

SHOCK OVERVIEW

Primary Care Paramedicine

Module: 15

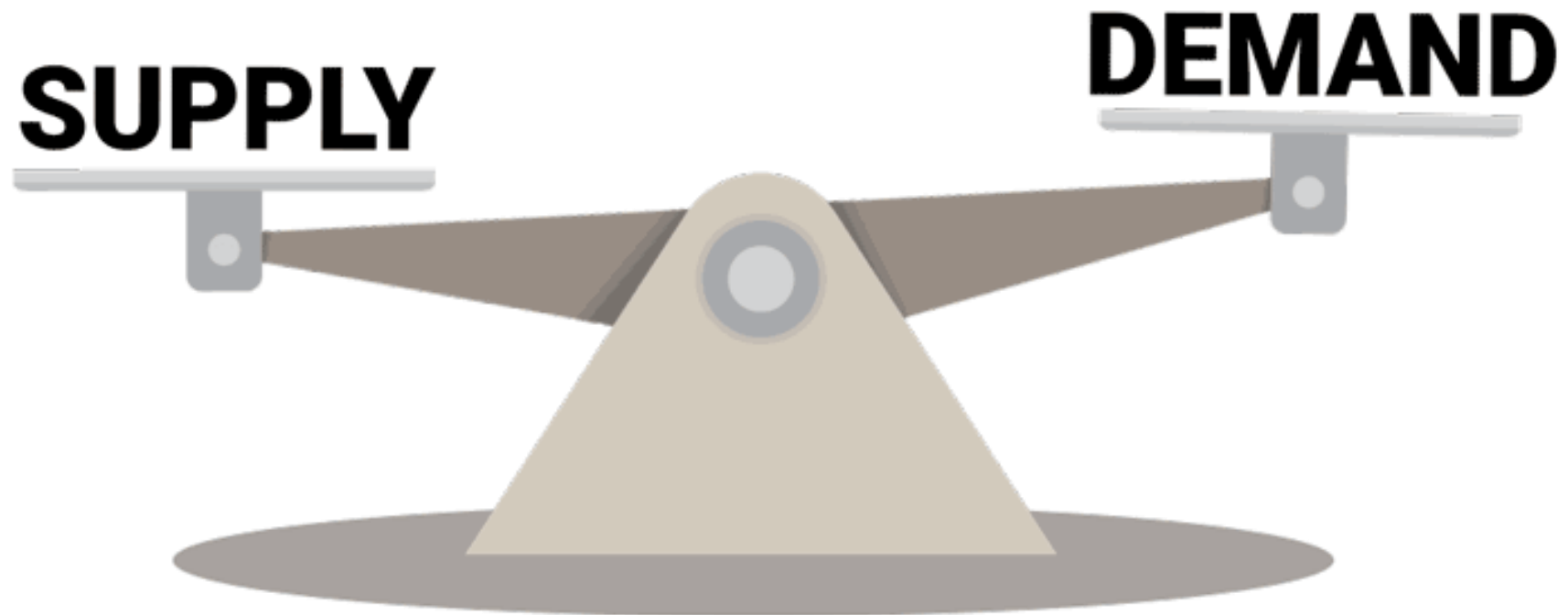
Section: 01



- 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.

- 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, SaO₂ 86% on room air.
- Broad differential diagnosis?

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



- Defined as circulatory insufficiency
- Imbalance between tissue oxygen demand, and tissue oxygen delivery
- Global tissue hypoperfusion
- How might the body attempt to compensate?



