Lesson 6

Undifferentiated Shock

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Objectives

On completion of this lesson, you should be able to:

1. Explain the pathophysiologic basis of shock.
2. List the common causes of shock.
3. Discuss the initial evaluation and stabilization of a patient with undifferentiated shock.
4. Explain the diagnostic strategies employed in the evaluation of undifferentiated shock.
5. Discuss treatment algorithms for the management of various shock states.

From the EM Model

1.0 Signs, Symptoms, and Presentations
1.3.42 Shock

Shock occurs when the circulatory system is unable to meet the body’s metabolic demand, ultimately leading to dysfunction of the vital organs and death. Despite recent medical advances, mortality for shock remains extremely high (50% in cardiogenic shock and 35% in septic shock), and untreated shock is almost uniformly fatal. The only variable consistently shown to improve outcomes in its management, regardless of cause, is time to identification and intervention. Shorter door-to-balloon times following myocardial infarction are associated with a decreased incidence of cardiogenic shock; early aggressive resuscitation and antibiotics are among the few interventions that can improve outcomes in patients with septic shock; and national standards stress the importance of the first hour of trauma management. The adept emergency physician must, therefore, be facile with the rapid recognition, diagnosis, and management of the multiple causes of the shocked state.

Case Presentations

Case One

A 59-year-old man is brought in by EMS. He awoke from sleep with chest pressure and shortness of breath. The emergency medical technician administered 325 mg of aspirin and a sublingual nitroglycerin tablet en route. The patient has a history of hypertension, a 10-pack/year history of smoking, and no allergies. On arrival, he is tachypneic, has coarse rales throughout both lungs, and has jugular venous distention (JVD) above the angle of the mandible. His skin is cool and diaphoretic; vital signs are blood pressure 85/42, heart rate 120, respiratory rate 32, and oxygen saturation 82%.

Case Two

A 64-year-old woman presents because of shortness of breath and fatigue. She awoke this morning with a feeling of suffocation and was unable to ambulate. She was recently diagnosed with breast cancer, but had been previously well. On arrival, she appears ill and is pale and diaphoretic. She has clear lung sounds, normal heart sounds, and no JVD. Her skin is cool to the touch, with weak distal pulses. Vital signs are blood pressure 84/56, heart rate 132, respiratory rate 26, temperature of 37.9°C (100.2°F), and oxygen saturation 88%. An ECG shows sinus tachycardia but is otherwise normal.

Case Three

A 32-year-old woman with no known medical history arrives via EMS following a motor vehicle collision. The patient was a restrained passenger in a vehicle traveling approximately 55 miles per hour when it hit a tree head on. The patient arrives on a stretcher with a cervical spine collar in place. Additional medical history is unobtainable. She is awake but confused, with a Glasgow Coma Scale score of 9 (EVM 2-3-4). Breath sounds are shallow but clear to auscultation bilaterally, and distal pulses are intact. Vital signs are blood pressure 78/46, heart rate 65,
Shock

Shock is a systemic circulatory insufficiency that results in an imbalance of tissue oxygen supply and demand. When circulating oxygen content does not meet tissue demand, a cascade of metabolic sequelae is triggered that ultimately results in cellular injury. This cellular cascade causes an ion imbalance, resulting in an overload of intracellular calcium. Toxic levels of cellular calcium induce free radical oxidative damage and impede mitochondrial ATP synthesis, muscle relaxation, and cardiac myocyte contractility. Acidification of the serum from anaerobic metabolism, lactic acid production, and renal failure further increase this calcium overload. In addition, acidosis independently diminishes skeletal and cardiac muscle contractility, decreases the effectiveness of circulating catecholamines, increases interstitial edema, and eventually results in cell membrane disruption and death. In specific types of shock, pro-inflammatory cytokine release causes cellular dysfunction and endothelial cell activation, producing nitric oxide and leading to generalized vasodilation.

The primary compensatory mechanism for the shock state is an increase in cardiac output via catecholamine and cortisol release. Selective arteriolar vasoconstriction diverts blood away from the skin, skeletal muscles, kidneys, and splanchnic organs, while constriction of venous capacitance vessels increases cardiac preload and focuses oxygen delivery to the heart and brain. The release of antidiuretic hormones and activation of the renin-angiotensin axis increase sodium and water reabsorption in an attempt to optimize intravascular volume. However – given sufficient stress – the system fails, resulting in acidosis, coagulopathy, inflammatory mediator release, and ultimately multiple organ dysfunction and death.

The differential diagnosis for undifferentiated shock can be broken down into four broad classifications: hypovolemic, cardiogenic, obstructive, and distributive. Understanding the pathophysiologic differences between these groups is critical to proper and timely management.

Hypovolemic shock results from a physiologic fluid status that is insufficient to adequately perfuse the body. This fluid deficit most commonly is caused by hemorrhage, ruptured abdominal aortic aneurysm, or ectopic pregnancy. However, hypovolemia also can result from gastrointestinal losses, as seen in severe vomiting and diarrhea. Although septic shock generally occurs through the distributive mechanism described below, it is important to note that cytokine release can cause capillary leakage into the interstitial space, resulting in a relative decrease in intravascular volume.

