Shock

Objectives
- Review the components of perfusion
  - Cardiac output
  - Perfusion at the capillary level
  - Perfusion and cellular metabolism
    - Aerobic metabolism and energy production

Objectives
- Identify the stages of shock and the signs and symptoms found in each stage
- Explain how each stage of shock impairs and damages cellular perfusion and energy production

Objectives
- Outline the various types of shock including their causes and signs and symptoms
- Discuss the general treatment strategies for shock

Review of Blood Flow and Perfusion

Cardiac output

Preload

Stroke Volume

Afterload
Perfusion Equations

Cardiac output = stroke volume × HR

BP = cardiac output × PVR

Capillary Sphincters

- Control the amount of blood flow and nutrient exchange at the capillaries
- Controlled by various mechanisms
  - Sympathetic nervous system
  - Local levels of CO₂ and lactic acid
  - Cellular demand for oxygen

Perfusion at the Cellular Level

1. Blood Flow
2. Step 1: CO₂
3. Step 2: CO₂
4. Step 3: CO₂
5. Step 4: CO₂

Perfusion and the Cell

- Cells generate their own energy to sustain life
- 2-Step Process for energy production
  - Anaerobic metabolism
    - Glucose breakdown into 2 molecules
  - Aerobic metabolism
    - Adds oxygen to the molecules to finish the energy production process

The 2-Step Process of Energy Production

1. Glucose breakdown
2. CO₂ release
3. ATP production
4. CO₂ release

Anaerobic metabolism as the only mechanism for energy production

1. Glucose breakdown
2. CO₂ release
3. ATP production
Differences in Metabolism

- Aerobic Metabolism
  - Net energy production of 36 ATP
  - More than enough to sustain all internal processes of the cell
  - Waste products
    - CO₂ and H₂O
    - Efficient elimination by the body

- Anaerobic Metabolism
  - Net energy production of 2 ATP
  - Back-up energy plan in case of hypoxia
  - Waste products
    - Lactic acid
    - Difficult to get rid of by the body

Case Study in Perfusion

You are assessing a 63 year-old female who complains of dizziness and weakness. She tells you that her heart pounds and she feels like she will pass out if she tries to stand up. She has been vomiting for the past 3 hours. Her vital signs (when supine) include a heart rate of 116, respiratory rate of 22 and BP of 136/82. Her skin is pale.

Case Study, Continued

- As you continue your assessment, you find that the patient has been vomiting bright red blood.
- Is she in shock?
  - If yes, then what stage of shock is she in?
- What are the assessment findings for compensated, decompensated and irreversible shock?

The Stages of Shock

- Compensated
  - 15-25% of fluid loss from the vessels
  - Signs are subtle
  - Patient may show signs of an adrenaline rush

- Decompensated
  - 25-35% of fluid loss from the vessels
  - The body cells are profoundly hypoxic
  - Classic signs of shock

- Irreversible
  - > 35% fluid loss from the vessels
  - Body cells die
  - All vital signs bottom out
### The Pathophysiology of Hypoperfusion and Shock

<table>
<thead>
<tr>
<th>Signs</th>
<th>Comp.</th>
<th>Decomp.</th>
<th>Irrevers.</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR</td>
<td>120-ish</td>
<td>140-ish</td>
<td>Slow</td>
</tr>
<tr>
<td>RR</td>
<td>Little change</td>
<td>Elevated</td>
<td>Agonal</td>
</tr>
<tr>
<td>LOC</td>
<td>Restless</td>
<td>Confused</td>
<td>Coma</td>
</tr>
<tr>
<td>SKIN</td>
<td>Pale, cool, peripheral sweating</td>
<td>Very pale, cold, sweating</td>
<td>Mottled, cold, waxen, no sweat</td>
</tr>
<tr>
<td>BP</td>
<td>Very little change</td>
<td>Decrease</td>
<td>none</td>
</tr>
</tbody>
</table>

### Compensated Shock
- Baroreceptors sense a drop in pressure in the arteries
- Medulla is stimulated
- Sympathetic NS Response
  - Clammy and pale skin
  - Increased HR

### The First Wave of Compensated Shock

<table>
<thead>
<tr>
<th>O2</th>
<th>CO2</th>
<th>Symp. NS Close Pre-capillary Sphincter</th>
</tr>
</thead>
</table>

### Case Study, Continued
- Just after you move the patient to the gurney, she tells you she is going to throw up.
- You hand her a trash can and she begins to vomit dark red blood.
- Approximately 250mL of blood is in the trash can when she is done vomiting.
- Her HR increases to 126 and her RR increases to 24. Her BP does not change. She is sweating and her skin is pale and very cool.

