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Shock Overview

DND Primary Care Paramedicine

Module: 05

Section: 14

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Objectives and Overview

- Case based approach to shock.
- Review of pathophysiology of shock.
- Differential diagnosis based approach to patients with shock.
- Solidify the differential diagnosis for shock patients.
- Introduction to treatment options for the various causes of shock.

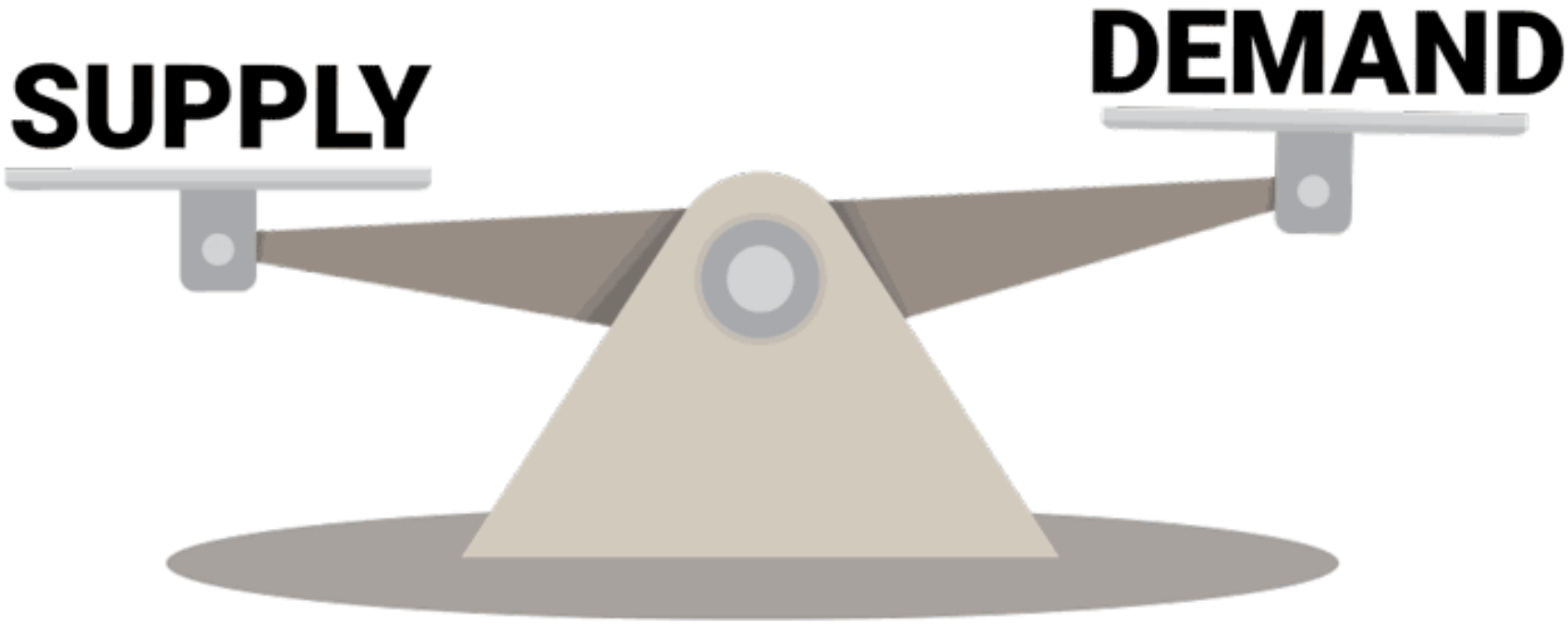
Case

- Dispatched for 65 y/o M with decreased LOC.
- 4 weeks post op partial colectomy for colon cancer.
- VON attending his dressings daily, arrived today to find him unresponsive, called 911.
- On arrival the patient is lying in bed with snoring respirations, does not respond to voice.
- VS: HR 150, BP 60 p, T 36°C, BGL 12.0 mmol/L, RR 36, SaO_2 86% on room air.
- Broad differential diagnosis?

What is Shock?

- Being extremely surprised?
- An altered LOC?
- Massive hemorrhage?
- Hypotension?
- Profound hypotension?
- Circulatory insufficiency?

Shock



Shock

- Defined as circulatory insufficiency
- Imbalance between tissue oxygen demand, and tissue oxygen delivery
- Global tissue hypoperfusion

- How might the body attempt to compensate?

Shock

- Given the global tissue hypoperfusion that occurs with shock, coupled with the bodies attempt to compensate, what signs and symptoms would you expect to see?

