Physiologic Effects of Acute Hemorrhage

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POS Rounds
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Hemorrhage

- Abnormal internal or external loss of blood

Do you have an appointment?
Hemorrhage – Points to consider

- Circulatory System
- Hemorrhage Classification
- Hemorrhage Control
- Stages of Hemorrhage
- Hemorrhage Assessment
- Hemorrhage Management
Circulatory System

- Delivery of nutrients and $O_2$ to tissues and cells
- Transportation of waste products produced by metabolism to liver and kidneys
- Delivery of $CO_2$ to lungs
Components of Circulatory System

- Heart or pump
- Blood vessels or pipes
- Blood or fluid
Cardiac Terminology

- Stroke Volume
- Preload
- Ventricular Filling
- Frank-Starling Mechanism
- Afterload
Terminology

- **Preload**
  - Represents filling of the ventricle
  - Volume of blood delivered to atria prior to ventricular diastole
  - *Dependent on venous return*

- **Afterload**
  - Amount of *resistance* heart must overcome to eject blood

- **Contractility**
  - Ability to contract, *inotropy*
  - *Frank Starling’s Law*
Frank – Starling Law =

- The force exerted/beat of heart is directly proportional to the length of fibers
Factors necessary for systemic O2 delivery

- Ability of O$_2$ to diffuse across alveolar membrane into blood stream
- Adequate number of RBC’s to transport O$_2$
- Adequate blood flow to transport RBC’s
- Ability of RBC’s to off-load O$_2$
Cardiac Output

- Volume of blood pumped in 1 minute
- SVxHR = CO
- Heart:
  - Parasympathetic Nervous System
    - Slows rate
    - Vagus Nerve
  - Sympathetic Nervous System
    - Increases rate
    - Cardiac Plexus
Blood Pressure

- Directly proportional to the product of the CO multiplied by SVR
- $BP = CO \times SVR$
- SVR, resistance to flow in the system (systemic vascular resistance)
Hemorrhage Classification

**CAPILLARY**
- Slow, even flow
- Bright red color

**VENOUS**
- Steady, slow flow
- Dark red color

**ARTERIAL**
- Spurting blood
- Pulsating flow
- Bright red color
Body composition - blood

- 60% of body weight is fluid
  - 7% circulating blood volume (CBV): Male
    - 5 L (10 units)
  - 6.5% CBV in women
    - 4.6 L (9-10 units)
Stages of Hemorrhage

**Stage 1**

- 15% loss of CBV
  - 70 kg pt = 500-750 mL
- **Compensation**
  - Vasoconstriction
  - Normal BP, Pulse Pressure, Respirations
  - Slight Elevation of Pulse
  - *Release of catecholamines*
    - Epinephrine
    - Norepinephrine
      - Anxiety, slightly pale and clammy skin
Stages of Hemorrhage

Stage 2

- 15-25% loss of CBV
  - 750-1250 mL

- *Early Decompensation*
  - Unable to maintain BP
  - Tachycardia & Tachypnea

(continued)
Stages of Hemorrhage

Stage 2

- Decreased pulse strength
- Narrowing pulse pressure
- Significant catecholamine release
  - Increase PVR
  - Cool, clammy skin & thirst
  - Increased anxiety and agitation
  - Normal renal output
- MAP
  - < 70 hypoperfusion
MAP

- Mean Arterial Pressure
- \( \text{Systolic} + \frac{2(\text{diastolic})}{3} \)
- Map should be maintained \( \geq 70 \)
Stages of Hemorrhage

Stage 3

- 25-35% loss of CBV
  - 1250-1750 mL

- Late Decompensation (Early Irreversible)
  - Compensatory mechanisms unable to cope with loss of Blood Volume

(continued)
Stages of Hemorrhage
Stage 3

• Classic Shock
  • Weak, thready, rapid PULSE
    • Narrowing pulse pressure = < MAP
  • Tachypnea
  • Anxiety, restlessness
  • Decreased LOC
  • Pale, cool and clammy skin
Stages of Hemorrhage

Stage 4

- >35% CBV Loss
  - >1750 mL
- **Irreversible**
  - Pulse: Barely palpable
  - Respiration: Rapid, shallow and ineffective
  - LOC: Lethargic, confused, unresponsive
  - GU: Ceases
  - Skin: Cool, clammy and very pale
  - Unlikely survival
## Stages of Hemorrhage

<table>
<thead>
<tr>
<th>Stage</th>
<th>Blood Loss</th>
<th>Vasoconstriction</th>
<th>Pulse Rate</th>
<th>Pulse Pressure/Strength</th>
<th>BP</th>
<th>Resp. Rate</th>
<th>Resp. Volume</th>
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<tbody>
<tr>
<td>1</td>
<td>&lt;15%</td>
<td>↑</td>
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<td>15-25%</td>
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<tr>
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<td>25-35%</td>
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<tr>
<td>4</td>
<td>&gt;35%</td>
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</table>

Average Blood Volume = 5 L
Stages of Hemorrhage

Concomitant Factors

- Pre-existing condition
- Rate of blood loss
- Patient Types
  - *Pregnant*
    - >50% blood volume than normal
    - Fetal circulation is impaired when mother is compensating
  - *Athletes*
    - Greater fluid and cardiac capacity
  - *Obese*
    - CBV is based on IDEAL weight (less CBV)