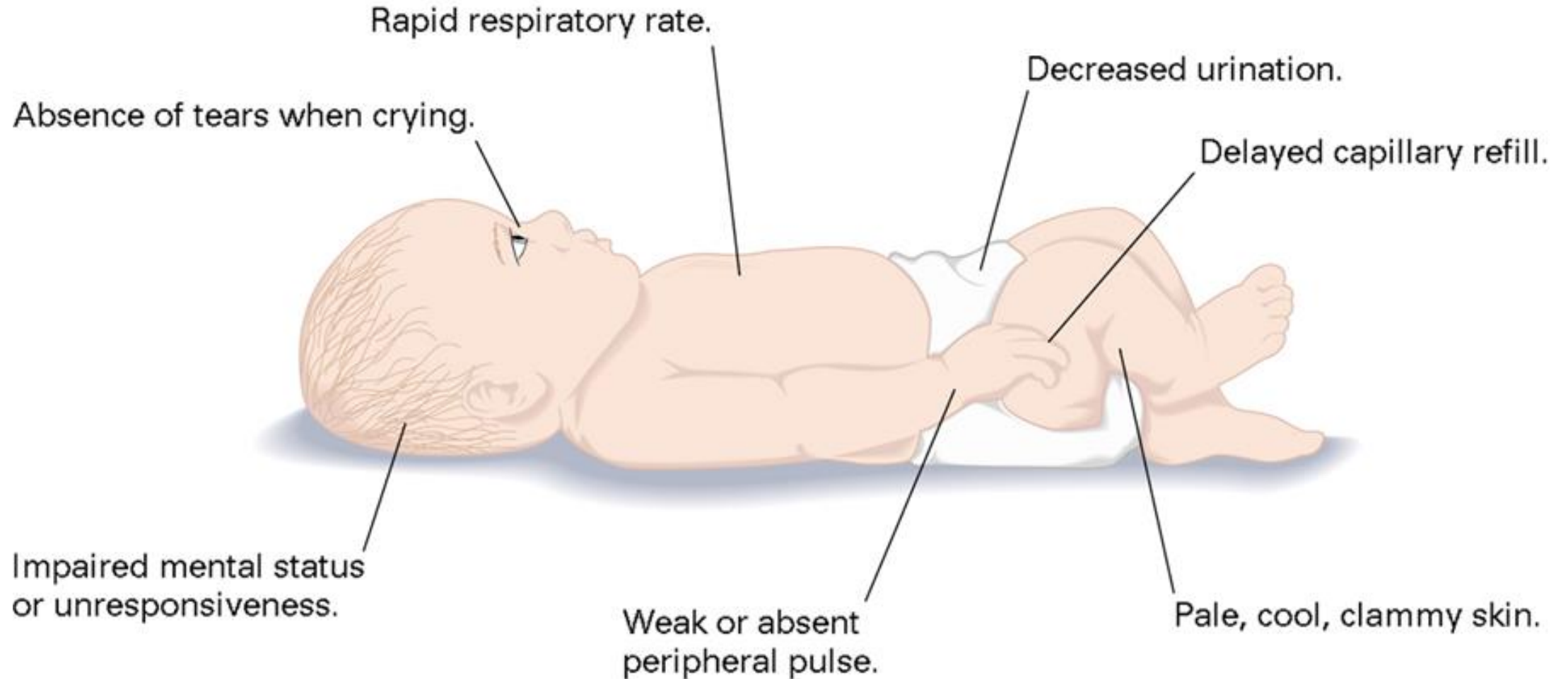
- Given the global tissue hypoperfusion that occurs with shock, coupled with the bodies attempt to compensate, what signs and symptoms would you expect to see?

Signs and Symptoms

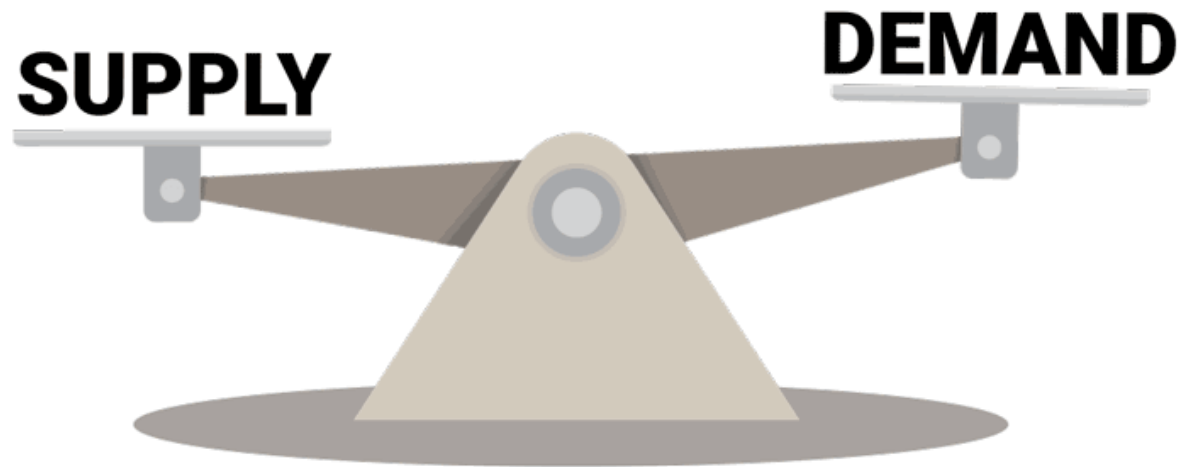
- Shortness of breath
- Tachypnea
- Hypotension
- Tachycardia with weak peripheral pulses
- Pale/cool/clammy skin
- Cold/mottled extremities
- Altered mental status
- Decreased urination

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

Hypoperfusion in a child



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



4 Broad Categories of Shock

Cardiogenic

Obstructive

- PE
- Tamponade
- Tension pneumothorax

Hypovolemic

- Hemorrhagic
- Non-hemorrhagic

Distributive

- Septic
- Anaphylactic
- Neurogenic
- Endocrine
- Toxicological

- **S**epsis
- **H**ypovolemia
- **O**bstructive
- **C**ardiogenic
- **A**napylactic
- **E**ndocrine
- **N**eurogenic
- **D**rugs

Shock Overview

CIRCULATORY PHYSIOLOGY REVIEW

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

$$CO = SV \times HR$$

$$L/min = L/beat \times \text{beats/minute}$$

- The amount of blood circulated from the heart in liters/minute.
- What determines stroke volume?
 - Preload
 - Contractility
 - Afterload

- 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

- The force generated by the myocardium on contraction
- Starling's Law (Rubber band theory)

- 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 = \uparrow resistance = \uparrow afterload = \downarrow stroke volume.

$$BP = CO \times PVR$$

(Recall $CO = HR \times SV$)