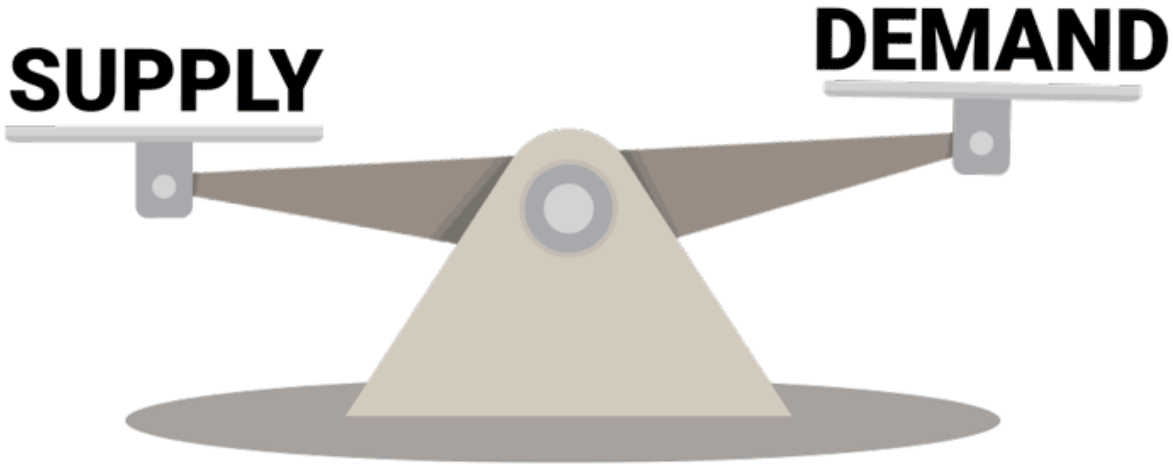
Signs and Symptoms

- Shortness of breath
- Tachypnea
- Hypotension
- Tachycardia with weak peripheral pulses
- Pale/cool/clammy skin
- Cold/mottled extremities
- Altered mental status
- Decreased urination

- Note that the extremities may be warm or red in distributive shock states

What Causes Shock?

- What circumstances might create a picture of circulatory insufficiency causing an oxygen supply vs. demand problem?



4 Broad Categories of Shock

- Cardiogenic
- Hypovolemic
  - Hemorrhagic
  - Non-hemorrhagic
- Obstructive
  - PE, tamponade, tension pneumothorax
- Distributive:
  - Septic
  - Anaphylactic
  - Neurogenic
  - Endocrine
  - Toxicological

Causes of Shock: "SHOCKEND"

- **S**epsis
- **H**ypovolemia
- **O**bstructive
- **C**ardiogenic
- Anapyla**K**tic
- **E**ndocrine
- **N**eurogenic
- **D**rugs

Shock Overview

CIRCULATORY PHYSIOLOGY REVIEW

Stroke Volume

- Stroke Volume (SV) is the amount of blood released from the heart per beat.

Cardiac Output

CO = SV X HR

L/min = L/beat X beats/minute

- The amount of blood circulated from the heart in liters/minute.

- What determines stroke volume?
  - Preload
  - Contractility
  - Afterload

Preload

- Is blood returned to the heart during diastole
- Preload is dependent on venous return
- Decreased venous return = reduce preload
- Reduced preload can reduce CO, unless there is compensation
- How might you compensate?
- Increased preload can increase stroke volume

Myocardial Contractility

- The force generated by the myocardium on contraction

- Starling's Law (Rubber band theory)

Afterload

- Pressure or “afterload” the left ventricle pumps against.
- Blood must overcome this resistance to achieve cardiac output.
- This is dependent on the degree of peripheral arterial vasoconstriction.
- Vasoconstriction = ↑ resistance = ↑ afterload = ↓ stroke volume.

What about Blood Pressure?

BP = CO X PVR

(Recall CO = HR x SV)

- PVR (peripheral vascular resistance)
  - May also be seen as systemic vascular resistance (SVR)
  - The resistance of blood flow by the force of friction between the blood and walls of the vessels

Blood Flow

- PVR is dependent on internal diameter of blood vessels and the viscosity of blood.
- Aorta and arteries do not significantly change diameter.
- Arterioles however can change lumen size by a factor of 5.
- Peripheral vasoconstriction can occur to increase blood pressure, vasodilation will decrease blood pressure.

