

# CURRENT

Diagnosis & Treatment



## EMERGENCY MEDICINE

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# Shock

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Shock is a state of severe systemic reduction in tissue perfusion characterized by decreased cellular oxygen delivery and utilization as well as decreased removal of waste byproducts of metabolism. Hypotension, although common in shock, is not synonymous to shock. One can have hypotension and normal perfusion, or shock without hypotension in a patient who is usually very hypertensive. Shock is the final preterminal event in many diseases. Progressive tissue hypoxia results in loss of cellular membrane integrity, a reversion to a catabolic state of anaerobic metabolism, and a loss of energy-dependent ion pumps and chemical and electrical gradients. Mitochondrial energy production begins to fail. Multiple organ dysfunction follows localized cellular death, and organism death follows. Despite recent advances in treatment, mortality remains high: >60% in cardiogenic shock and >35% in septic shock.

## PATHOPHYSIOLOGY OF SHOCK

One method of evaluating shock is to recall the determinants of systemic blood pressure. Blood pressure is determined by the formula  $BP = \text{systemic vascular resistance (SVR)} \times \text{cardiac output (CO)}$ , where  $CO = \text{heart rate (HR)} \times \text{stroke volume (SV)}$ .  $SV = \text{end diastolic volume (EDV)} - \text{end systolic volume (ESV)}$ . EDV is the filled ventricular volume prior to systolic contraction averaging about 100 cc in many adults. ESV is residual blood left in the ventricle after emptying during systole averaging about 40 cc. Therefore, the determinants of blood pressure are vascular resistance, HR, preload volume, and contractility (see Figure 9-1). SVR is the vascular "tone" and is a large determinant of diastolic blood pressure. EDV is largely determined by a preload volume that augments SV via Frank-Starling curves where increases in diastolic filling volumes increase CO. ESV is determined largely by cardiac contractility and it decreases as the heart ejects a greater percentage of its diastolic volume. For example, one can increase SV by increasing preload (EDV) with volume or decreasing ESV with increased contractility. The ejection fraction  $((EDV - ESV)/EDV)$  thus increases.

The initial derangement precipitating a state of shock might be (1) vasodilation (causing a decreased SVR) from sepsis, anaphylaxis, drugs, or cervical cord lesion, (2) extremes of HR, (3) loss of preload volume (causing de-

creased EDV) from blood or volume loss, or (4) loss of contractility (increasing the ESV) from heart failure. Compensatory mechanisms come into play and provide many of the clinical clues to early shock.

The initial insult of vasodilation from loss of SVR generally causes a compensatory tachycardia and thirst. Despite systemic tissue hypoxemia, the skin remains perfused and is warm initially. Blood or fluid loss (decreasing EDV) causes a reflex increase in SVR, which increases diastolic BP, narrowing the pulse pressure; increases sympathetic cholinergic sweating; and makes the patient pale, thirsty, and cool. As volume loss increases, tachycardia and hypotension ensue. Loss of contractility also is compensated by increases in SVR to maintain blood pressure with similar symptoms.

Once compensatory mechanisms fail, irreversible shock occurs with irreversible cell death, microcirculation plugging, and free radical generation. There is loss of autonomic regulation due to local nitric oxide vasodilator generation, and even with complete correction of blood volume (for example, in hypovolemic shock), tissue function and organ function are not restored, causing eventual death.