When shock occurs because of an intrinsic defect of the heart itself, it is classified as cardiogenic. Cardiogenic shock, defined as hypotension in the setting of a decreased cardiac index (<1.8 L·min⁻¹·m⁻²) and increased left heart filling pressures, occurs when there is insufficient cardiac output to adequately maintain blood pressure and tissue perfusion. Myocardial infarction; cardiac contusion; myocarditis; arrhythmia; heart failure; and toxins, including certain medications, can all result in cardiogenic shock. Mechanical causes such as valvular rupture or ventricular wall rupture are particularly devastating.

Obstructive shock occurs as a result of the physical obstruction of the heart or great vessels. Although obstructive shock often is categorized as a subset of cardiogenic shock, it is useful to think of the two as distinct entities. Obstructive shock occurs as the direct result of impaired diastolic filling or significantly increased cardiac afterload. Both cardiac tamponade and tension pneumothorax directly compromise diastolic filling, albeit by different mechanisms. Massive pulmonary artery embolism can impede right ventricular output, resulting in a significant increase in afterload; and right ventricular bowing into the left ventricle impedes left ventricular diastolic filling. Both result in decreased cardiac output.

Distributive shock occurs when there is normal circulating intravascular volume, but the capacity of the vascular system has increased secondary to generalized vasodilation. In the case of septic shock, a severe systemic inflammatory response is triggered, leading to diffuse peripheral vasodilation and a drop in cardiac afterload. Anaphylactic shock is the result of uncontrolled mast cell degranulation and vasodilatory cytokine release. Neurogenic shock occurs when a traumatic high spinal cord injury disrupts the sympathetic chain of the autonomic nervous system, resulting in a loss of vascular tone.
This type of shock often presents with a paradoxical bradycardia due to the now unopposed vagal stimulation of the heart.7

Finally, it should be understood that a clinical presentation of undifferentiated shock can be associated with multiple categories of shock. For example, a trauma patient with massive hemotherax might have a combination of obstructive shock from tamponade and the mechanics of thoracic tension, in addition to hypovolemic shock secondary to hemorrhage.

**CRITICAL DECISION**
What are the key components of a patient’s history in the evaluation of undifferentiated shock?

In patients with undifferentiated shock, attention should immediately focus on stabilization of the patient’s airway, breathing, and circulation. Although a thorough history can provide important information, this should occur only after, or in tandem with, initial stabilization.

Collateral history from EMS personnel or family members can be invaluable, especially in an altered or confused patient. A complete medication history should be obtained, including information on any new or changed medications. Particular attention should be given to any anticoagulants, diuretics, drugs of abuse, antihypertensive medications, or antiarrhythmic agents. A history of allergies also should be obtained.

Often, symptoms such as lightheadedness, weakness, palpitations, fatigue, and decreased urine output are associated with impending shock, but many of these findings are nonspecific. Recent profuse vomiting, diarrhea, or decreased oral intake can indicate hypovolemia. The patient should be questioned regarding recent melena, hematochezia, or hematemesis. Chest pain, increased dyspnea on exertion, orthopnea, and worsening volume overload should raise concern for a cardiac cause. Risk factors for pulmonary embolus should be elicited and any recent trauma should be addressed. Additionally, elicit any exposure to potential toxins, including but not limited to home medications, pesticides, toxic alcohols, and cyanide.

Patients in early sepsis can present with vague symptoms, including fevers, chills, and myalgias. They should be questioned further about symptoms such as cough or abdominal pain, which can help pinpoint infection. It also is important to gather information on any indwelling catheters, new rashes, retained tampons or other foreign bodies, recent operations, or other immunocompromising conditions.

**CRITICAL DECISION**
What are the critical physical examination maneuvers in the differentiation of various shock states?

**Vital Sign Evaluation**

Even though vital signs cannot consistently identify shock, it is essential to evaluate them early. Tachycardia is an appropriate compensatory response to the shocked state, but significant tachycardia, including cardiac arrhythmias, can itself result in decreased end diastolic volumes and decreased cardiac output. Likewise, significant bradycardia also can result in decreased cardiac output and systemic hypoperfusion. It should be noted that while vital signs can be useful in the initial identification of shock, there often is a poor correlation between blood pressure, heart rate, and cardiac output in severely hypotensive patients.11

Hypotension frequently is a late and concerning finding in shock. Blood pressures should be measured often using an appropriately sized blood pressure cuff. Although hypotension is commonly found, it is not always present, particularly in early shock.3 In the case of hemorrhagic shock, there seems to be significant variability in the relationship between blood loss and the clinical signs of hypoperfusion,12

and the clinical utility of the often-cited ATLS classification of hemorrhage shock table6 has been disputed.13 The shock index, defined as heart rate divided by systolic blood pressure, might be of more help in predicting disease severity.12,14,15 A shock index of 0.9 or greater is concerning and should prompt aggressive management.

A point-of-care blood glucose analysis often is included in the initial set of vital signs. Hyperglycemic crises and severe hypoglycemia should be identified and addressed promptly. Pulse oximetry (SpO2) monitoring, providing an estimate of circulating oxygen, also is a routine practice in the emergency department. A low SpO2 generally indicates hypoxemia or hypoventilation, which should be promptly addressed. Alternative devices, including transcutaneous oxygen and carbon dioxide monitoring16 and infrared spectroscopy17,18 have become increasingly available. However, while these devices show promise, existing data is insufficient to recommend their routine use.