- 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

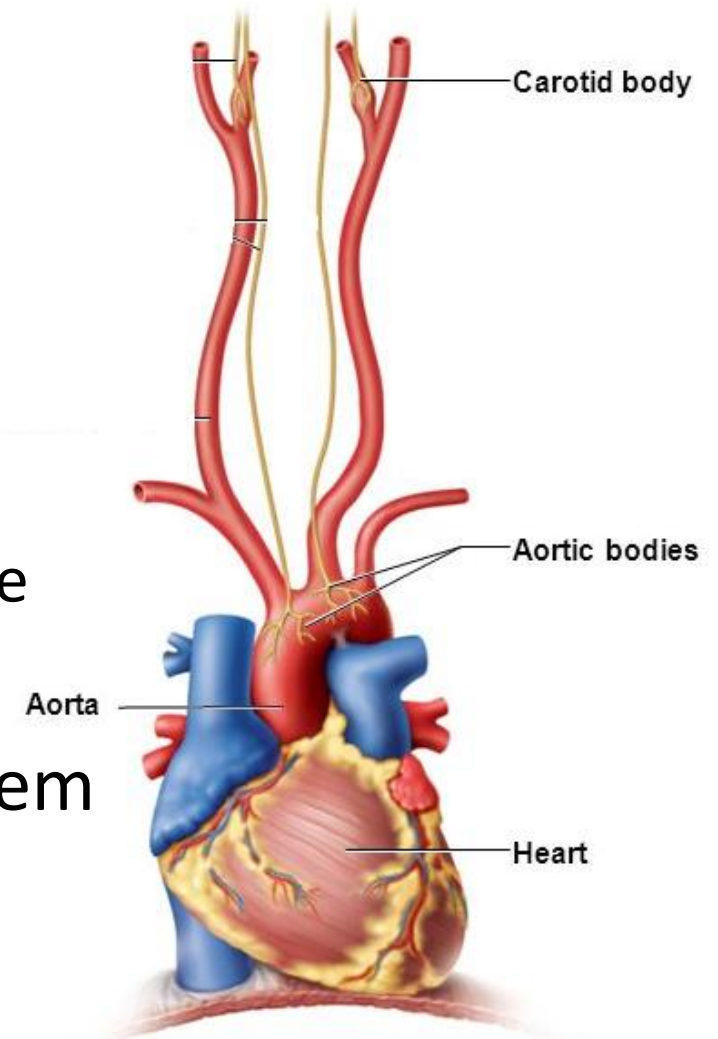
- PVR is dependent on internal diameter of blood vessels and the viscosity of blood.
- Aorta and arteries do not significantly change diameter.
- Arterioles however can change lumen size by a factor of 5.
- Peripheral vasoconstriction can occur to increase blood pressure, vasodilation will decrease blood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

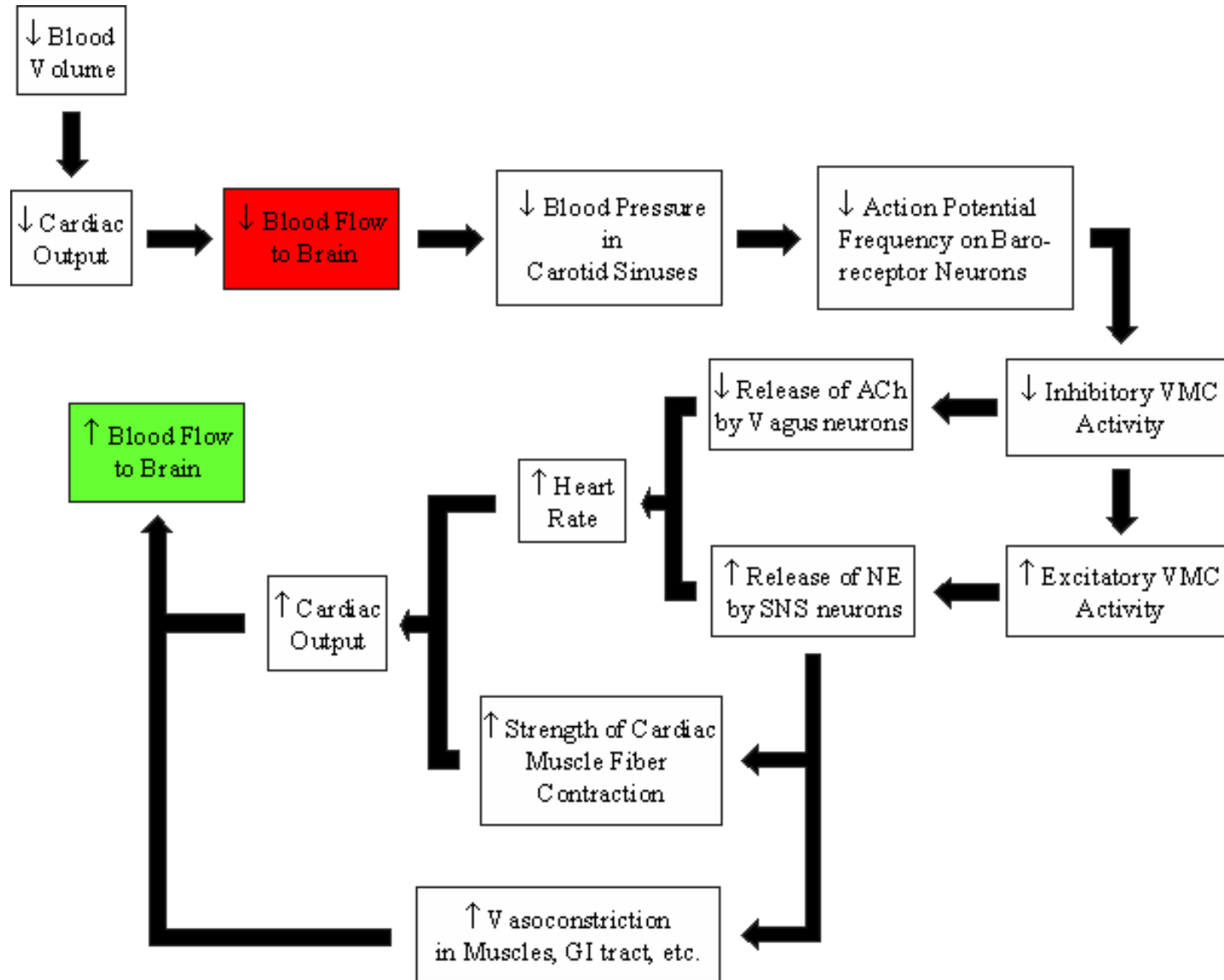
$$BP = CO \times PVR(\text{afterload})$$