## CAUSES OF SHOCK

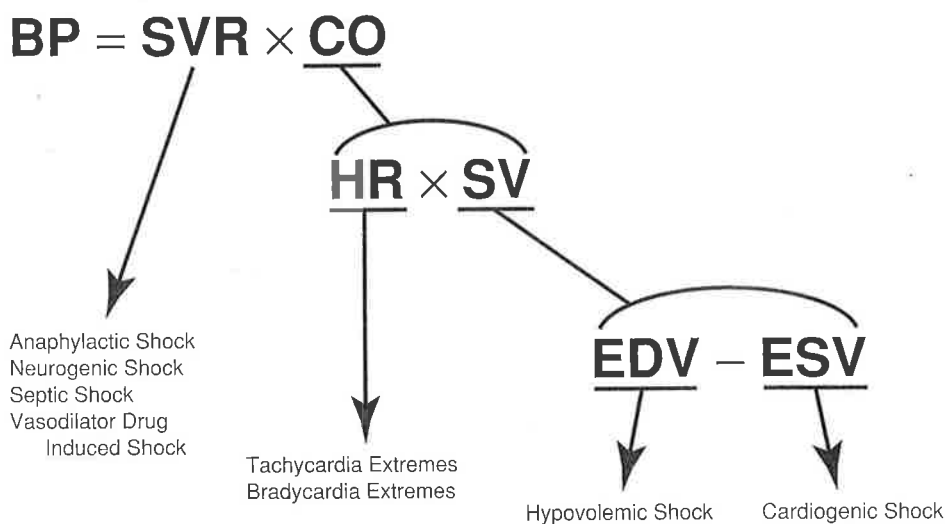
The major classical classification of shock includes (1) hypovolemic, (2) cardiogenic, (3) distributive, and (4) obstructive shock. The first three involve a primary derangement in EDV, ESV, and SVR, respectively, while obstructive shock is usually a problem with SV due to mechanical obstruction to preload. Causes of "obstructive shock" are sometimes classified as hypovolemic or cardiogenic. Common causes of each type are listed in Table 9-1.

## CLINICAL PRESENTATION

Unfortunately, there is no one clinical or biological test to determine shock. If compensatory mechanisms are functioning early in shock, one may not see hypotension but instead an anxious patient still maintaining a blood pressure. In these early stages (called preshock) symptoms can be subtle, but provide an opportunity for early intervention. Waiting for full-blown shock leads to a loss of precious time, and an aggressive proactive approach should be pursued.



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BP = blood pressure, SVR = systemic vascular resistance, HR = heart rate, SV = stroke volume, EDV = end diastolic volume (i.e. preload), ESV = end systolic volume (i.e. contractility)

Figure 9-1. Determinants of blood pressure.

During the early or preshock state, pale, cool, moist skin reflects compensatory elevated SVR in hypovolemic and cardiogenic shock. The pulse pressure narrows (with a slight decrease in systolic blood pressure and rise in diastolic blood pressure) and patient anxiety increases. Blood is shunted preferentially from "nonessential" skin and gastrointestinal (GI) tract to heart and brain. After a 20–30% volume loss in hypovolemic shock, tachycardia increases, urine volumes decrease with decreased renal blood flow, and the patient becomes more agitated. In cardiogenic shock, left-sided heart failure manifests as pulmonary edema and right-sided heart failure manifests as peripheral edema with elevated jugular venous distention (JVD).

In distributive shock, the primary problem is loss of vascular tone with erythematous warm skin despite hypotension. Tachycardic response is variable and early on, the heart may be hyperdynamic. This is the "warm patient in shock."

In full-blown shock, patients become agitated and finally decrease their mental status. Hypotension occurs and may be profound. The patient has tachypnea until respiratory failure occurs and has a metabolic acidosis due to elevated lactic acid from anaerobic metabolism. At the cellular level, tissue oxygen extraction is maximal and is reflected in decreased mixed venous  $O_2$  saturation. Multiple organ failure follows.

Irreversible shock follows if treatment is not aggressive.

### INITIAL EVALUATION OF SHOCK

In the initial evaluation of a patient in preshock, one must ask: Is the patient in shock or headed that way? It is ill advised to wait for severe hypotension in the patient who is still compensating for their shock before aggressively intervening. The clinician's first priority is to maintain vital functions while exploring the potential causes of shock. One should also consider early decontamination if the patient has been exposed to a toxin.

