Shock

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OBJECTIVES

- To understand Physiology of sustaining BP
- To learn about the classifications of shock
- To understand the consequences of the natural history of shock
- To be able to diagnose and plan appropriate treatments for different types of shock

Color Index:
-Doctor's Notes -Surgery Recall -Doctor's Slides+Davidson -Important -Extra

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Definitions

Cardiac Output (CO)	The amount of blood that the heart pumps in one minute - It is determined by how much blood the heart ejects with every beat (stroke volume (SV)) and how many times the heart beats in one minute (heart rate (HR)) - Cardiac output = stroke volume x heart rate (CO=SV x HR)
Systemic Vascular Resistance (SVR)	The total resistance that is present in the peripheral arterioles (it may also be called total peripheral resistance) - The radius of the peripheral arterioles is the main determinant of SVR ➔ Vasodilation (histamine release in anaphylactic shock) → decrease in SVR → decrease in mean arterial pressure ➔ Vasoconstriction (infusing the patient with dobutamine) → increases SVR → increases mean arterial pressure
Mean arterial pressure (MAP)	MAP= CO x SVR - In order to increase MAP we can either increase the SVR or increase the CO
Venous return (VR)	The amount of blood going back to the heart
Effect of sympathetic autonomic nervous system (SANS) on BP	- SANS stimulates beta adrenergic receptors in the heart → increase stroke volume and increase heart rate ➔ Leading to an increase in CO - SANS stimulated the adrenal medulla→ releases epinephrine → stimulates beta receptors in the peripheral resistance arterioles → vasoconstriction ➔ Leading to an increase in SVR - Bottom line: SANS increases CO and SVR → increases mean arterial pressure
Frank Starling	Frank Starling mechanism explains that when we stretch the myocardial tissue, we get an increased contractility - If we increase the end diastolic volume of the left ventricle → the left ventricular tissue stretches more → stronger contraction→ increased stroke volume → increase cardiac output → increased mean arterial blood pressure - Bottom line: an increase or decrease in venous return to the heart can affect the cardiac output and mean arterial pressure ➔ an increase in venous return to the heart increases the CO which increases the mean arterial pressure • Infusing a patient with normal saline → increases venous return → increases CO → increases mean arterial pressure ➔ A decrease in venous return decreases the cardiac output which decreases the mean arterial pressure • Blood loss → decreases venous return → decreases CO→ decreases mean arterial pressure ➔ These are controlled by the venous tone • SANS → increases venous tone → increases venous return → increases cardiac output → increases mean arterial pressure
Volume control	- Not all of the fluid (blood/plasma/sodium chloride) that is present in our circulatory system contributes to BP ➔ Because around 80% of this fluid is present in the large veins which do not contribute to blood pressure

Hemodynamic response to a drop in BP

- Decreased pressure on the baroreceptor present in the carotid sinus
  a. This tells the brain that there is decreased pressure in the body
  b. The brain then increases SANS firing which works on increasing blood pressure

- The opposite occurs when there is an increase in blood pressure
  - When examining the CVS you should not palpate both carotid arteries at the same time [1]
    - Applying pressure on both carotid sinuses → brain thinks that there is an increase in BP → decreases SANS → decreased BP → the patient might pass out

- What does the SANS do to increase BP?
  1. Stimulates beta adrenergic receptors in the heart → increasing cardiac output
     a. Increasing LV contractility → increasing stroke volume
     b. Increasing heart rate
     c. ↑SV & ↑HR→ ↑CO → ↑MAP

  2. Stimulates the release of epinephrine from the adrenal medulla
     a. Epinephrine stimulates beta adrenergic receptors in the peripheral resistance arterioles → vasoconstriction → ↑ systemic vascular resistance SVR→ ↑MAP

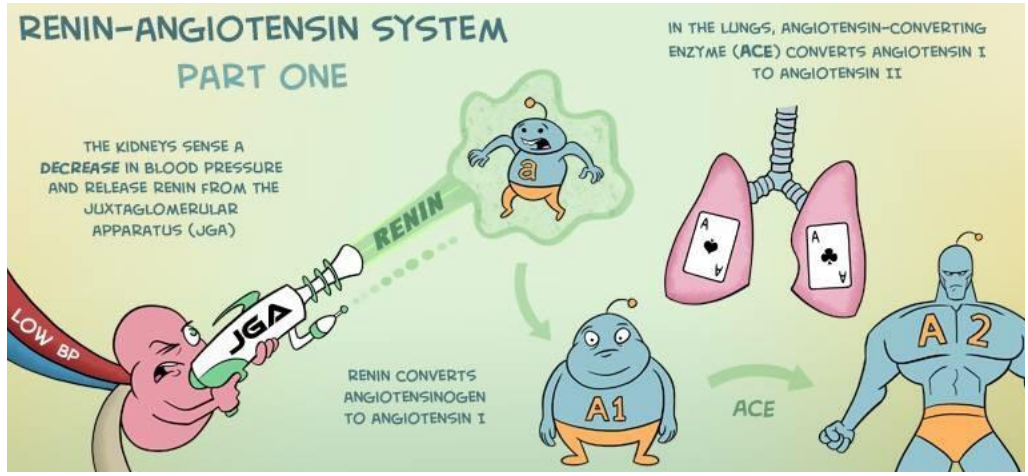
  3. Causes **veno**constriction which pushes the blood from the large veins into the heart → ↑venous return → ↑ stroke volume and CO

