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.”[1]

- 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 X Cardiac Output
- Cardiac Output = HR X 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

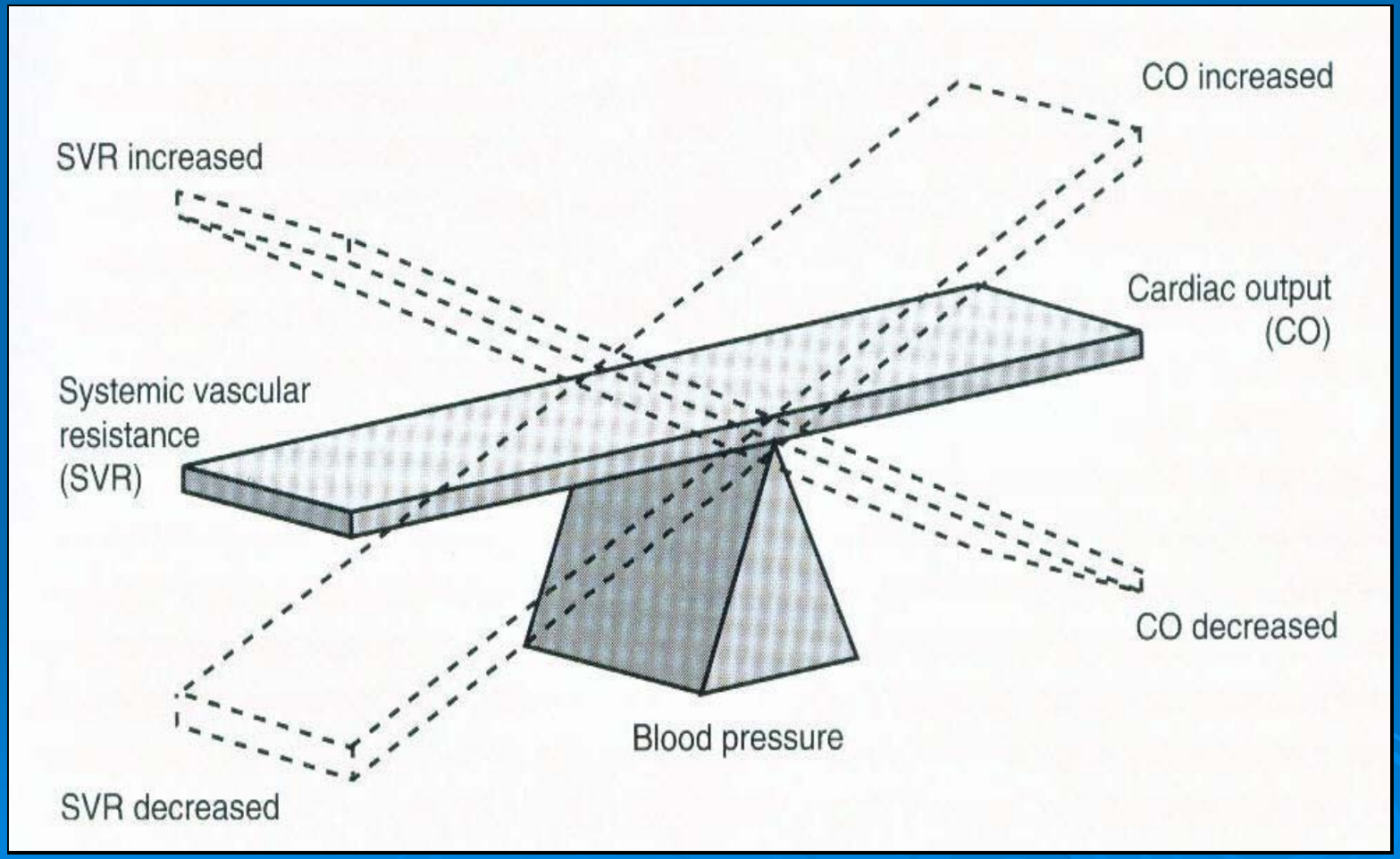
Compensatory Changes

- Sympathetic Nervous System – fight/flight
  - Vasoconstriction, ⇑HR, ⇑ contractility, ⇓ UOP
- Renin-Angiotensin-Aldosterone
  - Response to ⇓ BP/⇓ blood volume with ⇑ Na+
- Tissue Injury → 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



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

	Class I	Class II	Class III	Class IV
mL Loss	< 750	750-1500	1500-2000	> 2000
% Loss	< 15%	15-30%	30-40%	> 40%
HR	< 100	>100	>120	>140
BP	Normal	Normal	↓	↓
PP	- or ↑	↓	↓	↓
Fluids	Xloid	Xloid	Xloid + B	Xloid + B

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……

	Rapid Response	Transient Response	No Response
VS	Return to Normal	Transient improvement; recurrence of ↓ BP and ↑ HR	Remain abnormal
EBL	Minimal	Moderate and ongoing	Severe
Need for More Xloid	Low	High	High
Need for Blood	Low	Mod to High	Immediate
Blood Prep	Type & Cross	Type-specific	Emerg blood release
Need for OR	Possibly	Likely	Highly likely

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 – SVO_2
  - 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 ½ 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 Recomemdations

- Level 1
  - Standard hemodynamic parameters are not adequate
  - Supranormal DO_2 correlates with survival*
- Level 2
  - Time to normalization of base deficit, pHi, lactate is predictive
  - Persistently high base deficit or pHi may indicate complications
  - Base deficit predictive value is altered with alcoholics, hyperchloremic metabolic acidosis

Thank you

Questions?