**ABCs** Open the airway and maintain adequate ventilation with high flow oxygen. Airway adjuncts such as the nasopharyngeal airway may help, but one must anticipate worsening of the airway with time. If the patient cannot protect the airway, has a GCS score < 9 in trauma, has extremes of respiratory rate or is hypoxic despite supplemental oxygen, endotracheal intubation is indicated. Relieve any tension pneumothorax based on clinical grounds (below). Establish multiple short-length, large-bore peripheral IV access and place on a cardiac monitor in a critical care area of the ED. Central venous access and arterial catheter placement should be considered. Remove clothes and keep the patient warm. Next, try to determine if there is a readily

**Table 9-1.** Causes of shock

- 
- A. Hypovolemic shock
1. Blood loss
    - a. Traumatic hemorrhage
      1. Exsanguination (e.g. Scalp)
      2. Hemothorax
      3. Hemoperitoneum
      4. Fracture (femur & pelvis)
    - b. Nontraumatic hemorrhage
      1. GI bleed
      2. AAA rupture
      3. Ectopic pregnancy rupture
  2. Volume loss
    - a. Burns
    - b. Skin integrity loss (TEN)
    - c. Vomiting
    - d. Diarrhea
    - e. Hyperosmolar states (DKA)
    - f. Third spacing (e.g., Ascites)
    - g. Decreased intake
- B. Cardiogenic shock
1. Dysrhythmia
    - a. Bradycardias and blocks
    - b. Tachycardias
  2. Cardiomyopathy
    - a. Infarction
    - b. RV infarction
    - c. Dilated cardiomyopathy
  3. Mechanical
    - a. Valvular
      1. Aortic insufficiency from dissection
      2. Papillary muscle rupture from ischemia
    - b. Ventricular aneurysm rupture
    - c. Free wall ventricle rupture
- C. Distributive shock
1. Anaphylactic shock
  2. Septic shock
  3. Neurogenic shock
  4. Drug induced vasodilation
  5. Adrenal insufficiency
- D. Obstructive shock
1. Tension pneumothorax
  2. Pericardial disease
    - a. Pericardial tamponade
    - b. Constrictive pericarditis
  3. Massive pulmonary embolism
  4. Auto PEEP from mechanical ventilation
- 

recognizable and potentially reversible cause of the patient's condition?

### Is There a Reversible Condition?

**Is there traumatic blood loss?** Stop visible hemorrhage, look for cavitory bleeding in the chest (CXR,

ultrasound (U/S)), abdomen (FAST exam), or pelvis from pelvic fracture or disruption or long-bone fracture. If pelvic or long-bone fractures are present, immobilize these injuries and consider pneumatic antishock trousers (PAST) for additional stabilization.

**Is there nontraumatic blood loss?** Check for a pulsatile abdominal mass (abdominal aortic aneurysm, AAA) or perform bedside U/S of the abdomen; check for GI bleed (hematemesis or melena).

**Is there a dysrhythmia?** Monitor and obtain an ECG. Cardiovert an unstable tachydysrhythmia and begin external cardiac pacing for unstable bradydysrhythmias.

**Is there a tension pneumothorax?** Check for decreased unilateral breath sounds, tracheal deviation away from pneumothorax, and a hyperresonant hemithorax. Perform needle decompression followed by chest tube thoracostomy.

**Is there cardiac tamponade?** Check for JVD, muffled heart sounds, low ECG voltage, and electrical alternans and perform bedside U/S if available. Perform pericardiocentesis if U/S demonstrates a pericardial effusion in the symptomatic patient.

**Is there evidence of massive pulmonary embolism?** In the patient at risk for pulmonary embolism (PE), is there hypoxemia with signs of acute right ventricular overload (bedside echocardiography)? Consider thrombolytics or surgical intervention.