- Increased afterload increases \uparrow BP
- Decreased afterload decreases \downarrow BP

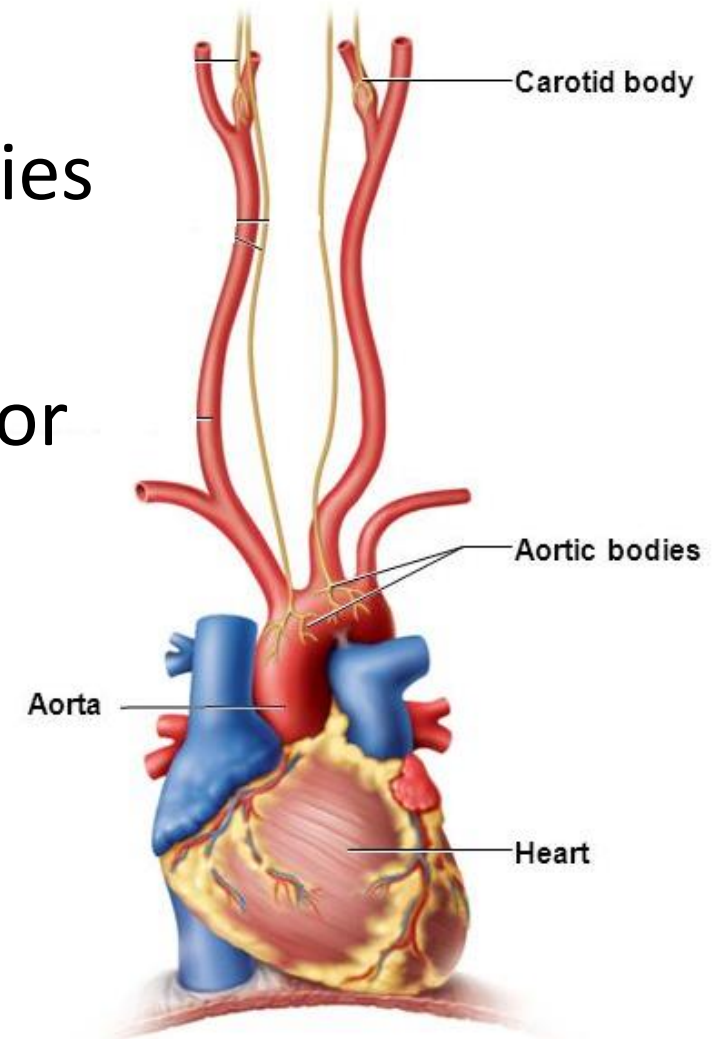
- How are the variables contributing to blood pressure controlled?
- How do we control things like heart rate, preload, contractility, blood vessel tone or SVR?

- 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 .

