



Shock

Dr. Aayad AlQahtani

Our notes in **green** –imp in **red**

Surgery Team 429

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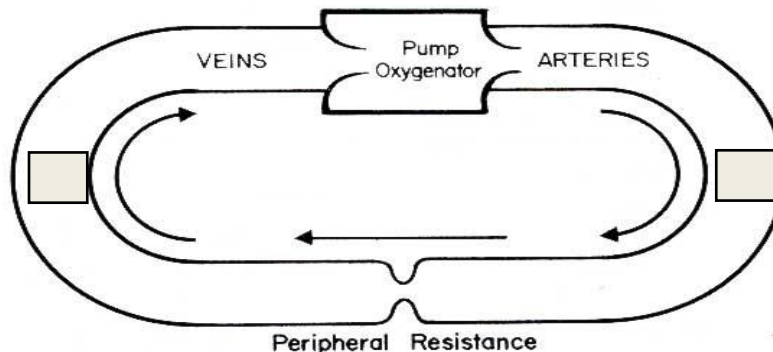
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OBJECTIVES:

- To understand Physiology of sustaining blood pressure
- 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

CHANGES IN MANY ELEMENTS REGULATE BP AND PERFUSION:

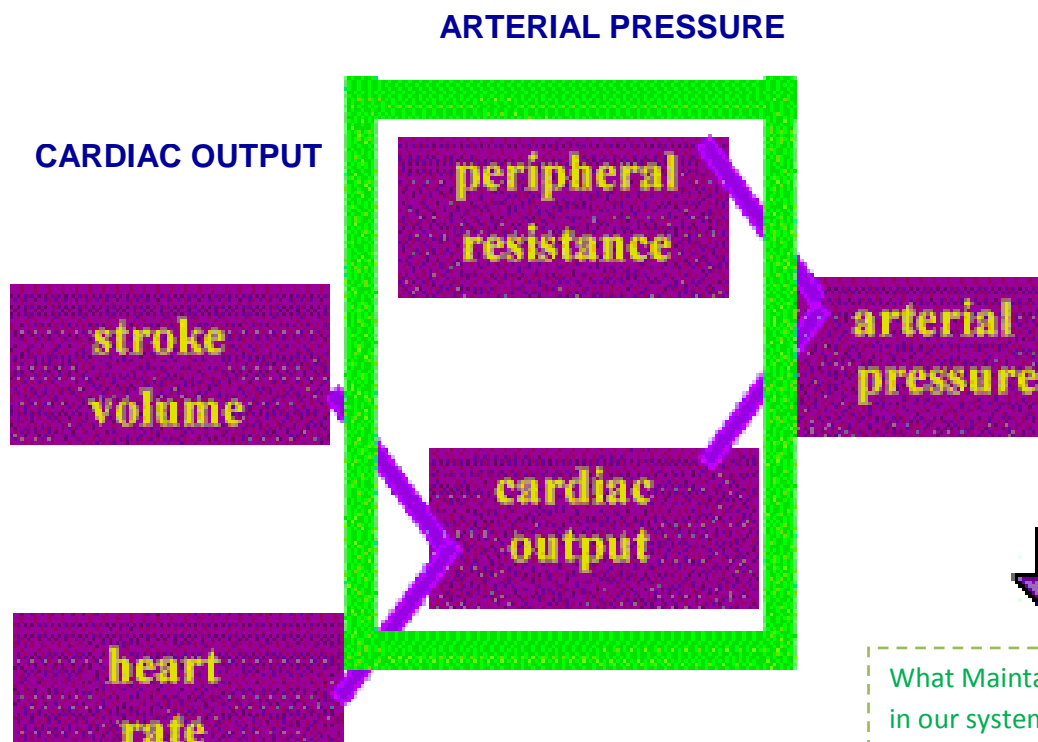
- ✓ Intravascular volume
- ✓ Heart
- ✓ Arteriolar bed
- ✓ Capillary exchange network
- ✓ Venules
- ✓ Venous capacitance circuit
- ✓ Large vessel patency



- **Decreased peripheral resistance**
 - *Decreased arterial blood pressure ($MAP = CO \times PR$)*
- **Increased peripheral resistance**
 - *Decreased venous return*
 - *Decreased EDV*
 - *Decreased SV*
 - *Decreased CO ($CO = HR \times SV$)*
 - *Decreased arterial blood pressure ($MAP = CO \times PR$)*

Heart Rate X Stroke Volume (*Decreased intravascular volume, decreased EDV*) =

Cardiac Output → **Cardiac Output X Peripheral Resistance** = **Arterial Pressure**



HOW DOES INTRAVASCULAR VOLUME AFFECT BP AND PERFUSION?

- Alters mean blood pressure
 - *Decrease in intravascular volume=decreased BP*
- Alters venous return to the heart
 - *Decrease in intravascular volume=*
 - *Decreased venous return=*
 - *Decreased end diastolic volume*
 - $CO = HR \times SV$
 - $CO \times SVR \approx MAP$
- How can intravascular volume be lost?

Examples:

- *Bleeding*
- *Failure to rehydrate*
- *Loss of third space fluids (sweating)*

What Maintain blood pressure in our system and artery is a pump which is the heart.

