Approach to the Patient with Shock

Chapter 314 | Part 8: Critical Care Medicine

KEY CLINICAL POINTS

1. Shock is defined as organ dysfunction resulting from an imbalance between cellular oxygen supply and demand, leading to cellular and tissue hypoxia.
2. Four major types of shock are classified by hemodynamic profiles: Distributive (low SVR), Cardiogenic (low CO), Obstructive (extracardiac mechanical issues), and Hypovolemic (low preload).
3. Shock progresses through three stages: Compensated (preshock), Shock (decompensated), and Irreversible.
4. Lactate is a product of anaerobic metabolism; elevated levels correlate with worse outcomes and are used for risk stratification.
5. The Shock Index (SI = HR/SBP) > 0.9 is a sensitive indicator of transfusion requirement and critical bleeding in hypovolemic shock.
6. The qSOFA score (SBP ≤ 100, GCS < 15) identifies patients with sepsis at risk of death or prolonged ICU stay; a score ≥ 2 indicates high risk.
7. Distributive shock is unique for having a compensatory increase in cardiac output (CO).
8. Obstructive shock involves extracardiac mechanical processes such as tamponade or pulmonary embolism.
9. ECMO provides circulatory and/or respiratory support for refractory hypoxemia or shock, with specific types including VA-ECMO, VV-ECMO, and ECCOR.
10. Early identification of shock is critical to prevent the transition from reversible organ dysfunction to irreversible multisystem organ failure.

1. DEFINITION & OVERVIEW

- **Definition:** Shock is the clinical condition of organ dysfunction resulting from an imbalance between cellular oxygen supply and demand, leading to cellular and tissue hypoxia.
- **Clinical Context:** A common reason for ICU admission; requires prompt identification and treatment to prevent irreversible organ dysfunction.
- **Stages of Shock:**
  - **Compensated Shock (Preshock):** Body utilizes physiologic responses to counteract the initial insult. No overt signs of significant organ dysfunction. Laboratory evaluation may show only mild elevation in creatinine, troponin, or lactate.
  - **Shock (Decompensated Shock):** Compensatory responses are overwhelmed; evidence of organ dysfunction (elevated lactate, oliguria, altered mental status).
  - **Irreversible Shock:** Organ dysfunction is permanent; patient progresses to multisystem organ dysfunction and death.

2. EPIDEMIOLOGY

- **Setting-Dependent Prevalence:**
  - **Emergency Department (ED):**
  - Hypovolemic: 30.8%
  - Septic: 27.2%
  - Distributive (nonseptic): 23.4%
  - Cardiogenic: 14%
  - Obstructive: 0.9%
  - **ICU Setting:**
  - Septic shock predominates (62%).
  - Hypovolemic: 16%
  - Cardiogenic: 15%
  - Obstructive: 2%
  - **Specialized ICUs:**
  - Medical ICU: High prevalence of distributive shock related to sepsis.
  - Cardiac ICU: 66% of shock patients are assessed as having cardiogenic shock.
- **Mortality Rates:**
  - Septic shock: 56.2% (90-day mortality in ED study).
  - Cardiogenic shock: 52.3% (90-day mortality in ED study).
  - Hypovolemic shock is associated with a lower mortality rate.

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3. ETIOLOGY & PATHOPHYYSICOLOGY

