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Kaushal Shah, Jarone Lee, Edited in association with Clark G. Owyang, Benjamin Christian Renne
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Shock

KENNETH R. L. BERNARD

Introduction

- **Shock** is a pathological state resulting from **inadequate delivery, increased demand, or poor utilization** of **metabolic substrates** (i.e., oxygen and glucose) which leads to **cellular dysfunction and cell death**. This leads to progressive acidosis, endothelial dysfunction, and inflammatory cascade that results in **end-organ injury**.
- Early in the course of shock, compensatory mechanisms may attempt to augment **cardiac output (CO)** and/or **systemic vascular resistance (SVR)** in an effort to improve tissue perfusion.
- Without treatment, those compensatory mechanisms are overwhelmed, leading to decompensated shock, multiorgan failure (MOF) and death.
- Shock can be conceptualized as a derangement of the three components of the circulatory system:
  - **the pump (the heart)**
  - **the fluid (intravascular volume)**
  - **the tank (blood vessels)**

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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, abdominal compartment syndrome

- Conventional mechanisms of shock are in turn classified as:
  - **cardiogenic/obstructive** (pump failure or outflow obstruction)
  - **hypovolemic** (fluid/blood loss)
  - **distributive** (tank malfunction) (Table 1.1).
- Shock may be multifactorial, encompassing more than one category (e.g., septic shock can also result in septic cardiomyopathy, adding a cardiogenic component to the shock state).

Presentation

- Shock can present in a variety of ways and, especially in the **early stages**, patients may report **very subtle, vague** or **generalized symptoms** such as restlessness, anxiety, fatigue, or slight confusion. In these early stages, patients may be in **compensated shock**.
- Without rapid intervention, patients quickly progress to the critical state of **decompensated** or **refractory shock** and will appear **in distress, pale, diaphoretic, tachypneic, tachycardic, hypotensive, and encephalopathic**. At this point, **MOF** and **irreversible tissue injury** is likely present.
- 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).

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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 pulse, 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, dyspnea	COPD, connective tissue disorder, malignancy	JVD, muffled heart sounds, asymmetric breath sounds, tracheal deviation

- A commonly held misbelief that often leads to delayed treatment and poorer outcomes is that **shock necessitates hypotension**. Not all patients with shock are hypotensive, and not all hypotensive patients are in shock.

Diagnosis and Evaluation

Epidemiology

- In all comers, etiology of shock from most to least common:
  - Septic >>> Hypovolemic > Cardiogenic/Obstructive

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Vital Signs

- No single vital sign in isolation is sufficient to diagnose or identify the possible etiology of shock.
  - Tachycardia, hypotension, tachypnea, fever should all raise suspicion for shock.
- Multiple **qSOFA (quick SOFA) criteria** predict poor outcomes and mortality in critically ill patients.
  - Altered mental status (GCS $<$ 15)
  - Respiratory rate $\geq$ 22
  - Systolic BP $\leq$ 100

Clinical Signs

- Shock should be suspected when patients present with a **constellation** of signs including **ill-appearance, confusion, tachycardia, tachypnea, hypotension, and poor urine output**.
- Warm extremities caused by vasodilation suggest distributive shock.
- Cold extremities caused by vasoconstriction suggest hypovolemic or cardiogenic shock.
- Jugular venous distention is usually present with cardiogenic/obstructive shock.
- A narrow pulse pressure (systolic BP - diastolic BP) suggests hypovolemic or cardiogenic shock.
- Dry mucous membranes, poor skin turgor, pallor, and low jugular venous pressure, are suggestive of hypovolemic shock.
- Tachypnea and Kussmaul breathing are often present with significant acidosis.
- Encephalopathy is often present with poor cerebral blood flow, renal failure, liver failure, hypoxia, and acidemia caused by shock states.
- Urticaria, facial or lip swelling, stridor and wheeze suggest anaphylaxis.

Laboratory Tests

- A complete blood count will identify leukocytosis or bandemia. It will also identify anemia (Hct $<$ 30% or Hgb $<$ 10). However, a normal value may be misleading in the acute stage of blood loss.