Once Again

- BP = CO x PVR(afterload)

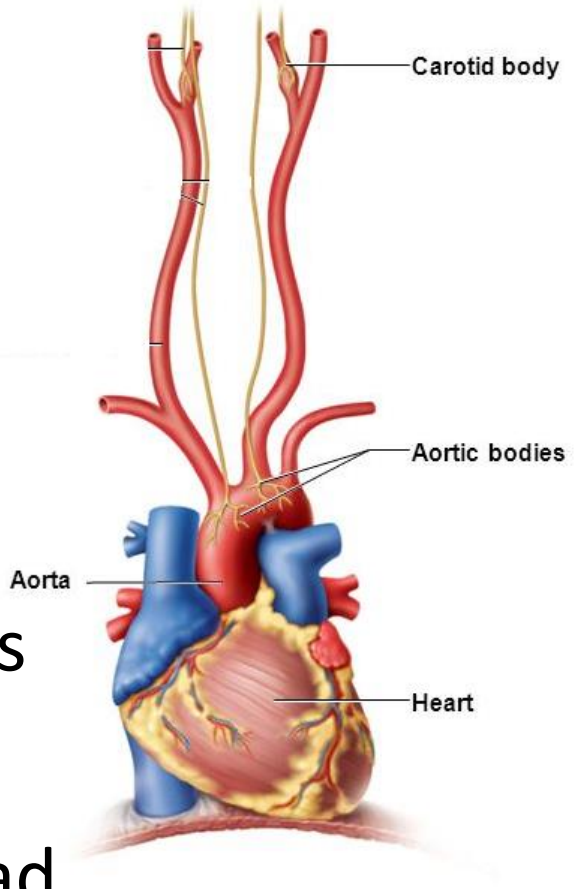
- Increased afterload increases ↑ BP
- Decreased afterload decreases ↓ BP

Blood Pressure

- How are the variables contributing to blood pressure controlled?
- How do we control things like heart rate, preload, contractility, blood vessel tone or SVR?

Baroreceptors

- Sensory fibers located in the aortic and carotid tissues
- Help control BP by two negative feedback mechanisms:
  - Lower BP in response to increased arterial pressure
  - Increase BP in response to decreased arterial pressure
- Increased pressure results in signals sent by baroreceptors through sympathetic nervous system to the brain to decrease heart rate, preload and afterload .



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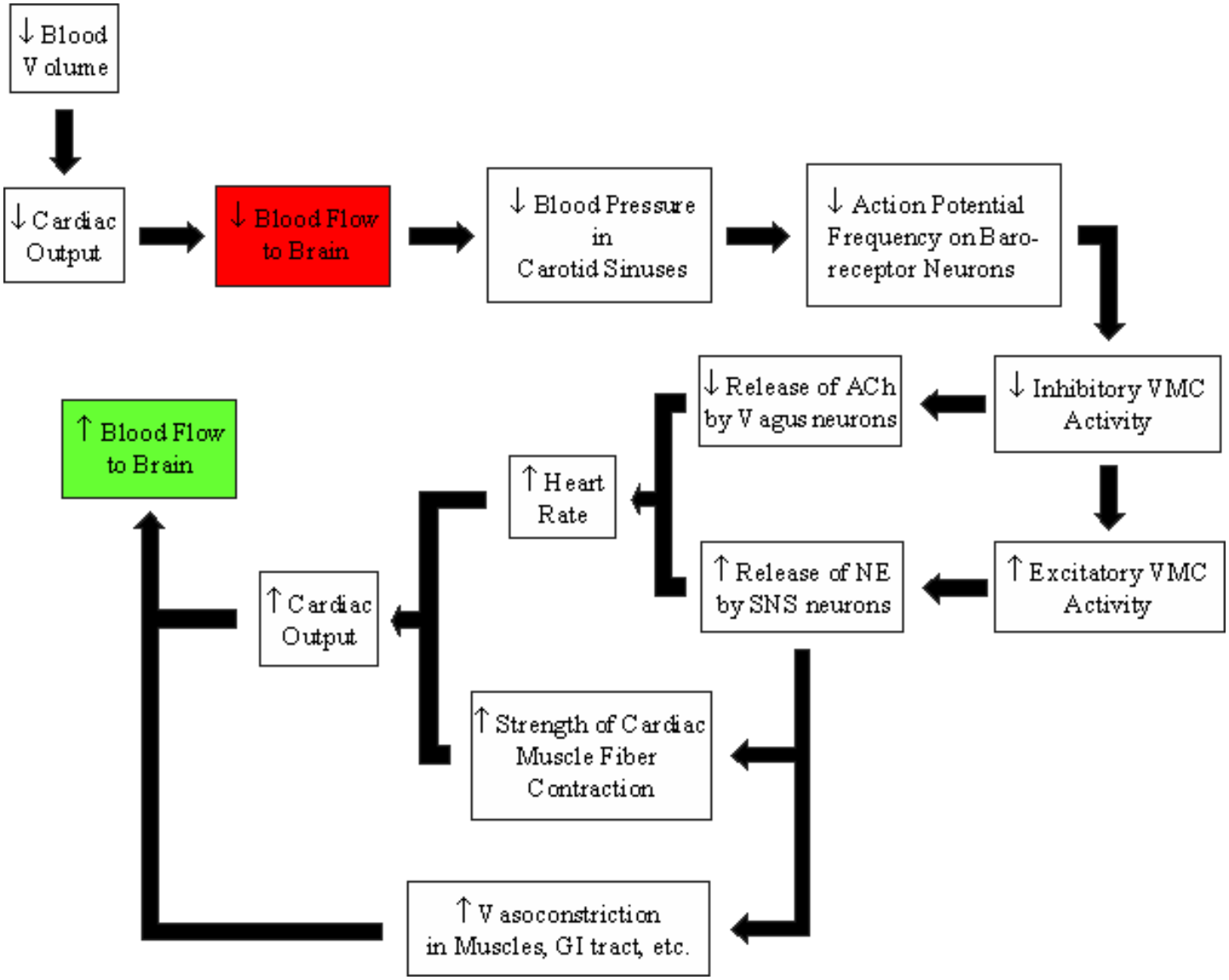
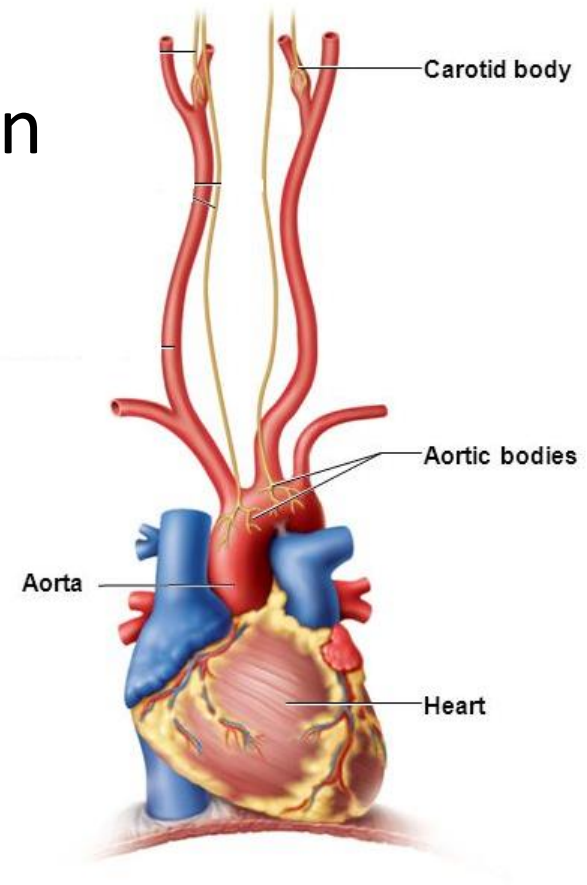
Baroreceptor Reflex During Hypovolemic Shock