- **Mechanism of Injury:**
  - Inadequate oxygen delivery → failure of aerobic metabolism.
  - Shift to anaerobic metabolism → pyruvate converted to lactate with significantly less ATP generation.
  - Decreased ATP → failure of Na+/K+ ATPase (consumes 20–80% of cell energy) → loss of osmotic, ionic, and pH homeostasis.
  - Calcium influx → activation of calcium-dependent phospholipases and proteases → cellular swelling and death.
- **Determinants of Oxygen Delivery (DO):**
  - Formula: DO = CO imes CaO_2
  - Cardiac Output (CO) components: CO = HR imes SV
  - Stroke Volume (SV) determinants: $SV \propto (Preload imes Contractility) / SVR$
  - Arterial Oxygen Content (CaO_2): $CaO_2 = (Hb imes 1.34 imes SaO_2) + (PaO_2 imes 0.003)$
- **Pathophysiology of Lactate:**
  - Produced from anaerobic glucose metabolism.
  - Elevated lactate correlates with worse outcome and is used for risk stratification.
- **Table 314-1: Physiologic Classification of Shock**
  - **Distributive:** Septic shock, Pancreatitis, Severe burns, Anaphylactic shock, Neurogenic shock, Endocrine shock, Adrenal crisis.
  - **Cardiogenic:** Myocardial infarction, Myocarditis, Arrhythmia, Valvular (i. Severe aortic valve insufficiency, ii. Severe mitral valve insufficiency).
  - **Obstructive:** Tension pneumothorax, Cardiac tamponade, Constrictive pericarditis, Pulmonary embolism, Aortic dissection.

– **Hypovolemic:** Hemorrhagic (i. Trauma, ii. GI bleeding, iii. Ruptured ectopic pregnancy, Gi losses, Burns, Polyuria), Nonhemorrhagic (i. Diabetic ketoacidosis, ii. Diabetes insipidus).

3.1 Hemodynamic Profiles

- **Distributive Shock:**
  – Primary defect: \downarrow SVR.
  – Compensation: \uparrow CO (unique among shock types).
- **Cardiogenic Shock:**
  – Primary defect: \downarrow CO due to primary cardiac problem.
  – Compensation: \uparrow SVR.
- **Obstructive Shock:**
  – Primary defect: \downarrow CO due to extracardiac pulmonary vascular or mechanical process.
  – Compensation: \uparrow SVR.
- **Hypovolemic Shock:**
  – Primary defect: \downarrow Preload (low CVP/PCWP) → \downarrow CO.
  – Compensation: \uparrow SVR.
- **Table 314-2: Hemodynamic Characteristics of the Major Types of Shock**
  – Distributive: CVP \downarrow, PCWP \downarrow, CO \uparrow, SVR \downarrow
  – Cardiogenic: CVP \uparrow, PCWP \uparrow, CO \downarrow, SVR \uparrow
  – Obstructive: CVP \uparrow, PCWP \downarrow \uparrow, CO \downarrow, SVR \uparrow
  – Hypovolemic: CVP \downarrow, PCWP \downarrow, CO \downarrow, SVR \uparrow

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3.2 Mixed and Undifferentiated Shock

- **Mixed Shock:**
  – Example: Sepsis-induced cardiomyopathy (Distributive + Cardiogenic).
  – Other conditions with reduced SVR: Pancreatitis, severe burns, liver failure.
- **Anaphylaxis:**
  – IgE-mediated; rapid onset of profound distributive shock.
  – Evidence of both venous and arterial vasodilation.
  – Up to 35% of circulating blood volume can extravasate within 10 min.
- **Neurological Injury:**
  – Brain/spinal cord injury → reduced SVR due to autonomic pathway disruption; pooling of blood in the venous system → decreased CO.
- **Adrenal Insufficiency:**
  – Related to chronic steroid use, medications (e.g., immune checkpoint inhibitors), or other pathologies (malignancy, infection, autoimmune).
  – Deficit in cortisol leads to vasodilation; aldosterone deficiency leads to hypovolemia.

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4. CLINICAL FEATURES

- **Vital Signs & Scoring:**
  – **Shock Index (SI):** HR/SBP

– Normal: 0.5–0.7.

– >0.9: Sensitive indicator of transfusion requirement and critical bleeding in hypovolemic shock; identifies risk for postintubation hypotension.

– **qSOFA Score:**

– Points awarded for: SBP ≤ 100 or altered mental status (GCS <15).

– ≥ 2 with concern for infection → significantly greater risk of death or prolonged ICU stay.

