**Chapter 7
Hypotension and shock**

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**Introduction**

Shock is a life-threatening physiological state characterized by decreased tissue perfusion and end-organ tissue dysfunction, and is a significant predictor for complications including death [1]. The presence of shock must be recognized and therapeutic interventions must be started early to prevent progression. Unfortunately, the identification and treatment of shock in the out-of-hospital setting are fraught with many difficulties and potential pitfalls. Patient assessment is often limited by the challenging out-of-hospital environment and lack of diagnostic and therapeutic options. The tools available for the diagnosis and treatment of shock in the field are limited. Even when shock is properly identified, the most appropriate out-of-hospital management is often unknown or the subject of great debate.

In the out-of hospital setting, the identification of shock relies primarily on the recognition of signs and symptoms, including tachycardia, poor skin perfusion, and altered mental status. Note that hypotension, arbitrarily defined at a systolic blood pressure of less than 90 mmHg, is not an adequate definition of shock and may not adequately reflect the onset of tissue hypoperfusion [2]. Unfortunately, the early stages of compensated shock, with only subtle alterations in physical findings, are easily overlooked or misinterpreted by out-of-hospital care providers. Physiological changes associated with age, pregnancy, or treatment for medical conditions, such as beta-blockers for hypertension, may also mask or alter the body’s compensatory responses. As a result, the patient with severe shock may present with near-normal vital signs.

**Pathophysiology**

Shock is a complex physiological 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 [3]. In general, the following relationships drive this process.

Blood pressure = Cardiac output × Peripheral vascular resistance

Cardiac output = Heart rate × 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 cardiac output and hence blood pressure secondary to decreased cardiac filling. Also, decreasing stroke volume may 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 extensive 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 [4]. 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 [5]. Thus, categorizing shock into four categories may help prehospital providers and EMS physicians organize their assessments and approaches ([Table 7.1](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c07.xhtml#c7-tbl-0001)). Accurate physical assessment is vital for the EMS provider to determine the etiology of the shock state ([Box 7.1](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c07.xhtml#c7-fea-0001)).

[**Table 7.1**](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c07.xhtml#R_c7-tbl-0001) Categories of shock

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| **Type of shock** | **Disorder** | **Examples** | **Comments** |
| --- | --- | --- | --- |
| Hypovolemic | Decreased intravascular fluid volume | 1. External fluid loss
	1. Hemorrhage
	2. Gastrointestinal losses
	3. Renal losses
	4. Cutaneous loss
2. Internal fluid loss
	1. Fractures
	2. Intestinal obstruction
	3. Hemothorax
	4. Hemoperitoneum
	5. Third spacing
 | Hypovolemic shock states, especially hemorrhagic shock, produce flat neck veins, tachycardia, and pallor |
| Distributive | Increased “pipe” size: peripheral vasodilation | A. Drug or toxin inducedB. Spinal cord injuryC. SepsisD. AnaphylaxisE. Hypoxia/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 embolismB. Tension pneumothoraxC. Cardiac tamponadeD. Severe aortic stenosisE. Venocaval obstruction | Obstructive shock states tend to produce jugular venous distension, tachycardia, and cyanosis |
| Cardiogenic | “Pump” problems | A. Myocardial infarctionB. ArrhythmiasC. CardiomyopathyD. Acute valvular incompetenceE. Myocardial contusionF. Myocardial infarctionG. Cardiotoxic drugs/poisons | Cardiogenic shock states tend to produce jugular venous distension, tachycardia, and cyanosis |