So what in the arteries can be effected as a result of low or high blood pressure

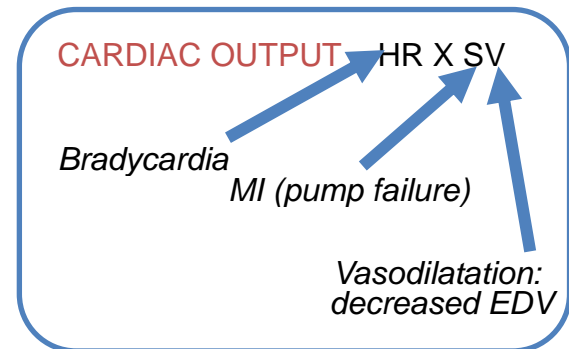
- Most important determine in arteries is the peripheral resistance which determine the BP

$Bp = Co \times \text{peripheral resistance}(PR)$

- so increase PR lead to increase BP
- decrease in the PR lead to decrease BP

HOW DOES CARDIAC FUNCTION ALTER BP AND PERFUSION?

- Cardiac output is the result of:
 - ✓ Heart rate
 - ✓ Contractility
 - ✓ Loading conditions
- Examples of changes that can alter cardiac output
 - ✓ Heart rate (bradycardia or tachycardia)
 - ✓ Contractility (MI or cardiomyopathy)
 - ✓ Load (histamine release: vasodilation)



HOW DOES THE RESISTANCE CIRCUIT: ARTERIOLAR BED AFFECT BP AND PERFUSION?

- Decreases in arteriolar tone produce:
 - ✓ Hypotension
 - ✓ Decreased perfusion to vital organs
- Increases in tone will prevent optimal cardiac performance (increased afterload=decreased contractility)

in the arterioles what determine the BP?

Increase in the permeability and the oncotic pressure ,

The oncotic pressure will increase due to increase in the present of protein in the blood vessel which lead to the movement of the fluid from extravascular to intravascular ,so more volume enter the system, this normal process we may loss it when we have sepsis, trauma ,cytokine and Systemic inflammatory response(SIR)

What is the oncotic pressure?

It is the osmotic pressure which the pressure result from the present of the protein

HOW DOES THE CAPILLARY EXCHANGE NETWORK AFFECT BP AND PERFUSION?

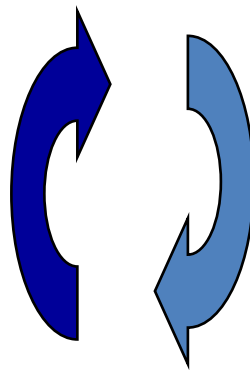
- Largest area of the vascular tree
- Site of exchange of nutrients, electrolytes and fluids
- Alterations in microvascular integrity (e.g., capillary leak syndrome) result in loss of intravascular volume
- Blockage of or shunting away from small vessels leads to decreased tissue perfusion

- **HOW DOES THE VENOUS CAPACITANCE CIRCUIT AFFECT BP AND PERFUSION?**

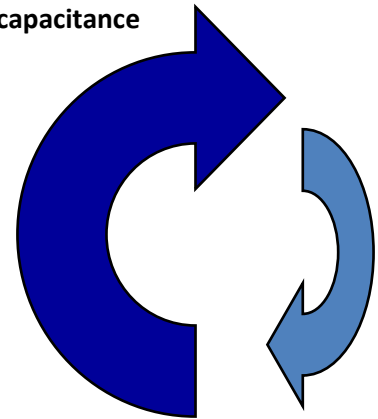
- Portion of the circulatory system contains 80% of the intravascular volume
- Decrease in effective circulating blood volume and MAP caused by:

- ✓ Decreases in venous tone

- ✓ Increases in venous vascular capacitance



Normal



INCREASED VENOUS CAPACITANCE

*Decreased effective blood volume
Decreased MAP*

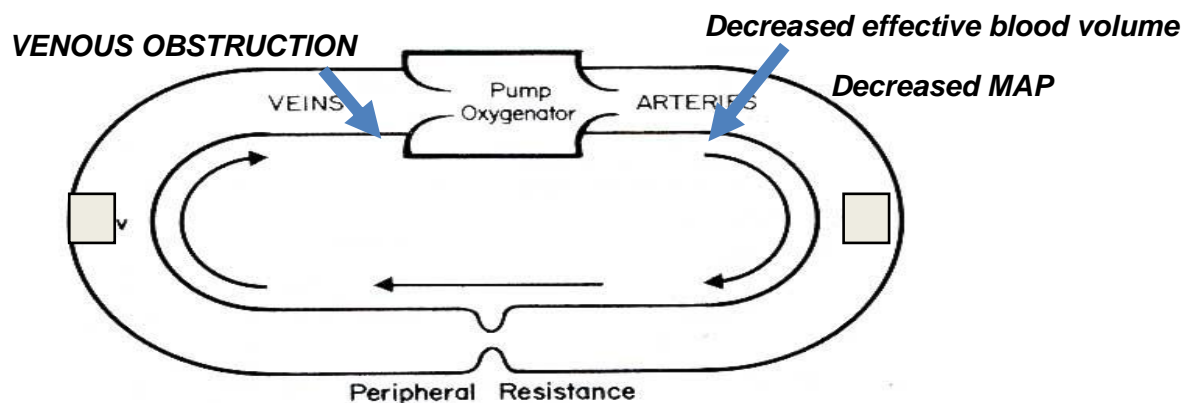
If we have vasodilatation there is what we called venous capacitance which is the capacity of the venue that will accumulate the volume, so some time if you look for the total volume in the body is the same but the distribution is different (volume stay in the system not go back to the heart) these shock called **redistributive shock**.