The patient’s core body temperature also can yield valuable information. Both hyperthermia and hypothermia suggest a systemic inflammatory response. Environmental factors leading to either can, in and of themselves, lead to shock. Both hypothermia and hyperthermia have been shown to result in significantly increased mortality,18 and steps should be taken to re-establish a normal body temperature immediately. Taken together, a temperature higher than 38°C (100.4°F) or lower than 36°C (96.8°F), a heart rate faster than 90 beats per minute, and a respiratory rate of more than 20 breaths per minute make up three of the four clinical parameters defining the systemic inflammatory response syndrome (SIRS). The fourth, a white blood cell count above 12,000 or less than 4,000 per mm3 or with more than 10% bands, is often measured in the patient’s laboratory evaluation.
The Physical Examination

The early diagnosis of shock is critical in reducing patient morbidity; however, recognition can be particularly difficult in its earliest stages. As with any patient who presents to the emergency department, a primary survey should be performed immediately on presentation. After evaluating patency and integrity of the airway, the primary survey should include auscultation of the lungs for appropriate ventilation and the presence of bilateral lung sounds. Any signs of overt hemorrhage should be immediately addressed, and bleeding controlled. Direct pressure should be applied if possible. In patients with severe limb hemorrhage, a tourniquet can be used if direct pressure is impossible or impractical. Tourniquets should be placed proximal to the injury and should be tightened to a pressure sufficient to stop hemorrhage. Tourniquet times of up to 2 hours are considered safe.19,20

After the primary survey, the physician should perform a diligent head-to-toe physical examination. Mental status often is diminished in shock because of cerebral hypoperfusion. This can range from mild disorientation to a profoundly altered mental state and loss of airway reflexes. Conjunctival pallor can indicate anemia from internal hemorrhage, and dry mucous membranes indicate hypovolemia. The neck veins should be examined for jugular venous distention, indicating increased right atrial pressures. The Kussmaul sign, a paradoxical increase in jugular venous pressure during inspiration, can indicate obstructive or cardiogenic shock.

Findings such as absent breath sounds over one lung suggest a tension pneumothorax, and a new cardiac murmur raises the possibility of valvular rupture or septic shock from infective endocarditis. Pulsus paradoxus, a paradoxical decrease in blood pressure during inspiration, or muffled heart sounds might indicate cardiac tamponade, but these findings are not of sufficient sensitivity to make a definitive diagnosis.21

The abdomen should be carefully palpated for signs of peritonitis, and the abdominal wall inspected for any signs of ecchymosis. An abdominal seatbelt mark should be searched for in cases of motor vehicle collisions. Edema and bruising surrounding the umbilicus (Cullen sign) or ecchymosis of the flanks (Grey Turner sign) might indicate an intra-abdominal catastrophe such as hemorrhagic pancreatitis, ruptured spleen, ruptured ectopic pregnancy, or a ruptured aneurysm of the abdominal aorta.22 Although a palpable mass in the mid-abdomen can suggest an aortic aneurysm, this finding is difficult to recognize in most patients.

The extremities also should be evaluated. Extremity paralysis can indicate a neurogenic etiology. Unilateral leg swelling, indicating venous thrombosis, can suggest obstructive shock from a pulmonary embolus. Delayed capillary refill time can indicate hypoperfusion, but typically is not an early finding in shock and can be found at baseline in the elderly and those with peripheral vascular disease. Bilateral peripheral edema might suggest a cardiogenic cause of shock. Cool peripheral skin temperature can indicate shock; however, there is poor correlation between peripheral temperature and cardiac index or systemic vascular resistance.23 Further, patients with distributive shock frequently will present with normally perfused extremities. A passive leg raise, transiently increasing venous return, is an easy bedside maneuver that can predict fluid responsiveness.24

CRITICAL DECISION
What additional evaluations, including laboratory studies and imaging, assist in differentiating the cause of the shocked state?

In addition to the physical examination, additional diagnostic measures help to differentiate the etiology of shock. These include the laboratory evaluation, ECG, radiographic imaging, ultrasonography, and advanced hemodynamic monitoring.

Laboratory Investigations

Serum analysis can yield valuable information about the cause and severity of the shock state. A point-of-care blood glucose analysis should be performed immediately in all patients presenting with undifferentiated shock, and a pregnancy test should be obtained in all women of childbearing age. In addition, a metabolic profile and CBC should be obtained. Cardiac biomarkers can prove useful, and blood cultures should be drawn if there is suspicion of a systemic infection. Likewise, a urine analysis and culture should be obtained. If a toxic ingestion for which a serum assay exists is suspected, this should also be tested. Blood should be tested for type and crossmatch in the case of suspected hemorrhagic shock. A coagulation profile should be considered, particularly if the patient is taking anticoagulants, and can have a role in estimating the severity of illness. Specific serum markers also should be considered and targeted to specific suspected endocrine emergencies, including a cortisol level in suspected adrenal crisis and thyroid stimulating hormone and T3 and T4 levels in suspected thyrotoxicosis or myxedema.