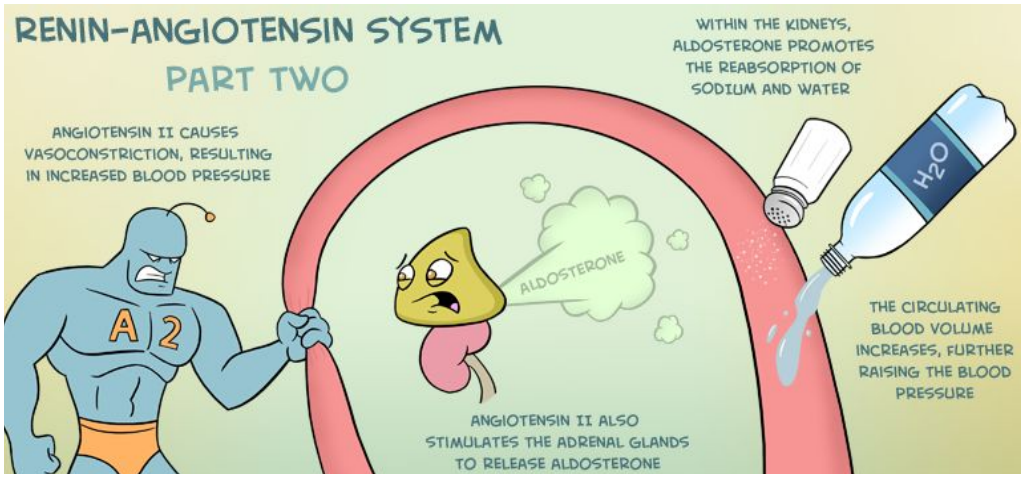
  4. Vasoconstriction of peripheral resistance arterioles also indirectly increases intravascular volume
     a. Vasoconstriction of arterioles → ↓amount of blood going to the capillaries → ↓capillary hydrostatic pressure→ resorption of fluid into the capillaries → more fluid inside the circulatory system
     b. Vasoconstriction of glomerular arterioles → ↓GFR → more time for proximal convoluted tubules to reabsorb Na^+ and H_2O→ increase fluid in the circulatory system
     c. This increased fluid in the circulatory system increases venous return→ increases CO → increases MAP
     d. Bottom line: SANS can also indirectly increase intravascular volume

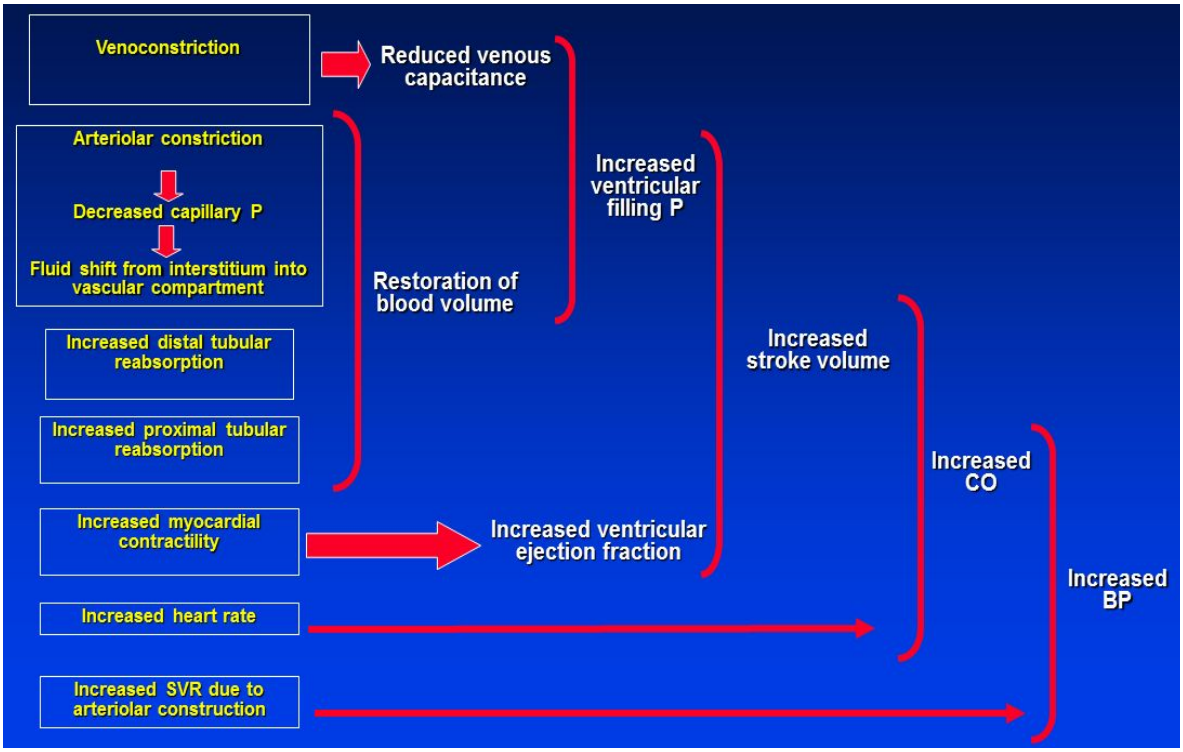
  5. Bottom line: SANS increases HR and SV, which both increase cardiac output. It also causes vasoconstriction of peripheral resistance arterioles which increases SVR. Both CO and TPR increased mean arterial pressure MAP