Chemoreceptor Reflexes

- Low arterial pressure stimulates peripheral chemoreceptor cells in carotid and aortic bodies

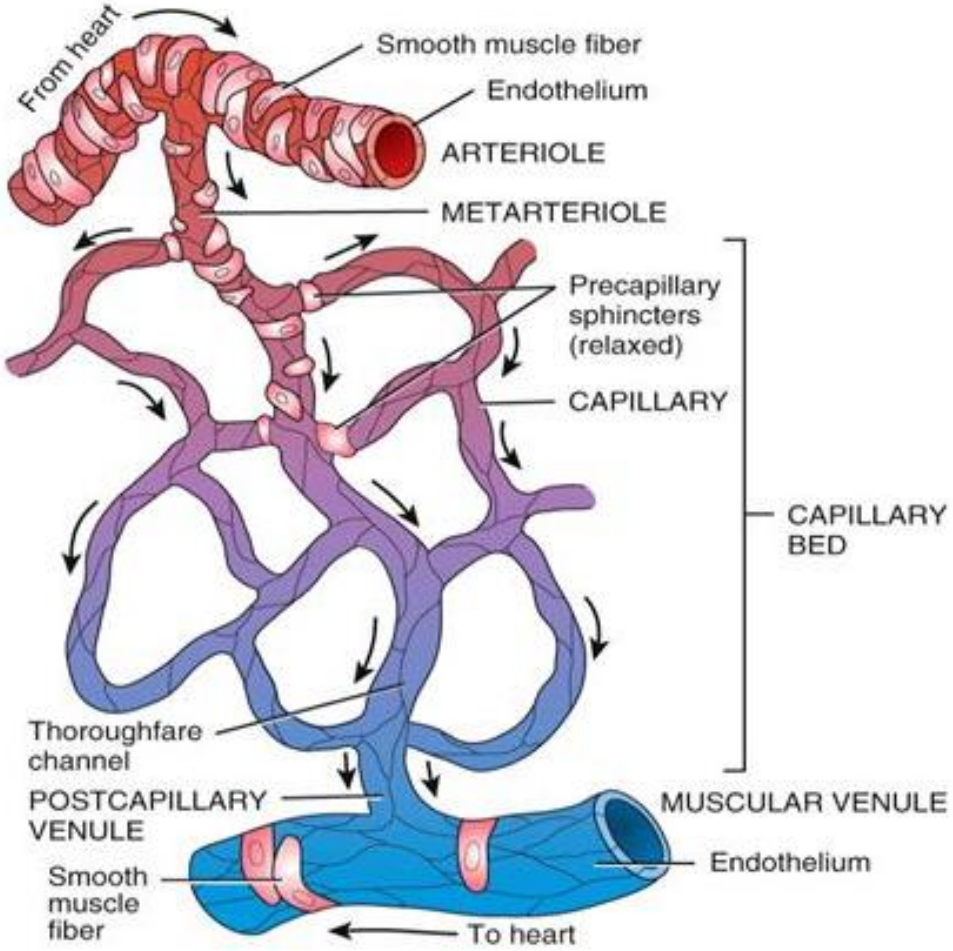
- If oxygen or pH decreases, stimulate vasomotor center of medulla