- **Laboratory Indicators:**

– **Alkaline Phosphatase:** Elevation may suggest biliary obstruction (source of infection in distributive shock).

– **Cardiac Enzymes:** Indicate myocyte damage (ischemia, myocarditis, or pulmonary embolism).

– **WBC Count:** Elevated count with left shift suggests infective process.

– **Hemoglobin/Hematocrit:** Reductions seen in hemorrhagic hypovolemic shock.

– **Urinalysis:** Sent to evaluate for pyuria.

- **Physical Examination Findings:**

– **Cardiogenic signs:** Rales on pulmonary auscultation, S3 gallop.

– **Obstructive signs:**

– Pulsus paradoxus and elevated JVP → Cardiac tamponade.

– Reduced breath sounds, tracheal deviation, or subcutaneous emphysema → Tension pneumothorax.

– **Hypovolemic signs:** Large ecchymosis (trauma/retroperitoneal bleed), rectal exam for GI hemorrhage.

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5. DIFFERENTIAL DIAGNOSIS

- **Identification of Shock Type:**

– Distinguish cardiogenic from obstructive shock via physical examination.

– Identify specific etiologies (e.g., pulmonary embolism, aortic dissection) through history and imaging.

- **Specific Clinical Scenarios:**

– **Anaphylaxis:** Rapid onset of distributive shock; potential for massive fluid extravasation.

– **Adrenal Insufficiency:** Suspect in patients with no response to fluids or in those with known steroid use.

– **Neurological Injury:** Suspect reduced SVR and pooling of blood in the venous system.

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6. INVESTIGATIONS & DIAGNOSIS

1. **Initial Laboratory Evaluation (Table 314-4):**

– 1. Lactate

– 2. Renal function tests

– 3. Liver function tests

– 4. Cardiac enzymes

– 5. Complete blood count (with differential)

– 6. PT, PTT, and INR

– 7. Pregnancy test

– 8. Urinalysis and urine sediment

– 9. Arterial blood gas (ABG) - if hypoxemia is present.

– 10. ECG
– 11. CXR

2. **Imaging & Monitoring:**
– **ECG:** Assess for arrhythmias, ischemia, or signs of pulmonary embolism/tamponade.
– **CXR:** Evaluate for infection, pulmonary edema, or pneumothorax.
– **Vascular Access:** Required for hemodynamic monitoring and fluid/medication administration.

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7. MANAGEMENT & TREATMENT

1. **Core Principles (Table 314-3):**
– 1. Recognize shock early.
– 2. Assess for type of shock present.
– 3. Initiate therapy simultaneously with the evaluation into the etiology of shock.
– 4. Involve all members of the multidisciplinary team.
– 5. Aim of therapy is to restore oxygen delivery (DO).
– 6. Identify etiologies of shock that require additional lifesaving interventions.

2. **Extracorporeal Gas Exchange (ECMO) (Table 313-4):**
– **VA-ECMO (venoarterial):**
– Mechanism: Deoxygenated blood drains via venous catheter to a pump and membrane; blood is then returned to the arterial system.
– Features: Circulatory and respiratory support.
– Technical Notes: Requires large vascular catheters (16–30 Fr); higher blood flow rates (2–6 L/min).
– **VV-ECMO (venovenous):**
– Mechanism: Deoxygenated blood drains via venous catheter to a pump and membrane; blood is then returned to the venous system.
– Features: Respiratory support only.
– **ECCOR (extracorporeal CO removal):**
– Mechanism: Venous catheter drains blood to a CO removal device; blood then returns via a venous catheter.
– Features: Partial respiratory support, CO removal only.
– Technical Notes: Requires smaller vascular catheters (14–18 Fr); lower blood flow rates (0.25–2 L/min).

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8. PROGNOSIS & COMPLICATIONS

• **Early Intervention:** The goal is to identify the patient with shock promptly and initiate therapy to prevent irreversible organ dysfunction.
– **Reversibility:** Organ dysfunction in early shock is often reversible with restoration of adequate oxygen supply.
– **Multisystem Organ Dysfunction:** If left untreated, shock progresses to an irreversible phase leading to death.