[1] Nicholas Talley 7th edition page 70

- Renin angiotensin aldosterone system is also activated but it takes time to show an effect. It is activated by increasing renin release from the kidney by the juxtaglomerular apparatus
  a. Renin converts angiotensinogen to angiotensin 1
  b. Angiotensin 1 is converted to angiotensin 2 by Angiotensin Converting Enzyme (ACE)
  c. Angiotensin 2 does two things
     - Vasoconstriction → increase SVR
     - Increases aldosterone release → Na^+ & H_2O retention → increase intravascular volume → increase venous return → increased cardiac output





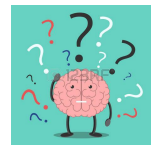


Shock

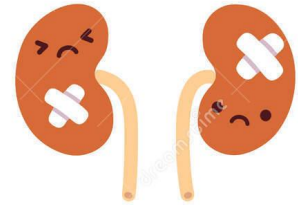
Shock is a state of altered tissue perfusion severe enough to induce derangements in normal cellular metabolic function

Signs and symptoms

- **Signs and symptoms of shock relate to decreased organ perfusion**
  - Note that these might be different depending on the type of shock. These are just some generalities and may differ depending on the specific type of shock (look at number 3 "skin changes" for an example)

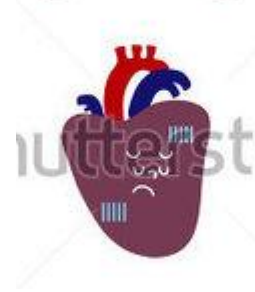


1. Altered mental status

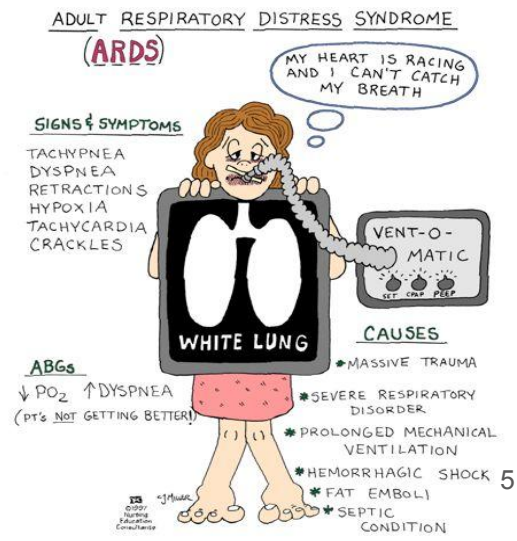


2. Decreased urine output (the most important sign of shock)
   - Might progress to acute tubular necrosis and acute kidney injury (renal failure)

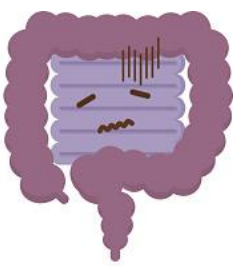
3. Skin changes
   - In case of hypovolemic and cardiogenic shock: the skin is cold and clammy
     - Because SANS is trying to restore BP by maximally constricting peripheral arterioles which leads to decreased perfusion of the skin
   - In case of septic , neurogenic and anaphylactic shock: the skin is warm
     - Because of dilatation of peripheral vascular resistance arterioles → more blood going to skin



4. Decreased blood pressure
5. Increased heart rate
6. ECG changes
   - If there is a cardiac cause of shock (massive MI) we might see specific ECG changes (ST elevation)
   - If the cause of shock is not related to the heart, we will likely see changes suggestive of myocardial ischemia due to decreased perfusion of the heart secondary to shock



7. Acute respiratory distress syndrome (ARDS)
8. Liver failure
9. Failure of intestinal barrier

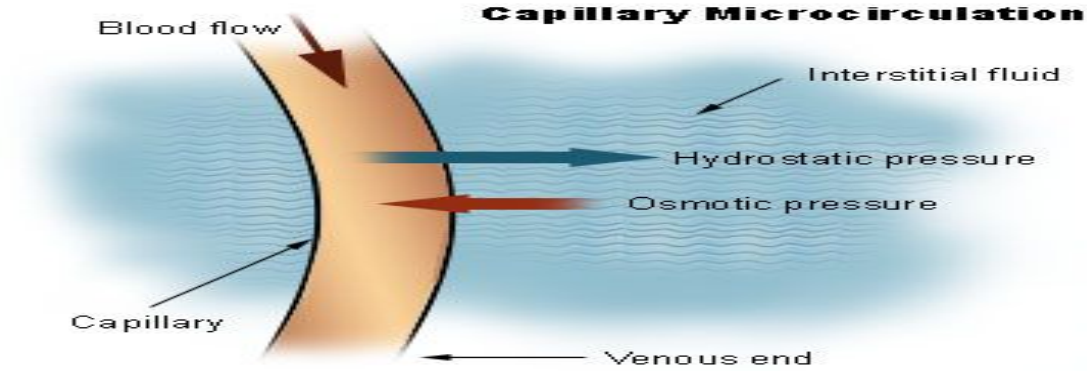


CAPILLARY LEAK SYNDROME

Capillary leak syndrome (also known as systemic capillary leak syndrome, SCLS, or Clarkson's disease) is an extremely rare medical condition characterized by self-reversing episodes during which the endothelial cells which line the capillaries are thought to separate for a few days, allowing for a leakage of fluid from the circulatory system to the interstitial space, resulting in a dangerous hypotension (low blood pressure), hemoconcentration, and hypoalbuminemia.