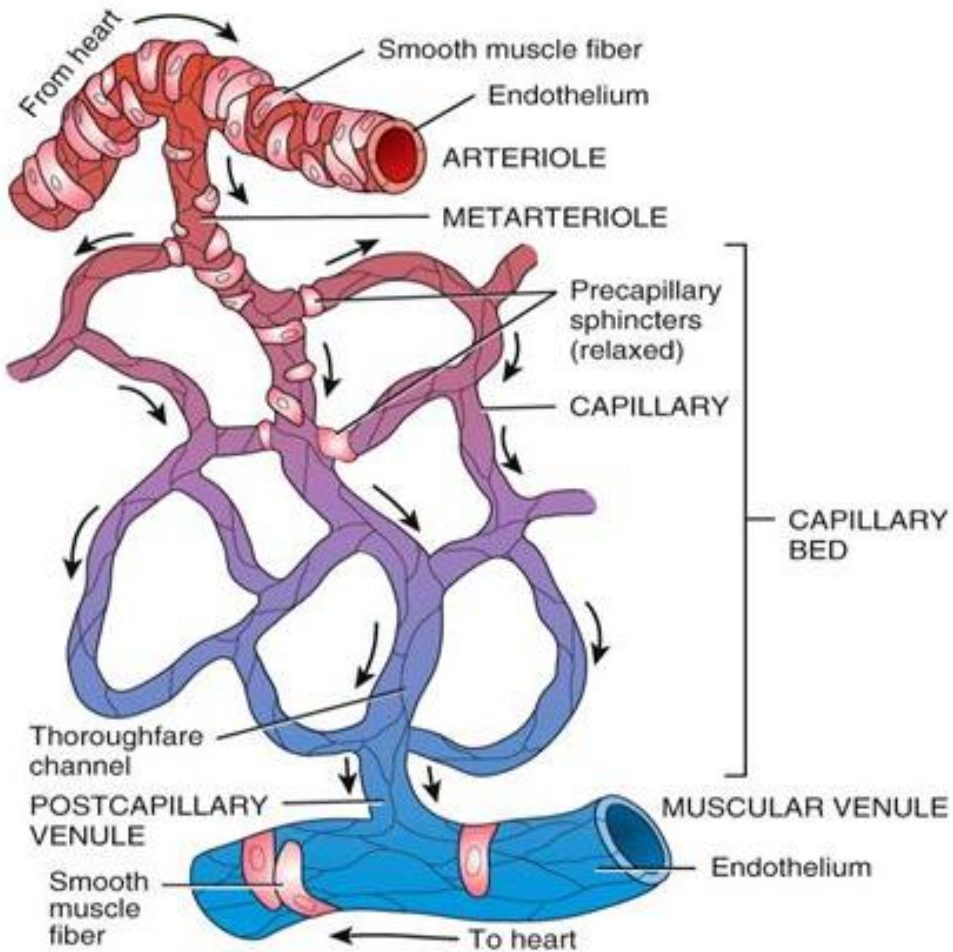




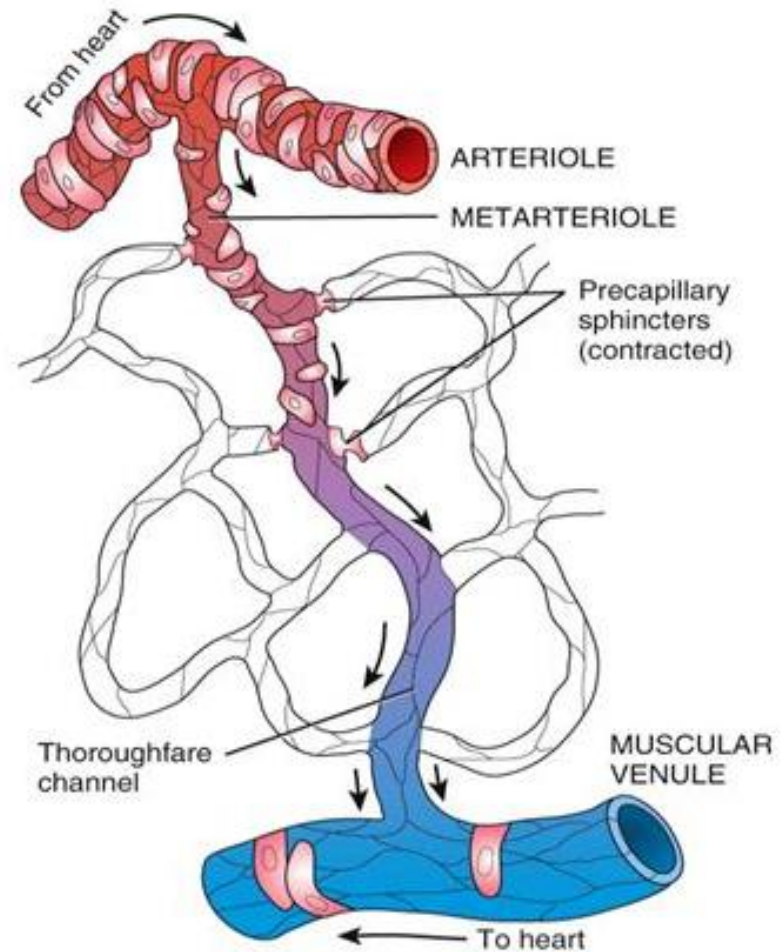
- Low arterial pressure stimulates peripheral chemoreceptor cells in carotid and aortic bodies
- If oxygen or pH decreases, stimulate vasomotor center of medulla