### Next Wave of Compensated Shock
- Sympathetic NS response continues
  - Adrenal hormones released
  - Chemoreceptors sense changes in CO2 and oxygen concentrations
  - Medulla stimulated again
    - Increase in RR
    - HR continues to rise
- Cellular Ischemic Phase
  - Cells transition from aerobic to anaerobic metabolism
  - Lactic acid begins to build
  - Blood begins to coagulate behind the closed pre-capillary sphincter
  - Post-capillary sphincter closes
Cellular Ischemic Phase

- CO2 and Acid
- Blood Flow
- O2

Post-capillary sphincter closes

Case Study
- After you load the patient into the ambulance, you reassess her condition. You find that she is quiet and less talkative with you. Her hands are cold.
  - HR – 132
  - RR – 28
  - BP – 126/70

Decompensated Shock
- Less blood flow and oxygen delivery impact more of the body
- Tissues in the core become hypoxic
- More shifts to anaerobic metabolism
- Organ function slows

Capillary Stagnation Phase
- Lactic acid build in the cells and leak into the capillaries
- Cell function drops dramatically
- Continued closure of post-capillary sphincters reduce preload
  - Cardiac output drops
  - Blood pressure begins to fall

Case Study
- The patient vomits blood again during transport. Another 250mL is lost.
- The patient becomes unconscious and is only responsive to painful stimuli. Her skin is cold and very sweaty. Her vital signs are:
  - HR - 124
  - RR – 20
  - BP – 114/52

Irreversible Shock
- The medulla stops working
- Sympathetic nervous system stimulation ceases
- Heart function drops
  - Drop in heart rate and contractility
- Vessels dilate
  - No more energy to constrict

Capillary Washout Phase
- Cell functions cease and cells die
- Potassium, acid dumps into the capillaries
- Large accumulations of acid force the opening of the capillary sphincters
- Sludge, clots and acid circulates throughout the entire body
- Other organ systems die off from this wave of destruction
Irreversible Shock and the Capillary Washout Phase

Why All This Detail?

- Cellular injury will occur when anaerobic metabolism is not reversed in a timely manner.
- Cellular injury and death will occur before obvious signs of shock are apparent to the EMT-I.
- Delays in recognition and aggressive management will increase the chances for long-term disability in the patient.

Shock Types

- Hypovolemic
- Cardiogenic
- Distributive
- Obstructive
- Neurogenic

Group Project

Spend the next 10 minutes discussing the following question. Tie the answers to the physiology of ventilation, respiration and perfusion.

- How do the following causes of shock lead the patient into compensated and decompensated shock?
  - Cardiogenic
  - Distributive
  - Obstructive
  - “Spinal”
Hypovolemic Shock

- Assessment findings
  - Trauma: MOI, signs of compensated shock
  - Medical
    - Vomiting, history suggesting fluid loss
    - Bleeding from the mouth or rectum
      - Bright or dark red
      - Coffee-ground emesis
      - Melena
  - Associated signs and symptoms
    - Dizziness or syncope with sitting or standing
    - Orthostatic changes in vital signs

- Dysfunctions in perfusion
  - Loss of plasma/fluid
  - Loss of red blood cells
    - Inability to deliver enough oxygen to the cells

- Treatment strategies
  - Increase the amount of circulating fluid
  - Minimize the loss of red blood cells in uncontrolled bleeding

Cardiogenic Shock

- Assessment findings
  - Chief complaint of chest discomfort, dyspnea or syncope/near syncope associated with altered heart rates
  - Signs of acute congestive heart failure
    - Pulmonary edema
    - Jugular venous distention
    - Orthopnea

- Dysfunctions in perfusion
  - Altered heart rates
    - Reduced preload in tachycardia
    - Reduction in timely cardiac output
  - Myocardial trauma
    - Damage to contractile and electrical cells
    - Ineffective pumping
  - CHF or severe AMI
    - Damage to contractile cells
    - Fluid and pressure backs up into the lungs