HOW DOES LARGE VESSEL PATENCY AFFECT BP AND PERFUSION?

- Obstruction of the systemic or pulmonic circuit will decrease ventricular ejection and systemic perfusion
- Venous obstruction will decrease venous return
- **Examples of obstructive shock:**
 - ✓ *Massive pulmonary embolism*
 - ✓ *Venous occlusion*

Another type of shock which is really happen is the **obstructive shock**. Here we don't have problem in the peripheral resistance but the artery here is completely suddenly block by a massive pulmonary embolism. It will block the pulmonary artery which will block the right ventricle that will lead to no volume reaching the left ventricle = no EDV.

Rare dieses to the artery enter to the right atrium (SVC & IVC), like tumor or lung cancer can block the SVC and that might result in shock.



DEFINITION OF SHOCK:

State of altered tissue perfusion severe enough to induce derangements in normal cellular metabolic function . *Shock: low perfusion that cause tissue hypoxia*

TYPES OF SHOCK imp ! : *MORE THAN ONE TYPE MAY BE PRESENT !!

| Type of Shock | Clinical causes | Primary mechanism |
|--|--|---|
| Hypovolemic | Volume loss | Exogenous blood, plasma, fluid or electrolyte loss |
| Cardiogenic | Pump failure | Myocardial infarction, cardiac arrhythmias, heart failure |
| Distributive " shock that will result in vasodilatation > vasodilatation or leak > lead to the movement of the blood outside the vessel > decrease the end diastolic volume." | Increased venous capacitance or arteriovenous shunting | Septic shock, spinal shock, autonomic blockade, drug overdose " Neuorogenic Anaphylactic Sepsis" |
| Obstructive | Extra-cardiac obstruction of blood flow | Vena caval obstruction, cardiac tamponade, pulmonary embolism, aortic compression or dissection |

The clinical signs and symptoms of shock relate to decreased organ perfusion:

- Mental status changes: *decreased cerebral perfusion*

- Decreased urine output: *decreased renal perfusion*

- Cold clammy extremities:

Decreased perfusion to the skin due to diverted blood flow

- EKG changes:

1. *May indicate myocardial ischemia*

2. *May be primary event (cardiogenic shock) or due to*

decreased myocardial perfusion due to shock from other causes

On different level of the body what happen?

Different part so different response :

Decrease blood to skin and muscle

Increase blood to brain, heart and adrenal gland

The most 2 important organs the body try to maintain is the heart and the brain .

Decrease urine output in the early stage of shock and cold extremities (because of vasoconstriction of skin vessels to maintain blood)

HEMODYNAMIC PARAMETERS THAT MAY INDICATE SHOCK:

- Heart rate

Initial tachycardia (attempt to increase CO)

- Rhythm

Regular and tachycardic

- Blood pressure

Low

- Cardiac output

Usually low

EFFECTS OF SHOCK AT THE ORGAN LEVEL *imp!*

- Kidney

Oliguric renal failure

- Liver

Liver failure

- GI tract

Failure of intestinal barrier (sepsis, bleeding)

- Lung

Capillary leak associated with or caused by sepsis and infection (ARDS = adult respiratory distress syndrome) Q: what happen to the lung in SIRS? ARDS (Imp)

HEMODYNAMIC RESPONSE TO SHOCK : Mechanisms for restoring cardiovascular homeostasis

