Circulatory shock
Types, Etiology, Pathophysiology

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Physiology of Circulation:
The Vessels

- 600,000 miles of vessels containing 5-6 liters of blood
- Vessel tone is controlled by the sympathetic and parasympathetic nervous system.
- Pre-capillary sphincters control blood flow through the capillaries in response to O₂ demand of the tissue.
- Preload is dependant on constant peripheral vascular resistance.
Microcirculation

-Responsive to local tissue needs
-Capillary beds can adjust size to supply undernourished tissue and bypass tissue with no immediate need
-Pre-capillary sphincters and post capillary sphincters open and close to feed or bypass tissues

Causes of Inadequate Perfusion

- Inadequate pump
  - Inadequate preload
  - Poor contractility
  - Excessive afterload
  - Inadequate heart rate
- Inadequate fluid volume
  - Hypovolemia
- Inadequate container
  - Excessive dilation
  - Inadequate systemic vascular resistance
Hemodynamic Parameters

- Systemic Vascular Resistance (SVR)
- Cardiac Output (CO)
- Mixed Venous Oxygen Saturation (SvO2)
- Central Venous Pressure (CVP)

Syncope

- Fainting - lack of blood flow to the brain
- Can be confused with a neurological condition (seizure)
- Difference
  - Episode begins in a standing position
  - Patient remembers feeling faint or lightheaded
  - Patient becomes responsive almost immediately after becoming supine
  - Skin is usually pale and moist
Shock

**definition**

Profound and widespread failure of the circulation leading to inadequate tissue perfusion (e.g. cellular hypoxia and injury and vital organ dysfunction)

Leads to Multiple Organ Dysfunction Syndrome (MODS)

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**Shock – Overview**

- The effects of tissue hypoperfusion are initially reversible, but lead to cellular hypoxia which can cause:
  - Cell membrane and ion pump dysfunction
  - Intracellular edema
  - Leakage of intracellular contents into the extracellular space
  - Inadequate regulation of intracellular pH
General Pathophysiology of Shock States

Inadequate perfusion
↓
Cell hypoxia
↓
Energy deficit
↓
Lactic acid accumulation and → Anaerobic metabolism
↓
Fall in pH
↓

Vasoconstriction
↓
Metabolic acidosis
↓

Failure of pre-capillary sphincters
↓
Cell membrane dysfunction and failure of sodium pump
↓
Intracellular lysosomes release digestive enzymes
↓
Influx of potassium
↓
Influx of sodium and water
↓
Toxic substances enter circulation
↓
Capillary endothelium damaged
↓
Further destruction, dysfunction and cell death

Classifications of Shock

1. Hypovolemic shock; (classic shock)
   - The most common form. It is a standard used to compare other forms of shock in differential diagnosis.
     - Hemorrhagic / Blood loss
     - Dehydration / Fluid loss

2. Extracardiac Obstructive Shock:
   - Pulmonary Embolism / Blocked pulmonary circulation
   - Tension Pneumothorax / Increased intrathoracic pressure
   - Cardiac Tamponade / Pressure on myocardium. Decreased preload.
Classifications of Shock

3. **Cardiogenic Shock:**
   - Heart (pump) Failure (40% of Myocardium damaged by AMI)

4. **Distributive Shock:**
   - **Neurogenic** / Spinal cord injury, drug overdose or poisoning which affects nervous systems ability to maintain vascular tone leading to vasodilation.
   - **Anaphylactic** / Vasodilation and fluid shifting from capillary to cell. Leads to micro clotting (hives) and smooth muscle contraction (bronchospasm)
   - **Septic** / vasodilation and fluid shifting due to overwhelming infection.
HYPOVOLEMIC SHOCK

- Shock due to loss of intravascular fluid volume
- Possible causes
  - Internal or external hemorrhage
  - Traumatic hemorrhage
  - Long bone or open fractures
  - Severe dehydration from GI losses
  - Plasma losses from burns
  - Diabetic ketoacidosis
  - Excessive sweating