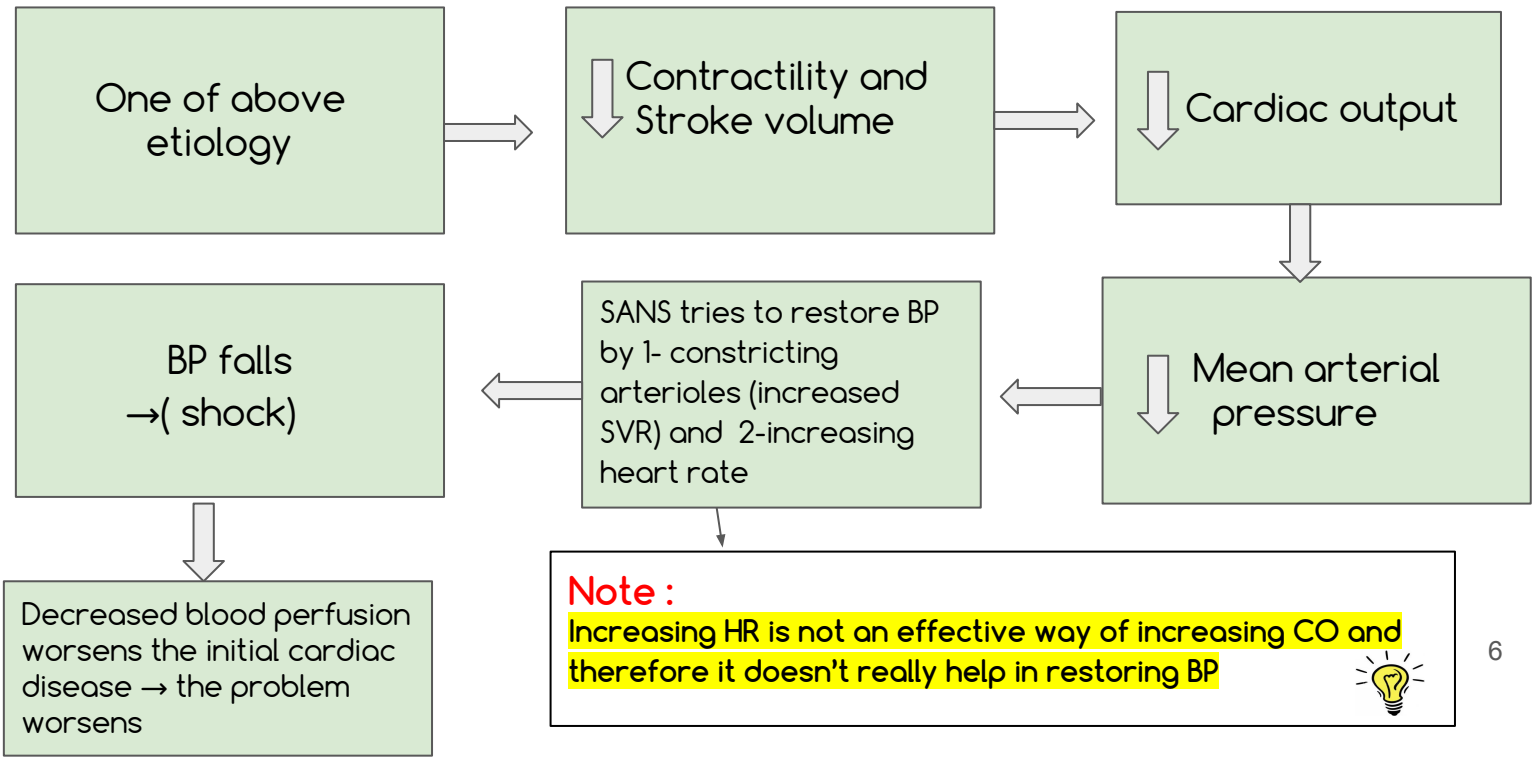
Signs and symptoms includes :

- Vasodilatation.
- A-V shunting,
- Maldistribution of flow.
- Increased capillary permeability + interstitial edema.
- Decreased oxygen extraction.
- Primary defect of oxygen utilization at cellular level .



