Section 1

General critical care

1

Shock

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Introduction

- Shock is the pathological state of circulatory collapse resulting in tissue hypoperfusion.
  - At a cellular level there is inadequate delivery of metabolic substrates (i.e., oxygen and glucose) to sustain aerobic metabolism. This leads to anaerobic metabolism and a buildup of lactic acid. As ATP stores are depleted, cell membrane integrity is lost, leading to cellular dysfunction and cell death.
  - At a systemic level, there is activation of compensatory mechanisms to augment cardiac output (CO) and systemic vascular resistance (SVR).
    - Blood flow is preferentially shunted to the brain and heart from splanchnic and renal vascular beds to the detriment of these organ systems.
    - Eventually, compensatory mechanisms are overwhelmed leading to the multiple organ dysfunction syndrome (MODS).
- The circulatory system can be thought of as having three major components: a pump (the heart), fluid (blood), and tubing (blood vessels). The etiologies of shock can be categorized as a malfunction of one or more of these components.
- Conventionally shock is classified as distributive (tubing malfunction), hypovolemic (fluid loss), cardiogenic (pump failure), obstructive (obstruction to the inflow or outflow of the pump), or undifferentiated (Table 1.1).
- A clinician must accurately identify the appropriate category as certain etiologies call for specific interventions (e.g., pericardiocentesis for cardiac tamponade).
- Treatment of shock is dependent on reversing tissue hypoxia by improving oxygen delivery and decreasing oxygen demand, as well as correcting the underlying cause.

Practical Emergency Resuscitation and Critical Care, ed. Kaushal Shah, Jarone Lee, Kamal Medlej, and Scott D. Weingart. Published by Cambridge University Press.

Table 1.1. Categories of shock and differential diagnosis

Distributive	Severe inflammatory response syndrome (SIRS) and sepsis, neurogenic, anaphylaxis, adrenal insufficiency/Addisonian crisis, drug or toxin reaction, hepatic failure
Hypovolemic	Hemorrhage (trauma, GI bleed, ruptured AAA), GI losses (diarrhea, vomiting, fistula), insensible losses, third spacing (pancreatitis, burns)
Cardiogenic	Myocardial infarction, myocarditis, arrhythmia, cardiac contusion, valve dysfunction, thyrotoxicosis, end-stage cardiomyopathy
Obstructive	Tension pneumothorax, cardiac tamponade, pulmonary embolism (PE), constrictive pericarditis, aortic coarctation, excessive PEEP or auto-PEEP

- Shock may be difficult to diagnose given subtle presentation in the early stages and rapid decline in late stages, and may encompass more than one category of shock (i.e., septic shock can also result in septic cardiomyopathy, adding a cardiogenic component to the shock state).
- A thorough history and examination along with certain diagnostic modalities can assist with categorizing shock.

Presentation

- A patient in shock presents with a wide variety of signs and symptoms related to both the precipitating event and the resultant cellular dysfunction.
- On presentation to the ED, a patient in shock may be very ill and have difficulty giving a history, which may necessitate obtaining collateral information from emergency medical services personnel, family, or friends.
- In general, a patient will appear toxic, in distress, with pale, clammy skin, with tachypnea, and with tachycardia as a result of the body's stress response to injury.
- Subtle clues from the patient's history including past medical conditions, preceding events, and appearance can help clinicians categorize a patient's shock state (Table 1.2).
- Patients often present in early shock, which will quickly progress from compensated to decompensated, and finally, refractory or irreversible shock.
  - A commonly held misbelief that often leads to delayed treatment and poorer outcomes is that shock necessitates hypotension.
  - Compensated or cryptic shock patients may appear relatively normal and asymptomatic as compensatory mechanisms resulting in tachycardia or vasoconstriction have yet to be overwhelmed.
  - Decompensated shock patients often appear ill, pale, diaphoretic, tachypneic, tachycardic, and with altered mental status.
  - The critical state refractory shock is recognized by manifestations of MODS such as obtundation or coma, refractory hypotension, renal failure, disseminated intravascular coagulation (DIC), and the acute respiratory distress syndrome (ARDS).