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



(a) Sphincters relaxed: blood flowing through capillaries

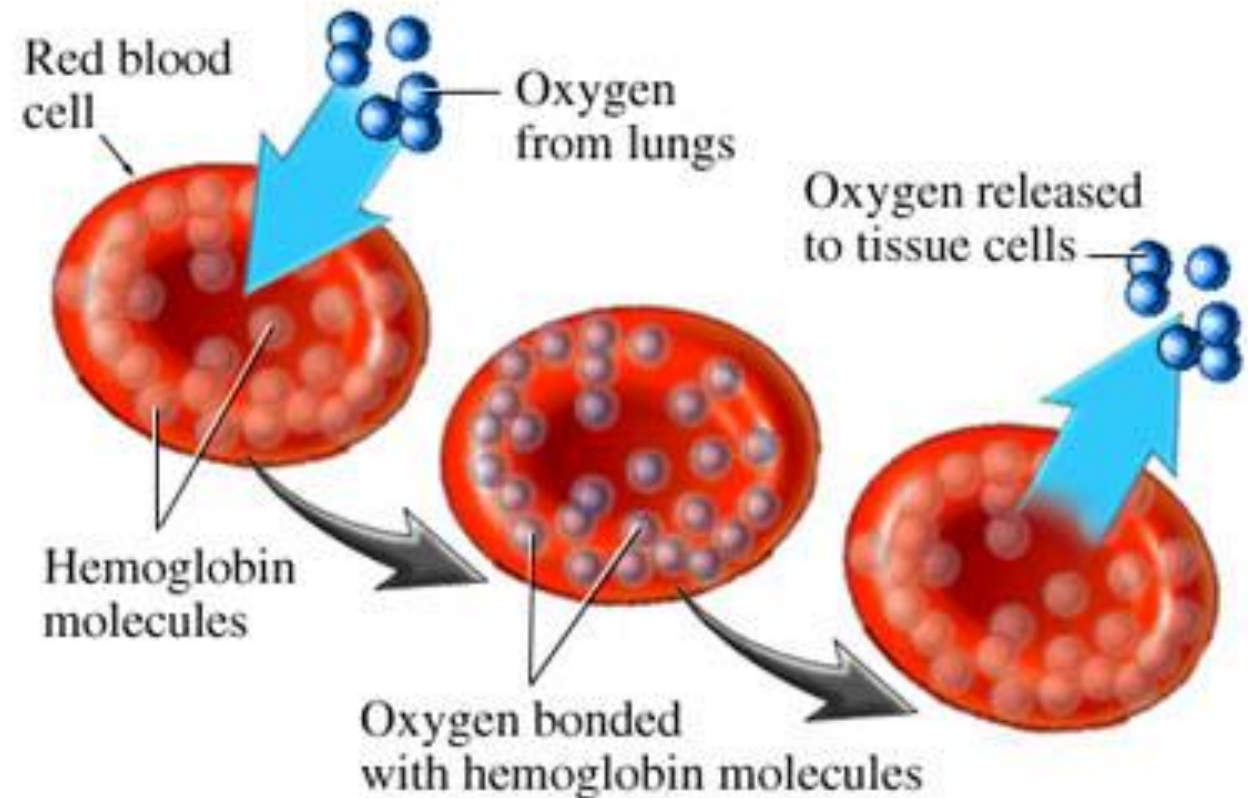


(b) Sphincters contracted: blood flowing through thoroughfare channel

- 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

- 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.

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



- 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

- 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?

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

- Examples:
 - Trauma
 - GI Bleeding
 - Ruptured aneurysm
 - Gynecologic bleeding, e.g. ruptured ectopic pregnancy.
- 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

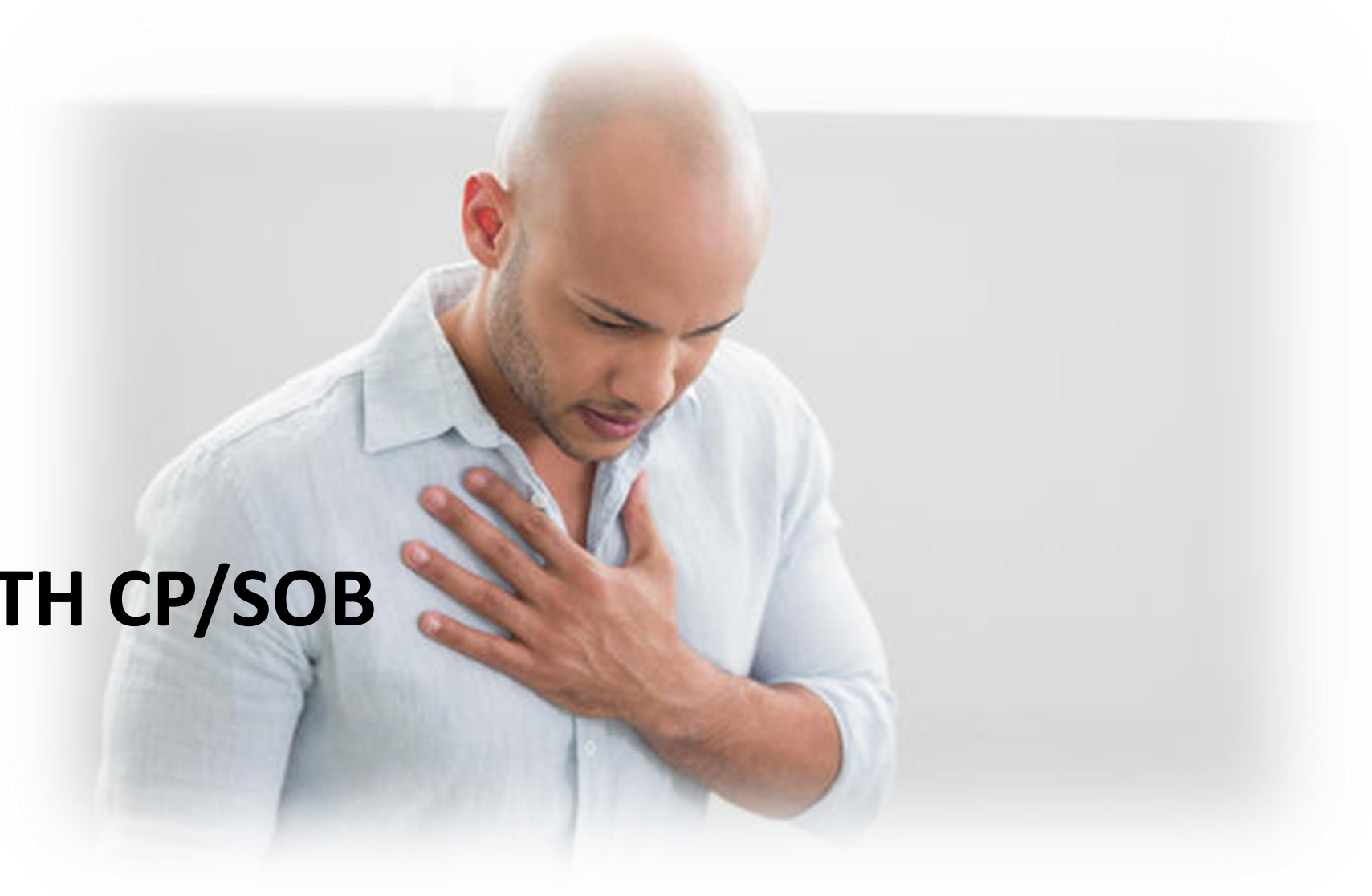
- History and physical exam critical to make diagnosis.
- Don't wait for late findings such as hypotension.
- Shock in trauma is always considered hemorrhagic until proven otherwise.
- In trauma, pts can lose life-threatening volumes of blood into the external world, as well as into the chest, abdomen, 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.