HYPOVOLEMIC SHOCK

- Also can result from internal third-space loss
- Possible causes
  - Bowel obstruction
  - Peritonitis
  - Pcreamitis
  - Liver failure resulting in ascites
CARDIOGENIC SHOCK

- Inability to pump enough blood to supply all body parts
- Primary cause is severe left ventricular failure (AMI, CHF)
  - Accompanying hypotension decreases coronary artery perfusion, worsening the situation
  - Other compensatory mechanisms-increased peripheral resistance, increased myocardial O2 demand -worsen situation

CARDIOGENIC SHOCK

- Other causes
  - Chronic progressive heart disease
  - Rupture of papillary heart muscles or intraventricular septum
  - End-stage valvular disease
- Patients may be normovolemic or hypovolemic
CARDIOGENIC SHOCK

- R.A.S. Activation
- ↑ CO
- ↓ Catecholamine Release
- ↑ SVR
- ↓ O₂ supply
- ↓ Peripheral & pulmonary edema
- ↑ Dyspnea

Impaired myocardial function

Myocardial O₂ demand

Volume/Preload

DISTRIBUTIVE SHOCK

- Shock resulting from inadequate peripheral resistance due to widespread vasodilation
- Common causes
  - Sepsis
  - Anaphylaxis
  - Spinal cord injury
  - Central nervous system injuries
  - Insulin overdose
  - Addisonian crisis
- No sympathetic response
DISTRIBUTIVE SHOCK

- **Sympathetic Tone**
- Or
- **Parasympathetic Tone**

- **Vascular Tone**

- **Tissue perfusion**

- **Massive Vasodilation**

- **Cardiac Output**

- **SVR & Preload**

**Distributive Shock Variants**

- **Anaphylactic Shock**
  - Mechanism: severe allergic reaction
  - Skin: hives, possible petechia. Urticaria, pallor, cyanosis
  - Blood pressure: abrupt fall in cardiac output
  - Respiration: rapid shallow, dyspnea with stridor, wheezes, crackles, leading to respiratory arrest
  - Other: swelling of mucus membranes/pulmonary edema
Distributive Shock

Variants

- **Septic shock**
  - Mechanism: overwhelming infection
  - Skin: varies from flushed pink (if fever is present) to pale and cyanotic. Purple blotches possible, peeling skin, general or on palms and soles of feet
  - Blood pressure: early—cardiac output increases but toxins prevent increase in BP. Late — drop in BP, hypotension
  - Respiratory: dyspnea with altered lung sounds
  - Other: high fever, (except in elderly and very young), Late sign is pulmonary edema

- **Neurogenic Shock**
  - Mechanism: vasodilation
  - Skin: areas of vasodilation, at first become warm, pink and dry. Later with pooling: pallor and cyanosis to the upper surfaces
  - Pulse: highly variable depending on injury or action of drug/poison: May be abnormally slow or abnormally fast,
  - Respiration: severely compromised: becoming slow, shallow, with abnormal patterns. Patient may lose stimulus to breath
  - Other: hypothermia.
Obstructive Shock (Extracardiac)

- Decreased diastolic filling
  - Tension pneumothorax
  - Pericardial tamponade
- Increased ventricular afterload
  - Massive PE

Differentiating Types of Shock (hemodynamic profiles)

<table>
<thead>
<tr>
<th>Physiologic variable</th>
<th>Preload</th>
<th>Pump function</th>
<th>Afterload</th>
<th>Tissue perfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical measurement</td>
<td>Pulmonary capillary wedge pressure</td>
<td>Cardiac output</td>
<td>Systemic vascular resistance</td>
<td>Mixed venous oxygen saturation</td>
</tr>
<tr>
<td>Hypovolemic</td>
<td>+</td>
<td>+</td>
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<tr>
<td>Cardiogenic</td>
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<tr>
<td>Distributive</td>
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Summary
SHOCK = Inadequate Tissue Perfusion

- **Mechanisms:**
  - Inadequate oxygen delivery
  - Release of inflammatory mediators
  - Further microvascular changes, compromised blood flow and further cellular hypoperfusion
  - Inadequate elimination of metabolic waste.