**Box 7.1 Signs and symptoms of shock**

* Cardiovascular
	+ Tachycardia, arrhythmias, hypotension
	+ Jugular venous distension in obstructive and cardiogenic shock states
	+ Tracheal deviation away from the affected side in tension pneumothorax
* Central nervous system
	+ Agitation, confusion
	+ Alterations in level of consciousness
	+ Coma
* Respiratory
	+ Tachypnea, dyspnea
* Skin
	+ Pallor, diaphoresis
	+ Cyanosis (in obstructive and cardiogenic shock cases), mottling
* **Evaluation**
* The diagnosis of shock depends on a combination of key historical features and physical findings in the proper clinical setting. For example, tachycardia and hypotension in an elderly patient with fever, cough, and dyspnea may represent pneumonia with septic shock. Hemorrhagic shock should 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 emergency medical technicians (EMTs) in a non-emergency setting [6]. The researchers suggest that when critical medical decisions will be based on the data gathered in the field, multiple assessments should be performed.
* Emergency medical services 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 [Box 7.1](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c07.xhtml#c7-fea-0001)). When available, adjunctive technologies can provide improved recognition and assessment of shock by demonstrating reductions in expired CO2, hypovolemia, obstruction, or poor contractility on ultrasound, and elevated serum lactate levels.
* Vital signs that fall outside of expected ranges must be correlated with the overall clinical presentation. Vital signs have a broad range of normal values and must be interpreted in the context of the individual patient. A petite 45 kg, 16-year-old female with lower abdominal pain with a reported blood pressure of 88 mmHg systolic by palpation may have a ruptured ectopic pregnancy, or may just be at her baseline blood pressure. An elderly patient with significant epistaxis may be hypertensive due to catecholamine release and vasoconstriction despite being relatively volume depleted. Consideration should be given to patient age, comorbid conditions, and medications that may affect the interpretation of vital signs.
* 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 [7]. Without an auscultated diastolic pressure, the pulse pressure (difference between systolic and diastolic pressure) cannot be calculated. A pulse pressure less than 30 mmHg or 25% of the SBP may provide an early clue to the presence of hypovolemic or obstructive shock. Conversely, a wide pulse pressure may be indicative of distributive shock [3]. Dividing the pulse rate by the systolic pressure typically produces a ratio of approximately 0.5 to 0.8, which is called the “shock index.” When that ratio exceeds 1.0, then a shock state may be present [8].
* Previously healthy victims of acute hypovolemic shock may maintain relatively normal vital signs with up to 25% blood volume loss [3]. Sympathetic nervous system stimulation with vasoconstriction and increased cardiac contractility may result in a normal blood pressure in the face of decreasing intravascular volume, especially in the pediatric population. In some patients with intraabdominal bleeding (e.g. ruptured abdominal aneurysm, ectopic pregnancy), the pulse may be relatively bradycardic despite significant blood loss [9].
* Emergency medical services personnel may equate “normal” vital signs with normal cardiovascular status [5]. 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. Following trends in the vital signs may also help identify shock before patients reach abnormal vital sign triggers. 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 [10].
* Prehospital hypotension may predict in-hospital morbidity and mortality in both trauma and medical patients [11–13]. Jones et al. noted a 30% higher mortality rate for medical patients with prehospital hypotension [11]. Other studies have shown similar findings in trauma patients with prehospital hypotension, even with subsequent normotension in the emergency department [12,13]. Therefore, hospital providers should consider any episode of prehospital hypotension as evidence of significant shock and the presence of a critical illness.
* Despite their questionable value, orthostatic vital signs are often evaluated in the emergency department, and occasionally in the field. A positive orthostatic vital sign test for pulse rate would result in a pulse increase of 30 beats per minute after 1 minute of standing [14]. Symptoms of lightheadedness or dizziness are considered a positive test. 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 euvolemia.
* Capillary refill, an easy test to perform in the field setting, is not a useful test for mild-to-moderate hypovolemia [15]. Moreover, environmental considerations, such as cold temperatures and adverse lighting conditions, also affect the accuracy of this technique for shock assessment. On-scene estimates of blood loss by EMS providers may influence therapeutic interventions, including fluid administration. However, studies suggest that providers are not accurate at estimating spilled blood volumes [16].
* Hypoxia is a common manifestation of shock states. Patients in various stages of exsanguination may not have sufficient blood volume to adequately perfuse the body with oxygen. Unfortunately, pulse oximetry alone cannot detect the adequacy of oxygen delivery. Pulse oximetry may fail to detect a pulse when blood flow is reduced [17,18]. Like pulse oximetry, capnography may also serve as an important tool in the evaluation and treatment of shock in the prehospital setting [19–22]. Capnography is the measurement of the exhalation 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 [23,24]. Additionally, changes in EtCO2 are virtually immediate when the airway is obstructed or the endotracheal tube becomes dislodged [25]. 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 [26–28]. EtCO2 is most useful as an indicator of perfusion when minute ventilation is held constant (e.g. when mechanical ventilation is applied) [20,26]. Under these conditions, changes in EtCO2 levels reliably indicate changes in pulmonary perfusion. In any patient suffering from a potential shock state, diminished EtCO2 should be a warning of the critical nature of the patient's problem.
* **Future technologies in the assessment of shock**
* Use of portable ultrasound in the field can facilitate the recognition of immediately life-threatening causes of shock including intraabdominal hemorrhage, cardiac tamponade, or an abdominal aortic aneurysm. Many EMS agencies, primarily air medical services, have deployed ultrasound for field evaluations, including the focused assessment by sonography in trauma (FAST) examination [29]. Ultimately, the EMS medical director must determine if the cost and time of acquiring equipment, training, and performing the skills translates into improved patient outcomes. The use of field ultrasound has the potential to worsen patient outcome if the procedure delays the time to definitive care, does not influence patient destination or care, or interferes with basic skills (e.g., airway maintenance).
* There is growing interest in the use of biomarkers that can be used to identify, monitor, and predict the outcome in shock [30]. Point-of-care testing devices make measurement of biomarkers in the field an attractive option. Elevation of the serum lactate may reflect anaerobic tissue metabolism in acute sepsis and shock [30–32]. In the emergency department, elevated lactate in the setting of infection indicates septic shock and the need for early sepsis therapy. Elevated point-of-care venous lactate is associated with increased mortality risk and the need for resuscitative care in trauma patients. Indeed, recent work by the RESUSCITATION OUTCOMES CONSORTIUM in prehospital trauma research indicates that lactate levels may rise before blood pressure drops, and that an elevated lactate level in the setting of trauma may be a useful predictor of a patient that will require aggressive resuscitation. Serial lactate measurements may indicate the progress of ongoing resuscitation [33].