**Is there overt anaphylaxis?** Look for angioedema, laryngeal edema with stridor, wheezing, and hives.

**Is the spinal cord injured?** Check for a motor/sensory level of paralysis/anesthesia. Trauma patients should be logrolled maintaining cervical spine precautions to examine the back for injury and check for gross rectal bleeding or loss of rectal tone.

**Is the problem with SVR?** If the skin is warm, despite hypotension, think sepsis, neurogenic, anaphylactic shock, or medication overdose ( $\beta$ -blocker or calcium channel blocker overdose).

### OBTAIN HISTORY CLUES TO THE CAUSE OF SHOCK

There may be historical clues to the patient's condition. Check for potential unsuspected trauma, unsuspected or known pregnancy, new medications, allergies, overdose, or depression. Look for potential drug interactions such as those of sildenafil and nitroglycerin. Obtain a travel history (SARS) and tampon-use history (toxic shock syndrome); chest pain and dyspnea may imply acute coronary syndrome or PE. Fever or hypothermia might signify sepsis.

## GIVE A FLUID CHALLENGE

Unless the patient is in severe pulmonary edema from cardiogenic shock, a fluid challenge of 20 cc/kg of isotonic crystalloid is a reasonable next step after the ABCs have been evaluated and overt causes addressed as above.

## OBTAIN APPROPRIATE LABS

Important early labs include complete blood count, coagulation studies, electrolytes, BUN, creatinine, arterial blood gas, and serum lactate. Note that the *venous* blood gas and *pulse oximetry* may be inaccurate in shock. In septic shock, obtain pan cultures. Obtain urinalysis in all patients and perform urine pregnancy testing in all women of child-bearing age. Type and crossmatch the patients for packed RBCs. In cardiogenic shock, obtain cardiac enzymes.

## OBTAIN APPROPRIATE IMAGING

A chest radiograph and ECG are valuable initial examinations with further testing dictated by clinical suspicion. Bedside U/S can have a key role with evaluation of pericardial fluid and hemoperitoneum but complex imaging studies should wait until the patient is resuscitated.

Hopefully, by this time you have some idea as to the nature of the type of shock and further therapy and evaluation can be tailored to the potential cause. Signs of a successful resuscitation include improvement in BP, improved mentation, decreasing lactate, resolving metabolic acidosis, urine output  $> 1$  cc/kg/h, and improved skin perfusion.

## SPECIFIC SHOCK CONDITIONS

### Hypovolemic Shock

The treatment objectives in hypovolemic shock are to stop the bleeding or fluid loss and to replace the blood or fluid. In traumatic hemorrhage, direct pressure is usually effective in stopping external hemorrhage. Shock from hemothorax or hemoperitoneum requires urgent operative intervention. Massive blood loss from a pelvic fracture might improve with manual stabilization in a sling (or sheet tied around the pelvis), pneumatic anti-shock trousers, or embolization via angiography. Femur fractures should be splinted with an external traction device.

A crystalloid infusion of normal saline or lactated Ringer's solution of 20 cc/kg should be given with general resuscitation measures described above. There has been no demonstrated advantage of albumin or other colloids over crystalloids. Hypertonic (7.5%) saline infusion (with or without dextran) has shown promise but is still largely of unproven benefit. Crystalloids continue to have the advantage of cost and availability.

**Table 9-2.** Signs of a successful initial resuscitation.

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1. Improved blood pressure
  2. Improving level of consciousness
  3. Improving peripheral perfusion
  4. Decreasing tachycardia
  5. Decreasing lactate
  6. Normalizing pH
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Blood and/or blood substitutes should be given early in hemorrhagic shock patients not readily responding to crystalloid infusion of 40 cc/kg. Choice is based on the time frame: For immediate need in an unstable patient, use un-crossmatched O-negative packed RBCs. If time allows, use type-specific red blood cells or typed and crossed packed RBCs. It is worthwhile knowing at your institution the time frames for when these various blood products are available.

