ETCO₂ are virtually immediate when the airway is obstructed or the endotracheal tube becomes dislodged.²³ ETCO₂ concentration may be influenced by factors other than ventilation. For example, ETCO₂ levels are reduced when pulmonary perfusion decreases in shock, cardiac arrest, and pulmonary embolism.^{24–26} ETCO₂ is most useful as an indicator of perfusion when minute ventilation is held constant (e.g., when mechanical ventilation is applied).^{18,24} Under these conditions, changes in ETCO₂ levels reliably indicate changes in perfusion. In any patient suffering from a potential shock state, diminished ETCO₂ should be a warning of the criticality of the patient.

Future Technologies in the Assessment of Shock

Preliminary work has been performed using ultrasound scanning in the field. Ultrasonography may potentially assist in shock resuscitation by facilitating recognition of intra-abdominal hemorrhage, cardiac tamponade, hypovolemia, or an abdominal aortic aneurysm. Selected air medical agencies have pioneered the use of ultrasound for field care, including the Focused Assessment by Sonography in Trauma (FAST) examination.²⁷

Serum lactate may reflect anaerobic tissue metabolism in acute sepsis and shock.²⁸ In the emergency department, elevated lactate in the setting of infection indicates septic shock and the need for early goal-directed sepsis therapy. A handheld fingerstick lactate meter exists, but the correlation with arterial or venous lactate levels remains unclear.²⁹

In summary, although technology may be the future solution, the current evaluation of the potential shock victim in the out-of-hospital setting is challenging due both to limited assessment capability in this environment as well as fewer diagnostic tools. Both the provider and the direct medical oversight (DMO) physician must be cautioned on placing too much emphasis on a single set of vital signs or a limited assessment.

GENERAL APPROACH TO SHOCK

All treatment approaches to shock must include the following basic principles:

1. Establish and maintain ABCs.
2. Maintain adequate oxygen saturation (SaO₂ > 94%) and ensure adequate ventilation.
3. Control blood and fluid losses.
4. Monitor vital signs, ECG, oxygen saturation, and capnography.
5. Prevent additional injury or exacerbation of existing medical conditions.
6. Protect the patient from the environment.
7. Attempt to determine the etiology of the shock state.
8. Determine need for early definitive care.
9. Notify and transport to an appropriate facility.

Once these basic principles are addressed, the field team should attempt to identify the etiology of the shock state. Often the etiology and the initial management options are clear from the history. For example, the out-of-hospital treatment of a young previously healthy college student with hypotension secondary to severe vomiting and diarrhea includes IV fluids. The treatment of cardiogenic shock in an unresponsive elderly patient with ventricular tachycardia (VT) requires prompt cardioversion. A patient suffering from severe anaphylaxis after an insect sting requires fluids and vasopressors (epinephrine). An elderly man from a nursing facility with an indwelling urinary catheter with signs of shock, fever, and tachycardia is likely experiencing septic shock. Occasionally, the primary problem may be strongly suspected but not readily treatable in the field (e.g., pulmonary embolism). Less frequent, but most difficult to manage, is the patient in shock without an obvious cause.

With the understanding of the limited treatment options in the out-of-hospital setting (i.e., fluids, inotropic

agents, and vasopressors), field treatment may be individualized for the four categories of shock: hypovolemic, distributive, obstructive, and cardiogenic.

Hypovolemic Shock

The treatment of hypotension and shock caused by hypovolemia is relatively straightforward. External bleeding should be controlled. Fluid replacement via vascular access is a mainstay of treatment. In the United States, crystalloids are the fluid of choice for the initial field resuscitation of the hypovolemic patient.³⁰ The amount of fluids that should be provided, however, remains controversial.^{30–38}

Distributive Shock

The treatment of distributive shock involves the combination of vasoactive medications, which constrict the dilated vasculature, and fluids, which fill the expanded vascular tree. Commonly used vasoactive medications in the out-of-hospital setting for distributive shock include epinephrine, norepinephrine, and dopamine. Although epinephrine is easily administered via several routes (intramuscular [IM], endotracheal, or IV bolus or drip), the drug has significant side effects. Norepinephrine and dopamine have side effects similar to epinephrine and must be administered via drip infusion. Continuous infusions may be difficult to maintain without special infusion pumps.

