

Shock: Presentation Handout

Adapted from Harding et al., 2023

CATEGORIES OF SHOCK

- **Cardiogenic:** Systolic/diastolic dysfunction (MI, cardiomyopathy, structural factors)
 - **Hypovolemic:**
 - Absolute: External blood/fluid loss (hemorrhage, vomiting, diarrhea)
 - Relative: Fluid shifts to extravascular space (burns, sepsis—third spacing)
 - **Distributive:** Maldistribution of blood flow (neurogenic, anaphylactic, septic)
 - **Obstructive:** Physical obstruction to blood flow (PE, cardiac tamponade, tension pneumothorax)
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STAGES OF SHOCK

Initial Stage

- Metabolism switches from aerobic to anaerobic
- Lactic acid buildup (requires oxygen to be removed by liver)
- Not clinically apparent

Compensatory Stage

- Body activates neural, hormonal, biochemical compensatory mechanisms to attempt to maintain homeostasis
- Clinical presentation reflects body's compensatory responses
- Classic sign: Hypotension from ↓ CO
- SNS activation: vasoconstriction, release of epinephrine/norepinephrine
- Blood flow maintained to heart and brain

Progressive Stage

- Compensatory mechanisms fail
- Fluid shifts out of capillaries into interstitial space
- Mental status changes
- Organ dysfunction begins

Refractory Stage

- Severe intravascular volume loss
- Severe hypotension and hypoxemia
- Waste products accumulate from organ failure
- Recovery unlikely

CLINICAL MANIFESTATIONS BY SYSTEM

CARDIOVASCULAR SYSTEM

Compensatory:

- Tachycardia (heart pumps faster to maintain CO)
- ↑ Contractility
- Narrowed pulse pressure
- Coronary artery dilation

Progressive:

- ↓ BP (MAP <60 mm Hg)
- Dysrhythmias (myocardial ischemia)
- Myocardial ischemia
- ↑ Capillary permeability → further fluid shift
- ↑ SVR (increased cardiac workload)

Refractory:

- Profound hypotension
- Bradycardia (paradoxical—reflects cardiac damage)
- Inadequate CO for vital organs

RESPIRATORY SYSTEM

Compensatory:

- Tachypnea (hyperventilation to blow off CO₂)
- ↑ Minute ventilation
- ↑ Physiological dead space

Progressive:

- ARDS develops (acute respiratory distress syndrome)
- ↑ Capillary permeability from inflammatory mediators
- Pulmonary edema (fluid in interstitial space → alveoli)
- Moist crackles on auscultation
- ↓ Lung compliance
- Type I pneumocytes destroyed
- Type II pneumocytes dysfunctional → ↓ surfactant production

- Alveoli collapse

Refractory:

- Respiratory failure
 - Refractory hypoxemia (doesn't respond to $\uparrow O_2$)
 - Severe hypoxemia
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GASTROINTESTINAL SYSTEM

Compensatory:

- $\downarrow$ Blood supply to GI tract
- $\downarrow$ GI motility
- Hypoactive bowel sounds
- Risk for paralytic ileus

Progressive:

- Ischemic gut
- Breakdown of protective mucosal barrier
- Stress ulcers (erosive ulcers)
- GI bleeding
- **Bacterial translocation** (bacteria cross from GI tract into bloodstream)
- Can trigger/worsen sepsis

Refractory:

- Profound ischemic gut
 - May be irreversible
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HEPATIC SYSTEM

Compensatory:

- $\downarrow$ Hepatic perfusion

Progressive:

- Hepatocyte death from ischemia
- $\uparrow$ Liver enzymes (ALT, AST, GGT)
- Liver dysfunction (may exist before clinically evident)
- Impaired protein synthesis
- Cannot metabolize medications $\rightarrow$ risk of drug toxicity

- Cannot clear bilirubin → jaundice
- Cannot convert ammonia to urea → ↑ NH_3 (contributes to confusion)
- Cannot convert lactate to glucose → lactic acidosis

Refractory:

- Liver cannot maintain adequate glucose → hypoglycemia
 - Metabolic acidosis from waste accumulation (NH_3 , lactate, CO_2)
 - ↓ Plasma oncotic pressure (cannot synthesize albumin) → fluid leaks to interstitial space
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NEUROLOGICAL SYSTEM

Compensatory:

- Anxiety, restlessness
- Confusion
- Change in LOC
- Apprehension

Progressive:

- ↓ Cerebral perfusion pressure
- ↓ Cerebral blood flow
- ↓ Responsiveness to stimuli
- Delirium

Refractory:

- Unresponsive
 - Areflexia (loss of reflexes)
 - Fixed, dilated pupils (indicates severe brain injury)
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RENAL SYSTEM

Compensatory:

- ↓ Renal blood flow
- RAAS activation:
 - Renin release → vasoconstriction
 - Aldosterone release → Na^+ / H_2O reabsorption
 - ADH release → water retention
- Kidneys still functioning, just trying to preserve volume

Progressive:

- Acute tubular necrosis (prolonged ischemia → actual kidney damage)
- Oliguria (<0.5 mL/kg/hr)
- ↑ BUN and creatinine
- BUN:Cr ratio >20:1
- ↓ Urine sodium
- ↓ Urine osmolality/specific gravity (damaged tubules cannot concentrate urine)
- Cannot excrete H⁺ ions → metabolic acidosis worsens
- Hyperkalemia

Refractory:

- Complete kidney failure
 - Anuria (no urine production)
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INTEGUMENTARY SYSTEM**Compensatory:**

- Pale and cool (vasoconstriction to preserve core organs)
- OR warm and flushed (in distributive shock—e.g., septic)

Progressive:

- Cold and clammy
- Diaphoresis (from SNS activation)

Refractory:

- Mottled skin (patchy purple discoloration)
- Cyanotic (severe hypoxemia)
- Mottling advancing proximally = very poor prognosis

KEY DIAGNOSTIC LABS

Lab	Finding	Significance
Lactate	↑	Key marker of tissue hypoperfusion; byproduct of anaerobic metabolism
ABGs	Respiratory alkalosis → Metabolic acidosis	Early: hyperventilation. Late: lactic acid accumulation
BUN/Creatinine	Both ↑; ratio >20:1	Prerenal kidney injury (reversible if perfusion restored)
Urine Specific Gravity	↑ early; fixed at 1.010 late	Early: ADH action. Late: renal failure (cannot concentrate urine)
Liver Enzymes	↑ (ALT, AST, GGT)	Hepatocyte destruction in progressive shock
Glucose	↑ early; ↓ late	Early: SNS/cortisol/liver glycogen release. Late: depleted stores + liver dysfunction

NURSING MANAGEMENT

Overall Goals:

- MAP ≥65 mm Hg
- Restore adequate tissue perfusion
- Recovery of organ function
- Prevention of complications

Key Nursing Interventions:

- **Neurological:** Assess orientation, LOC q1-2h (best indicator of cerebral blood flow)
 - **Cardiovascular:** Monitor HR, BP, hemodynamic parameters; treat hypotension (fluids, vasopressors); continuous ECG
 - **Respiratory:** Assess rate, depth, rhythm q15-30 min; auscultate breath sounds; pulse oximetry; ABGs
 - **Renal:** Hourly urine output (oliguria = <0.5 mL/kg/hr); monitor BUN/creatinine
 - **Temperature:** Monitor q4h if normal, q1h if abnormal; manage fever (NSAIDs, acetaminophen, cooling)
 - **Skin:** Assess temperature, color, cyanosis, diaphoresis; capillary refill
 - **GI:** Auscultate bowel sounds q4h; assess distension; check stool for occult blood
 - **Hygiene:** Balance skin integrity needs with oxygen demands; use clinical judgment
 - **Emotional Support:** Recognize anxiety, fear, pain; address with medications and compassionate care
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COMPLICATIONS

MODS (Multiple-Organ Dysfunction Syndrome)

- Failure of ≥ 2 organ systems
 - Homeostasis cannot be maintained without intervention
 - **Mortality:** 40% (2 organs) $\rightarrow$ 100% (7 organs)
 - Most common cause: sepsis
 - Results from uncontrolled inflammatory response and prolonged hypoperfusion
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ARDS (Acute Respiratory Distress Syndrome)

- Develops in **14% of patients with sepsis; 45% mortality**
- Inflammatory mediators $\rightarrow$ $\uparrow$ pulmonary capillary permeability
- Fluid moves into interstitial space $\rightarrow$ alveoli
- Type I pneumocytes destroyed
- Type II pneumocytes dysfunctional $\rightarrow$ $\downarrow$ surfactant production
- Alveoli collapse $\rightarrow$ $\uparrow$ shunt, worsening V/Q mismatch
- Requires aggressive pulmonary management with mechanical ventilation

Reference:

Harding, M. M., Kwong, J., Roberts, D., Hagler, D., & Reinisch, C. (Eds.), & Tyerman, J., & Cobbett, S. L. (2023). *Lewis's medical-surgical nursing in Canada: Assessment and management of clinical problems* (5th ed.). Elsevier.