Cardiogenic shock

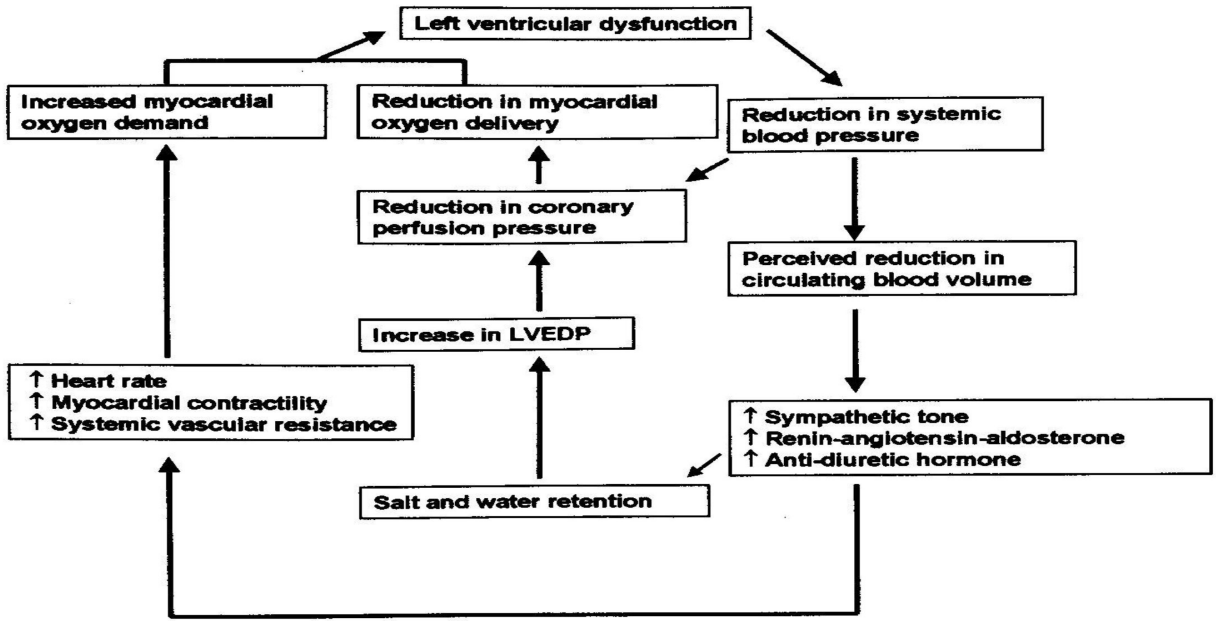
- Low perfusion due to pump failure.
- Most commonly caused by an acute myocardial infarction (a severe MI)
  Other causes: arrhythmia, cardiac tamponade, tension pneumothorax ((pneumothorax compresses the heart and it can't fill) , Papillary muscle dysfunction and valve failure.



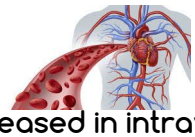
- Signs and symptoms include : Dyspnea, Rales , Pulsus alternans (increased the pulse with greater filling following a weak pulse), Loud Pulmonic component of S2 ,Gallop rhythm, cold skin and pulmonary edema.
- The vital signs associated are : Hypotension , decreased cardiac output, elevated central venous pressure, decreased urine output and tachycardia.
- Cardiogenic shock is not treated with IV fluids because the heart is not pumping properly and this might exacerbate the problem.

Clinical scenario the doctor mentioned during the lecture:

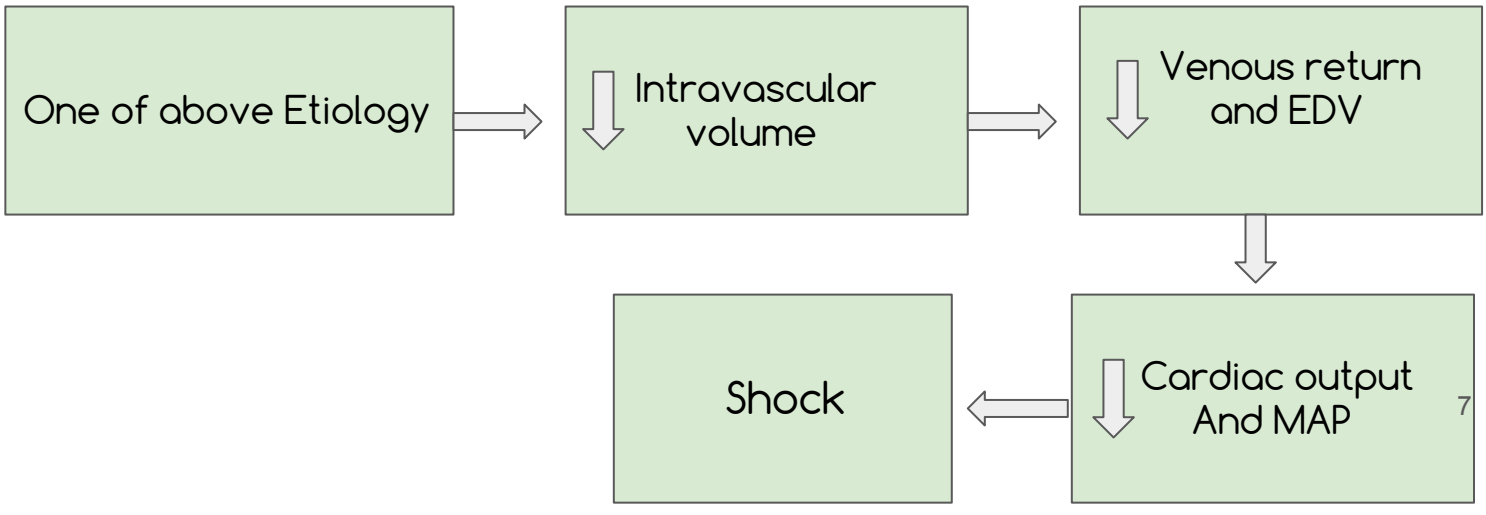
You were called in as an intern because a patient is not passing urine. Their BP is low (in shock). The first thing you should do is determine whether the patient has a cardiogenic shock or any other type of shock. Take a good history and look for signs of pump failure (increased JVP, abnormal heart sounds). This is important because cardiogenic shock is not treated with IV fluids and other types of shock are mainly treated with IV fluids.