- Oxygenation!
- Volume support/IV fluids (normal saline)
 - What is permissive hypotension?
- Try to stop the fluid loss:
 - Symptom control for vomiting (e.g. antiemetic)
 - Control the bleeding if hemorrhagic (how?)
 - Tranexamic acid if hemorrhagic.
- What about inotropes/vasopressors?

Questions about Hemorrhagic Shock?

Case:

25 Y/O M WITH CP/SOB



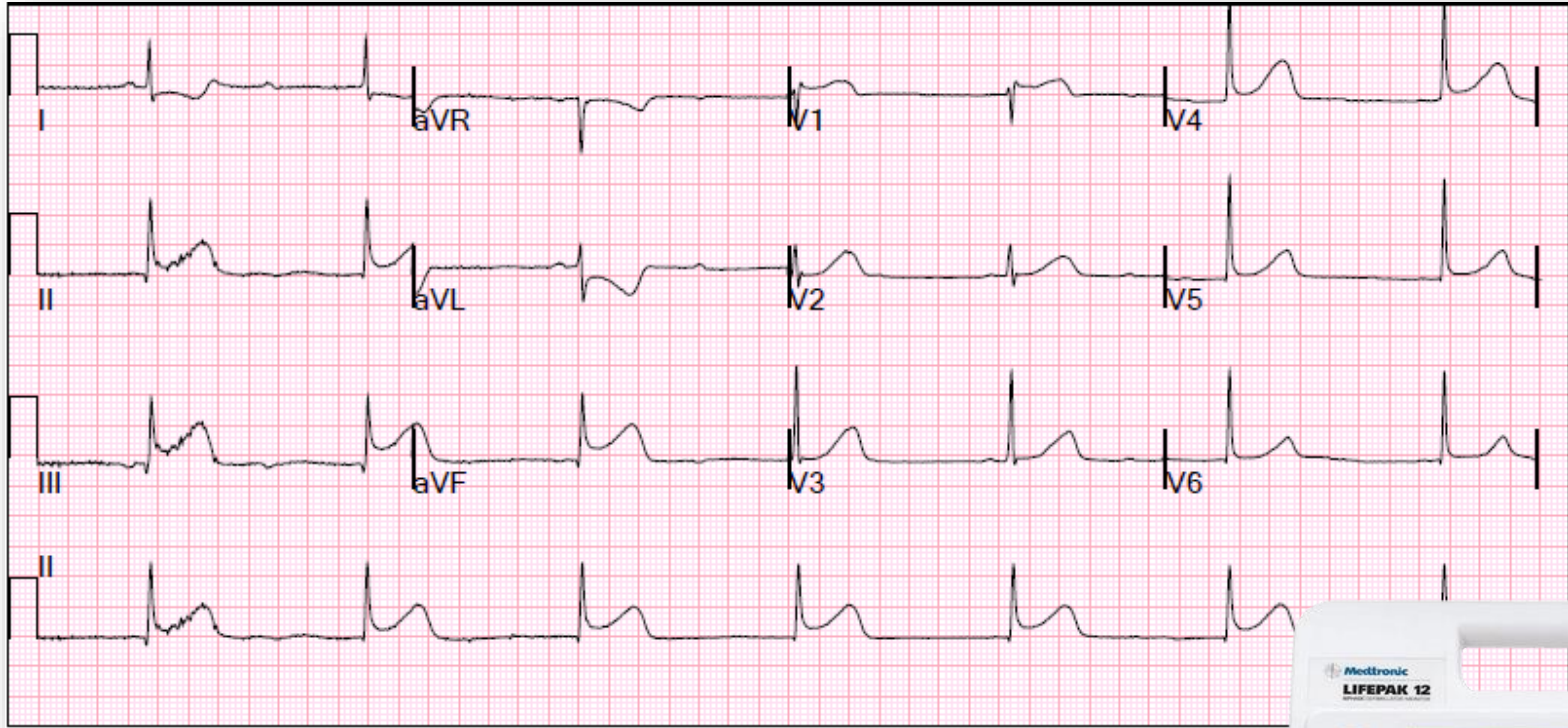
- Recall the causes:
 - PE
 - Tension pneumothorax
 - Cardiac tamponade
- How will you know when to suspect obstructive shock?
- How does obstructive shock occur in terms of pathophysiology?