- **Clinical Manifestations:**
  - Multiple organ failure
  - Hypotension

METABOLISM
OF POOR PERFUSION STATES

- Metabolism is anaerobic
- Glucose breaks down into pyruvic acid, but not enough oxygen is present to enter into the Krebs cycle
- Pyruvic acid accumulates, degrades into lactic acid, which also accumulates along with other metabolic acids
- Cells die; tissues die; organs fail; organ systems fail; death ultimately ensues
Cellular Response to Shock

- Impaired cellular metabolism
- Anaerobic metabolism
- Impaired glucose usage
- Stimulation of clotting cascade & inflammatory response
- Intracellular Na⁺ & water
- Na⁺ Pump Function
- Cellular edema
- Vascular volume
- Tissue perfusion
- O₂ use

Stages of Shock: Classic Shock Syndrome

1. **Compensated** - body is able to compensate and maintain tissue perfusion.

2. **Progressive (uncompensated)** - body begins to lose its ability to compensate - inadequate perfusion begins.

3. **Irreversible** - Cell and tissue damage result in multi-system organ failure leading to death.
**COMPENSATED SHOCK**

- Body defense mechanisms attempt to preserve major organs
  - Precapillary sphincters close, blood is shunted
  - Increased heart rate and strength of contractions
  - Increased respiratory function, bronchodilation
  - Decreased skin perfusion
  - Altered mental status

**COMPENSATED SHOCK**

- Will continue until problem solved or shock progresses to next stage
- Can be difficult to detect with subtle indicators
  - Tachycardia
  - Decreased skin perfusion
  - Alterations in mental status
- Some medications such as β-blockers can hide signs and symptoms
UNCOMPENSATED SHOCK

- **Physiological response**
  - Precapillary sphincters open, blood pressure falls
  - Cardiac output falls
  - Marked increase in heart rate
  - Rapid, thready pulse
  - Blood surges into tissue beds, blood flow stagnates
  - Red cells stack up in rouleaux
  - Agitation, restlessness, confusion

UNCOMPENSATED SHOCK

- Easier to detect than compensated shock
  - Prolonged capillary refill time
  - Marked increase in heart rate
  - Rapid thready pulse
  - Agitation, restlessness, confusion
IRREVERSIBLE SHOCK

- Compensatory mechanisms fail, cell death begins, vital organs falter
- Patient may be resusitated but will die later of ARDS, renal and liver failure, sepsis

Shock - Mortality

- Despite extensive research, mortality rates remain high
  - **Hypovolemic**: variable, depends on etiology and time to treatment
  - **Cardiogenic**: 60-90%
  - **Distributive** (Septic): 35-40%
**Shock - Management**

- Place the victim in shock position
- Keep the person warm and comfortable
- Turn the victim’s head to one side if neck injury is not suspected

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**Systemic Inflammatory Response Syndrome (SIRS)**

- Defined as when generalized inflammation occurs and threatens vital organs

- Causes: massive tissue injury, burns, and pancreatitis, severe infections or sepsis

- Effects: endothelium is damaged and allows fluid to leak into the body tissues, results in poor perfusion of blood to organs

- Body is in a hypermetabolic state
Multiply Organ Dysfunction Syndrome (MODS)

- Defined: when 2 or more organ systems are failing at one time
- Is caused by the immune system's uncontrolled response to severe illness or injury
- Common cause of death of patients in the ICU, with mortality of 50%
- Identifying and acting quickly can help survival
- Can develop quickly following surgery, trauma, or severe burns or slowly in the case of an infection

Treatment for SIRS/MODS

- Critical care
- Goals
  - Prevent and treat infections
  - Maintain tissue oxygenation
  - Provide nutritional and metabolic response
  - Support failing organs
Hemodynamic Monitoring

- Goal is to optimize tissue oxygenation
- Lactate
- CVP
- Arterial pressure
- Urine output
- Pulse oximetry

Thank you!