In summary, although technology may offer future value, 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 medical oversight 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. Perform the initial assessment.
2. Deal with issues identified in the initial assessment such as airway, breathing, and circulation issues, including active external bleeding.
3. Determine need for early definitive care.
	* Hemorrhage control and volume resuscitation
	* Needle thoracostomy
	* Electrical therapy for dysrhythmia
	* Invasive airway management
4. Maintain adequate oxygen saturation (SaO2 > 94%).
5. Ensure adequate ventilation without hyperventilating.
6. Monitor vital signs, ECG, oxygen saturation, capnography, and lactate (if available).
7. Prevent additional injury or exacerbation of existing medical conditions.
8. Protect the patient from the environment.
9. Determine the etiology of the shock state, and treat accordingly.
10. Notify and transport to an appropriate facility.

Often the etiology of the patient’s shock state 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. Occasionally, the primary problem may be strongly suspected but not readily diagnosable or 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 (primarily fluids, inotropic agents, and vasopressors), field treatment may be individualized for the four categories of shock: hypovolemic, distributive, obstructive, and cardiogenic.

## Hypovolemic shock

Hypovolemic shock is the result of significant loss of intravascular volume resulting in hypotension. The many etiologies of hypovolemic shock include external fluid loss and shifting of fluids from the vascular system to a non-vascular body compartment. The treatment of hypotension and shock caused by hypovolemia is relatively straightforward. External bleeding should be controlled. Fluid replacement via vascular access is the mainstay of treatment. Unfortunately, the ideal fluid for the resuscitation of hypovolemic shock and the amount of fluids that should be provided remains controversial [34–46].

