Traumatic Shock: Pathophysiology and Management

Shands Trauma Tracks
Goals/Objectives

- Review Shock and Types of Shock
- Review Mechanisms/Features of Hypovolemic Shock and Physiologic Response
- Discuss Monitoring/Management of the Patient in Hypovolemic Shock
Shock

- **Definition:** “A clinical syndrome in which the peripheral blood flow is inadequate to return sufficient blood to the heart for normal function, particularly transport of oxygen to all organs and tissues.”

- **Consequence:** Inadequate tissue oxygenation to meet tissue oxygen requirements

1, From: Taber’s Cyclopedic Medical Dictionary, 17th Edition
Forms of Shock

- **Cardiogenic** – loss of contractility
- **Distributive** – loss of vascular tone
  - Neurogenic, septic, anaphylactic
- **Obstructive** – relative decreased blood volume (preload)
  - Tension pneumo, cardiac tamponade
- **Hypovolemic** – loss of preload
  - Hemorrhagic
Physiologic Considerations

- Shock represents a failure of Oxygen Delivery (DO2) to meet Oxygen Consumption
- In the care of the patient in shock, we attempt to manipulate DO2
  - DO2 = Oxygen Content $\times$ Cardiac Output
  - Cardiac Output = HR $\times$ SV
Why is this Important? Metabolism!

- Aerobic Metabolism produces 36 ATP via the Krebs Cycle
  - ATP is the energy source of the cell

- Anaerobic Metabolism produces 2 ATP and produces lactate as a byproduct
  - Lactate can decrease cardiac function
  - Metabolic acidosis
Anaerobic Metabolism

Decreased ATP

Loss of Na-K Pump

Cellular Swelling, Loss of function

Lysosomes rupture, Auto-digestion

Cell death
Stages of Shock

- **Compensatory** – VS are maintained
  Once BP falls

- **Progressive** – compensatory mechanisms no longer support organ systems; requires increased levels of support

- **Irreversible**
  - Multiple Organ Dysfunction Syndrome
  - Refractory State
Response
Compensatory Changes

- **Sympathetic Nervous System – fight/flight**
  - Vasoconstriction, $\uparrow$HR, $\uparrow$ contractility, $\downarrow$ UOP

- **Renin-Angiotensin-Aldosterone**
  - Response to $\downarrow$ BP/$\downarrow\downarrow$ blood volume with $\uparrow$ Na+

- **Tissue Injury $\rightarrow$ cytokines**
  - Vasodilation, vasoconstriction, capillary permeability
Blood Pressure

- Body Systems mobilize to maintain homeostasis – BP is one of the homeostatic goals (other goals are pH, osmolality, ionic neutrality)
- Perfusion DOES NOT EQUAL BP
- Perfusion (Cardiac Output) will suffer to maintain BP – i.e. vasoconstriction
Body Maintains Blood Pressure

- SVR increased
- Systemic vascular resistance (SVR)
- SVR decreased
- Blood pressure
- Cardiac output (CO)
- CO increased
- CO decreased
Hemorrhagic Shock

- Hemorrhage is the most common cause of shock in the injured patient.
- Resuscitation requires:
  - Rapid hemostasis
  - Appropriate fluid replacement
- Resuscitation is complete when:
  - Oxygen debt is repaid
  - Tissue acidosis is eliminated
  - Normal aerobic metabolism is restored in all tissue beds
Blood Loss

- **Initial signs** - ↑HR, ↓PP, ↓capil refill
  - Beta blockers, cardiac reserve, athletes
- **Pulse Pressure = sBP - dBP**
- **Decreased sBP**
  - ↓ sBP with contractility, fluids
  - ↓ sBP with ↓ Stroke Volume
- **Increased dBP**
  - ↓ dBP from vasoconstriction
Blood Loss with Injury Type

- Long bone fx – assume substantial blood loss
- Scalp lacerations – bleed a lot
- Pelvic instability or distended abdomen – assume bleeding in retroperitoneum or abdominal cavity
- Penetrating trauma – pure blood loss
- Blunt trauma – mimics septic shock more than hypovolemic (↓ SVR)
## Blood Loss Classes