The concept of "permissive hypotension" requires rapid surgical intervention and remains unproven, although overly aggressive fluid resuscitation might dislodge clots, interfere with the clotting cascade, and exacerbate bleeding. The goal of resuscitation should be to maintain a reasonable perfusion while aggressively stopping the source of bleeding.

Nontraumatic hemorrhagic shock from ectopic pregnancy or AAA rupture requires operative intervention. Patients with GI bleeding should have a nasogastric tube placed to reduce gastric size and monitor bleeding. Proton pump inhibitor, H<sub>2</sub> blockade (for gastric bleeding), octreotide infusion (for variceal bleeding). Endoscopy will likely be necessary for any patient exhibiting signs of shock and GI bleeding. Shock from fluid loss or third spacing should respond to crystalloid infusion.

Monitor for signs of successful resuscitation (Table 9-2), but realize that many patients suffering hemorrhagic shock require operative intervention.

### Cardiogenic Shock

Cardiogenic shock from tachydysrhythmias should be treated with cardioversion. Bradydysrhythmias require immediate transcutaneous pacing, although atropine may be tried first for sinus bradycardia or second-degree type I (Wenckebach) block. Myocardial infarction remains the most common cause of cardiogenic shock, complicating 6-8% of all acute myocardial infarctions (MIs) (1% in the ED and 5% later in the course). Overall, 4% of ST segment elevation myocardial infarction (STEMI) patients and 2-3% of non-ST segment elevation myocardial infarction (NSTEMI) patients develop cardiogenic shock. Mortality remains high for these

patients and the incidence is increasing. Age, prior MI, signs of overt shock, oliguria, left coronary thrombus, and poor ejection fraction correlate with mortality.

Treatment of cardiogenic shock includes general supportive measures of oxygen, aspirin, heparin, and "gentle" fluid challenges (250 cc) if there is no overt pulmonary edema. Glycoprotein IIb/IIIa inhibitors have shown a risk/benefit in favor of treatment. Pulmonary artery catheter insertion is necessary to guide therapy and further fluid/pressor therapy.

Early mechanical ventilation has been shown to decrease mortality and should be considered to decrease the work of breathing and reverse acidosis. This can be done invasively with endotracheal intubation or noninvasively via continuous positive airway pressure.

Vasopressors are often needed in addition to the above mentioned measures. Dopamine, titrated to hemodynamic response (hopefully with pulmonary artery catheter data guidance), can be started at 5  $\mu\text{g}/\text{kg}/\text{min}$ , increasing to 15  $\mu\text{g}/\text{kg}/\text{min}$ , where alpha vasoconstrictor effects predominate. There is no role for "renal dose" dopamine in the management of patients with shock. Dobutamine, which causes mild vasodilation and increased contractility, can also be used (often in concert with dopamine).

Unfortunately, systemic thrombolysis has had disappointing results in the treatment of cardiogenic shock probably due to poor coronary perfusion limiting thrombolytic agent delivery to the affected area. Percutaneous coronary intervention (PCI) has been shown to reduce mortality from 62% to 38%. Intra-aortic balloon pump (IABP) insertion augments diastolic blood pressure, improving coronary artery perfusion. If pressors do not stabilize the patient, IABP should be used to buy time awaiting PCI.

An exciting, but investigatory treatment option is N-monomethyl-L-arginine (L-NMMA), a nitric oxide synthase inhibitor, which in a small study reduced mortality from 67% to 27%.

Right ventricular infarction, often occurring in the setting of an inferior MI involving the right coronary artery, can be complicated by hypotension. Infarction of the right ventricle causes the ventricle to stiffen, requiring a greater preload to stretch the noncompliant muscle. Decreased right ventricle function decreases CO to the left ventricle via the lungs causing hypotension, especially if given a vasodilator like nitroglycerin. Inferior MIs do not usually cause pump failure, and hypotension in this setting should be examined with right-sided ECG leads and a fluid challenge. Right ventricular infarction is confirmed when 1 mm of ST elevation is noted in lead V4R or when the ST-segment elevation in lead III exceeds the ST elevation in lead II. Pump failure due to valvular or mechanical problems requires immediate consultation with a cardiothoracic surgeon.