Obstructive Shock

Obstructive causes of shock are often difficult to diagnose and treat. If possible, the obstruction should be resolved, such as by decompression of a tension pneumothorax. However, when the primary problem cannot be treated successfully in the field (e.g., massive pulmonary embolus or cardiac tamponade), fluids may be helpful in increasing preload and temporarily overcoming the obstruction.

Cardiogenic Shock

Cardiogenic shock requires individualized treatment. Cardiogenic shock from severe dysrhythmias should be treated with appropriate chemical or electrical therapy. “Pump failure” is often difficult to diagnose and to treat without invasive monitoring. Adult patients without obvious pulmonary edema may benefit from fluid challenges of approximately 150 to 300 ml

of crystalloid. An improvement in the patient’s condition suggests that enhancing preload would be beneficial. A worsening of the patient’s condition with a fluid challenge or the presence of obvious pulmonary edema on initial evaluation suggests that fluid therapy would not be helpful. In such settings, treatment with inotropic agents or pressors, such as dopamine or dobutamine, would be more appropriate. The provider and DMO physician must realize that drips are often difficult to manage in the field and must be monitored closely.

Among the causes of cardiogenic shock are beta-blocker and calcium channel blocker toxicity. These agents block sympathomimetic receptors, impairing the body’s normal compensatory responses. These patients present with profound bradycardia and shock, often refractory to sympathomimetic treatment and fluid challenges due to the receptor blockade. An appropriate drug agent is IV glucagon, which facilitates heart rate stimulation and vasoconstriction through alternative cellular receptors, and which many EMS agencies carry for use in hypoglycemic patients.

Shock of Unclear Etiology

In a few disconcerting conditions the primary etiology for the patient’s shock state may not be obvious. The principal treatment decision is whether or not to give fluids. In hypovolemic, distributive, and obstructive shock, fluids are an appropriate initial treatment for hypotension. Some cases of cardiogenic shock will respond to fluids. However, fluids should not be given to patients in cardiogenic shock with profound pulmonary edema. Fluids are also not appropriate when cardiogenic shock has been precipitated by a treatable arrhythmia. In other cases, response to fluid challenges should dictate whether additional fluid challenges should be given or whether a trial of a sympathomimetic agent should be used.

Occasionally shock will be refractory to initial attempts at resuscitation. This may reflect the need for definitive care in the hospital (e.g., thoracotomy, laparotomy). If after vigorous field treatment the patient remains hypotensive, other etiologies for the hypotension must be considered. Refractory hypotension may be the result of inadequate volume replacement, inadequate oxygenation, cardiac tamponade, tension pneumothorax, acidemia, myocardial infarction, or medications, as previously discussed.

SHOCK INTERVENTIONS

Fluids

The treatment of shock must be customized to the individual EMS agency and geographic location. In the urban setting with short transport times, the victim of a penetrating cardiac wound probably benefits most from airway maintenance and rapid transport to the hospital. IV access could be attempted en route if it did not delay delivery to definitive care.³⁵ On the other hand, with longer transport times in the rural setting, a similar patient might benefit from carefully titrated crystalloid volume infusion during the transport. Fluids could be initiated while the patient is en route to the hospital, thereby prolonging neither scene time nor time until definitive care.³⁸ In the patient who presents a difficult IV access problem, intraosseous infusions may be attempted.

The ideal quantity of fluids to administer in the out-of-hospital setting is not known, especially in the trauma victim with uncontrolled hemorrhage. However, when rapid fluid infusion is required, fluids should be infused with either pressure bags or manual pressure applied to the IV bag.³⁹ Older trauma algorithms indicate the use of 2 L IV fluid on all major trauma victims. However, many patients may require much larger volumes. Conversely, some patients may require much smaller volumes.

Isotonic crystalloids are currently the fluid of choice for out-of-hospital resuscitation in the United States.³⁶ Some air medical services carry O-negative blood for administration to victims of hemorrhagic shock. Several centers have studied hypertonic saline, colloids, and artificial blood substitutes as alternatives to isotonic saline.^{40,41} Problems with these alternative fluids include cost, allergic reactions, coagulopathy, hypernatremia, and lack of demonstrated benefit versus isotonic crystalloids.⁴² As a result, none of the alternative fluids has gained widespread acceptance. However, the military uses several alternatives when treating shock.⁴³

IV administration of fluids is a gold standard treatment that has a long tradition in the care of critically ill patients. The route of IV administration depends on many factors, including the severity of the patient's illness and the available cannulation sites. Extremity veins provide the typical routes of venous access. External jugular veins are also useful sites in many patients. Few EMS systems use central venous access.