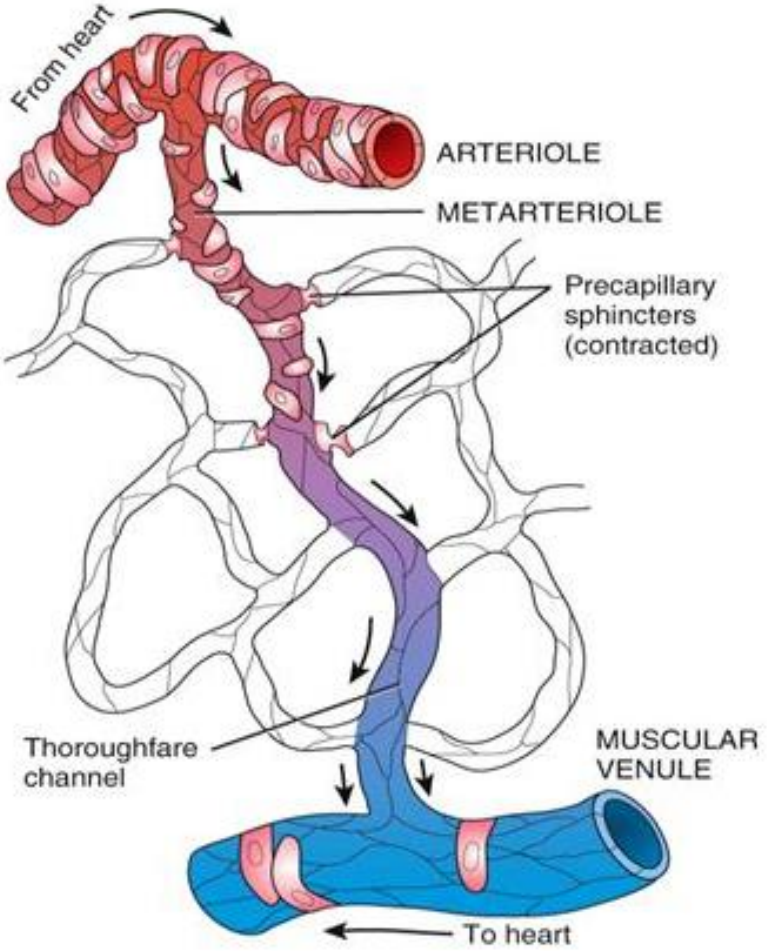
Systemic pressure effects of the Vasomotor Center (VMC)

- Venous
  - Venous system can vasoconstrict to increase preload and SV
- Arterial
  - Arterial system can vasoconstrict to increase afterload and BP as well

Microcirculation



(a) Sphincters relaxed: blood flowing through capillaries



(b) Sphincters contracted: blood flowing through thoroughfare channel

Microcirculation

- Systems/factors that affect microcirculation:
  - Local control by tissues
  - Nervous control of blood flow
  - Baroreceptors
  - Chemoreceptors
  - CNS ischemia response
  - Hormonal response
  - Adrenal-medullary response
  - Renin-angiotensin-aldosterone mechanism
  - Vasopressin
  - Reabsorption of tissue fluid