Table 1.2. Presentations of shock

Category	History	Past medical history	Appearance
Distributive	Fever, chills, headache, dyspnea, wheezes, stridor, meningismus, malaise, myalgias, cough, dysuria, diarrhea	Immunocompromised, allergies, adrenal insufficiency	Diaphoretic, distressed, flushed, warm skin
Hypovolemic	Poor intake, excessive vomiting or diarrhea, GI bleed or evidence of trauma	Coagulopathy (acquired or inherited), upper or lower GI bleed	Rapid and weak pulses, cool skin, delayed capillary refill, tachypneic, dry mucous membranes, poor skin turgor
Cardiogenic	Syncope, dyspnea, chest pain, palpitations	Coronary artery disease, myocardial infarction, dysrhythmia, congestive heart failure	Tachypneic, jugular venous distension, new murmur, delayed capillary refill, wheezes, rales, cool skin, murmur
Obstructive	Trauma	COPD, connective tissue disorder	*See Cardiogenic, Beck's triad, asymmetric breath sounds, tracheal deviation

Diagnosis and evaluation

- **Vital signs** are nonspecific.
  - Any single vital sign in isolation is not helpful in diagnosing shock or the possible etiology of shock.
  - Shock should be suspected when patients present with a constellation of signs including ill-appearance, tachycardia, tachypnea, hypotension, and oliguria.
  - Tachycardia is seen in the hyperdynamic state of shock. However, bradycardia may also be present in the setting of drug overdose (e.g., beta-blockers, calcium channel blockers, digoxin).
  - Hypotension is usually a late finding in a previously healthy individual, and noninvasive blood pressure monitoring can be inaccurate. Also, normotension in a previously hypertensive patient can be indicative of shock.
- **Signs of shock** are a result of the compensatory mechanisms, organ dysfunction, and the precipitant etiology. They can be helpful in providing clues to the cause of shock.

- Warm extremities can be caused by vasodilation present in distributive shock.
- Cold extremities can be caused by vasoconstriction present in hypovolemic or cardiogenic shock.
- Jugular venous distension in the setting of shock can be caused by cardiogenic or obstructive shock.
- Low jugular venous pressure is indicative of hypovolemic shock.
- Other findings will be present in specific causes of shock:
  - Absent breath sounds in tension pneumothorax
  - Muffled heart sounds in cardiac tamponade
- Signs of organ dysfunction include oliguria/anuria, encephalopathy and hypoxia.

- **Laboratory tests:**
  - A complete blood count will identify leukocytosis or bandemia in the setting of SIRS. It will also identify anemia (Hct <30% or Hgb <10). However, a normal value may be misleading in the acute stage of blood loss.
  - Serum chemistry will assess renal function, hydration status, and detect electrolyte derangements.
  - Cardiac biomarkers will identify myocardial injury.
  - Lactate and base deficits are markers for tissue hypoperfusion.
  - Arterial or venous blood gases will identify oxygenation or ventilation disorders and severe acid–base disturbances.
  - Urine or serum hCG (human chorionic gonadotropin) should be obtained in female patients of child-bearing age to evaluate for potential ruptured ectopic pregnancy as a source of hemorrhage and shock.
- **Electrocardiogram (ECG):**
  - Useful for the early diagnosis of acute coronary syndromes, arrhythmias, or electrolyte disturbances.
- **Imaging:**
  - **Chest radiography** may show edema, effusion, consolidation, pneumothorax, or an enlarged mediastinum and cardiac silhouette.
  - **Pelvic radiography** as a screening tool in blunt trauma may reveal a clinically significant pelvic fracture as a source of hemodynamic instability.
  - **Point-of-care ultrasonography (US)** can be very useful in the management of undifferentiated shock.
    - The Focused Assessment with Sonography in Trauma (FAST) examination can quickly identify free fluid in the abdomen as a potential source of bleeding in a hemodynamically unstable patient.
    - Cardiac views can reveal pericardial effusion and tamponade physiology.
    - Bedside echocardiography can also be used to assess for globally reduced ventricular function, a severely enlarged right ventricle (RV), or preload responsiveness by evaluating the inferior vena cava (IVC).