The intraosseous route for vascular access has been described and used for generations. This was a

common form of vascular access during World War II, though it became a less popular route in the post-war era with the rising use of IV cannulation. Recent innovations have made intraosseous access rapid and easy for most patients. Intraosseous access has become so important as a method of vascular access that it is supported by a position statement of the National Association of EMS Physicians.⁴⁴ Various devices are available, and EMS medical directors must work with their systems to determine the most appropriate device for use by their providers.

The control of external hemorrhage is essential for maintaining vascular volume. Direct pressure is usually sufficient to control external bleeding. Recent military experience suggests that tourniquets should be used early and liberally.⁴⁵ An assortment of topical hemostatic materials (placed directly on the bleeding wound) also exist. Initial versions of these products produced significant heat through their exothermic chemical reaction.⁴⁶ The role of hemostatic agents in EMS care is currently uncertain.

Ventilation

The patient in shock may require assisted ventilation. Venous return requires a relative negative pressure in the right atrium to ensure return of blood to the heart. Assisted ventilation using any of the typical techniques (such as bag-mask ventilation, endotracheal intubation, or any of the "alternate airways") results in an increase in airway pressure, raising intrathoracic pressure. Patients in shock from any cause are extremely sensitive to increases in intrathoracic pressure. Recent studies in a swine hemorrhagic shock model showed that even modest increases in the rate of positive pressure ventilation significantly reduce both brain oxygenation and brain blood flow.⁴⁷

Emergency EMS personnel must carefully control the rate of assisted positive pressure ventilation in the shock patient, as overventilation is very common. Generally speaking, a one-handed squeeze on the ventilation bag at a rate of approximately once every 8 seconds is reasonable in the adult, producing a minute ventilation of about 5 L/min.

Vasopressor Agents

Administration of vasoactive medications is often required to reverse systemic hypoperfusion from distributive or cardiogenic shock. These agents increase cardiac inotropy, chronotropy, and/or vasoconstriction.⁴⁸

Although a wide variety of vasoactive agents are available in the hospital, the drugs carried by prehospital services are limited by local, regional, or statewide protocols or regulations. In general, most services carry epinephrine and dopamine. Dobutamine, norepinephrine, and vasopressin may also be included in the drug armamentarium of some services.

The choice of vasopressor depends on the suspected underlying pathologic process and the patient's response to therapy. Unfortunately, in the out-of-hospital setting, the etiology of the shock state is often unclear, and close monitoring of vital signs is difficult. The administration of vasoactive agents in the field is fraught with many other potential pitfalls such as the difficulty of calculating weight-based drug dosages. Rescuers should use calculators or templates or seek DMO when initiating drug infusions. Accurate medication administration may be facilitated through portable IV infusion pumps.

Other Drug Agents

Other agents used for shock resuscitation include corticosteroids, antibiotics, albumin, inotropic agents, recombinant human activated protein C, and dextran.^{49,50} The role of these agents in out-of-hospital shock management remains undefined. It would be reasonable to administer steroids to shock victims with known adrenal insufficiency or chronic steroid use and refractory hypotension.

CONTROVERSIES

Shock Science

The lack of definitive studies on the treatment of shock in the out-of-hospital setting leaves the EMS medical director without clear guidelines for evaluating and treating these patients. One international study is examining the use of hypertonic saline for the treatment of hemorrhagic shock due to trauma. Out-of-hospital treatment is largely based on anecdotal reports, limited scientific studies, personal experience, and extrapolation from hospital-based pathways.^{39,51,52} As a result, considerable controversy exists with respect to many areas of the treatment of shock (especially traumatic shock) in the out-of-hospital setting.