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9. SPECIAL CONSIDERATIONS

• **Pregnancy:** Routine pregnancy test in all patients with undifferentiated shock.

– **Infection:** High index of suspicion for infection in all cases of undifferentiated shock; obtain blood, urine, and sputum cultures.

10. KEY PEARLS & CLINICAL TRAPS

- **Shock Definition:** Focus on the imbalance of oxygen supply vs. demand → cellular hypoxia.
  – **Distributive Shock:** Unique because it features a compensatory increase in Cardiac Output (CO).
  – **Lactate:** A tool for risk stratification and monitoring response to therapy.
  – **Shock Index:** HR/SBP > 0.9 is highly sensitive for identifying patients needing immediate transfusion or having critical bleeding.
  – **qSOFA:** A rapid assessment scale; a score ≥ 2 with concern for infection indicates high risk of death.

REFERENCE TABLES

TABLE 313-4

Main Types and Key Features of Extracorporeal Gas Exchange

TERM	DESCRIPTION	KEY FEATURES	IMPORTANT TECHNICAL NOTES
VA-ECMO (venoarterial extracorporeal membrane oxygenation)	Deoxygenated blood drains via venous catheter to a pump and membrane oxygenator; blood is then returned to the arterial system	Circulatory and respiratory support	Requires large vascular catheters (16–30 Fr) Higher blood flow rates (2–6 L/min)
	Deoxygenated blood drains via venous catheter to a pump and membrane oxygenator; blood is then returned to the venous system	Respiratory support	
ECCOR (extracorporeal CO removal) **2 2**	Venous catheter drains blood to a CO removal 2 device; blood then returns via a venous catheter	Partial respiratory support, CO removal only 2	Requires smaller vascular catheters (14–18 Fr) Lower blood flow rates (0.25–2 L/min)
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TABLE 314-1

Physiologic Classification of Shock Distributive

- Distributive Septic shock Pancreatitis Severe burns Anaphylactic shock Neurogenic shock Endocrine shock Adrenal crisis Cardiogenic Myocardial infarction Myocarditis Arrhythmia Valvular i. Severe

aortic valve insufficiency ii. Severe mitral valve insufficiency Obstructive Tension pneumothorax Cardiac tamponade Constrictive pericarditis Pulmonary embolism Aortic dissection Hypovolemic Hemorrhagic i. Trauma ii. GI bleeding iii. Ruptured ectopic pregnancy GI losses Burns Polyuria i. Diabetic ketoacidosis ii. Diabetes insipidus

TABLE 314-2

Hemodynamic Characteristics of the Major Types of Shock

TYPE OF SHOCK	CVP	PCWP	CARDIAC OUTPUT	SYSTEMIC VASCULAR RESISTANCE
Distributive	↓	↓	↑	↓
	↑	↑	↓	
Obstructive	↑	↓↑	↓	↑
	↓	↓	↓	

TABLE 314-3

Key Principles in the Treatment of Shock 1. Recognize shock early 2. Assess for type of shock present 3. Initiate…

- 1. Recognize shock early 2. Assess for type of shock present 3. Initiate therapy simultaneous with the evaluation into the etiology of shock 4. Involve all members of the multidisciplinary team 5. Aim of therapy is to restore oxygen delivery 6. Identify etiologies of shock that require additional lifesaving interventions

TABLE 314-4

Initial Laboratory Evaluation of Undifferentiated Shock 1. Lactate 2. Renal function tests 3. Liver function tests 4.…

- 1. Lactate 2. Renal function tests 3. Liver function tests 4. Cardiac enzymes 5. Complete blood count (with differential) 6. PT, PTT, and INR 7. Pregnancy test 8. Urinalysis and urine sediment 9. Arterial blood gas 10. ECG 11. CXR