- Redistribution of blood flow

Attempt to preserve perfusion to vital organs

- Augmentation of cardiac output

Increased heart rate

Increased peripheral resistance

- Restoration of intravascular volume

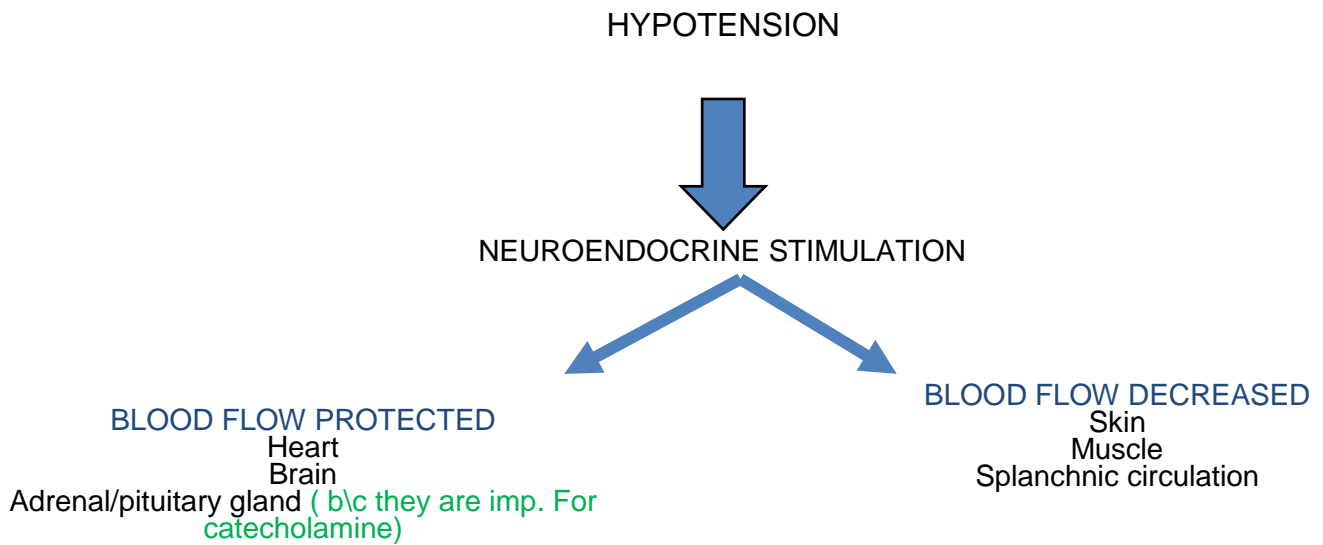
The organ which significantly will contribute to the shock response is the kidney? How?!!

The kidney is part of the solution not the problem when the body response to the shock , it will reabsorbed the volume and salt and returned it to the systems that will maintain intravascular volume .

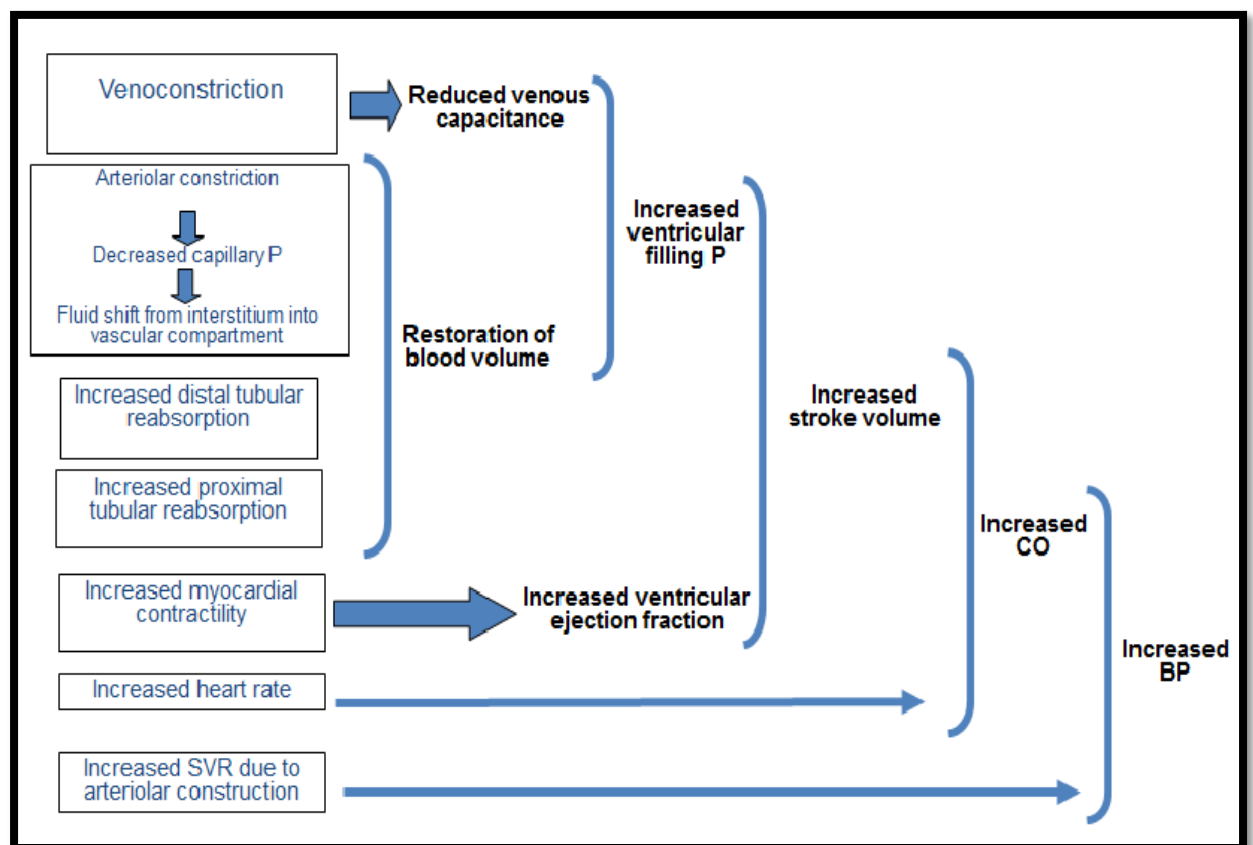
So if you have pts with low blood pressure start to think where is the problem in this equation

$$CO=SV*HR$$

HEMODYNAMIC RESPONSE TO SHOCK : REDISTRIBUTION OF BLOOD FLOW



HEMODYNAMIC RESPONSE TO SHOCK:



CARDIOGENIC SHOCK:

DECREASED CARDIAC FUNCTION

- Decreased ventricular function
 - ✓ Myocardial infarction
 - ✓ Pericardial tamponade
 - ✓ Tension pneumothorax
- Ineffective cardiac contraction
 - ✓ Primary arrhythmias

How do you know if it is cardiogenic or not?