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- Serum chemistry will assess renal function, hydration status, and detect electrolyte derangements.
- Cardiac biomarkers can identify myocardial injury.
- Lactate and base deficits may reflect tissue hypoperfusion (though are not specific for this); these markers are most useful as markers of effectiveness of a resuscitation.
- Arterial or venous blood gases will identify oxygenation or ventilation disorders and severe acid–base disturbances.
- Coagulation studies can identify coagulopathy associated with hemorrhagic shock and/or shock-related hepatic synthetic dysfunction.
- Urine or serum human chorionic gonadotropin (hCG) should be obtained in female patients of childbearing age for pregnancy.

Electrocardiogram (ECG)

- Key to identify and diagnose acute coronary syndromes, malignant 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 trauma may reveal a clinically significant pelvic fracture as a source of hemodynamic instability.
- Point-of-care ultrasonography (US) is a cheap, readily available bedside tool that can be very useful in the identification and management of undifferentiated shock.
    - Several protocols such as: The Focused Assessment with Sonography in Trauma (FAST), Rapid Ultrasound in Shock and Hypotension (RUSH), and the Abdominal and Cardiac Evaluation (ACES) protocols were developed to assist in the bedside diagnosis of shock to identify organ dysfunction, fluid responsiveness, and potentially guide specific interventions e.g., pericardiocentesis. The standard anatomical views are described below:
        - Cardiac views can reveal pericardial effusion, assess ventricular function, and identify valvular dysfunction or tamponade physiology.

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  - Views of the inferior vena cava (IVC) can assess volume responsiveness.
  - Lung views can quickly identify pneumothorax, consolidation, presence of pulmonary edema or effusions.
  - Abdominal views will identify free fluid.
  - Views of the thoracic and abdominal aorta can identify dissection or aneurysm.
  - Lower extremity vascular ultrasound can identify deep venous thrombosis suggesting coagulopathy and increasing suspicion of pulmonary embolism.
- Computed tomography (CT) scanning may be helpful in identifying the source of **undifferentiated shock**, identifying etiologies such as pulmonary embolism, aortic dissection, intra-abdominal sepsis, or trauma.

Invasive Hemodynamic Monitoring

- An arterial line, central venous catheter, or pulmonary artery catheter can further differentiate shock by determining myriad hemodynamic variables.
- A central venous catheter placed into a central vein allows for the administration of vasoactive medications and measurements of a central venous pressure (CVP) and central venous oxygen saturation ($Scv0_2$).
- An arterial catheter allows for accurate, real-time measurement of pulse pressure and mean arterial pressure (MAP).
- A pulmonary artery catheter (PAC) is an invasive catheter placed in the pulmonary artery that allows a clinician to directly measure PCWP (surrogate for left atrial pressure), pulmonary artery pressure, CO, CI, and SVR. Together, these data can help differentiate the underlying shock profile (Table 1.3). However, the use of a PAC is controversial and rarely performed in the emergency department.

Critical Management

Brief Summary Checklist

- ☑ ABCs
- ☑ Fluids or blood as indicated

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Table 1.3 Differentiating categories of shock

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

- ☑ Vasopressors if concern for shock and MAP < 65
- ☑ Labs
- ☑ Imaging (US, XR, or CT) to investigate cause
- ☑ Targeted interventions

Principles of shock management are **focused on restoring and maintaining adequate tissue perfusion** and providing specific interventions to **reverse the underlying cause**.

General approach with the ABCs (airway, breathing, circulation):

- **Airway**
  - Airway maneuvers such as repositioning, shoulder roll, and jaw thrust.
  - Airway adjuncts (nasal pharyngeal/oral airway), supraglottic device, or intubation to secure airway if necessary.
- **Breathing**
  - Administer supplementary oxygen with goal Sp02 $> 90\%$.
  - Positive pressure support to decrease work of breathing and improve ventilation as necessary.
- **Circulation**
  - Assess peripheral pulses and telemetry heart rate: provide pacing, cardioversion, defibrillation as necessary.
  - Obtain peripheral IV (PIV), central IV, or interosseous (IO) access immediately.
  - If hypovolemic or hemorrhagic shock is suspected, administer **20–30 mL/kg of isotonic crystalloid** and/or blood products (as applicable).
  - Consider **inotrope/pressor** support for distributive or cardiogenic/obstructive shock or undifferentiated but volume-unresponsive patients (Table 1.4):