The benefit of a prehospital procedure must be weighed against potential risks. A major pitfall

associated with shock treatment is that resuscitative interventions may delay definitive care.⁵³ For victims of myocardial infarction, for example, Pantridge and Geddes demonstrated that some aspects of definitive care, such as defibrillation and arrhythmia management, can and should be delivered in the field.⁵⁴ However, for trauma victims with internal hemorrhage, definitive care can only be provided in the hospital. Any field procedure that significantly delays delivery of definitive care must have proven value. For example, pneumatic antishock garments (PASG) were implemented in clinical EMS practice without supporting evidence, and then a formal assessment revealed that PASG actually worsens outcomes.⁵⁵

Treatment of Hemorrhagic Shock

Hemorrhage is a common cause of shock in the trauma victim. Based on animal studies, treatment schemes for hemorrhagic shock in the past have included aggressive fluid resuscitation and the use of PASG to restore normal blood pressure.³⁶ However, field clinical trials have suggested that volume resuscitation before controlling hemorrhage may be detrimental.^{30–34,36,37} Possible mechanisms for worse outcomes include dislodgement of clot, dilution of clotting factors, decreased oxygen-carrying capacity of the blood, and exacerbation of bleeding from injured vessels in the thorax or abdomen.^{32,33,36}

Studies in Houston and San Diego suggest that mortality following traumatic hemorrhage is not influenced by prehospital administration of fluid.^{32,34} Survival to hospital discharge rates were not significantly different for patients receiving fluids versus patients not receiving fluids in the field. Both studies were performed in systems with relatively short scene and transport times.

Currently, field providers in most clinical settings are taught to administer only enough IV or intraosseous fluid replacement as to restore a peripheral pulse or to reach a systolic blood pressure of 80 to 90 mm Hg. The optimum target blood pressure for these patients remains undefined. Excess fluid administration can create other problems in the out-of-hospital setting. Trauma victims with isolated head injuries who receive excess fluids may develop worsened cerebral swelling. In addition, excess fluids may precipitate congestive heart failure in susceptible individuals.

Intravascular access itself may present its own set of challenges. Several investigators have examined

the amount of time required to initiate interventions in the field, primarily in trauma victims, but these studies send conflicting messages. Smith et al. analyzed 52 cases of prehospital multiple trauma, finding that IV insertion time exceeded transport time in all cases, over one fourth of the IV attempts were unsuccessful, and only 1 L of fluid was infused in the most critical patients.³⁸ Conversely, studies led by Jacobs, Honigman, and Eckstein each found that on-scene time did not correlate with the number of prehospital procedures performed, including intubation, PASG application, or IV insertion.⁵⁶⁻⁵⁸ In the study by Jacob et al., ALS interventions did not delay transport time to the hospital as compared with BLS units.⁵⁸ Eckstein et al. described a 3.9 times higher adjusted survival rate for patients receiving IV fluids in the field versus those not receiving fluids.⁵⁶ Other investigators have described the feasibility of starting IV access while en route to the hospital, coining the term *zero-time* prehospital IV.⁵⁹

The majority of IV fluid studies took place in urban settings with primarily penetrating trauma victims and rapid transport times. The effectiveness of IV fluids for similar patients in the rural and wilderness settings remain undefined.

PROTOCOLS

A treatment protocol for treating shock in the field should address the following factors:

1. Establishing and maintaining the status of ABCs.
2. The definitive care permitted for these patients.
3. Transport to the hospital when appropriate.

4. Resources to be used in the field.

5. Skills of the various levels of prehospital care providers in the field.

Protocols developed for the out-of-hospital treatment of shock must consider the heterogeneity of the disease state, the limited assessment and treatment options, and the environment in which the protocols will be applied. Protocols for the inner city may not be appropriate for the rural setting. The level of training and clinical experience of the providers must also be considered. Ideally, medical oversight would use evidence-based medical decision making when developing treatment protocols. It is strongly recommended that the EMS medical director draw from best practices for the establishing of clinical protocols addressing the evaluation and treatment of shock that optimizes the resources of the area of medical oversight.

SUMMARY

Shock must be correlated with the patient's clinical condition, age, size, and present and past medical history. Providers must identify signs of decreased tissue perfusion when assessing for the presence of shock. Treatment modalities for shock are limited in the field, but include bleeding control, fluid administration, inotropic agents, and careful control of assisted ventilation. Although the mainstay of shock treatment is IV fluids, approaches should be individualized for different clinical scenarios. The potential benefits of shock care interventions must be weighed against the potential risks of delaying definitive care.

