

NOTES SHOCK

SHOCK

osms.it/shock

PATHOLOGY & CAUSES

- Global inadequate tissue perfusion
 - Extremely low blood pressure (BP) \rightarrow end-organ failure

TYPES

 Hypovolemic shock, cardiogenic shock, obstructive shock, distributive shock

Hypovolemic Shock

- General clinical manifestations
 Reduced preload with suspected cause
- Variable presentation based on etiology of fluid loss
- Hemorrhage, evidence of trauma
 - Internal bleeding into thoracic/peritoneal/ retroperitoneal space
- Nonhemorrhagic fluid loss
 - Decreased tissue perfusion
 - Elevated blood urea nitrogen, serum

creatinine concentration (non-specific, i.e. seen in all forms of shock)

- Abnormal potassium levels
- Metabolic acidosis/alkalosis
- Hematocrit, serum albumin concentration → reduction in plasma volume increases concentration

Cardiogenic Shock

- General clinical manifestations
 - Hypotension, manifestations of pulmonary edema
- Subtypes of cardiogenic shock
 - Myopathic: find specific cause via ECG/ lab values/chest radiograph
 - Arrhythmogenic: caused by arrythmia

Obstructive Shock

- General clinical manifestations
 - Low preload; obstruction of blood flow outside the heart
 - Cardiac tamponade, pulmonary embolism, tension pneumothorax



Figure 18.1 Illustration summarizing the causes and effects of hypovolemic, cardiogenic, and distributive shock.

Distributive Shock

- General clinical manifestations
 - Hypotension without reduced preload, fluid overload
- Subtypes of distributive shock
 - Septic: caused by infection
 - Anaphylactic: allergic reaction → respiratory distress, vomiting, abdominal pain, chest pain, dysrhythmia, collapse
 - Neurogenic: pain at site of spinal fracture, evidence of spinal injury (loss of sensation, paralysis, loss of reflexes)
 - Endocrine: adrenal crisis (nonspecific symptoms, eg. anorexia, nausea, vomiting, abdominal pain, fatigue, lethargy, weakness, fever, confusion, coma); confirmation of adrenal insufficiency

RISK FACTORS

- Dependent upon type
- Septic shock most common in United States, followed by cardiogenic, hypovolemic, other forms of distributive/ obstructive shock
- Hypovolemic shock from gastrointestinal (GI) losses/dehydration most common in low-income countries

STAGING

Initial

Cellular, not clinically apparent

Compensatory

- Neural, hormonal, biochemical compensation to maintain homeostasis; inadequate perfusion → autonomic nervous system attempts to compensate
 - Sympathetic nervous system

| CAUSES OF SHOCK | | | | | | |
|-----------------|-----------------------|-------------------------|--|--|--|--|
| | SUB-TYPE | PHYSIOLOGIC CHANGE | CAUSE | | | |
| HYPOVOLEMIC | Hemorrhagic | Low preload | Trauma | | | |
| | Non-hemorrhagic | Low preload | Vomiting, pancreatitis, diarrhea bowel obstruction, severe burns, fistula drainage, diabetes insipidus | | | |
| CARDIOGENIC | Myopathic | Low contractility | Myocardial infarction, acute decompensation of any etiology of chronic heart failure, blunt cardiac injury, myocarditis | | | |
| | Arrhythmogenic | Low HR, Low preload | Arrhythmia | | | |
| OBSTRUCTIVE | Obstructive | Low preload | Cardiac tamponade, pulmonary embolism, tension pneumothorax | | | |
| DISTRIBUTIVE | Septic | Low SVR | Infection | | | |
| | Neurogenic | Low SVR, Low HR | Trauma | | | |
| | Anaphylactic | Low SVR | Allergic reactions: insect bites or stings, drugs, allergies, IV contrast | | | |
| | Endocrine/hypoadrenal | Low SVR, Low preload | Adrenal insufficiency | | | |

vasoconstriction, ↑ contractility

 Release of catecholamines, vasopressin, angiotensin II → ↑ vasoconstriction, ↑ retention water, sodium → ↑ SVR, ↑ blood volume → ↑ BP → ↑ perfusion

Progressive

 Compensation fails, requires aggressive interventions to prevent multiple organ dysfunction syndrome

Irreversible

 Decreased perfusion (vasoconstriction, decreased cardiac output) → anaerobic metabolism; profound hypotension, hypoxemia, organ failure; recovery unlikely

SIGNS & SYMPTOMS

- Altered mental state, decreased peripheral pulse, tachycardia, hypotension
- Varies by type and subtype of shock (see table below)

DIAGNOSIS

DIAGNOSTIC IMAGING

Chest radiography

- Clear in hypovolemic/obstructive shock
 from pulmonary embolism
- Pneumonia
 - Septic shock
- Pneumothorax
 - Obstructive shock
- Pulmonary edema
 - Cardiogenic shock/ARDS

Pulmonary artery catheterization

- Hemodynamic measurements can be helpful
- Measure cardiac output, systemic vascular resistance, pulmonary artery occlusion pressure, right atrial pressure, mixed venous oxyhemoglobin saturation
- Rarely necessary to identify etiology of shock

Ultrasound/echocardiography

• Allows visualization of altered cardiac function

- Preserved/hyperdynamic left ventricle = distributive shock
- Point-of-care ultrasond
 - Examination of heart → cause of cardiogenic shock, obstructive shock