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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	0.25–0.75 micrograms/kg per minute

- Target a MAP $\geq$ 65 mmHg to ensure proper perfusion of the vital organs.
- **Norepinephrine**, an inopressor, is the agent of choice in septic shock.
- Vasopressin, a pure vasopressor, can be added in cases of refractory septic shock.
- Dobutamine, an inodilator, is the preferred agent in decompensated heart failure in patients who are normotensive or mildly hypotensive. It may need to be given in conjunction with vasopressors due to its mechanism as an inodilator.
- Epinephrine, an inopressor with higher inotropic properties than norepinephrine, may be preferable to dobutamine in suspected mixed septic and cardiogenic shock. Consider as primary vasopressor in pediatric cases.

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- Consider systemic glucocorticoids (e.g., hydrocortisone 50 mg every 6 hours), in cases of shock not responding to volume resuscitation and/or multiple vasopressors.

Targeted Interventions

- Antibiotics and source control for suspected sepsis.
- Needle decompression or tube thoracostomy for tension pneumothorax.
- Pericardiocentesis for cardiac tamponade.
- Hemorrhage control, e.g., tourniquet, direct pressure, Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA), pelvic binder.
- Resuscitative thoracotomy in trauma.
- Systemic thrombolytics for suspected massive pulmonary embolism.
- Advanced cardiac life support (ACLS) protocols for unstable arrhythmia management.
- Revascularization procedure for acute coronary syndrome.
- IM Epinephrine, albuterol, antihistamines, glucocorticoid for anaphylaxis.
- Systemic glucocorticoid for suspected adrenal crisis.
- Antipyretics for fever control.

Assessing Resuscitation Efforts

- After the initial resuscitation phase or specific intervention is administered, it is important to continually assess resuscitation efforts and maintain a structured approach to reassessing the patient.
- Physiologic responses suggesting effective resuscitation are improvement in heart rate, urine output, mental status, and pulse pressure, though in isolation none of these is a reliable surrogate for volume status.
- Be aware that static markers such as CVP have limited validity and utility in assessing volume status.
- One well-validated method to assess the adequacy of volume resuscitation is the **passive leg raise maneuver.** With an arterial line in place to reliably indicate pulse pressure (SBP-DBP), transition the patient from head elevated 45°and legs flat to head flat and legs 45° elevated; an increase in pulse pressure of 10 mmHg with this change in position

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suggests volume responsiveness and may indicate further crystalloid or blood product resuscitation as is applicable.
- Improving lactate and base deficit levels suggest appropriate response to resuscitation in hemorrhagic shock.

Sudden Deterioration

- Be alert for changes in mental status, loss of pulse oximetry plethysmographic waveform, or unobtainable blood pressures; suspect cardiovascular collapse, check peripheral and central pulses, and consider early inotrope/pressor support until stabilized.
- Refractory shock requiring multiple vasopressor agents should prompt a systematic consideration of causes:
  - **Correct?** Is the blood pressure correct? Are vasopressor agents correctly infusing?
  - **Control?** Is there a need for urgent septic or hemorrhagic source control?
  - **Confounders?** Are certain medications (e.g., sedatives) worsening hypotension?
  - **Cortisol?** Is the patient adrenally insufficient and requiring stress dose steroids?
  - **Cardiac?** Is there a missed cardiac etiology such as massive pericardial tamponade?
  - **Calcium?** Is the ionized calcium low (a frequent occurrence during hemorrhagic shock resuscitation)?
  - **Compartment?** Is there abdominal compartment syndrome causing IVC compression?

Special Circumstances

Pediatric Patients

- Recognition of shock is challenging due to variations in age-dependent vital signs, difficulty in assessing mental status, and the nonspecific symptoms such as irritability and poor feeding.

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