Shock

- Recall that

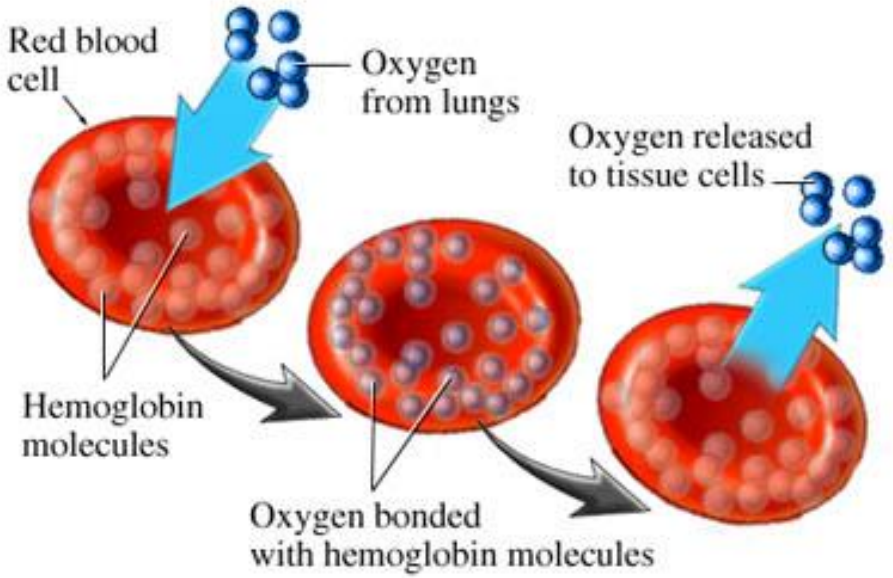
  Shock = global hypoperfusion

  Shock = oxygen demand>supply

- We have discussed the variables that maintain blood pressure and these are essential components of adequate perfusion.
- You also require adequate hemoglobin and oxygen delivery to tissues to maintain adequate tissue perfusion.

Oxygen

- Binds to hemoglobin and diffuses across capillary membrane.
- 97 - 100 % of hemoglobin is saturated in the normal setting.



Fick's Principle

- Recognized the five conditions required for effective movement of oxygen within the body
  - Adequate FiO_2
  - Appropriate O_2 diffusion from alveoli to capillaries
  - Adequate numbers of RBC's
  - Proper tissue perfusion
  - Effective tissue off loading

Let’s focus on differential diagnosis in terms of causes of shock

CASES

CASE:
71 Y/O M WITH SYNCOPE

CASE:
47 Y/O F WITH ABDOMINAL PAIN

CASE:
25 Y/O F FROM MVC

Shock Overview

HYPOVOLEMIC SHOCK

Hypovolemic Shock

- State caused by internal or external loss or deficit of fluid.
- 2 types:
  - Hemorrhagic
    - May be due to bleeding internally or externally, e.g. major trauma, spontaneous internal bleeding, etc.
  - Non-hemorrhagic
    - Due to vomiting, diarrhea, poor intake/dehydration, internal fluid shifts (e.g. burns).
- How might a patient in hemorrhagic shock present?

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Hemorrhagic Hypovolemic Shock

	Stage I	Stage II	Stage III	Stage IV
Blood loss	Less than 15%	15-30%	30-40%	More than 40%
Heart Rate	Normal	Tachycardic (above 100)	Tachycardic (above 120)	Tachycardic (above 140)
Blood Pressure	Normal; slight rise in diastolic pressure	Orthostatic changes	Decreased systolic blood pressure (below 90)	Profoundly decreased systolic blood pressure (less than 80)
Respirations	Normal	Slight tachypnea	Moderate tachypnea	Marked tachypnea; respiratory collapse
Capillary Refill Time	Less than 2 seconds	More than 2 seconds; clammy skin	Usually more than 3 seconds; cool, pale skin	More than 3 seconds; cold, mottled skin
Mental Status	Normal or slightly anxious	Mildly anxious or agitated	Confused, agitated	Obtunded
Bowel Sounds	Present	Hypoactive	Absent	Absent
Urinary Output	More than 30 mL/hr	20-30 mL/hr	Less than 20 mL/hr	None

Causes of Hemorrhagic Hypovolemic Shock?

- Examples:
  - Trauma
  - GI Bleeding
  - Ruptured aneurysm
  - Gynecologic bleeding, e.g. ruptured ectopic pregnany.
- In trauma, what body cavities can you lose enough blood to develop shock?

Causes of Non-Hemorrhagic Hypovolemic Shock?

- Vomiting
- Diarrhea
- Decreased intake of fluids
- Dehydration
- Osmotic diuresis (e.g. with hyperglycemia)
- Fluid shifts into “third space” e.g. burns

Hypovolemic Shock Pearls

- History and physical exam critical to make diagnosis.
- Don’t wait for late findings such as hypotension.
- Shock in trauma is always considered hemorrhagic until proven otherwise.
- In trauma, pts can lose life-threatening volumes of blood into the external world, as well as into the chest, abdomen, pelvis and thigh cavities.
- Intracranial bleeding does not cause shock. A shocky head injured patient is assumed to be bleeding into one of the above spaces.

Hypovolemic Shock: Management

- Oxygenation!
- Volume support/IV fluids (normal saline)
  - What is permissive hypotension?