1- SOB

2-rest JVP[†]

3-lower limb edema

→ CLINICAL FINDINGS:

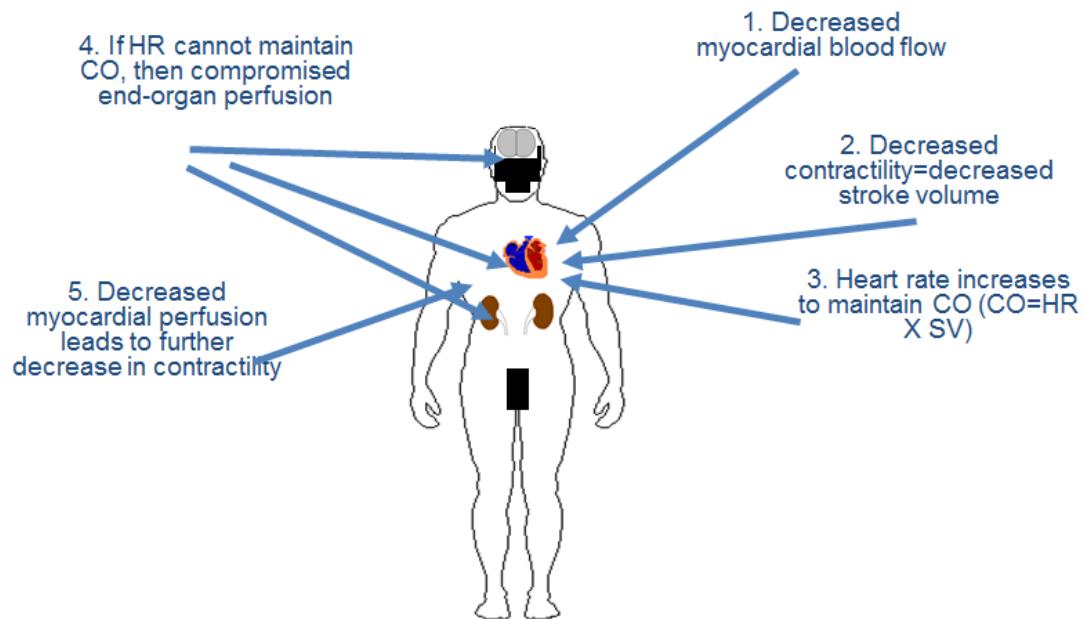
- Hypotension
- Tachycardia
- Tachypnea
- Oliguria

- Caused by the progressive loss of myocardium
- Usually due to an acute myocardial infarction
- When the total amount of myocardium affected reaches a critical point, myocardial function begins to deteriorate
- While stroke volume decreases, the heart rate increases in an effort to maintain cardiac output ($CO = SV \times HR$)
- But increased HR is limited and CO falls to levels that are inadequate to support end-organ function
- Coronary perfusion decreases and this in turn causes progressive myocardial ischemia with progression of myocardial injury

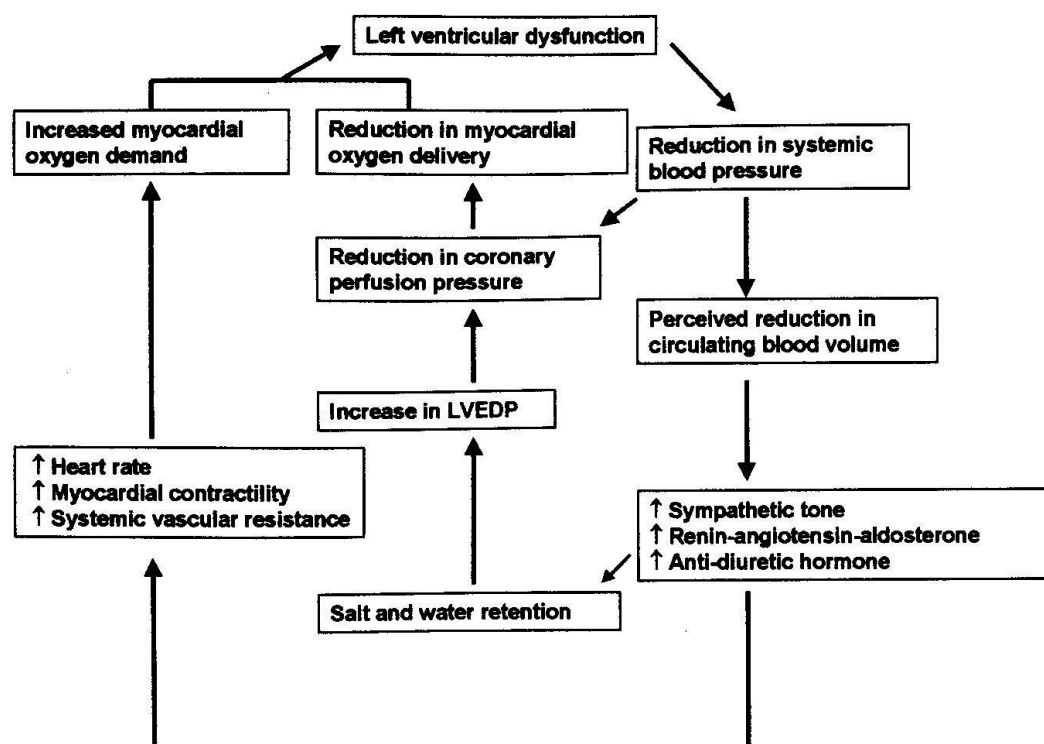
-In cardiogenic the volume is not the problem.