If the patient is in respiratory distress, a blood gas analysis can be useful. Venous blood gas analysis – less invasive than arterial blood gas analysis – provides an excellent correlation to arterial values for pH, bicarbonate, and partial pressure of carbon dioxide (PCO2)23,20 and is the preferred initial technique, especially if the patient does not have any acute oxygenation or ventilation issues.

The mixed-venous oxygen saturation (Svo2), obtained via a central venous catheter, or central venous oxygen saturation (Scvo2), obtained via a pulmonary artery catheter, also can help guide resuscitative therapy in septic shock.27
An ScvO₂ value above 70% or an Svo₂ value above 65% is desirable; failure to meet these goals despite adequate fluid resuscitation should prompt the consideration of vasoactive agents or blood transfusion.²⁷ However, such analysis requires the placement of an invasive catheter that otherwise might not be indicated. Further, recent studies have suggested that improvement in serum lactate levels might be equivalent to invasive measures in guiding resuscitative efforts in septic shock.²⁸

An elevated serum lactate indicates an overall shock state, and a higher lactate level suggests a sicker patient with a higher risk of death.²⁸,³⁰ A serum lactate level can be useful as an indicator of overall illness severity, but its use in septic shock has been the most extensively evaluated. Lactate should be measured, and any result above normal should be aggressively addressed, with particular concern for patients with a level above 4 mmol/L.²⁷ Although guidelines recommend reducing lactate to normal levels within 6 hours, a reduction of 10% in 2 hours is an alternative strategy.²⁸ The approach to achieving this target will depend on specific patient parameters, but will generally include fluid resuscitation and administration of vasoactive agents or blood products, as discussed below.

**Electrocardiography**

The ECG can provide important information regarding the cause of shock. ST-segment elevations indicate complete coronary artery occlusion and resultant cardiogenic shock. Evidence of right heart strain, including negative T waves in the lateral precordial leads or right bundle-branch blocks, or the finding of an S₁Q₃T₃ or S₁S₂S₃ pattern can indicate pulmonary artery obstruction from pulmonary embolism or critical pulmonary hypertension. Electrical alternans, despite having a low sensitivity,³¹ suggests cardiac tamponade. The ECG also can help identify toxic or metabolic derangements. A wide QRS complex with a terminal R wave is suggestive of a sodium channel-blocker overdose such as with cyclic antidepressants.³¹ Bradycardia in the setting of a wide QRS or a long QT suggests electrolyte abnormalities, and a new heart block suggests myocardial infarction or ingestion of an antiarrhythmic nodal blocking agent.

**Plain Radiography**

Plain radiography can be useful in certain situations, but should not delay empiric intervention in a hemodynamically unstable patient. Chest radiographs can reveal a pneumothorax or hemothorax; pulmonary edema is suggestive of cardiogenic shock. A wide mediastinum (generally wider than 8 cm) suggests an aortic dissection, and evidence of pneumonia suggests a source of septic shock.

**Ultrasonography**

Ultrasonography is a fast, simple, and effective means of obtaining vital information at the bedside.³² Two protocols developed to aid in the differentiation of the shock state are the abdominal and cardiac evaluation with sonography in shock (ACES) examination and the rapid ultrasonography for shock and hypotension (RUSH)³⁴ examination. These protocols take slightly different approaches, but focus on the same general principles. The heart is evaluated for adequate wall motion; right ventricular bowing, suggesting elevated right ventricular pressures from an obstructive pathology; and evidence of diastolic right ventricular collapse, suggesting cardiac tamponade. A hyperdynamic left ventricle generally is seen in hypovolemia or in the setting of vasoactive medications. A hypodynamic ventricle often points to a cardiac cause of shock, toxins, or sepsis. The inferior vena cava (IVC) is evaluated for diameter and collapse as an indicator of fluid tolerance. The IVC diameter is measured at 2 to 3 cm from the right atrial border on both inspiration and expiration; collapse of 50% or more suggests fluid tolerance.³⁵ Then, the abdomen is evaluated for free fluid. Although as little as 200 mL of intra-abdominal free fluid can be seen using ultrasonography,³⁶ the ability of ultrasound to identify free fluid in the retroperitoneal space is limited. The aorta should be scanned along its course to the bifurcation, and measurements taken in both long and short axis. A diameter of more than 3 cm is concerning for aneurysm.³⁷ Finally, the lungs are examined for pneumothorax or pleural fluid.

This series of evaluations easily can be remembered with the pneumonic HIMAP – heart, IVC, Morison pouch, aorta, and pneumothorax.³⁴ The RUSH examination adds an evaluation for deep vein thrombosis and groups the examinations into three domains – the “tank,” the “pump,” and the “pipes,” which refer to volume status, the heart, and vascular tone, respectively.