Antman EM et al.: ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction—executive summary: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1999 Guidelines for the Management of Patients With Acute Myocardial Infarction). *Circulation* 2004;110:588. [PMID: 15358047]

Berger PB et al.: Impact of an aggressive invasive catheterization and revascularization strategy on mortality in patients with cardiogenic shock in the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO-I) Trial. An observational study. *Circulation* 1997;96(1):122-127. [PMID: 9236426]

Cotter G et al.: LINCOS: L-NAME (a NO synthase inhibitor) in the treatment of refractory cardiogenic shock: A prospective randomized study. *Eur Heart J* 2003;24(14):1287-1295. [PMID: 12871685]

Hochman JS et al.: Early revascularization in acute myocardial infarction complicated by cardiogenic shock. SHOCK Investigators. Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock. *N Engl J Med* 1999;341(9):625-634. [PMID: 10460813]

Hochman JS, Menon V: Clinical manifestations and diagnosis of cardiogenic shock complicating acute myocardial infarction. In: Rose BD (ed). *UpToDate*. UpToDate, Waltham, MA, 2005.

## Anaphylactic Shock

One of the most frightening causes of shock is the patient in anaphylaxis where the airway is obstructing, ventilation is compromised with bronchospasm, and the blood pressure is low. Treatment should be aggressive and proactive. The airway is at risk in anaphylaxis due to angioedema, tongue or laryngeal edema. Patients with anaphylaxis are likely to be among the most challenging with regard to airway management. These patients can rapidly deteriorate and become "can't intubate, can't ventilate" airway disasters. In rapidly advancing airway obstruction, early intubation is advised. In these patients, because of difficulties with standard intubation techniques, a surgical airway may be needed. Patients with bronchospasm should receive aggressive treatment with  $\beta$ -agonist aerosol or epinephrine (see below). Systemic vasodilation causes hypotension, and aggressive fluid resuscitation is needed using crystalloids.

Treatment includes removal of any known antigen, early administration of epinephrine,  $\beta$ -agonist aerosol, H1 and H2 histamine receptor blockade, and steroids. For mild to moderate symptoms, epinephrine can be given 0.3-0.5 mg SQ or preferably IM (pediatrics use 0.01 mg/kg/dose). For life-threatening symptoms, use 0.5-1 cc of 1:10,000 solution IV slowly. For a safer and titratable infusion, mix 1 mg epinephrine in 250 D<sub>5</sub>W for a 4  $\mu\text{g}/\text{mL}$  drip and start at 1-10  $\mu\text{g}/\text{min}$  titrating to response. Albuterol by nebulizer may also be used as a bronchodilator. Diphenhydramine 50 mg IV or IM (pediatric 1-2 mg/kg) and ranitidine 50 mg IV (or famotidine

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20 mg) provide histamine blockade. A stress dose of hydrocortisone 100 mg completes the primary treatment. Mild cases, after a 6-hour observation, can be discharged with a prescription for a self-injector of epinephrine for future life-threatening symptoms. Patients need to be encouraged to use this potential lifesaving self-treatment. Moderate to severe cases require admission.

Sampson HA et al.: Symposium on the definition and management of anaphylaxis: Summary report. *J Allergy Clin Immunol* 2005;115:584. [PMID: 15753908]

## Septic Shock

Septic shock is a clinical syndrome complicating infections caused by an exaggerated release of inflammatory mediators causing widespread organ dysfunction. The hallmark is the systemic inflammatory response syndrome (SIRS) (two or more of (1) temperature  $>38^{\circ}\text{C}$  or  $<36^{\circ}\text{C}$ , (2) HR  $>90$  bpm, (3) respiratory rate  $>20$  breaths per minute, or  $\text{PaCO}_2 <32$  (4) WBC  $>12,000$  cell/ $\text{mm}^3$  or  $<4000$  cell/ $\text{mm}^3$  or  $>10\%$  bands). In septic shock, SIRS is associated with decreased SVR with an early hyperdynamic compensation followed by impaired contractility from myocardial depressants and hypoxemia.