-The only shock that you suppose to not give volume is cardiogenic otherwise the pt. will have pulmonary edema (b\c the ventricle is not functioning →all volume will go to the RT ventricle then to lung) !

-the treatment will depend on the cause



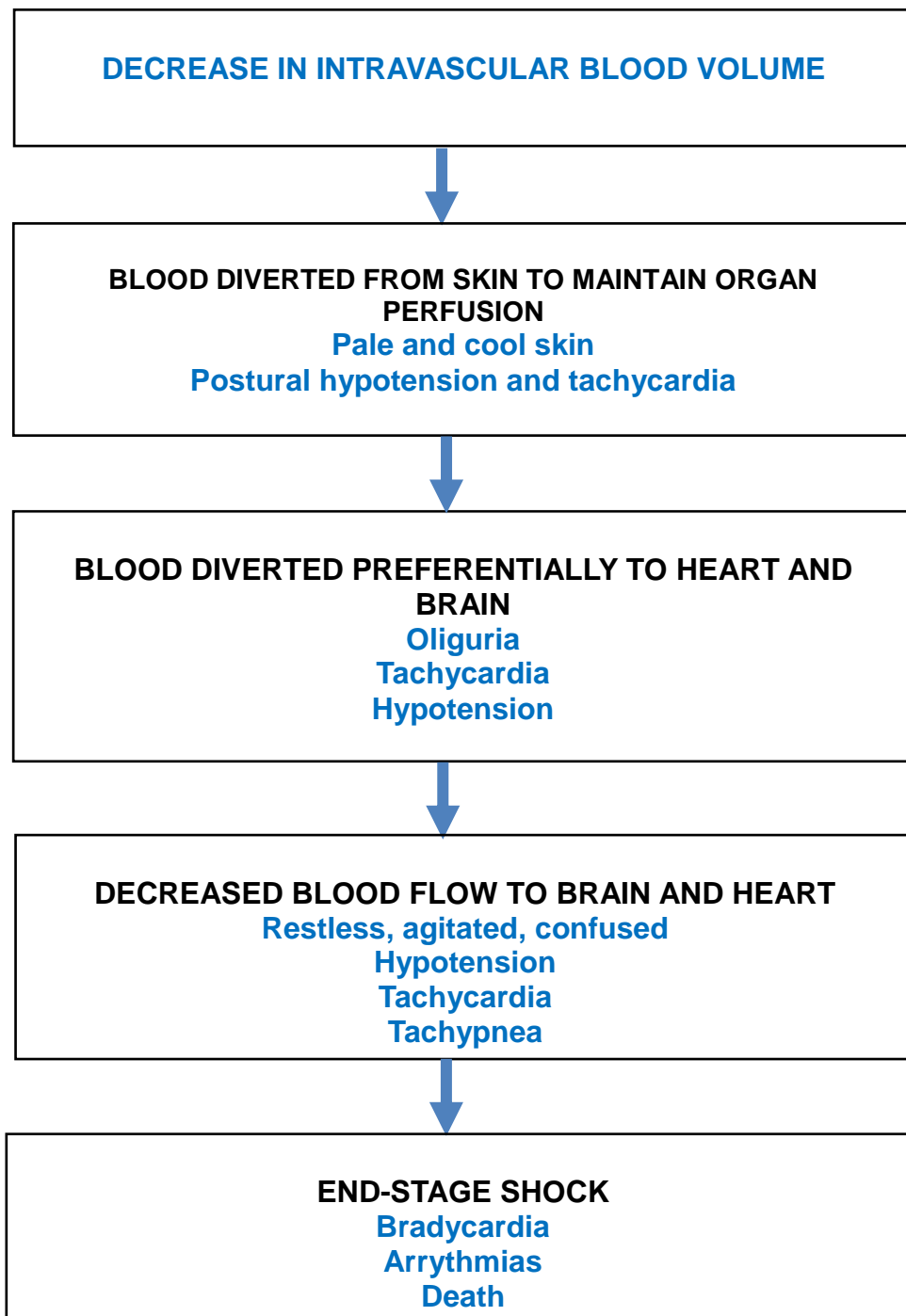
EVENTS IN CARDIOGENIC SHOCK



HYPOVOLEMIC SHOCK

DECREASE IN INTRAVASCULAR BLOOD VOLUME (Hemorrhage. Vomiting, Diarrhea ,Fluid sequestration "Intraluminal – bowel obstruction, Intraperitoneal – pancreatitis, Interstitial – burns")