Ultrasonography of the lungs also can be a useful adjunct by identifying B-lines that indicate pulmonary edema, which suggests cardiogenic shock. If seen during empiric fluid loading of the hypotensive patient, B-lines can indicate that the patient is no longer fluid tolerant.³⁹ Lung ultrasonography is superior to plain films in identifying pneumothoraces.⁴⁰

In cases of suspected trauma, the focused abdominal sonography for trauma (FAST) examination is useful in identifying intra-abdominal hemorrhage.⁴¹ This examination includes views of the Morison pouch in the right upper quadrant, the splenorenal space in the left upper quadrant, suprapubic views of the pelvis, and a subxyphoid cardiac view. Although the FAST examination is useful in the evaluation of hypotensive trauma patients, the protocols outlined above offer a more complete evaluation in the case of undifferentiated shock.
Advanced Hemodynamic Monitoring

Intravascular volume status, cardiac output, tissue perfusion, and overall hemodynamic status can be assessed using advanced hemodynamic monitoring, although many of these methods are controversial. The gold standard of hemodynamic monitoring is the Swan-Ganz catheter; however, its use should be restricted to experienced practitioners in ICUs and in highly selected populations.42

Invasive blood pressure monitoring should be considered in the management of severe shock, particularly with concomitant vasopressor use or at the extremes of blood pressure measurements.43

Invasive blood pressure monitoring also should be considered if peripheral blood pressure readings are inaccurate or difficult to obtain. Central venous pressure (CVP), obtained via pressure transduction from a central venous catheter, also is frequently monitored in the critically ill. Recently, however, CVP has fallen out of favor as an accurate estimate of fluid status in the emergency department.44 The use of ultrasonography to measure the diameter and collapsibility of the inferior vena cava provides an alternative method for assessing intravascular fluid status and fluid responsiveness,32 particularly at the extremes of distention or collapsibility. Sonography also can be used to estimate fluid responsiveness through the measurement of brachial artery peak velocity, in which the peak velocity of blood flow through the brachial artery is measured by Doppler ultrasound during inspiration as compared to expiration. However, accurate measurement requires a mechanically ventilated patient.45

CRITICAL DECISION
What are the important principles in treating the various etiologies of shock?

A patient with suspected shock should undergo a primary survey immediately on arrival, including evaluation of the airway and hemodynamic status. The patient should be promptly moved to the resuscitation area and placed on

<table>
<thead>
<tr>
<th>Table 1: Recommended Empiric Antibiotic Regimens for Patients with Severe Sepsis and Septic Shock Temporary</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Source</strong></td>
</tr>
</tbody>
</table>
| **Skin and soft tissue** (Impetigo, cellulitis, erysipelas, necrotizing fasciitis) | - *S. aureus*, *S. pyogenes*, Strep species | - Strep and MSSA coverage:  
  - Nafcillin, 2 g IV q 24 hrs  
  - Cefazolin, 1 g IV q 8 hrs  
  - Ceftriaxone, 1-2 g IV q 24 hrs  
  - Cefotaxime, 1-2 g IV q 8 hrs  
  - Clindamycin, 600 mg IV q 8 hrs |
|  | - Consider *Vibrio vulnificus*, *Aeromonas hydrophila*, anaerobic streptococci, *clostridia*, polymicrobial for necrotizing infections | - MRSA coverage:  
  - Clindamycin, 600 mg IV q 8 hrs  
  - Linezolid, 600 mg IV q 12 hrs  
  - Vancomycin, 15 mg/kg IV q 12 hrs |
|  |  | - Necrotizing infections:  
  - Imipenem-cilastatin, 1g IV q 6-8 hrs  
  - Meropenem, 1g IV q 8 hrs  
  - Ertapenem, 1g IV q 24 hrs  
  - Ampicillin-sulbactam, 1.5-3g IV q 6-8 hrs OR piperacillin-tazobactam 3.375g IV q 6-8 hrs PLUS clindamycin, 600 mg IV q 8 hrs PLUS ciprofloxacin, 400 mg IV q 12 hrs  
  - Cefotaxime, 2 g IV q 6 hrs PLUS metronidazole 50 mg IV q 6 hrs OR clindamycin, 600 mg IV q 8 hrs |
| **Pneumonia** [Community-acquired pneumonia (CAP), healthcare-associated pneumonia (HCAP)] | - CAP: *S. pneumoniae*, *Mycoplasma pneumoniae*, *Haemophilus influenzae*, *Chlamydophila pneumoniae*, *Legionella*, viruses  
  - HCAP: *Pseudomonas*, *E.coli*, *Klebsiella pneumoniae*, *Acinetobacter*, MRSA | - CAP coverage:  
  - Respiratory fluoroquinolone  
  - Beta-lactam PLUS macrolide  
  - Beta-lactam PLUS doxycycline  
  - HCAP coverage:  
  - Antipseudomonal cephalosporin OR antipseudomonal carbapenem OR beta-lactam/beta-lactamase inhibitor PLUS  
  - Antipseudomonal fluoroquinolone OR aminoglycoside PLUS  
  - MRSA coverage (linezolid or vancomycin) |

*Note: All necrotizing infections must include MRSA coverage.*
a cardiac monitor. Intravenous access should be obtained rapidly with large-bore catheters (14- to 18-gauge). Intraosseous access is a viable alternative if intravenous access is unobtainable. Although the tibial insertion site might be more familiar, a humeral insertion provides an alternative location with similar infusion capability; infusion rates of more than 5 liters per hour in an adult patient are possible. Ventilatory support might be necessary in the event of significant respiratory distress or arrest. Noninvasive positive-pressure ventilation has been used successfully for a variety of conditions, particularly in cardiogenic pulmonary edema. However, invasive ventilation via endotracheal tube might become necessary in the hypotensive patient, especially in cases of decreased mental status. It should be noted, however, that increased intrathoracic pressures in mechanical ventilation can decrease cardiac output and potentially worsen hypotension, particularly in patients requiring high end-expiratory pressures.