- Try to stop the fluid loss:
  - Symptom control for vomiting (e.g. antiemetics)
  - Control the bleeding if hemorrhagic (how?)
  - Tranexamic acid if hemorrhagic.
- What about inotropes/vasopressors?

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Questions about Hemorrhagic Shock?

CASE:
25 Y/O M WITH CP/SOB

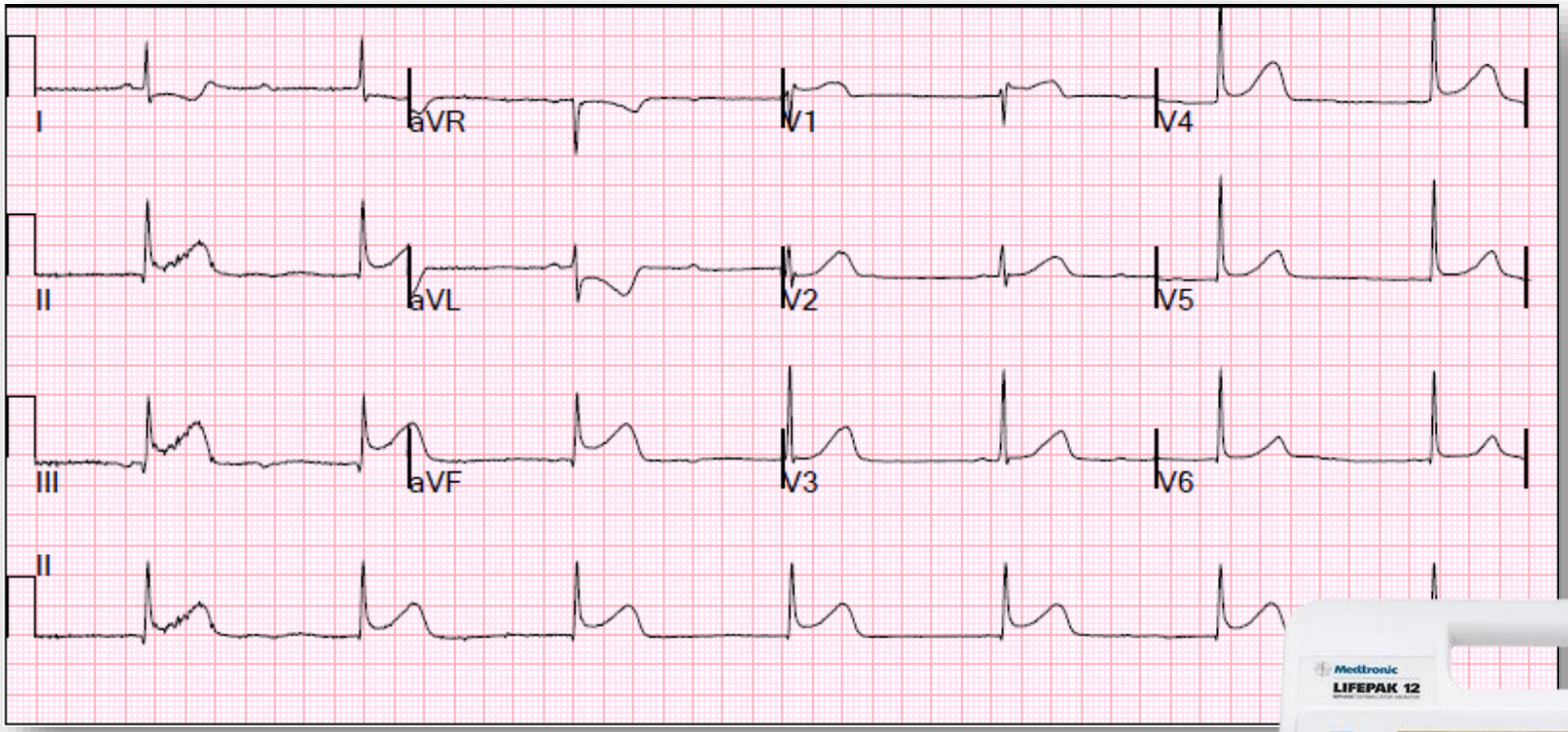
Obstructive Shock

- Recall the causes:
  - PE
  - Tension pneumothorax
  - Cardiac tamponade
- How will you know when to suspect obstructive shock?
- How does obstructive shock occur in terms of pathophysiology?

Obstructive Shock: Treatment

- Oxygenation!
- Volume support, IV NS bolus, then reassess.
- Specific care (in addition to above):
  - PE: anticoagulation or in extreme cases thrombolysis (usually in ED).
  - Tension pneumothorax: needle decompression.
  - Cardiac tamponade: tx underlying cause, pericardiocentesis (in ED).
- What about inotropes/vasopressors?