Hypovolemic shock



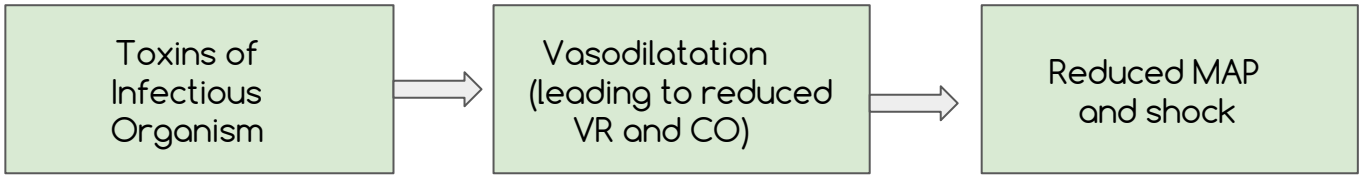
- Low perfusion due decreased in intravascular volume.
- Causes: diarrhea, hemorrhage, vomiting, burns (due fluid loss), pancreatitis, bowel obstruction , dehydration or crush Injury.



- Can be caused by capillary leak syndrome where the capillaries leak their fluid into the interstitium → decreased intravascular volume → hypovolemia
- Signs and symptoms: 1-Early stage :Orthostatic (postural) hypotension, Mild tachycardia , anxiety, diaphoresis decreased pulse pressure (systolic - diastolic) with increased diastolic pressure (due vasoconstriction) and warm extremities -> All symptoms and signs of early stage due compensatory mechanism
  2-Late Stage :Changed In mental status, Hypotension , oliguria , pale and cold skin and tachycardia.
- Treated by Intravenous fluid (Isotonic)
- The effectiveness of treatment is evaluated by : urine output, BP, heart rate , mental status, warmth extremity, capillary refill and body temperature.

Septic shock

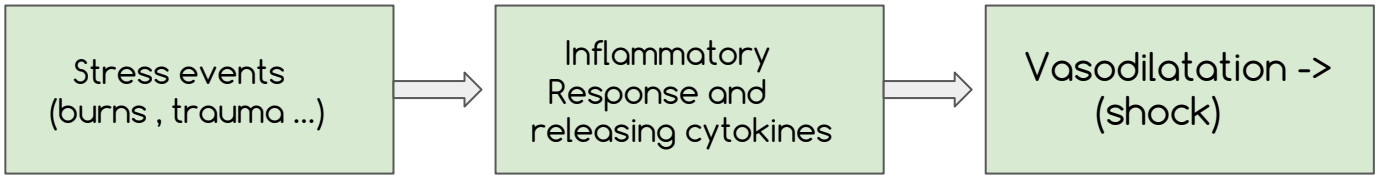
- Documented infection with hypotension



- Most common etiology is Gram negative septicemia, less common gram positive and fungus.
- The risk factors of septic shock are any factors that increases risk of infection such as : (DM, trauma, immunosuppression, hematologic disease and corticosteroids)
- Signs and symptoms: 1-initial : warm skin, full pulses and normal urine output.
  2- delayed : poor urine output, mental status changes, cold skin and hypotension
- Associated vital signs : Fever, tachycardia and hyperventilation.
- Lab findings : 1- Early : hyperglycemia, glycosuria , respiratory alkalosis , hemoconcentration and leukopenia.
  2- Late : acidosis , leukocytosis and elevated lactic acid
- Complication : Multiple organ failure , Disseminated intravascular coagulation and death
- Treatment By : Intravenous fluids, Antibiotics (empiric, then by cultures), drainage of infection (if there is abscess), pressors and zygris (if needed)