The choice of medication used for intubation in the shocked patient also warrants consideration. In a patient with suspected septic shock, etomidate can cause a decrease in circulating cortisol levels, although this has not been shown to have a measurable effect on mortality rates and should not preclude the use of etomidate as a sedative agent. In the hypotensive patient, a decreased dose of sedative should be used, and strong consideration should be given to the use of ketamine because of its sympathetic stimulation. The chosen paralytic dose should be increased (time to onset of action escalates in the setting of hypoperfusion).

After initial stabilization, the patient often requires empiric management before a definitive

**Table 1 (Continued).**
Recommended Empiric Antibiotic Regimens for Patients with Severe Sepsis and Septic Shock

<table>
<thead>
<tr>
<th>CNS (Meningitis, encephalitis)</th>
<th>0-3 month: <em>E. coli</em>, Group B <em>Strep, Listeria</em></th>
<th>0-3 months: <em>Cefotaxime</em>, 50 mg/kg q 8 hrs <strong>PLUS</strong> ampicillin, 50 mg/kg q 8 hrs</th>
<th>0-3 months: <em>E. coli</em>, Group B <em>Strep, Listeria</em></th>
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<tr>
<td></td>
<td>Adult: <em>S. pneumoniae, N. meningitidis</em>, Listeria (&gt;50 yo)</td>
<td>Adult: <em>Ceftiraxone</em>, 2 g IV q 12 hrs <strong>PLUS</strong> vancomycin, 500-750 mg IV q 6 hrs</td>
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<td>Adult: <em>Ampicillin</em>, 2 g IV q 4 hrs for listeria coverage if &gt;50 years old</td>
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<td></td>
<td></td>
<td><em>Consider acyclovir</em></td>
<td><em>Consider acyclovir</em></td>
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<td>Genitourinary (UTI, pyelonephritis)</td>
<td><em>E. coli, Proteus mirabilis, Klebsiella pneumoniae, Staphylococcus saprophyticus</em></td>
<td><em>Ciprofloxacin, 400 mg IV q 12 hrs</em></td>
<td><em>Ciprofloxacin, 400 mg IV q 12 hrs</em></td>
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<td><em>Levofoxacin, 750 mg IV q 24 hrs</em></td>
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<td><em>Ampicillin, 2 g IV q 6 hrs <strong>PLUS</strong> Gentamicin 2 mg/kg load THEN 1.7-2 mg/kg q 8 hrs</em></td>
<td><em>Ampicillin, 2 g IV q 6 hrs <strong>PLUS</strong> Gentamicin 2 mg/kg load THEN 1.7-2 mg/kg q 8 hrs</em></td>
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<td><em>Ampicillin-sublactam, 3 g IV q 6 hrs</em></td>
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<td><em>Extended spectrum cephalosporin</em></td>
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<td><em>Carbapenem</em></td>
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<td>Intra-abdominal (Biliary, appendicitis, diverticulitis, colitis, abscess, spontaneous bacterial peritonitis)</td>
<td><em>E. coli, Strep species, Klebsiella species, Pseudomonas, Proteus, Clostridium species, Acinetobacter, Staphylococcus aureus, Enterobacteriaceae, Bacteroides fragilis, anaerobes</em></td>
<td><em>Single agent:</em></td>
<td><em>Single agent:</em></td>
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<td><em>Enterococcus: coverage recommended for severe infections</em></td>
<td><em>Imipenem-cilastatin 250-500 mg IV q 6-8 hrs</em></td>
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<td><em>Meropenem 1g IV q 8 hrs</em></td>
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<td><em>Piperacillin-tazobactam 4.5 g IV q 8 hrs</em></td>
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<td><em>Combination therapy:</em></td>
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<td><em>Ciprofloxacin, 400 mg IV q 12 hrs</em></td>
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<td><em>Levofoxacin, 750 mg IV q 24 hrs</em></td>
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<td><em>Ampicillin, 2 g IV q 6 hrs <strong>PLUS</strong> Gentamicin 2 mg/kg load THEN 1.7-2 mg/kg q 8 hrs</em></td>
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<td></td>
<td><em>Carbapenem</em></td>
<td><em>Carbapenem</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Metronidazole 500 mg IV q 6-8 hrs</em></td>
<td><em>Metronidazole 500 mg IV q 6-8 hrs</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Add vancomycin if concern for MRSA</em></td>
<td><em>Add vancomycin if concern for MRSA</em></td>
</tr>
<tr>
<td>Without an obvious source</td>
<td>*Children &gt;3 months: <em>S. pneumonia, N. meningitidis, S. aureus, H. influenza</em></td>
<td><em>Children &gt;3 months:</em></td>
<td><em>Children &gt;3 months:</em></td>
</tr>
<tr>
<td></td>
<td><em>Adults: Gram-negative bacilli, S. aureus, streptococci, others</em></td>
<td><em>Ceftiraxone, 75-100 mg/kg IV q 24 hrs</em></td>
<td><em>Ceftiraxone, 75-100 mg/kg IV q 24 hrs</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Neutropenic adults:</em></td>
<td><em>Neutropenic adults:</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Vancomycin, 1 g IV q 12 hrs <strong>PLUS</strong></em></td>
<td><em>Vancomycin, 1 g IV q 12 hrs <strong>PLUS</strong></em></td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Imipenem, 1 g IV q 8 hrs OR cefepime, 2 g IV q 8 hrs OR ceftazidime, 3 g IV q 6 hrs</em></td>
<td><em>Imipenem, 1 g IV q 8 hrs OR cefepime, 2 g IV q 8 hrs OR ceftazidime, 3 g IV q 6 hrs</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>+/- aminoglykoside</em></td>
<td><em>+/- aminoglykoside</em></td>
</tr>
</tbody>
</table>