- 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





- 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.

- 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

- 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?

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

- 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?

- 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:
 - $>38\text{ }^{\circ}\text{C}$ or $<36\text{ }^{\circ}\text{C}$
 - HR >90
 - RR >20
 - WBC >12 or <4

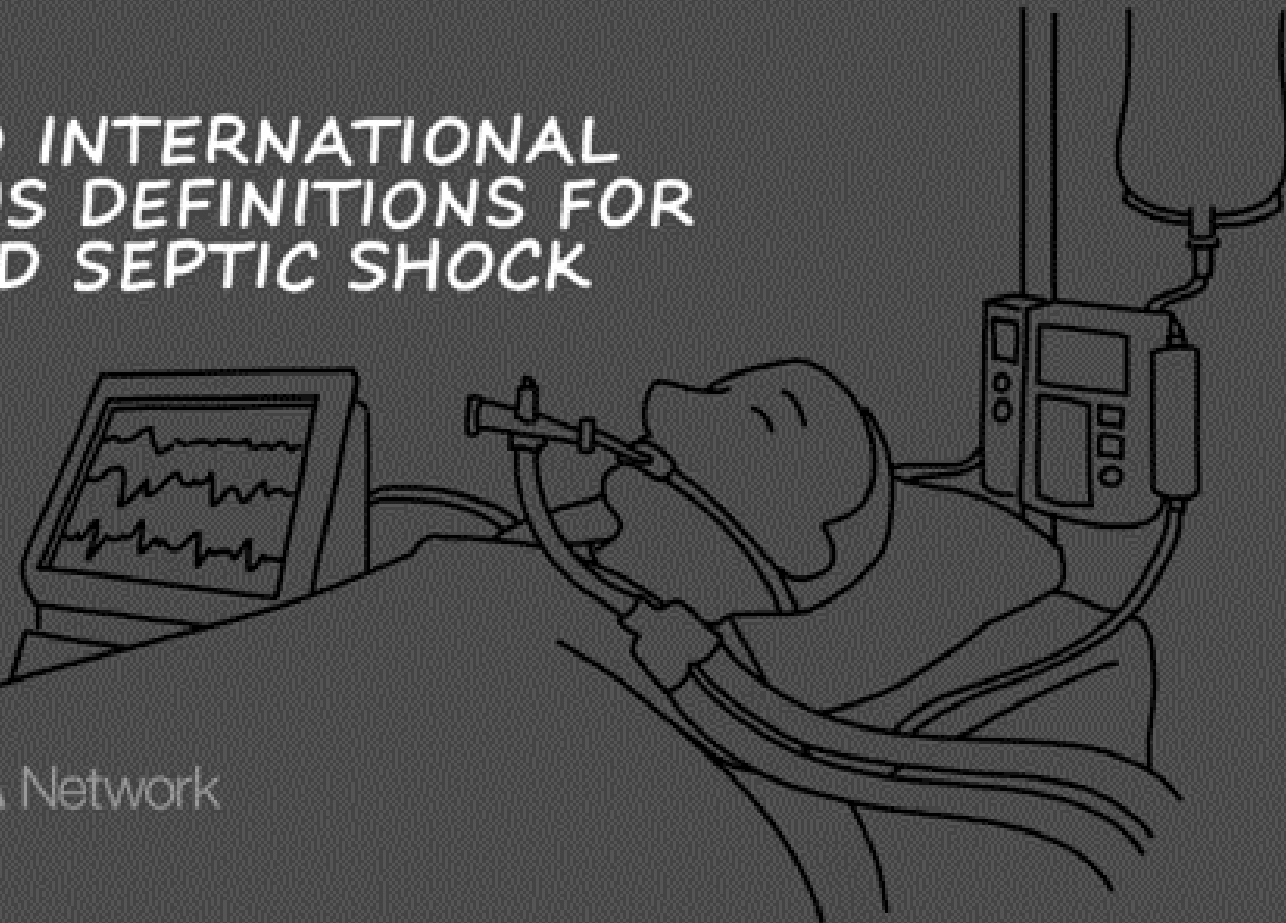
- Sepsis: SIRS due to infection.
- Severe sepsis: Sepsis associated with hypoperfusion, organ dysfunction or hypotension.
- Septic shock: Severe sepsis that does not respond to adequate fluid resuscitation.

THE THIRD INTERNATIONAL CONSENSUS DEFINITIONS FOR SEPSIS AND SEPTIC SHOCK (SEPSIS-3)

JAMA



The JAMA Network



- 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)

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

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

- 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?

- 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.

- 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.

- 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.

- 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.

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