Gram-negative rods are the classic cause of septic shock but increasingly gram-positives and fungal infections contribute. The cause might also be a toxemia from staphylococcal or streptococcal infection. Importantly, up to 10% of patients have no known source of infection.

Patients with trauma, wounds, diabetes, extremes of age, and those whose immune systems are depressed by chemotherapy, cancer, or renal disease are at greatest risk. Menstruating females and patients with wounds are at risk for toxic shock syndrome caused by TSST 1 (toxic shock syndrome toxin 1) from tampons or from wounds. These patients have SIRS, hypotension, and an erythematous rash. Frequent occult sites of infection include the biliary tree, urinary tract, retroperitoneum, and perirectal areas. A lumbar puncture will reveal meningitis.

Treatment begins with the general principles outlined above. Patients are resuscitated, given goal-directed therapy, targeted antimicrobials, and, if present, drainage of any abscess. Early endotracheal intubation should be done to decrease the work of breathing and ensure oxygen delivery. Etomidate, which is often used in emergency intubations, should probably be avoided although data are scarce. Etomidate inhibits glucocorticoid synthesis, and some patients in septic shock have a relative adrenocortical insufficiency that might be theoretically worsened by etomidate.

Goal-directed therapy requires central venous and arterial monitoring, sedation with or without paralysis, optimization of central venous pressures (CVP) to 8–12 mm Hg first with fluids, optimization of mean arterial pressure (MAP) with fluid and vasopressors, and optimization of mixed venous oxygen content by initiation of vasopressor therapy or transfusion to a hematocrit  $>30$ . Dopamine is commonly used as the initial vasopressor but norepinephrine, with its alpha activity, might be a better choice. Vasopressin shows some promise, but data are limited.

Antibiotics should be given within the first hour of recognizing sepsis; appropriate cultures should be obtained prior to antibiotic administration, covering the most likely pathogens based on the most likely site and immune competence of the patient. A third or fourth generation cephalosporin is a reasonable choice for an immunocompetent patient. Anaerobic coverage is helpful in intra-abdominal infections and adding a macrolide for pneumonia is reasonable. In areas of high resistance, vancomycin is added. Immune incompetent patients require overlapping coverage for gram-positive and gram-negative aerobes and anaerobes and possibly viral or fungal causes.

Removal of any abscess or foreign body is important and might necessitate amputation, foreign body removal or incision, and drainage. High-dose steroids have been shown to be potentially harmful in septic shock, but increasing evidence show that some patients in septic shock have a degree of relative adrenal insufficiency and has prompted treatment with a stress dose of hydrocortisone (300 mg hydrocortisone/day) or a formal cosyntropin challenge.

Recombinant human-activated protein C (drotrecogin alfa) has been promising as an adjunctive treatment for septic shock in those with APACHE II scores  $>25$ , but at significant cost and risk of bleeding. Unfortunately, giving antiendotoxin antibodies or nitric oxide synthase inhibitors has not been shown to be effective.

Bernard GR et al.: Efficacy and safety of recombinant human activated protein C for severe sepsis. *N Engl J Med* 2001;344(10):699–709. [PMID: 1123677]

Rivers E et al.: Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med* 2001;345(19):1368–1377.

Stapeczynski JA: Septic Shock. In: *eMedicine Hypovolemic Shock*. Available at: [www.emedicine.com](http://www.emedicine.com).

Sibbald WJ, Mandel J: Management of severe sepsis and septic shock in adults. In: Rose BD (ed). *UpToDate*. UpToDate, Waltham, MA, 2007.