Table 1.3. Differentiating categories of shock

	CVP	$ScvO_2$	CI	SVR
Distributive	↓	↑ or ↓	↑ or ↓	↓
Hypovolemic	↓	↓	↓	↑
Cardiogenic	↑	↓	↓	↑
Obstructive	↑	↓	↓	↑

  - The extended FAST examination with lung views may reveal a pneumothorax or pleural effusion.
  - Additionally, bedside US of the abdomen can identify a ruptured abdominal aortic aneurysm (AAA) as the cause of shock.
- **Conventional echocardiography (transthoracic or transesophageal)** will identify ventricular dysfunction, regional wall motion abnormalities, valvular pathology, aortic pathology, tamponade physiology, or RV strain pattern suggestive of massive pulmonary embolism or other causes of right heart failure.
- **Computed tomography (CT) scanning** may be helpful in identifying the source of shock in specific cases such as pulmonary embolism, aortic dissection, intra-abdominal sepsis, or intra-abdominal hemorrhage.
- **Invasive hemodynamic monitoring** by way of an arterial line, central venous catheter, or pulmonary artery catheter can further differentiate shock categories by determining values such as mean arterial pressure (MAP), central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), cardiac output (CO), cardiac index (CI), systemic vascular resistance (SVR), and oxygen transport variables. Different categories of shock have different hemodynamic profiles (Table 1.3).
  - **Central venous catheter:** a catheter placed into a central vein, either the internal jugular, subclavian, or femoral vein. It allows one to administer vasoactive medications and obtain the following measurements (a femoral line does not allow for accurate measurements):
    - **CVP** is an indicator of volume status and cardiac pump function. The target range in septic patients is 8–12 mmHg.
    - **Central venous oximetry:** the **$ScvO_2$** value is an indicator of tissue oxygenation and utilization. It is obtained by sampling blood from the superior vena cava (SVC) via a central venous catheter. Low central venous oxygen saturation (<70%) suggests that tissues are extracting more oxygen because of hypoperfusion and hypoxia.
    - **Arterial line:** an invasive catheter placed in the artery (common sites are radial or femoral) that allows for direct measurement of arterial blood pressure. It is more accurate for determining MAP and is helpful in a patient who requires multiple blood draws.
    - **Pulmonary artery catheter (PAC):** an invasive catheter placed in the pulmonary artery that allows a clinician to directly measure PCWP (surrogate

for left atrial pressure) and pulmonary artery pressure to assess pump function. It also allows one to measure other hemodynamic parameters including CO, CI, and SVR. The use of a PAC has not been shown to provide a mortality benefit. This procedure is almost never performed in the emergency department.

Critical management

- **Principles of shock management** include specific therapy for treating the underlying cause, and general therapy to manage the shock syndrome.
  - Specific therapies also referred to as source control include
    - Antibiotics in sepsis
    - Operative repair of traumatic injuries
    - Thrombolysis in massive PE
    - Revascularization in acute coronary syndrome
    - Pericardiocentesis in tamponade
    - Tube thoracostomy in tension pneumothorax
- The general treatment of shock is focused on restoring and maintaining adequate organ perfusion. This is accomplished by increasing oxygen delivery and decreasing oxygen demand.
- **ABCs (airway, breathing, circulation):**
  - As with all critically ill patients, secure the airway early if necessary. If the situation permits, rapid sequence intubation (RSI) is the method of choice.
  - Ensure adequate oxygenation with a goal SaO_2 of >90%.
  - Obtain large-bore intravenous (IV) access to allow infusion of crystalloids or transfusion of blood products. If peripheral IV access fails, consider central venous access if time permits, or emergent intraosseous line placement.
- **Increase oxygen delivery:**
  - **Volume resuscitation:**
    - In some categories of shock, patients have a decreased intravascular volume as a result of blood or fluid loss, or because of vascular dilation or leakage.
    - 20–30 mL/kg bolus of normal saline or lactated Ringer's solution is the preferred initial resuscitation treatment. This bolus may have to be repeated if the patient does not respond adequately.
    - In hemorrhagic shock from trauma, crystalloid administration should be limited and blood products such as packed red blood cells (PRBCs), fresh frozen plasma (FFP), and platelets should be transfused during resuscitation. Excessive crystalloid administration can dilute the important blood constituents that are already depleted and lead to worse outcomes.
    - **Vasopressors (Table 1.4):**
    - Hypotensive patients who do not respond to volume resuscitation may benefit from vasopressor support.