Focused assessment and sonography for trauma (FAST)

• Fast ultrasound examination for hemopericardium, intra-abdominal bleeding; rule out/in hypovolemic shock

Hemodynamic monitoring

- Via central venous catheters
- Elevated central venous pressure, low mixed venous oxygen saturation = cardiogenic shock

LAB RESULTS

Elevated serum lactate

• Early indicator, reflective of poor tissue perfusion

Renal, liver function tests

- Elevated blood urea nitrogen (BUN), creatinine, transaminases indicate endorgan damage
 - May help point to cause (acute hepatitis, chronic cirrhosis)

Coagulation studies, D-dimer level

 Elevated fibrin split products, elevated
 D-dimer level, low fibrinogen level = severe shock

Cardiac enzymes, natriuretic peptides

 Elevated troponin, creatine phosphokinase, N-terminal pro-brain natriuretic peptide, brain natriuretic peptide = cardiogenic shock due to ischemia/pulmonary embolism

Complete blood count, differential

- High hematocrit
 - Hemoconcentration from nonhemorrhagic hypovolemic shock
- Anemia, bleeding
 - Hemorrhagic shock
- Elevated eosinophil
 - Allergy, anaphylactic shock
- Leukocytosis
 - Septic shock, not specific; more common

in septic shock, may also occur in other types of shock as sign of poor prognosis

Coagulation studies, D-dimer level

- Elevated prothrombin time, international normalized ratio, activated partial thromboplastin time
 - Septic shock, other issues (e.g. sepsis, systemic inflammatory response syndrome); elevated D-dimer levels common in septic shock

Peripheral O₂ sat via pulse oximetry

- Hypoxemia
 - Obstructive, cardiogenic shock

Urinalysis

Infection, septic shock

Material gram stain from infection sites

Septic shock

Blood culture

 identifies causative microbe in case of septic shock; directs targeted antibiotic therapy

OTHER DIAGNOSTICS

History & physical

 Low blood pressure, tachycardia, tachypnea, signs of poor end-organ perfusion (low urine output, confusion, loss of consciousness), weak pulse, cool skin, metabolic acidosis, hyperlactatemia

Shock index

- Heart rate divided by systolic pressure
 - Normal range 0.5–0.8
 - If index higher, increased suspicion of underlying state of shock
 - Most useful for isolated hypotension/ tachycardia

ECG

- Arrhythmia, ST segment changes consistent with ischemia
- Low-voltage ECG
 - Pericardial effusion
- Arrhythmia
 Arrhythmogenic cardiogenic shock
- Ischemia
 - Myopathic cardiogenic shock

BLOOD PRESSURE CHANGES IN SHOCK

| | PRELOAD (PWP) | PUMP FUNCTION (CO) | SVR | TISSUE PERFUSION (Sv02) |
|---|------------------|-----------------------|-----|-------------------------------|
| HYPOVOLEMIC | Ļ | Ļ | ¢ | Ļ |
| CARDIOGENIC | Ŷ | Ļ | Ŷ | Ļ |
| DISTRIBUTIVE | Ļ | ↑ (early), ↓ (late) | Ļ | Ť |
| OBSTRUCTIVE (PULMONARY EMBOLISM, HYPERTENSION, TENSION PNEUMOTHORAX) | Ļ | Ļ | ¢ | Ļ |
| OBSTRUCTIVE (PERICARDIAL TAMPONADE) | î | Ļ | ſ | Ļ |



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Treatment for shock Airway: ensure clear airway, possibly intubate

- Breathing: assist individual in breathing, mechanical ventilation/sedation
- Circulation: administer fluids (e.g. isotonic crystalloid)
- Delivery of oxygen: monitor lactate levels
- Endpoint resuscitation (specific to septic shock)

TREATMENT

• See chart for a detailed summary of treatments for different forms of shock

OTHER INTERVENTIONS

Surviving sepsis campaign guidelines

- End resuscitation when urine output 0.5ml/ kg/hr, central venous pressure (CVP) 8–12 mmHg, mean arterial pressure (MAP) 65–90mmHg, central venous oxygen concentration > 70%, normalize lactate levels
 - CVP 8–12mmHg (recent literature shows CVP poorly predicts fluid responsiveness, poor marker of adequate resuscitation)

TREATMENTS FOR SHOCK

| | SUB-TYPE | TREATMENT | |
|--------------|-----------------------|--|--|
| HYPOVOLEMIC | Non-hemorrhagic | - Fluid resuscitation - Prevent hypothermia caused by fluid resuscitation | |
| | Hemorrhagic | - Same treatment as non-hemorrhagic shock - Packed red blood cell transfusion | |
| CARDIOGENIC | Cardiogenic | - Diuretics - Inotropes - Intra-aortic balloon pump - ACE inhibitors - Hydralazine | |
| | Obstructive | - Treat underlying cause | |
| DISTRIBUTIVE | Septic | - Antibiotics - Fluid resusitation - Vasopressors | |
| | Anaphylactic | - Epinephrine - Antihistamines - Fluid resusitation | |
| | Neurogenic | - Fluid resusitation - Vasopressors - Corticosteroids | |
| | Endocrine/hypoadrenal | - Corticosteroids | |