Kwan I, Bunn F, Roberts I: WHO Prehospital Trauma Care Steering Committee. Timing and volume of fluid administration for patients with bleeding. *Cochrane Database Sys Rev* 2003;(3):CD002245. [PMID: 12917926] (Review)

## Neurogenic Shock

Loss of vascular tone due to paralysis from a cervical cord spinal lesion can cause hypotension and shock. In trauma, however, any patient, even with paralysis, should be assumed to have an alternate source of hemorrhage before assigning the hypotension to neurogenic shock. Loss of feedback loops of autonomic ganglia cause less reflex tachycardia even in the face of hypotension. Neurogenic shock should not be confused with spinal shock, which is due to transient spinal cord dysfunction after injury manifest as loss of spinal reflexes such as the bulbocavernosus. Spinal shock clouds the prognosis from cord injury until spinal reflexes return.

Clinically, patients with neurogenic shock present with warm skin, hypotension (often marked if the patient is tilted in reverse Trendelenburg), and a variable tachycardia response (in a patient with a cervical spine level of injury). Treatment revolves around an aggressive evaluation of other potential causes of shock and includes a fluid challenge of  $20 \text{ cc/kg} \times 2$ . A reasonable endpoint is a mean arterial pressure  $> 90 \text{ mm Hg}$ . If volume replacement is unsuccessful, vasopressors with alpha activity should be given. The key point is to not assume that the cause is only neurogenic shock until all other sources of traumatic shock have been excluded.

## Drug-Induced Vasodilation

$\beta$ -Blockers and calcium channel blocker overdose or overuse can precipitate hypotension and shock. Hypotension and warm skin without any compensatory tachycardia is the hallmark of presentation. Glucagon 5–10 mg, IV, followed by an infusion of 2–5 mg/h will improve  $\beta$ -blocker toxicity and calcium channel blocker toxicity. Calcium gluconate 10% will improve calcium channel toxicity at a dose of 10–20 cc. Either cause of shock may require atropine or pacing. Of course, general decontamination with charcoal is helpful as well as fluid resuscitation.

## Obstructive Shock

Tension pneumothorax, pericardial tamponade, and massive pulmonary embolism are often termed *obstructive shock*, although all three impair ventricular filling and CO.

Tension pneumothorax is a clinical not radiographic diagnosis characterized by unilateral decreased breath sounds, unilateral chest hyperresonance, and tracheal deviation in the setting of respiratory distress and shock. Treatment is immediate needle decompression followed by chest tube thoracostomy placement.

Pericardial tamponade likewise should be considered early in the evaluation of undifferentiated shock. Patients with blunt or penetrating chest trauma can rapidly decompensate with minimal bleeding into the pericardium, while those with uremia and cancer usually develop an effusion over time. Symptoms include hypotension, elevated right side pressures (JVD) pulsus paradoxus (a fall in systolic blood pressure in inspiration), and Kussmaul's sign (increased jugular venous pressure on inspiration). Bedside U/S is extremely sensitive in detecting pericardial fluid and can be instrumental in guiding pericardiocentesis, although in the patient in extremis, blind pericardiocentesis might be lifesaving.

Massive pulmonary embolism presents as chest pain, syncope, tachypnea, and hypotension with signs of acute right ventricular overload with JVD and ECG changes. Fluid administration might worsen right ventricular failure and should be given only cautiously. Blood pressure should be augmented with an appropriate vasopressor such as norepinephrine 0.5–1  $\mu\text{g}/\text{min}$  titrated to response. Immediate surgical embolectomy is sometimes effective but not usually feasible. Shock complicating pulmonary embolism is an indication for thrombolytics if no other contraindication exists.

Gaieski D, Manaker S: General evaluation and differential diagnosis of shock in adults. In: Rose BD (ed). *UpToDate*. UpToDate, Waltham, MA, 2005.