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)

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

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.
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 (brochospasm)
   - **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
  - Preactatitis
  - 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
- \( \uparrow \text{CO} \)
- \( \uparrow \text{Myocardial O}_2 \text{ demand} \)
- \( \uparrow \text{Dyspnea} \)
- \( \uparrow \text{SVR} \)
- \( \uparrow \text{Catecholamine Release} \)
- \( \downarrow \text{Volume/Preload} \)
- Impaired myocardial function
- \( \uparrow \text{Peripheral & pulmonary edema} \)

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

Distributive Shock Variants

- **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>
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<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>
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<tr>
<td>Hypovolemic</td>
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<td>+ or ++</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

- Reduced \( \text{O}_2 \) use
- Reduced tissue perfusion
- Impaired cellular metabolism
- Impaired glucose usage
- Reduced ATP synthesis
- Impaired Na\(^+\) pump function
- Increased intracellular Na\(^+\) & water

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

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

- **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 resuscitated 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

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!