Table 1.4. Vasopressors and inotropes

Agent	Receptors	Mechanism of action	Effective dose
Vasopressors			
Epinephrine	α, β	Vasoconstriction, inotropy, chronotropy	1–10 micrograms/minute
Norepinephrine	$\alpha_1 > \beta_1$	Vasoconstriction, mild inotropy and chronotropy	2–30 micrograms/minute
Phenylephrine	α_1	Vasoconstriction	10–300 micrograms/minute
Vasopressin	V_1	Vasoconstriction	0.01–0.04 U/minute
Dopamine	D, α, β	Inotropy and chronotropy at lower doses, vasoconstriction at high doses	2–20 micrograms/kg/minute
Inotropes			
Dobutamine	$\beta_1 = \beta_2$	Inotropy, chronotropy, vasodilation at high doses	2–20 micrograms/kg per minute
Milrinone	Phosphodiesterase-inhibitor	Inotropy, chronotropy, vasodilation at high doses	0.375–0.75 micrograms/kg per minute

- A MAP of ≥65 mmHg should be targeted to ensure proper perfusion of the vital organs.
- Norepinephrine is the vasopressor of choice in septic shock.
- Epinephrine or vasopressin can be added to norepinephrine in cases of refractory septic shock.

- **Inotropes (Table 1.4):**
  - Patients with low cardiac output secondary to decreased myocardial contractility or cardiogenic shock benefit from inotropic support to improve tissue perfusion.
  - Dobutamine is the preferred agent in decompensated heart failure in patients that are normotensive or mildly hypotensive. It may need to be given in conjunction with vasopressors.
  - Dopamine use is controversial as recent studies have suggested increased mortality in cardiogenic shock due to its arrhythmogenic properties.
- **Blood products:**
  - Hemoglobin is the primary mode of delivering oxygen to tissues and should be transfused in patients with hemorrhagic shock, anemia, or low central venous saturation (<70%).

- In the hemorrhagic shock patient, PRBCs should be transfused based on the patient's clinical status, as hemoglobin and hematocrit levels may not accurately represent blood loss early on.
- In the nonbleeding patient with shock, goal hematocrit levels are controversial but lie somewhere between 21% and 30%.

- **Decrease oxygen demand:**
  - Treat fever with antipyretics to decrease metabolic demand and insensible losses.
  - Initiate mechanical ventilation early to decrease the work of breathing, prevent aspiration, improve oxygenation, and manage acidosis.
- **Assessing resuscitation efforts:**
  - Once appropriate intravenous access, definitive airway, and hemodynamic monitors have been acquired, it is imported to continually assess resuscitation efforts and maintain a goal-oriented approach.
  - Volume resuscitate to achieve a CVP of 8–12 mmHg for optimal preload in nonintubated patients, and 12–15 mmHg in intubated patients. Using CVP as a preload surrogate is controversial as studies have shown that CVP may not be indicative of preload responsiveness. Alternative techniques to CVP monitoring are described in the following chapter.
  - Maintain a MAP >65 mmHg by using vasopressors once the patient is appropriately volume-resuscitated.
  - Keep $ScvO_2$ >70% by administering PRBCs if the patient is anemic, and/or initiating inotropes.
  - Serially assess lactate levels. Clearance of lactate (a decrease of ≥10% of the original value 2–3 hours after initiation of resuscitation) or correction of the base deficit are reliable adjuncts to the measurement of $ScvO_2$.