## Distributive shock

Distributive shock, characterized by a decrease in systemic vascular resistance, is associated with abnormal distribution of microvascular blood flow [47]. Causes of distributive shock include sepsis, anaphylaxis, medication overdose, and acute neurological injury. 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 (e.g. intramuscular, intravenous bolus or infusion), the drug has significant side-effects. Norepinephrine infusions are associated with a lower incidence of cardiac dysrhythmias than either dopamine or epinephrine [48]. In addition, recent studies of cardiogenic shock suggest increased mortality associated with dopamine [49]. However, 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), intravenous fluids may be helpful in increasing preload and temporarily improving the condition.

## Cardiogenic shock

Causes of cardiogenic shock include arrhythmias, valvular heart disease, cardiotoxic agents, and myocardial infarction. As a result, cardiogenic shock requires individualized treatment. Cardiogenic shock from severe dysrhythmias should be treated with appropriate electrical or pharmacological therapy. “Pump failure” is often difficult to diagnosis and to treat without invasive monitoring. Adult patients without obvious pulmonary edema may benefit from fluid challenges of approximately 200–300 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 modest 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 dobutamine or norepinephrine, would be more appropriate. Intravenous infusions are often difficult to manage in the field without an infusion pump and must be monitored closely.

The causes of cardiogenic shock also can include 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. Alternative therapies may include IV glucagon or calcium, which facilitates heart rate stimulation and vasoconstriction through alternative cellular receptors, and which many EMS agencies carry for the treatment of hypoglycemia.

## Shock of unclear etiology

In a few disconcerting situations, the primary etiology for the patient’s shock state may be difficult to determine. The primary treatment decision is whether or not to give fluids. In hypovolemic, distributive, and obstructive shock, fluids are an appropriate initial treatment for hypotension, given the important caveats mentioned above regarding that in the setting of uncontrolled hemorrhage, indiscriminate administration of large volumes of IV fluids may not improve patient outcome. Some cases of cardiogenic shock will respond to fluids. However, fluids should not be given to patients in cardiogenic shock with pulmonary edema. Fluids are also not appropriate when cardiogenic shock has been precipitated by a treatable arrhythmia. Response to fluid challenges (where appropriate) 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, including adrenal suppression, hypothyroidism, or toxidromes. In some cases patients with profound acidosis will not respond to vasopressors or inotropes, as their receptors are pH dependent. Administration of sodium bicarbonate at 1 mEq/kg may improve perfusion by buffering acidosis and increasing vasopressor activity. Use of vasopressin to supplement other vasopressors may also improve perfusion as it increases systemic vascular resistance even during acidosis. In cases of refractory shock or adrenal suppression, administration of steroids may also be of benefit. Hydrocortisone is ideal for this purpose, as patients may benefit from both mineralocorticoid and glucocorticoid properties. Methylprednisolone is far more widely available in the prehospital environment and may have some limited utility in refractory shock. Patients exposed to potent cellular toxins such as cyanide or hydrogen sulfide may also present with refractory shock, prompting therapy with agent-specific antidotes.

## Shock in the pediatric population

The recognition and management of shock in the pediatric population follow the same general principles as in adults, with a few notable exceptions [50]. Children in shock more commonly present with a low cardiac output and a relatively high systemic vascular resistance (SVR). This has been described as “cold shock,” as opposed to the low-SVR state or “warm shock” frequently seen in adults. Children presenting in distributive shock usually require more aggressive fluid resuscitation with volumes of 60 cc/kg or more [51]. If children fail to respond to the initial fluid resuscitation, epinephrine is preferred as the first-line vasopressor in order to counter the relatively low cardiac output seen in pediatric shock. Additional support for patients with low SVR and wide pulse pressure may be provided with norepinephrine or vasopressin. Dobutamine may provide inotropic and chronotropic support in patients with very low cardiac output and improve delivery of oxygen to tissues.