*Dosing is based on a typical 70-kg patient with normal renal function.*
diagnosis is made. Interventions should be made based on the likelihood of the particular cause of shock. When the history, physical examination, or ancillary testing suggests a specific toxidrome, targeted antidotes should be provided. Suspected adrenal insufficiency initially should be treated with dexamethasone, as it does not interfere with subsequent evaluation. In the case of known adrenal insufficiency, dexamethasone or hydrocortisone can be administered.

Patients with a cardiac cause of shock require treatment depending upon the suspected condition. If the ECG demonstrates ST-segment elevations consistent with STEMI, the patient should be taken immediately for cardiac revascularization or given thrombolytic therapy. Emergency department interventions should not delay revascularization. If a massive pulmonary embolus is suspected, immediate thrombolysis is warranted in a hemodynamically unstable patient, although a definitive diagnosis is preferred if the patient's clinical condition allows. The most well-described adult dosing regimen is alteplase, 10-mg bolus, followed by 90 mg over the subsequent 100 minutes, in addition to heparin; however, other agents and dosing regimens have been used.

If there is evidence of significant ongoing hemorrhage, blood products should be promptly transfused in accordance with institutional protocol. Hypotensive resuscitation has demonstrated benefits in patients with hemorrhagic shock. Patients should receive blood and blood products to maintain physiologic parameters of adequate perfusion, but should not be resuscitated to normal arterial blood pressures. Although typed and crossmatched blood products are preferred, uncrossmatched blood should be administered until crossmatched products are available. If the patient is taking any anticoagulant medications, these agents should be reversed.

Some circumstances require prompt invasive or surgical interventions. Needle thoracostomy can provide temporary decompression of a tension pneumothorax. When pleural space cannot be reached with standard catheters, tube thoracostomy should be performed in all patients with suspected tension physiology. Pericardiocentesis is indicated when cardiac tamponade is confirmed by bedside echocardiography. Bradycardia can necessitate transcutaneous pacing or placement of a transvenous cardiac pacing wire, depending on the cause. An identifiable, surgically correctable source of septic shock should be addressed promptly, including incision or débridement of soft tissue infections, laparotomy for bowel perforation or ischemia, and washout of infected joints.

In a patient with presumed sepsis, broad-spectrum antibiotics should be administered as early as possible, preferably within the first hour (Table 1). Empiric treatment should be initiated against all suspected pathogens – be they bacterial, viral, or fungal. Combination drug therapy is strongly encouraged for patients who

Table 2.
A Summary of Cardio- and Vasoactive Medications

<table>
<thead>
<tr>
<th>Agent</th>
<th>Receptor of Action</th>
<th>Dosing</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dobutamine</td>
<td>β1 &gt;&gt; β2</td>
<td>5 - 40 mcg/kg/min</td>
<td>Strong inotropy, moderate chronotropy; mild vasodilation; increased myocardial oxygen demand. Arrhythmogenic.</td>
</tr>
<tr>
<td>Dopamine</td>
<td>Low: DA Moderate: β1 High: α1</td>
<td>2 – 20 mcg/kg/min</td>
<td>Norepinephrine precursor; positive inotropy and chronotropy at low to moderate doses; vasoconstriction at higher doses</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>Low: β1, β2 High: α1</td>
<td>1 - 10 mcg/min Push dose: 5 – 20 mcg every 2 – 5 min</td>
<td>Strong inotropy and chronotropy; potential to induce myocardial infarction</td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>β1, β2</td>
<td>2 – 10 mcg/min</td>
<td>Strong inotropy and chronotropy with minimal effect on vasculature; some decrease in systemic vascular resistance; neutral cardiac output</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>α1 &gt;&gt; β1, β2</td>
<td>2 - 30 mcg/min</td>
<td>Significant vasoconstriction, minimal chronotropy; possible reflex bradycardia; potential to induce tachyarrhythmia and myocardial infarction</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>α1</td>
<td>40-200 mcg/min Push dose: 50 – 200 mcg every 2 – 5 min</td>
<td>Vasopressor with minimal cardiac effect; potential to cause significant reflex bradycardia</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>V1, V2</td>
<td>0.01 - 0.04 units/min</td>
<td>Minimal coronary and cerebral vasoconstriction; dose-dependent increase in systemic vascular resistance; preservation of effect in acidosis</td>
</tr>
</tbody>
</table>
are critically ill or neutropenic, or if there is a possibility for multidrug-resistant pathogens. At least two sets of blood cultures should be obtained prior to antimicrobial administration, provided the testing does not delay treatment. Anti-infective therapy should be narrowed as soon as susceptible pathogens are isolated.\(^{27}\)