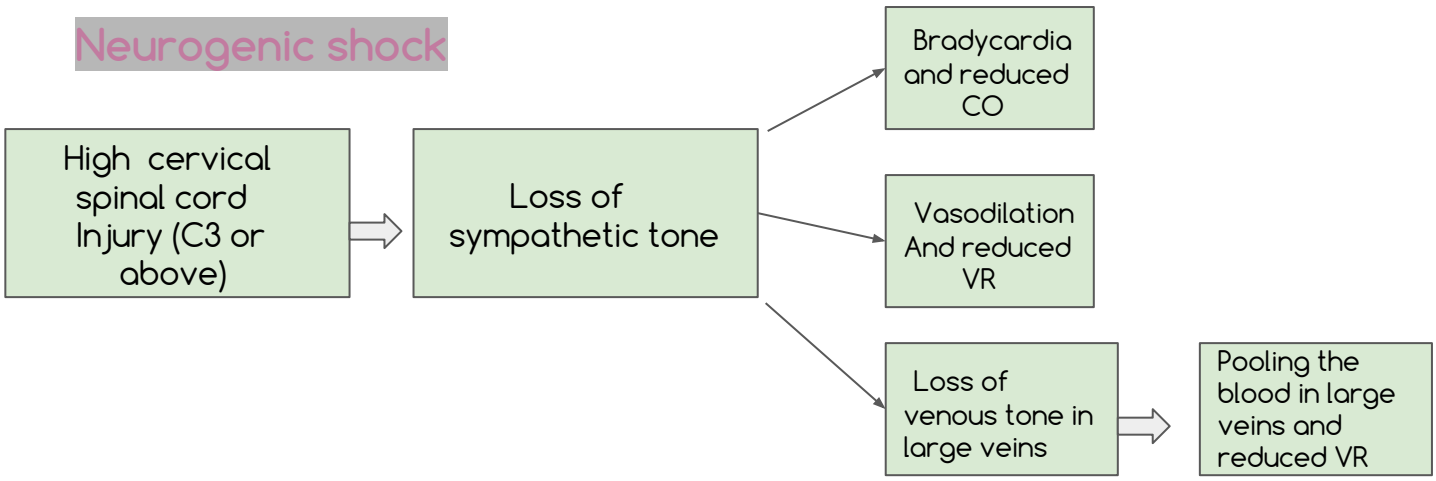
Systemic Inflammatory response syndrome

- Similar to septic shock but with the absence of an infective agent



- Criteria (2 or more to call it SIRS):
  - Temperature above 38 or below 36.
  - Heart rate more than 90.
  - Respiratory rate more than 20 or Pco2 less than 34 mmhg(4.3kpa).
  - WBC more than 12000 or less than 4000 with more than 10% bands..

Neurogenic shock



- Loss of perfusion due loss of sympathetic tone.
- The special thing about this one (hallmark) is presence of bradycardia with hypotension.
  - Normally it is the opposite because if there is hypotension the SANS must work to increase HR and incease CO
  - Arterial and venous dilatation causing hypotension.
  - Bradycardia as a result of unopposed vagal tone
- Causes : complete transection of spinal cord, partial cord injury with spinal shock and spinal anesthesia.
- Management
  1. Assess the airway
  2. Stabilize the entire spine
  3. Volume resuscitation (infuse the patient with normal saline to increase BP)
  4. Rule out other causes of shock (maybe the patient is bleeding due to the trauma that affected the spine or whatever)
  5. High dose corticosteroids (to reduce edema in spine)

> **Note**
> Spinal shock is complete flaccid paralysis immediately following spinal cord injury , may or may not be associated with circulatory shock.
>
> Septic, anaphylactic and neurogenic shock are called distributive shock due all have vasodilatation and normal blood volume.

Obstructive shock

- Low perfusion due obstruction of pulmonary or systemic circuit.
- Obstruction occurs in either : arteries or veins.
- Etiology :
  a. Cardiac tamponade→ heart chambers cannot fill → decreased CO → shock
  b. Massive pulmonary embolism → decreased blood going to lungs → decreased blood going to left ventricle → decreased CO → shock
  c. Others : Vena caval obstruction,, aortic compression or dissection.

Respiratory failure in shock

- Occurs due increase oxygen demand (especially In trauma, sepsis and hypovolemia) -> Hyperventilation -> Respiratory fatigue -> Respiratory failure.
- Presentation : respiratory acidosis, lethargy, coma and hypoxia.
- Treatment : Primary resuscitation , oxygen and mechanical ventilation (If needed)

Principles of resuscitation

1. Maintain ventilation
2. Enhance perfusion
3. Treat the underlying cause

- Oxygen delivery to tissues depends on how much blood is going to the tissue (CO) and how much oxygen is present in the blood
    a. CO
        i. HR (can increase with inotropes (drugs that increase HR)
        ii. SV (can increase with inotropes or fluids)
    b. Oxygen content
        i. Hemoglobin (can increase with blood transfusion)
        ii. Saturation of O_2 (S_aO_2)
        iii. Arterial pressure of oxygen (P_aO_2)

Summary (very important)

* Means this is the primary insult, here is where the problem started
Example: in cardiogenic shock, the primary insult is a decrease in cardiac output, thus the * is there

Type	Central venous pressure [2]	Cardiac output	Systemic vascular resistance[3]
Hypovolemic [4]	↓[5]	↓*	↑
Cardiogenic	↑	↓*	↑
Septic	↓ or ↑[6]	↑[7]	↓*
Neurogenic	↓[8]	↓	↓
Trumatic	↓ or ↑	↓ or ↑	↓ or ↑
Hypoadrenal	↓ or ↑	↓ or ↑	↓ or ↑

2 It is the pressure in the venae cavae: if heart is pumping well → there shouldn't be lots of blood in the vena cava → normal CVP (if heart not pumping well→ increased CVP)

3 If peripheral resistance arterioles dilated → decreased SVR, if constricted → increased SVR

4 We have lost a high amount of blood → decreased venous return → deceased CO

5 Patient is hypovolemic → there is no enough blood in the body to raise the central venous pressure

6 Depending if the patient went into heart failure or not (look at 7)

7 Because the initial insult is a decrease in SVR, the SANS will work on the heart to increase CO. Initially, it does increase the CO but with time the patient might get into high output heart failure and the cardiac output will decrease. So in the early stages of septic shock we might have an increased CO and we might get a decreased CO in later stages