CASE:
57 Y/O M WITH SOB


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II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Cardiogenic Shock

- Impaired pump function of the heart muscle causing insufficient perfusion of body tissues.
- Causes?
- Always place patient on monitor and do EKG.
- History and signs of pump failure?
- When do you suspect this?
- Mortality is very high!
- Management is challenging.

Cardiogenic Shock: Challenging Management!

- Oxygenation!
- Volume support, cautious and small IV fluid bolus, with frequent reassessments.
- Why the caution with IV fluid?
- Vasopressors/inotropes (ACP scope)
- Address the underlying cause (ACP):
  - Treat brady or tachy dysrhythmias.
  - Treat cardiac ischemia, timely reperfusion e.g. PCI/TNK for STEMI.

CASE:
85 Y/O F WITH CONFUSION

Distributive Shock

- Unlike hypovolemic shock, the actual blood volume hasn't significantly changed.
- The problem is blood vessel dilation secondary to the underlying cause.
- This causes a "relative hypovolemia".
- These patients are hypoperfused, but classically warm and pink in the extremities.
- Why?

Distributive Shock

- Types of distributive shock:
  - Neurogenic
  - Anaphylactic
  - Septic
  - Toxicologic
  - Endocrine
- Or, NASTE!!!

Distributive: Septic Shock

- May be signs to suggest infection – like what?
- Severe infection causes “full body” inflammatory response.
- Inflammatory mediators cause widespread vasodilation.
- What is SIRS?
- What is infection vs bacteremia vs SIRS vs sepsis vs severe sepsis vs septic shock?

Definitions/Progression

- Infection: invasion of host by microorganisms with associated response.
- Bacteremia: bacteria in the blood stream.
- SIRS: Systemic inflammation due to a number of possible causes (including infection).
  - 2 or more:
    - >38 °C or <36 °C
    - HR >90
    - RR >20
    - WBC >12 or <4

Definitions/Progression

- Sepsis: SIRS due to infection.
- Severe sepsis: Sepsis associated with hypoperfusion, organ dysfunction or hypotension.
- Septic shock: Severe sepsis that does not respond to adequate fluid resuscitation.

Septic Shock

- What is early goal directed therapy?
- Time sensitive condition!
- Early treatment = improved outcomes, every minute counts!
- Management:
  - Oxygenation
  - IV Fluids, be aggressive
  - Vasopressors (ACP)
  - Early antibiotics! (ED)

Distributive: Anaphylactic Shock

- History is important.
- Contact with allergen.
- Causes histamine release.
- Histamine & other mediators cause:
  - Vasodilation
  - Leaking of blood vessels
  - Bronchocontriction.
- Clinical presentation?

Anaphylactic Shock Management

- Oxygenation!
- Airway monitoring.
- Bronchodilators as needed for wheeze/SOB.
- Antihistamines (ie diphenhydramine).
- Epinephrine IM, (ACP IV prn if refractory).
- IV fluid support as needed.

Distributive: Neurogenic Shock

- Due to acute spinal cord injury.
- Disrupts the sympathetic nervous system – how does this cause a problem for perfusion?
- Clinically what does this picture look like/when do you suspect this?
- What is the difference between neurogenic shock and spinal shock?

Neurogenic Shock: Treatment

- Oxygenation!
- C-spine immobilization.
- IV fluid support.
- Vasopressors/inotropes/chronotropes if no response to IV fluid (ACP)
- Assume traumatic shock is associated with hemorrhagic shock until proven otherwise.
- Keep mind open to multiple etiologies.

Distributive: Endocrine Causes of Shock

- Less common, but you will see this.
- Severe hyper or hypothyroidism can present in acute crisis with shock.
- Adrenal insufficiency can also present with shock. Which patients are at risk for this?
- Treat supportively with oxygenation and IV fluid support.
- Definitive management in ED.

Distributive: Toxicologic Causes of Shock

- Relatively common
- Many medications and recreational drugs can affect heart rate and vasomotor status.
- Examples?
- PCP treatment is supportive, with oxygenation and IV fluid support.
- Specific therapies may include cardiac pacing, vasopressors/inotropes, antidotes, drug specific interventions, GI or dialysis decontamination.

General Principles For All Causes of Shock

- Recognize symptoms early prior to hypotension, this is a late finding.
- PCP care will include maximizing oxygenation, and cautious IV fluid support with ongoing reassessments between boluses.
- Call for help when ALS interventions are warranted.
- Always maintain your ddx for shock, and recognize more that one cause may occur at once.

4 Broad Categories of Shock

- Cardiogenic
- Hypovolemic
  - Hemorrhagic
  - Non-hemorrhagic
- Obstructive
  - PE, tamponade, tension pneumothorax
- Distributive:
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