<table>
<thead>
<tr>
<th></th>
<th>Class I</th>
<th>Class II</th>
<th>Class III</th>
<th>Class IV</th>
</tr>
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<tbody>
<tr>
<td>mL Loss</td>
<td>&lt; 750</td>
<td>750-1500</td>
<td>1500-2000</td>
<td>&gt; 2000</td>
</tr>
<tr>
<td>% Loss</td>
<td>&lt; 15%</td>
<td>15-30%</td>
<td>30-40%</td>
<td>&gt; 40%</td>
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<tr>
<td>HR</td>
<td>&lt; 100</td>
<td>&gt;100</td>
<td>&gt;120</td>
<td>&gt;140</td>
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<tr>
<td>BP</td>
<td>Normal</td>
<td>Normal</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>PP</td>
<td>- or ↑</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Fluids</td>
<td>Xloid</td>
<td>Xloid</td>
<td>Xloid + B</td>
<td>Xloid + B</td>
</tr>
</tbody>
</table>
Blood Loss Classes

- VS are NOT the most sensitive indicators of fluid loss.
- End-Organ perfusion parameters (UOP, mentation, skin signs) are better indicators.
- Class/Volume of Blood Loss is NOT used to determine resuscitation.
- However, failure of parameters to return to normal should cause suspicion of ongoing losses.
- Response to initial fluid resuscitation is used to determine plan of action......
<table>
<thead>
<tr>
<th>VS</th>
<th>Rapid Response</th>
<th>Transient Response</th>
<th>No Response</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Return to Normal</td>
<td>Transient improvement; recurrence of ↓BP and ↑HR</td>
<td>Remain abnormal</td>
</tr>
<tr>
<td>EBL</td>
<td>Minimal</td>
<td>Moderate and ongoing</td>
<td>Severe</td>
</tr>
<tr>
<td>Need for More Xloid</td>
<td>Low</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>Need for Blood</td>
<td>Low</td>
<td>Mod to High</td>
<td>Immediate</td>
</tr>
<tr>
<td>Blood Prep</td>
<td>Type &amp; Cross</td>
<td>Type-specific</td>
<td>Emerg blood release</td>
</tr>
<tr>
<td>Need for OR</td>
<td>Possibly</td>
<td>Likely</td>
<td>Highly likely</td>
</tr>
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</table>
Initial Resuscitation

- Administer 2L of isotonic xloid ASAP
  - NS, LR, Plasmalyte
  - NS can cause hyperchloremic acidosis

- Rapid Responders
  - Complete resuscitation
  - No evidence of ongoing fluid/blood loss
  - No perfusion deficits
Less Favorable Responses

Degree of instability depends on:
- Ongoing losses
- Ability to compensate

Remember, BP can be misleading.

Remember, HR, BP, PP, UOP can underestimate blood loss

Keep looking at THE WHOLE PICTURE
Transient Responders

- These patients show an initial response and then show signs of ongoing loss and perfusion deficits
- Class II or III hemorrhage OR can be due to a bleed with a rebleed
- Give fluids and look for losses
- Consider early blood transfusion
Non-Responders

- Due to a life-threatening hemorrhage
- Goal is to find the site of fluid losses
- All these patients require blood transfusions
- Need to administer uncrossmatched blood
Resuscitation Strategies/Monitoring
Fluid Resuscitation

- **Standard of Care = Crystalloid**
  - Can find studies using colloids, hypertonic
  - None of these $\uparrow$O$_2$ carrying capacity
  - Hemodilution – can worsen DO$_2$
  - Fluid Overload - $\downarrow$ cardiac performance

- **Blood Transfusions**
  - Only fluid that $\uparrow$O$_2$ carrying capacity
Crystalloid (Xloid)

- **3:1 Rule**
  - Rough estimate – replace 3 mL of crystalloid for each mL of blood loss

- **Na⁺ levels**
  - LR = 130; also has K⁺, Ca++, lactate
  - NS = 154
Hypertonic/Dextran

- Hypertonic (3%, 7.5% saline) causes fluid shift from IS and IC to IV
  - Intracellular dehydration
- Dextran (large glucose molecule) – maintains IV volume
  - Risk of rebleeding, short-lived
- Watch fluid overload – esp elderly
- More effective with TBI
Colloids

- Albumin, hespan
- Increased osmotic pull into the IV space
  - Colloid osmotic pressure (COP)
- Questioning research results
  - Hespan may reduce reperfusion injury
  - Albumin – helpful, no effect, harmful
Do we need fluids?