Fluids should be administered liberally, guided by appropriate measures of fluid responsiveness as discussed above. Although limited data supports the preferential use of either Ringer’s lactate or normal saline, large volumes of normal saline can cause a hyperchloremic (nonanion gap) metabolic acidosis. In the case of hemorrhagic shock, packed red blood cells should be administered in lieu of crystalloid. The use of colloid solutions offers no additional benefit and should be avoided.\(^{58}\)

Vasoactive agents might be necessary based on the hemodynamic profile of the presumed cause of the shock state (Table 2). In the case of anaphylactic shock, for example, the drug of choice is epinephrine; and in cardiogenic shock, the addition of dobutamine to agents such as norepinephrine can be beneficial. Dopamine has an increasingly limited role in the management of shock and should be used with caution.\(^{59}\)

The lowest possible vasopressor dose should be used to maintain adequate perfusion. Patients with neurogenic shock often will require blood pressure support; however, the associated bradycardia need not be treated.

**Case Resolutions**

**Case One**

Immediately on arrival, the patient who awoke with chest pressure and shortness of breath was placed on noninvasive positive-pressure ventilation, and a cardiac monitor was applied. An ECG showed ST-segment elevations in the inferior leads with reciprocal depressions. The cardiac catheterization team was activated; and while the cardiac lab was being prepared, bedside echocardiography was performed, showing focal hypokinesis and poor contractility. Despite some improvement in oxygen saturation, the patient ultimately required endotracheal intubation. A central line was placed, and the patient was started on norepinephrine and dobutamine. In the cardiac catheterization lab, a right coronary artery occlusion was identified and stented. An intra-aortic balloon pump temporarily was placed for hemodynamic support. The patient recovered well in the ICU and was discharged several days later.

**Case Two**

The patient in this case received supplemental oxygen via nasal cannula, and her oxygen saturation improved to 94%. She was placed on a cardiac monitor, intravenous access was obtained, and a bolus of normal saline was administered. Bedside ultrasonography revealed a distended IVC without respiratory variation. No free fluid was seen in the abdomen, and the abdominal aorta was of normal caliber. No appreciable pneumothorax was identified. Echocardiography demonstrated no pericardial effusion, but the right ventricle was noted to be distended with a bowed septum and there was poor cardiac contractility. Repeat blood pressure was 60/42, and the patient’s mental status began to deteriorate. A central venous catheter was placed and norepinephrine was administered. After discussion with the family and an evaluation for contraindications, systemic thrombolytic agents were given. The patient’s hemodynamic status eventually improved and thoracic computed tomography (CT) angiography confirmed the diagnosis of pulmonary embolus.

**Case Three**

In the case of the woman injured in a motor vehicle collision, a second large-bore intravenous line was placed and the institutional massive transfusion protocol was initiated for presumed internal hemorrhage. The patient remained difficult to arouse and was intubated for airway protection. Bedside ultrasonography revealed no evidence of pneumothorax, pericardial effusion, or free fluid in the peritoneum. Blood was transfused with a goal mean arterial pressure of 55 to 65, while preparations were made to transport her to radiology for a CT scan. ECG, chest radiograph, and pelvis radiograph in the trauma bay were all unremarkable. The patient maintained her blood pressure through the CT scan, which revealed an unstable C3-C4 cervical spinal fracture. The patient remained persistently hypotensive despite fluid resuscitation. Bedside ultrasound of the IVC did not show any respiratory variation in vessel diameter, and she was given a vasopressor infusion prior to being taken to the operating room for neurosurgical intervention.

**Summary**

Shock is a common presentation that is associated with high morbidity and mortality. Undifferentiated shock requires rapid identification and management, often empirically, and treatment should be tailored to the suspected cause. Several diagnostic tools can help determine the cause of the shocked state, complementing the history and physical examination. Limited bedside ultrasonography has an ever-increasing role in the emergency department and should be used liberally. Serum lactate levels have shown continued utility, while noninvasive measures of fluid responsiveness and tolerance are rapidly usurping the use of central venous pressure measurements. Undifferentiated shock can provide a substantial diagnostic dilemma, but diligent evaluation and careful utilization of appropriate diagnostic measures and interventions can help the astute emergency physician improve clinical outcomes.


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**The Critical ECG**

A 67-year-old woman with the sensation of a rapid heart beat

Atrial fibrillation, rate 140, aberrant ventricular conduction. When the rhythm is irregularly irregular, the most common causes are atrial fibrillation, multifocal atrial tachycardia, and atrial flutter with variable AV conduction. Distinct P waves or flutter waves are absent, excluding the latter two possibilities. The QRS complexes are wide, suggesting aberrant ventricular conduction. Aberrant conduction can be the result of a bundle branch block, metabolic abnormality (eg, hyperkalemia), accessory pathway (eg, WPW), or a non-specific intraventricular conduction delay. In the absence of a full 12-lead ECG, it is difficult to specify the exact cause of the aberrant conduction in this case.