Shock

Introduction

Shock is a broad term that describes a physiologic state where oxygen delivery to the tissues is inadequate to meet metabolic requirements, causing global hypoperfusion. It may also be thought of as an imbalance between tissue oxygen supply and demand.

Objectives

After completion of this module, the student will be able to:

- understand the main types and physiology of shock
- identify the shock patient, and target diagnostic testing and treatment accordingly

Shock may be described as compensated (normal blood pressure with inadequate perfusion) or uncompensated (hypotension and inability to maintain normal perfusion). Thus, a patient with normal vital signs may still be in shock.
Types of Shock

<table>
<thead>
<tr>
<th>Type</th>
<th>Physiology</th>
<th>Examples</th>
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</thead>
<tbody>
<tr>
<td>Hypovolemic (most common)</td>
<td>Decreased circulatory volume</td>
<td>Hemorrhage or fluid loss</td>
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<tr>
<td>Cardiogenic</td>
<td>Impaired heart pump function</td>
<td>Acute coronary syndrome, valve failure, dysrhythmias</td>
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<td>Distributive</td>
<td>Pathologic peripheral blood vessel vasodilation</td>
<td>Sepsis, anaphylaxis, neurogenic</td>
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<tr>
<td>Obstructive</td>
<td>Non-cardiac obstruction to blood flow</td>
<td>Pulmonary embolus, tension pneumothorax, tamponade</td>
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Understanding the primary cause is important to guide treatment, but the end results of shock (circulatory collapse and end organ damage) are the same for all.

Classic Presentation

Signs of shock may be subtle or obvious. In the classic patient, a brief history will provide clues to the diagnosis:

- Obvious bleeding, from trauma or an anatomic source (GI, vaginal, or ENT) suggests hypovolemic shock, as does decreased PO intake or fluid loss due to vomiting, diarrhea, excess urination or other conduit (ostomy).
- Chest pain, shortness of breath, leg swelling, or syncope may precede the development of shock due to a cardiac (ACS, CHF) or obstructive (PE) cause.
- Sudden onset of hives, face or body swelling whether associated with a known trigger or not can signal anaphylactic (distributive) shock.
- Signs of infection such as fever with cough, abdominal pain, or headache may indicate sepsis.
- In some cases, however, non-focal, vague symptoms such as weakness, altered mental status, or malaise may be the only presenting signs of any of the types of shock.
Physical Exam findings are also variable. Blood pressure alone should not be used as the sole marker to determine shock. Early shock may present with normal or even elevated blood pressure, and normal heart rate; but, if left untreated, tachycardia and hypotension will follow. Hypoperfused patients often exhibit cool, pale or cyanotic skin with decreased capillary refill and dry mucous membranes; confusion, altered mental status or coma; thready pulses or tachypnea. In cardiogenic shock, arrhythmias, jugular venous distention, and dependent edema may be present.

The shock index is easily calculated (heart rate divided by systolic blood pressure) and can provide clues to the severity of the patient’s condition. A normal index ranges from 0.5-0.7; repeated values >1.0 indicate decreased left ventricular function and are associated with higher mortality.

Diagnostic Testing

Although a wide array of labs are generally ordered, the suspected etiology of shock should guide diagnostic testing. Studies should determine whether end organ damage (neurologic, cardiac, renal, GI) is present. In shock patients, the following tests should be considered:

- CBC and coagulation studies (to determine anemia/blood loss, infection, hypocoagulability)
- electrolytes
- BUN/creatinine and urinalysis; hepatic function panel (to assess liver and renal function)
- chest x-ray, EKG
- lactate (to gauge the degree of hypoperfusion)
- urine pregnancy test
- More invasive testing is often required: arterial blood gas for O2/pH; central venous oxygen measurement, systemic vascular resistance, and cardiac output may be measured through special central venous catheters

If a particular type of shock is suspected, further studies may be directed accordingly:

- infectious etiology (sepsis) – blood, sputum, urine, pelvic, or wound cultures; head CT and lumbar puncture; targeted imaging (US/CT)
- cardiogenic – cardiac enzymes and echocardiogram
- obstructive – CT or V/Q scan (PE), echo (tamponade)

How do I make the diagnosis?
Shock should be strongly considered in ill-appearing patients with vital sign abnormalities (particularly tachycardia and hypotension), altered mental status, or signs of organ hypoperfusion. In most cases, the clinical picture should guide decision making – is there significant bleeding/anemia? Signs of infection? Cardiac signs or symptoms? However, certain disease processes can be confounding and mixed cases may occur. Although it may require invasive monitoring techniques, understanding shock physiology can help point to the primary cause:

<table>
<thead>
<tr>
<th>Type</th>
<th>HR</th>
<th>CVP</th>
<th>Contractility</th>
<th>SVR</th>
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<tbody>
<tr>
<td>Cardiogenic</td>
<td>↑</td>
<td>↑</td>
<td>↓↓</td>
<td>↑</td>
</tr>
<tr>
<td>Hypovolemic</td>
<td>↑</td>
<td>↓↓</td>
<td>±↑</td>
<td>↑</td>
</tr>
<tr>
<td>Distributive (Sepsis)</td>
<td>↑</td>
<td>↓↓</td>
<td>±</td>
<td>↓</td>
</tr>
<tr>
<td>Obstructive</td>
<td>↑</td>
<td>±↑</td>
<td>±</td>
<td>↑(tamponade, PE) ↓(tension PTX)</td>
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Laboratory values should be examined, but do not wait for results to begin your treatment. Common results include:

- anemia (decreased hemoglobin/hematocrit), disorders of platelets or coagulation studies
- elevated or depressed white blood count with left shift
- elevated lactate level or decreased serum bicarbonate (suggests a shift to anaerobic metabolism and tissue hypoperfusion)
- evidence of end organ damage: elevated creatinine, abnormal liver function tests, ARDS/edema on chest x-ray, arrhythmia/ischemia on EKG or abnormal cardiac enzymes, ischemic neurologic changes on CT/MRI

**Treatment**

Treatment should begin emergently – early therapy has been shown to significantly improve mortality in certain kinds of shock. Start with the ABCs – airway control is paramount; intubation should be strongly considered for airway protection, oxygenation, decreasing metabolic load by removing the work of breathing and optimization of respiratory status. Keep in mind, though, that many drugs used in intubation, as well as positive pressure ventilation itself, can have negative hemodynamic effects. Ensuring proper oxygenation is critical for all of the etiologies of shock. Arterial oxygen saturation should be maximized, and central venous oxygenation levels targeted above 70%.
Circulatory support is crucial – obtain IV access through large bore peripheral lines or a central venous catheter (which can help for rapid fluid and medication delivery, as well as provide invasive monitoring). In most kinds of shock, crystalloid fluids (normal saline or Ringer’s lactate) should be given as boluses. Be careful with rapid fluid administration to the patient in cardiogenic shock with pulmonary edema. Blood products may be necessary in certain types of hemorrhagic shock. If volume resuscitation does not improve the patient’s hemodynamic status, vasoactive medications such as epinephrine, norepinephrine, dopamine, and vasopressin may be used.

Careful monitoring of fluid status is encouraged, using a urinary catheter, intraarterial blood pressure measurements, and central venous pressure monitoring.

Aggressive treatment of the underlying cause of shock is warranted:

- Hypovolemia due to hemorrhage may warrant surgical or interventional control.
- Sepsis syndromes should be treated with early goal-directed therapy (maximization of oxygen delivery, careful hemodynamic monitoring) and aggressive antibiotic treatment.
- Cardiogenic shock may necessitate emergent angiography or surgical procedures (bypass, valve repair, IABP).
- Obstructive shock due to PE often requires anticoagulation or thrombolysis, whereas when due to cardiac tamponade emergent drainage of the pericardial fluid may be necessary.

Resuscitation of a shock state is thought to be successful when the following occurs:

- normalization of hemodynamic state (BP, HR, and urine output)
- lactate decreases by half in the first couple of hours
- normal volume status restored
- maximal tissue oxygenation
- resolution of acidosis and return to normal metabolic parameters

Disposition

Patients in shock should be quickly and aggressively treated in the ED to prevent later morbidity and mortality, and usually require admission to an ICU setting under the supervision of an intensivist and other consulting services as needed.

Despite proper treatment, the mortality rates from shock can exceed 50 percent.
Pearls & Pitfalls

- Shock is generally associated with ill-appearing, hypotensive and tachycardic patients, but the clinical picture can be far more subtle.
- Understanding the different types of shock and their physiologic features can help to target appropriate treatment.
- Shock is a true emergency and should be treated quickly and aggressively in the ED to decrease morbidity. Do not wait until labs and/or studies return to begin resuscitation; remember the ABCs!

References