CLINICAL VIGNETTES

Case 1

Paramedics report that they are caring for a 65-year-old male complaining of abdominal pain and dizziness on standing. This man has a history of an abdominal aortic aneurysm, coronary artery disease, and recent prostate surgery, and he has an indwelling urinary bladder catheter. The patient is taking oral antibiotics and has no allergies. The patient is alert and oriented with a blood pressure

of 60 mm Hg palpable, pulse rate of 95 beats/min, and respiratory rate of 16 breaths/min. Medics note that he is pale and diaphoretic. Their evaluation is remarkable for clear lung fields and no evidence of jugular vein distention (JVD) or peripheral edema. The abdomen is slightly distended and tender. ECG monitor shows sinus rhythm at a rate of 95. The medics are 25 minutes from the nearest hospital and are requesting orders from DMO.

How Would You Direct the Management of This Patient?

Although the parties involved in this case were rightly concerned about a leaking abdominal aortic aneurysm, other etiologies of hypotension and shock in this patient may include a perforated abdominal viscus, myocardial infarction, gastrointestinal bleeding, and sepsis. Fluid therapy would be appropriate for hypovolemic, cardiogenic, distributive, or obstructive shock with no signs of fluid overload. Suspecting an abdominal hemorrhagic catastrophe, the DMO physician should instruct the field personnel to expedite transport. IV access should be established en route. A fluid challenge of 300 to 500 ml of crystalloid should be rapidly infused under pressure with frequent evaluation for the presence of a radial pulse and/or a palpated systolic blood pressure of 80 to 90 mm Hg. Serial fluid challenges may be administered according to this plan. The patient should be reevaluated frequently, ECG monitoring should be started, a 12-lead ECG should be taken and evaluated, and the operating room and surgical team at the receiving hospital should be notified. Additional standard protocol measures include the monitoring of pulse oximetry, capnography, and dextrose level. A secondary survey should be completed by the providers to ascertain any other conditions that may be present.

Case 2

A 65-year-old man with a history of hypertension, coronary artery disease, and myocardial infarction was working on his roof on a hot, sunny day. He struck a beehive with his hammer and suffered a fall approximately 6 feet from the roof. On arrival of the two-person paramedic crew, the patient was found unresponsive on the ground. A primary survey was performed, and the airway was secured by endotracheal intubation. During their report to medical oversight, the paramedics noted that the patient was relatively bradycardic with a heart rate 65 beats/min, blood pressure of 60 mm Hg systolic, and clear and equal lung sounds. Secondary survey revealed no signs of external or obvious sources of internal bleeding, urticaria, facial swelling, internal or external trauma, or arrhythmias. The paramedics estimate a 20-minute transport time to the nearest trauma center.

How Would You Direct the Management of This Patient?

The etiologies of hypotension in this patient are numerous, but the initial evaluation and treatment is independent of the etiology. The patient showed no signs of fluid overload, and therefore interventions to be performed by the paramedics en route to the hospital should include large-bore IV access or intraosseous access and the administration of a fluid challenge of at least 300 ml of an isotonic crystalloid. Response to these interventions would direct further therapy. The patient remained hypotensive despite repeated fluid challenges (200-ml trials infused rapidly under pressure for a total of 1 L). A dopamine drip was initiated with moderate improvement of the patient's blood pressure and perfusion. However, the patient remained unresponsive and without spontaneous movement. As in the previous case, additional standard protocol measures include the monitoring of pulse oximetry, capnography, and dextrose level. Evaluation at the trauma center revealed a cervical spine fracture and ECG changes that were suggestive of an inferior wall myocardial infarction to the emergency department team.

Case 3

Paramedics have initiated transport of a 25-year-old female who cut both of her wrists in an apparent suicide attempt. On arrival of the paramedics at the scene, the patient was awake, but drowsy, with active bleeding from both wrists. The field team estimates a 900-ml blood loss on scene. The bleeding is now controlled with direct pressure, and two large-bore IV catheters have been established. The patient's present systolic blood pressure is 60 mm Hg. Normal saline IV lines are running wide open.

How Would You Direct the Management of the Intravenous Fluids for This Patient?

This patient is suffering from hypovolemic shock and requires fluid resuscitation. In this case, the hemorrhage is controlled, and fluids should be administered at a wide open rate with pressure applied to the IV fluid bag to increase the flow. Unlike the uncontrolled hemorrhage model in which aggressive fluid administration may lead to increased bleeding, the bleeding here is controlled. Therefore, fluid volume should be rapidly replaced to normalize blood pressure.

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