Special circumstances

- **Pediatric patients:**
  - Recognition of shock is difficult due to variations in age-dependent vital signs, difficulty in assessing mental status, and the nonspecificity of early manifestations of shock such as irritability and poor feeding.
  - Shock should be suspected in children that have signs of poor perfusion such as delayed capillary refill, dry mucous membranes, absent tears, or are ill-appearing.
  - Children have strong compensatory mechanisms and by the time they are hypotensive may already be in an irreversible state of shock.
- **Pregnant patients:**
  - Management is made more difficult due to changes in maternal physiology and due to the considerations for both maternal and fetal well-being.
  - Shock may be caused by pregnancy-specific diagnoses such as peripartum hemorrhage, pulmonary embolism, peripartum cardiomyopathy, or supine hypotensive syndrome.

- Usual monitoring modalities are still employed in addition to cardiotocographic monitoring of the fetus.
- The first resuscitative maneuver, while securing the ABCs, is to have the patient lie in the left lateral decubitus position. This alleviates pressure on the IVC allowing increase venous return to the heart.

- **Geriatric patients:**
  - Elderly patients experience significantly more morbidity and mortality from all causes of shock due to their limited ability to augment cardiac output and maintain vascular tone.
  - Elderly patients often have multiple comorbidities or use multiple medications that distort the diagnosis and management of shock.

REFERENCES

Cheatham ML, Block EFJ, Promes JT, Smith HG, Dent, DL, Mueller DL. Shock: an overview. In: Irwin RS, Rippe JM, eds. *Irwin & Rippe's Intensive Care Medicine*. 5th edn. Philadelphia: Lippincott Williams & Wilkins; 2003.

Gaieski D. Shock in adults: types, presentation, and diagnostic approach. *UpToDate*: Wolters Kluwer Health [updated 2012 December 17]. Available from: www.uptodate.com/contents/shock-in-adults-types-presentation-and-diagnostic-approach (accessed April 30, 2013).

Jones AE, Jeffrey A. Shock. In: Marx JA, Hockberger RS, Walls RM, Adams J, Rosen P, eds. *Rosen's Emergency Medicine: Concepts and Clinical Practice*. 7th edn. Philadelphia, PA: Mosby Elsevier; 2010.

Maier RV. Approach to the patient with shock. In: Longo DL, Fauci AS, Kasper DL, *et al.* eds. *Harrison's Principles of Internal Medicine*. 18th edn. New York: McGraw-Hill; 2012.

Rivers E, Amponsah D. Shock. In: Wolfson AB, Hendey GW, Hendry PL, *et al.*, eds. *Harwood-Nuss' Clinical Practice of Emergency Medicine*. 4th edn. Philadelphia: Lippincott Williams & Wilkins; 2005.

2

Monitoring

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Introduction

Monitoring and understanding of monitoring devices are necessary for proper management of the critically ill patient. The goal of hemodynamic and respiratory monitoring is to ensure and maintain adequate tissue perfusion. This chapter will review the basic methods and principles of monitoring in the emergency setting.

Basic monitoring

Several aspects of a critically ill patient's status can be obtained from the telemetry monitor (Table 2.1).

- Three electrodes (white, black, and red) are used in 3-lead ECG systems, allowing for multiple views of the heart. Lead II is typically displayed on the cardiac monitor. For accurate ECG tracing, the electrodes should be applied in the following manner:
  - The white electrode is placed just below the clavicle on the right shoulder.
  - The black electrode is located on the left clavicle near the shoulder.
  - The red electrode is connected to the left pectoral muscle near the apex of the heart.
- Heart rate is measured as number of beats per minute (bpm). The normal heart rate range is 60–100 bpm.
- Blood pressure is measured as systolic pressure over diastolic pressure.
  - *Systolic pressure* is the peak pressure in the arteries. This occurs when the ventricles contract.

Practical Emergency Resuscitation and Critical Care, ed. Kaushal Shah, Jarone Lee, Kamal Medlej, and Scott D. Weingart. Published by Cambridge University Press.