- Treatment strategies
  - Base treatment on the particular dysfunction
    - Bradycardia: atropine
    - Tachycardia: vagal maneuver, oxygen and fluid
    - Myocardial trauma: oxygen, fluid bolus if no signs of pulmonary edema
    - MI/CHF: oxygen; other medications if BP allows for their administration

Distributive Shock

- Occurs with any mechanism suggesting vasodilation and “leaky” vessels
- Examples:
  - Anaphylaxis
  - Septic shock
  - Certain drug overdoses
Distributive Shock
- Assessment findings
  - Signs of a sympathetic nervous system response
    - Increase in HR, RR
    - Clammy or diaphoretic skin
  - Warm, flushed skin
    - Typical finding
  - Other “adventitious” signs
    - Systemic swelling and bronchoconstriction
    - Hives
- Dysfunction in perfusion
  - Slow movement of red blood cells to the tissues
    - Dilated vessels are unable to move fluid as effectively to the cells
  - “Leaky” vessels encourage fluid to move out of the vascular system
- Treatment strategies
  - Fluid boluses – septic shock
  - Specific treatments for anaphylaxis and overdose

Obstructive Shock
- Physical obstruction of blood flow or ventilation
  - Examples
    - Significant chest wall trauma
    - Tension pneumothorax
    - Cardiac tamponade
    - Pulmonary embolus
- Dysfunction in perfusion
  - Chest wall trauma
    - Inability to ventilate adequately
  - Tension pneumothorax
    - Compression of lung tissue and kinking of vena cava
  - Cardiac tamponade
    - Pressure against the ventricles that reduces cardiac output
  - Pulmonary embolus
    - Obstruction of the pulmonary artery
      - Inefficient loading of RBC at the lungs

Pulmonary Embolus
Obstructive Shock

- Treatment strategies
  - Early recognition and transport to a trauma or critical care hospital
  - Recognition is often difficult
  - Use of MOI or patient history may assist in recognition
  - Fluid boluses
  - Aggressive ventilation

Neurogenic Shock

- Also known as Spinal Shock
- Associated specifically with spinal trauma
- Rare occurrence

- Assessment findings
  - Mechanism of injury
    - Falls: landing feet-first or head-first
    - Penetrating trauma to the back with neurological deficits
  - Loss of function below the site of injury
  - Loss of sympathetic nervous system function
    - Relative bradycardia and hypotension
    - Warm, flushed skin
    - Loss of bladder control
    - Priapism

- Dysfunction in perfusion
  - Similar to distributive shock
  - Loss of sympathetic tone
    - Reduction of heart rate, cardiac output and peripheral vascular resistance

- Treatment strategies
  - Oxygen
  - Large-bore IV with fluid boluses
  - PASG
  - Atropine with slow rates

General Shock Management

- Airway and Ventilatory Support
  - Oxygen as needed to support perfusion
    - High-flow with any stage of shock!
  - Assistance with ventilation should be based on MOI assessment and level of respiratory distress
  - Consider PEAD with unresponsive patients
  - Suction as necessary

- Circulatory Support
  - Control bleeding
    - External
    - Internal
    - Bleeding from the nose or ears after head trauma
  - EKG monitoring
  - IV Therapy
    - Selection of administration sets
    - Rate of administration
    - Indications
General Shock Management

• PASG
  • Increases systemic vascular resistance via direct compression of blood vessels
    • Increases arterial blood pressure above the garment
  • But is it useful?
    • Profound hypotension/hypovolemia

  Could this treatment help our patient?

PASG

• Indications
  • Hypotensive patient with an unstable pelvis
  • Decreased systemic vascular resistance that cannot be corrected by other means
  • Local standing orders

PASG

• Contraindication
  • Pulmonary edema

• Precautions
  • Advanced pregnancy
    • Avoid inflation of the abdominal compartment
  • Impaled object/evisceration of the abdomen
  • Ruptured diaphragm
  • Cardiogenic shock

Summary

• Recognize shock in its earliest stage(s)
• Try to keep the cells from anaerobic metabolism!!
  • Lots of oxygen
  • Other therapies to support cardiac output
    • Tailor your treatments towards the primary causes of the patient’s shock

Summary

• Do what you can to get the patient to definitive care
• Medical causes for shock should be handled as aggressively as the traumatic causes for shock