Following initial treatment with fluids and vasoactive agents, pediatric patients may also benefit from adjunctive therapies for shock [50]. Early airway management should be considered, as children may use up to 40% of their cardiac output to support the work of breathing. Ketamine is the preferred induction agent as it preserves cardiac output and will not result in the hypotension or adrenal suppression potentially seen with other induction agents. Hydrocortisone should be administered to children with adrenal insufficiency. Transport to an appropriate facility with pediatric critical care should be an important consideration.

## 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 hemorrhage control, airway maintenance, and rapid transport to the hospital. IV or IO access could be attempted en route if it will not delay delivery to definitive care [43]. 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. Fluid delivery could be initiated while the patient is en route to the hospital, thereby prolonging neither scene time nor time until definitive care [44]. In the patient who presents a difficult IV access problem, IO infusions may be attempted. Placing the IO needle in the humeral head may result in faster infusion rates than the proximal tibia.

The ideal fluid for use in the field would be small in volume, portable, non-allergenic, inexpensive, and would not interfere with clotting factors [34]. Unfortunately, this ideal fluid has yet to be discovered. Isotonic crystalloids are currently the fluid of choice for out-of-hospital resuscitation in the United States [1,35,38,44]. They are inexpensive and widely available but may contribute to dilutional coagulopathy, hyperchloremic acidosis, and hypothermia when given in large volumes.

Whole blood would arguably provide the greatest benefit as a resuscitation fluid in the setting of hemorrhagic shock but is impractical due to issues of cost storage and availability. Use of blood products in the out-of-hospital environment is limited to a few air medical services which carry O-negative blood for administration to victims of hemorrhagic shock. Prehospital administration of plasma and factor concentrates is being investigated [52]. Several centers have studied hypertonic saline, colloids, and artificial blood substitutes as alternatives to isotonic saline [52,53]. Problems with these alternative fluids include high cost; increased risks including allergic reactions, kidney injury, coagulopathy, and hypernatremia; and lack of demonstrated benefit versus isotonic crystalloids [36–38,54,55]. As a result, none of the alternative fluids has gained widespread acceptance.

The optimal volume of fluids to administer in the out-of-hospital setting is not known, especially in the trauma victim with uncontrolled hemorrhage [35,39–46]. Older trauma algorithms indicate the administration of 2 liters IV fluid for all major trauma victims. Evidence suggests, however, that attempts at normalization of blood pressure with a large volume of fluids in the patient with uncontrolled hemorrhagic shock may be deleterious to patient outcome. Complications may include acidosis, dislodgment of blood clots, and dilution of clotting factors [56]. In such a patient, it appears that the best course is to give sufficient crystalloid to maintain a peripheral pulse, pending the delivery of the patient to the appropriate facility [40–42].

Administration of IV 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 IO route for vascular access has been used for generations. This was a common form of vascular access during World War II, though it became a less popular route in the postwar era with the rising use of IV cannulation. IO access has become so important as a method of vascular access that it is supported by a position statement from the National Association of EMS Physicians [57]. In patients *in extremis* or cases in which peripheral access is not immediately available, IO access may be preferred. Various devices are available, and EMS medical directors must work with their systems to determine the most appropriate device for use by their providers.

Controlling external hemorrhage is essential for maintaining vascular volume. Direct pressure is usually sufficient to control external bleeding. Military and civilian experience suggests that tourniquets should be used early and liberally [58]. An assortment of topical hemostatic materials to be placed directly on the bleeding wound also exists [58–61]. The hemostatic dressing must be applied in conjunction with direct pressure to be effective. Pelvic binders may compress bleeding pelvic vessels while reducing the internal volume available for hemorrhage into the pelvis.

Tranexaminic acid (TXA) is a lysine derivative which blocks fibrinolysis. It has long been used to control hemorrhage during surgery [2]. In a randomized controlled study, TXA demonstrated an ability to reduce mortality from traumatic hemorrhage if administered within 3 hours of the time of injury [62]. Some prehospital systems are beginning to use TXA to treat hemorrhagic shock. Evidence for its benefit is found in its early administration, with late administration of TXA being associated with worsening outcomes [63].