8 Loss of venous tone on the veins → decreased pressure in the venae cavae

Anaphylactic shock (NOT Includes in slides)

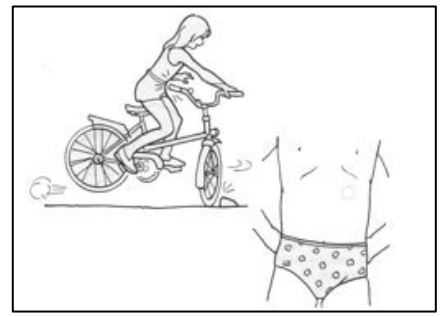
This is a severe systemic hypersensitivity reaction following exposure to an agent (allergen) triggering the release of vasoactive mediators (histamine, kinins and prostaglandins) from basophils and mast cells.
Anaphylaxis may be immunologically mediated (allergic anaphylaxis), or non-immunologically mediated (non-allergic anaphylaxis).
Anaphylactic shock results from vasodilation, intravascular volume redistribution, capillary leak and a reduction in cardiac output.
Common causes of anaphylaxis include drugs (e.g. neuromuscular blocking drugs, β-lactam antibiotics), colloid solutions, radiological contrast media, foodstuffs (peanuts, tree nuts, shellfish, dairy products), hymenoptera stings and latex.

Treatment: "BASE"
Benadryl (diphenhydramine), **Aminophylline**, Steroids, Epinephrine

Cases from doctor

Case 1: 10 years old female.Fell off bike riding down a hill. Initially well but 4 hours later complained of abdominal pain and left shoulder pain.

Vital signs: BP: 90/60, Pulse rate : 120 (tachycardic), Respiratory rate 30 (tachypneic),
Temperature :100.1, O_2 **saturation** 95% (low)
General examination : pale and anxious
LUNG: clear to auscultation
Cardiac: tachycardic with murmur best at base
Abdomen : diffuse tenderness w/o peritonitis or mass
Hb : 7.5 (low)
Abdominal CT: splenic laceration with free peritoneal fluid

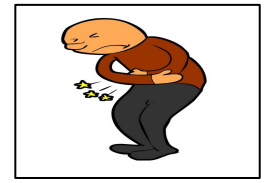


The case presentation of : *CIRCULATORY SHOCK*

Case 2 : 17 years old male, diving into water.
Vital signs : BP 90/60 (low), Pulse rate :110 (High), Respiratory rate : 24 (high)
Physical examination : paralysis below C5
Cervical X-ray: C5 fracture
Hemodynamics:
Central venous pressure Decreased
Cardiac output Decreased
Systemic vascular resistance Decreased

The case presentation of : *Neurogenic shock*

Case 3 : 15 years old male, 4 day history of abdominal pain, N/V and anorexia
Vital signs: BP : 70/60 (low), Pulse rate : 130 (high), Respiratory rate 28 (high)
Temperature :102.4, O_2 **saturation** : 99%
General examination: moderate distress from abdominal pain
Cardiac : tachycardia
Abdomen: diffuse tenderness w peritonitis
WBC : 19,600 (high), 90% segs
Hb : 14.2
Hemodynamics: *Cardiac output Increased and systemic vascular resistance Decreased*
Diagnosis *: Perforated appendicitis*

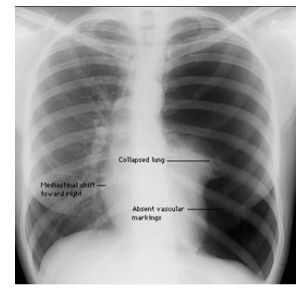


The case presentation of : *septic shock*

Cases from doctor cont.

Case 4 : 17 year old male. Training for track team.

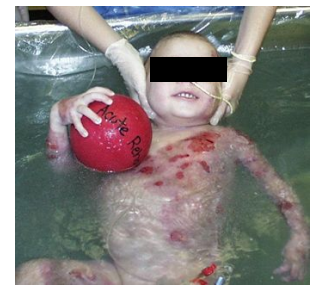
Vital signs: BP: 70/50 (low), Pulse rate : 140 (high), Respiratory rate 35 (high)
O_2 saturation : 88%
Physical examination : absent breath sounds in L lung field, distended neck veins
DX: tension pneumothorax
Hemodynamics:
Central venous pressure Increased
Cardiac output Decreased
Systemic vascular resistance Normal



The case presentation of : *CARDIOGENIC SHOCK*

Case 5 : 3 years old male. Clothes ignited from roaster at Thanksgiving.

Vital signs: BP: 60/60 (low), Pulse rate : 170 (high), Respiratory rate : 35 (high),
Temperature :102.4, O_2 saturation : 89%
General examination : moderate distress
Lung: tachypneic, clear to auscultation
Cardiac: tachycardic, regular
Skin: 60% TBSA partial and full thickness burn
DX: 60% TBSA burn
Hemodynamics:
Cardiac output Decreased
Systemic vascular resistance Increased



The case presentation of : *HYPOVOLEMIC SHOCK (LOSS OF FLUID INTO INTERSTITIUM) due to CAPILLARY LEAK*

Thank you