→ DECREASE IN CARDIAC OUTPUT AND TISSUE PERFUSION



To treat it give:

Volume + stop
the cause

SEPTIC SHOCK:

To treat it give:

Volume +
antibiotic

SEVERE INFECTION W RELEASE OF MICROBIAL PRODUCTS
Release of vasoactive mediators



HYPERDYNAMIC STATE
Peripheral vasodilation
Increased cardiac output
Fever, tachycardia, tachypnea, warm skin



MAINTENANCE OF
INTRAVASCULAR VOLUME
Hyperdynamic shock

FAILURE TO MAINTAIN
INTRAVASCULAR VOLUME
Hypodynamic shock
Cool skin, tachycardia, hypotension,
oliguria

DIAGNOSING SHOCK STATE BASED ON HEMODYNAMIC PARAMETERS:

| TYPE | Central Venous Pressure | Cardiac Output | SVR |
|--------------------|-------------------------|------------------------|------------------------|
| Hypovolemic | Decreased | Decreased | Increased |
| Cardiogenic | Increased | Decreased | Normal or Increased |
| Septic | Decreased or increased | Increased | Decreased |
| Traumatic | Decreased | Decreased or increased | Decreased or increased |
| Neurogenic | Decreased | Decreased | Decreased |
| <u>Hypoadrenal</u> | Decreased or increased | Decreased or increased | Decreased or increased |

What the different between the SIRS and the sepsis?

In sepsis: there is an agent (Bacteria, viruses)

SIRS: there is only the inflammatory response

But both will result an increase the permeability so volume will be drag out of the intravascular system and that will lead to shock lead to low BP

Systemic Inflammatory Response Syndrome (SIRS):

- The patients demonstrate a similar response as sepsis but without infective agents.
- The criteria are : (two or more to call it SIRS)
 - Temperature >38 or <36 (in sepsis it could be hypothermia OR hyperthermia !)
 - Heart rate >90
 - RR >20 or a pco2 <34 mmHg (4.3 kpa)
 - WBC $>12,000$ Or $<4,000$ with more than 10% bands

Neurogenic Shock:

It is a shock that result from a high spinal cord injury (e.g Cervical spine injury) > so it's Because of trauma in the spinal cord, the injury be in level T2 or above

- This will result in loss of sympathetic tone
- Loss of sympathetic tone will result in:
 - Arterial and venous dilatation causing hypotension.
 - Bradycardia as a result of unopposed vagal tone.

The typical feature is hypotension with bradycardia (non- neurogenic patient must have tachycardia as a result of shock)

Management of neurogenic shock:

- Assessment of airway
- Stabilization of the entire spine
- Volume resuscitation
- R/O other causes of shock
- High dose corticosteroids.

PRINCIPLES OF RESUSCITATION:

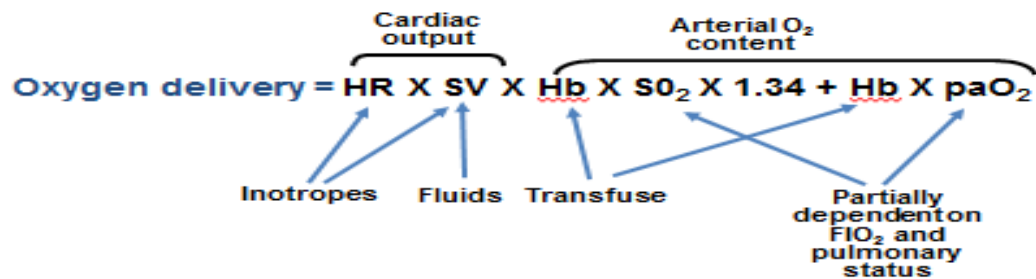
- Maintain ventilation: ensure oxygen delivery
- Enhance perfusion
- Treat underlying cause

We do the ABC:

A→airway B→breathing C→circulation (pulse)

If you find pulse in the : 1- radial the BP will be near 90 ,, 2- femoral →80 ,, 3-carotid → 60

TREATMENT OF SHOCK ENHANCING PERFUSION/OXYGEN DELIVERY:



SUMMARY:

- Shock is an altered state of tissue perfusion severe enough to induce derangements in normal cellular function
- Neuroendocrine*, hemodynamic and metabolic changes work together to restore perfusion
- Shock has many causes and often may be diagnosed using simple clinical indicators
- Treatment of shock is primarily focused on restoring tissue perfusion and oxygen delivery while eliminating the cause