### 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 supraglottic devices, results in an increase in airway pressure, raising intrathoracic pressure. Patients in shock from any cause are extremely sensitive to increases in intrathoracic pressure. Studies in a swine hemorrhagic shock model showed that even modest increases in the rate of positive pressure ventilation significantly reduce brain blood flow and oxygenation [64]. 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 for an adult, producing a minute ventilation of about 5 L/min. Minute ventilation should be adjusted to ensure an EtCO2 between 35 and 45 cmH2O.

### Vasopressor agents

Administration of vasoactive medications may be required to reverse systemic hypoperfusion from distributive or cardiogenic shock. These agents increase cardiac inotropy, chronotropy, and/or vasoconstriction [65]. Although a wide variety of vasoactive agents is available in the hospital, the drugs carried by prehospital services are limited by local, regional, or state-wide 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 pathological 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 direct medical oversight, where an experienced clinician in a more controlled setting can perform important calculations. When available, particularly during interfacility or air medical transport, portable IV infusion pumps should be used to ensure accurate and precise medication administration.

### Other drug agents

Other agents used for shock resuscitation include corticosteroids, antibiotics, colloids, inotropic agents, recombinant human activated protein C, and dextran [66,67]. 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 treating these patients. 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 an out-of-hospital procedure must be weighed against potential risks. A major pitfall associated with shock treatment is that resuscitative interventions may delay definitive care [68]. 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 [69]. However, for trauma victims with uncontrolled 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 anti-shock garments (PASG) were implemented in clinical EMS practice without supporting evidence, and then a formal assessment revealed that PASG actually worsened patient outcome in certain circumstances, particularly thoracic injury [70].

### Treatment of hemorrhagic shock

Hemorrhage is a common cause of shock in the trauma victim. Field clinical trials have suggested that volume resuscitation before controlling hemorrhage may be detrimental [35,39–42,44,45]. Possible mechanisms for worse outcomes include dislodgement of clot, dilution of clotting factors, decreased oxygen-carrying capacity of the blood, hyperchloremic metabolic acidosis, and exacerbation of bleeding from injured vessels in the thorax or abdomen [40,41,44].

Studies in Houston and San Diego suggest that mortality following traumatic hemorrhage is not influenced by prehospital administration of fluid [40,42]. 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.

As discussed above, currently field providers in most clinical settings are taught to administer only enough IV or IO fluid replacement to restore a peripheral pulse or to reach a systolic blood pressure of 80–90 mmHg. However, the optimum target blood pressure for these patients remains undefined. 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 or lead to impaired immune response following severe injury.

Conversely, the benefit of limited volume resuscitation has been derived from military and urban data with a predominance of penetrating injuries and young, healthy patients. This population may be more tolerant of hypovolemic resuscitation and benefit from relative hypotension while reducing the risk of clot dislodgment. However, patients with blunt injury and limited organ reserve due to comorbid illness or age may be intolerant of hypotensive resuscitation. A multicenter trial evaluating limited crystalloid resuscitation versus standard aggressive resuscitation coordinated by the Resuscitation Outcomes Consortium (ROC) is currently under way [71].

Attempts at establishing intravascular access in critically injured trauma victims may delay time to definitive care, especially in the urban setting [39,56,72–74]. The majority of IV fluid studies have taken place in urban settings primarily with penetrating trauma victims and rapid transport times. The effectiveness of IV fluids for similar patients in the rural and wilderness settings remains undefined. The subject remains controversial, with several studies providing mixed messages [39,56–59,72–76].

**Protocol development**

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

1. Performing the initial assessment.
2. The definitive or life-saving interventions appropriate for these patients.
3. Access to definitive care without unnecessary prehospital delay.
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 directors 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 establishing clinical protocols addressing the evaluation and treatment of shock.

**Conclusion**

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.