Table 2.1. Basic readings on the telemetry monitor

3-Lead or 5-lead ECG system
Heart rate
Blood pressure
Mean arterial pressure
Respiratory rate
Oxygen saturation
Temperature

  - *Diastolic pressure* is the minimum pressure of the arteries. This occurs when the ventricles are filled with blood.
- Noninvasive blood pressure measurements can be obtained via auscultatory or oscillometric measurements.
  - Oscillometric blood pressure is used with telemetry monitoring. Proper cuff size is essential for accurate readings.
- Mean arterial pressure (MAP) is the average arterial pressure during a single cardiac cycle and can be used as indicator of adequate tissue perfusion. MAP = (⅔ diastolic pressure) + (⅓ systolic pressure).
  - MAP = (cardiac output (CO) × systemic vascular resistance (SVR)) + central venous pressure (CVP).
  - Normal MAP can range between 70 and 110 mmHg.
- Respiratory rate is a measure of the total number of breaths per minute.
- Temperature can be measured via oral, tympanic, axillary, esophageal, or rectal routes. Rectal and esophageal temperatures are more indicative of a patient's core temperature.

Respiratory monitoring

Pulse oximetry

- Pulse oximetry provides continuous measurement of a patient's oxygenation status.
- Measurements are obtained via sensors placed on the patient's fingertip, earlobe, or forehead. These sensors use two light-emitting electrodes.
  - The first emits red light that has a wavelength of 660 nm.
  - The second emits infrared light that has a wavelength of 905, 910, or 940 nm.
  - The difference in absorption of the red light and infrared light determines the oxyhemoglobin to deoxyhemoglobin ratio. This measurement corresponds to the pulse oximeter saturation estimate (SpO_2).
- Pitfalls:
  - Once the arterial oxygen saturation (SaO_2) falls below 70%, the pulse oximetry readings are no longer accurate.

- Pulse oximetry cannot reliably detect hypoxemia in the setting of carbon monoxide poisoning because carboxyhemoglobin has similar light absorption to that of oxyhemoglobin at 660 nm.
- It is also inaccurate in the setting of methemoglobinemia as both red light and infrared light are equally absorbed by methemoglobin. This results in a fixed SpO_2 around 85% regardless of the true SaO_2.
- Oxygenated blood that is redistributed from central to peripheral circulation can cause a delay in saturation readings known as pulse oximetry lag. This lag can be as long as 2–3 minutes in critically ill patients secondary to reduced blood flow.
- A proper waveform must be visualized on the monitor in order to be able to properly determine the SpO_2.
- Hypothermia and peripheral vasoconstriction can lead to inaccurate or unobtainable values.

End-tidal carbon dioxide ($EtCO_2$)

- Capnography measures the partial pressure or concentration of expired carbon dioxide (CO_2), the $EtCO_2$. This concentration is a function of the production of CO_2 at the tissue level and delivery of CO_2 to the lungs by the circulatory system. Therefore, capnography provides important information regarding ventilation, circulation, and metabolism.
- The normal expired CO_2 level is around 5%, which is approximately 40 mmHg.
- There are two different $EtCO_2$ monitoring modalities that are used in the emergency department (ED).
  - Colorimetric capnometry is a filter generally used to confirm endotracheal tube (ET) placement in the trachea.
    - The filter attaches to the ET tube and displays the change in concentration of carbon dioxide by color change.
    - In certain colorimetric capnometers, detection of carbon dioxide is exhibited by filter color change from purple to yellow. This is a semiquantitative mode of monitoring. The color ranges are as follows: purple = $EtCO_2$ <0.5%; tan = $EtCO_2$ 0.5–2%; and yellow = $EtCO_2$ >2%.
    - Pitfalls:
      - The filter can turn yellow when exposed to acidic material such as stomach contents, or medications including lidocaine and epinephrine.
      - In patients in cardiac arrest, color change may not be seen even with tracheal intubation because of the low $EtCO_2$ value resulting from poor or absent blood flow.
  - Quantitative capnography uses infrared technology to provide a continuous numerical value and a continuous waveform display of $EtCO_2$. This measure is plotted graphically on a monitor.
- Some ED applications of quantitative $EtCO_2$ monitoring are listed below.