(continued)
Stages of Hemorrhage
Concomitant Factors

**Children**
CBV 8-9% of body weight
Poor compensatory mechanisms
TREAT AGGRESSIVELY

**Elderly**
Decreased CBV
Medications: BP, & Anticoagulants
Compensation

- Respiratory
- Cardiovascular
- Sympathetic NS activation
- Neuroendocrine Response
- Transcapillary refill
Respiratory Compensation

- Chemoreceptors located in carotid body and aortic arch
  - Communicate respiratory center via CN IX, X
  - \( \text{PaO}_2 < 50 \text{mmHg} \), hypoxemia
  - \( \text{PaCO}_2 \) increased, hypercarbia
  - Acidosis

- Increased rate, depth or respirations
Respiratory Control

- Increased blood CO2
- Decreased blood O2
- Decrease CSF pH (acidosis)
- Mast cells release histamine
Histamine Release

- Eventually:
- Vasodilation
  - Increased venous capacitance
  - Blood pooling
- Increased vascular permeability
  - Leaking into tissues
  - Edema
Circulation

- **Vascular Control**
  - Increased sympathetic tone results in increased vasoconstriction

- **Microcirculation**
  - Blood flow in the arterioles, capillaries and venules
  - Sphincter Functioning

![Diagram of microcirculation](image_url)

a. Blood directed to the tissue.
b. Blood bypassing the tissue.
The Body’s Response to Blood Loss

- Greater Loss
  - \(\uparrow\) Cellular Ischemia
  - \(\downarrow\) Capillary Microcirculation
  - \(\uparrow\) Possibility of Capillary Washout
    - Buildup of lactic acid and CO\(_2\)
    - Relaxation of post capillary sphincters
    - Release of byproducts into circulation
      - PROFOUND METABOLIC ACIDOSIS
PNS & SNS always act in balance

Baroreceptors: Monitor BP

- Location
  - Aortic Arch
  - Carotid Sinuses
- Send Impulses to the Medulla
  - Cardioacceleratory Center
    - SNS: controls release of E and NE
    - Cardioinhibitory Center
    - PNS: controls the vagus nerve
- Vasomotor Center
  - Arterial and Venous tone
Cardiovascular System Regulation

- Chemoreceptors
  - Monitors level of CO$_2$ in CSF
  - pH CSF
  - Monitors level of O$_2$ in blood
Antidiuretic Hormone (ADH)
- aka: Arginine Vasopressin (AVP)
- Released
  - Posterior Pituitary
  - Drop in BP or Increase in serum osmolarity
- Action
  - Increase in peripheral vascular resistance
  - Increase water retention by kidneys
  - Decrease urine output
  - Splenic vasoconstriction
    - 200 mL of free blood to circulation
Angiotensin II

- Released
  - Primary chemical from Kidneys
  - Lowered BP and decreased perfusion
- Action
  - Converted from Renin into Angiotensin I
  - Modified in lungs to Angiotensin II
    - 20 minute process
    - Potent systemic vasoconstrictor
    - 1 hour duration
    - Causes release of ADH, Aldosterone and Epi
Aldosterone

- Release
  - Adrenal Cortex
  - Stimulated by Angiotensin II
- Action
  - Maintain kidney ION balance
  - Retention of sodium and water
  - Reduces insensible fluid

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Glucagon

> Release
  Alpha Cells of Pancreas
  Triggered by Epi

> Action
  Causes liver and skeletal muscles to convert glycogen into glucose
  Gluconeogenesis
Neuroendocrine Response

- ACTH (adrenocorticotropic hormone) secreted by pituitary
- Stimulates adrenal cortex to produce aldosterone and cortisol
- Aldosterone causes reabsorption of Na & H2O in kidney
- Kidney releases renin when cells of juxtaglomerular apparatus (JGA) are hypoperfused
- Renin accelerates conversion angiotensin to angiotensin I
- Lung tissue converts angiotensin I to angiotensin II, potent vasoconstrictor and stimulates release aldosterone
Cortisol

- Stimulates protein synthesis
- Adrenal medulla secretes epi and NE
- Vasopressin (ADH) released by posterior pituitary in response to increased osmolality
- Causes distal renal tubules to increase H2O absorption
Scenarios

- **Mild Hemorrhage (500 mL)**
  - Decreased central blood volume
  - Venous return and decreased CO
  - Decreased arterial pressure
    - Causes: 1. Small increase in peripheral resistance
    - 2. Small decrease in pulse pressure
Scenarios

- **Greater Hemorrhage (800mL)**
  - Increased HR and contractility
  - Constriction of resistance and capacitance vessels
    - skin, splanchnic, renal, skeletal muscles
  - Renal Vasoconstriction reduces GFR
Plasma Volume Maintenance

1. **Vasoconstriction**
   - Decreases circulatory capacity
   - Decreases capillary pressure

2. **Sympathetics**
   - General vasoconstriction
   - Reduced renal blood flow decreases GFR
   - Decreased Atrial Stretch causes reflex release of ADH
3. Aldosterone and vasopressin
   - Decreases Na⁺ and water excretion

Factors which restore plasma volume:

> Reduced cap pressure and renal vasoconstriction
> Increased vasopressin and aldosterone levels