- “Permissive Hypotension”
  - Studied mostly with penetrating trauma
- Large fluid resuscitations
  - Cause hemodilution
  - Prevent clot formation
- TBI: hypotension doubles mortality
- Elderly: low cardiac reserve......
  ..ischemia...death
What are best indicators for blood transfusion?

- Persistent tissue hypoxia despite fluid resuscitation
- Significant metabolic acidosis, even if BP is stable
  - Especially if acidosis persists after fluids
- SVO$_2$ or CVO$_2$ < 55%
Hct is NOT a useful indicator

- Hematocrit = % of rbc to circulating volume
- Acute bleeds – lose cells and volume equally
  - May maintain normal Hct
- Better to use serial Hcts than absolute
- If Hct is low – tells you something
- If Hct is normal – tells you very little
- Time delay of lab procedures
Platelets

- Non-Trauma: plt of 20,000 is sufficient to prevent spontaneous bleeding
- Trauma: consider if plt < 100,000 or evidence of ongoing bleeding
- Less predictable is platelet FUNCTION
- Patient history – ASA, NSAIDS
- CHI: increased risk of bleeds due to damaged neural tissue
FFP

- Fresh Frozen Plasma = coagulation factors
- Transfusions deplete coagulation factors
- Patients with decreased hepatic function – can’t mobilize additional coagulation factors
- If 10 units of PRBCs – coagulation becomes paramount (not proven, intuitive)
Hypothermia

- Ongoing Concern with Trauma
  - Injury Site – air temp, “wet” area
  - Nosocomial – room temp, cold fluids

- Prevention:
  - Warming lights, Huggy Bear, Thermostat
  - Keep body and head covered
  - Fluid warmers – warm to 39 degrees

- Active Rewarming – PD, CTs, vents
Hypothermia

- Deadly Triad
  - Hypothermia
  - Acidosis
  - Coagulopathy

- Decreased cellular oxygen extraction
- Decreased cardiac contractility
- Decreased platelet function
- TBI pts benefit from hypothermia
Endpoints of Resuscitation

- Problem: “adequate resuscitation” still leaves occult hypoperfusion and ongoing tissue acidosis (compensated shock)
- Traditional markers underestimate resuscitation: HR, BP, PP, UOP
- Look at acidosis, oxygen extraction, end organ function
Resuscitation End-Points

- **Global**
  - Cardiac volumes and indexes – EDV, LVSWI
  - Extraction values – SVO₂
  - Base deficit
  - Lactate

- **Regional**
  - pHᵢ
  - Skeletal
**Base Deficit**

- ABG states either “deficit” or “excess”
- Reflects TOTAL buffering system of the blood (HCO$_3$ is only $\frac{1}{2}$ the total) – amount of buffering needed for systemic acidosis
- pH level is less specific because it includes compensation effects
- Bicarb levels correlate with base deficit
Lactate

- Produced from anaerobic metabolism
- Time to normalize lactate levels also shows prognostic value
- Normal hepatic function metabolizes lactate in LR; LR does not cause lactic acidosis
pHi

- Intramucosal pH (not gastric pH)
- Mucus-producing cells in gut are very oxygen dependent
  - Decreased oxygen delivery causes increased acidosis
  - Canary test – first sign of hypoperfusion
- EAST: early indicator of complications
- Requires specialized NG tube
Newer Measurements

- Transcutaneous O$_2$ and CO$_2$ levels
  - CO$_2$ used as marker of cellular metabolism end-product
- Intramuscular measures
- Sublingual CO$_2$
- Near infrared spectroscopy
  - Simultaneous measurement of pH, pO2, pCO$_2$
  - Also shows mitochondrial function
EAST – 2003 Recommendations

- **Level 1**
  - Standard hemodynamic parameters are not adequate
  - Supranormal $DO_2$ correlates with survival*

- **Level 2**
  - Time to normalization of base deficit, pH, lactate is predictive
  - Persistently high base deficit or pH may indicate complications
  - Base deficit predictive value is altered with alcoholics, hyperchloremic metabolic acidosis
Thank you

Questions?