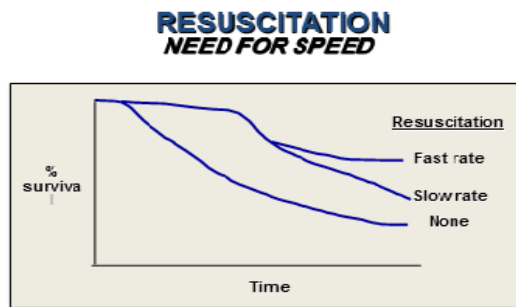
b/c u r graduated from new block system u should know that> as what dr said :p

- epinephrine: more on beta which found mostly in the heart
- Nor-epinephrine: more on alpha which found mostly in vessels

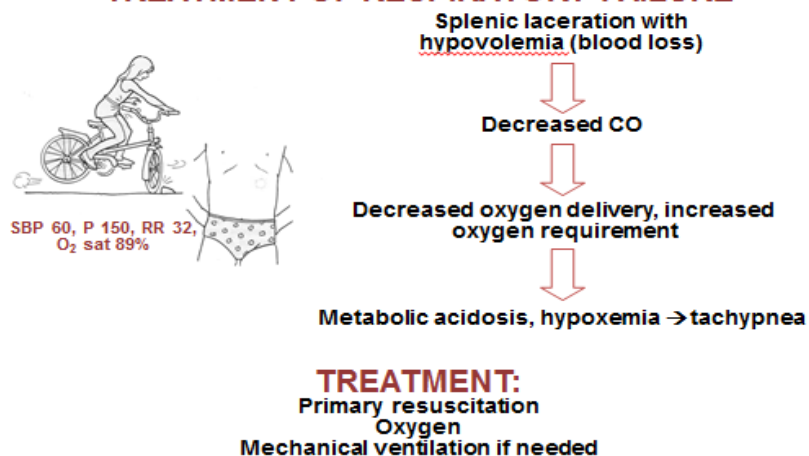
CASE PRESENTATIONS

-CIRCULATORY SHOCK:

- 10 yo female
- Fell off bike riding down a hill. Initially well but 4 hrs later complained of abd pain and L shoulder pain
- **VS:** BP 90/60, P 120 (tachycardic), RR 30 (tachypneic), T 100.1, O₂ sat 95% (low)
- **GEN:** pale, anxious
- **LUNG:** clear to auscultation
- **COR:** tachycardic with murmur best at base
- **ABD:** diffuse tenderness w/o peritonitis or mass
- **Hb** 7.5 (low)
- **Hemodynamics:**
 - *Central venous pressure* *Decreased*
 - *Cardiac output* *Decreased*
 - *Systemic vascular resistance* *Decreased*
- **ABD CT:** splenic laceration with free peritoneal fluid



TREATMENT OF RESPIRATORY FAILURE



NEUROGENIC SHOCK:

- 17 yo male
- Diving into water
- **VS:** BP 90/60 (low), P 110 (high), RR 24 (high)
- **PE:** paralysis below C5
- **Cervical X-ray:** C5 fracture
- **Hemodynamics:**
 - Central venous pressure Decreased
 - Cardiac output Decreased
 - Systemic vascular resistance Decreased

SEPTIC SHOCK:

- 15 yo male
- 4 day history of abdominal pain, N/V and anorexia
- **VS:** BP 70/60 (low), P 130 (high), RR 28 (high), T102.4, O₂ sat 99%
- **GEN:** moderate distress from abd pain
- **COR:** tachycardic
- **ABD:** diffuse tenderness w peritonitis
- **WBC** 19,600 (high), 90% segs, Hb 14.2
- **Hemodynamics:**

Cardiac output Increased

Systemic vascular resistance Decreased

DX: perforated appendicitis

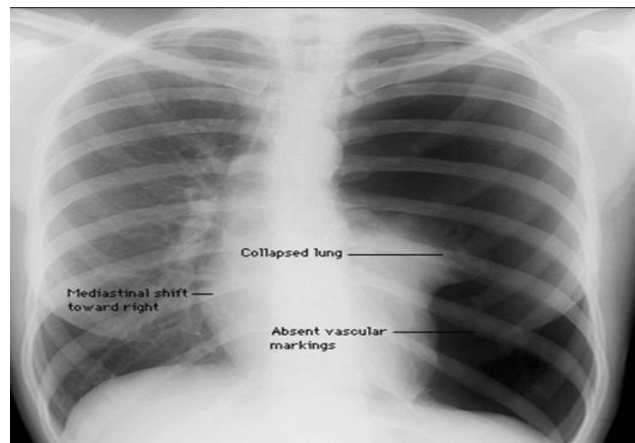
CARDIOGENIC SHOCK:

- 17 yo male
- Training for track team
- **VS:** BP 70/50 (low), P 140 (high), RR 35 (high), O₂ sat 88%
- **PE:** 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



CAPILLARY LEAK:

- 3 yo male
- Clothes ignited from roaster at Thanksgiving
- VS: BP 60/60 (low), P 170 (high), RR 35 (high), T102.4, O₂ sat 89%
- GEN: moderate distress
- LUNG: tachypneic, clear to auscultation
- COR: tachycardic, regular
- SKIN: 60% TBSA partial and full thickness burn
- Hemodynamics:

Cardiac output Decreased

SVR Increased

DX: 60% TBSA burn

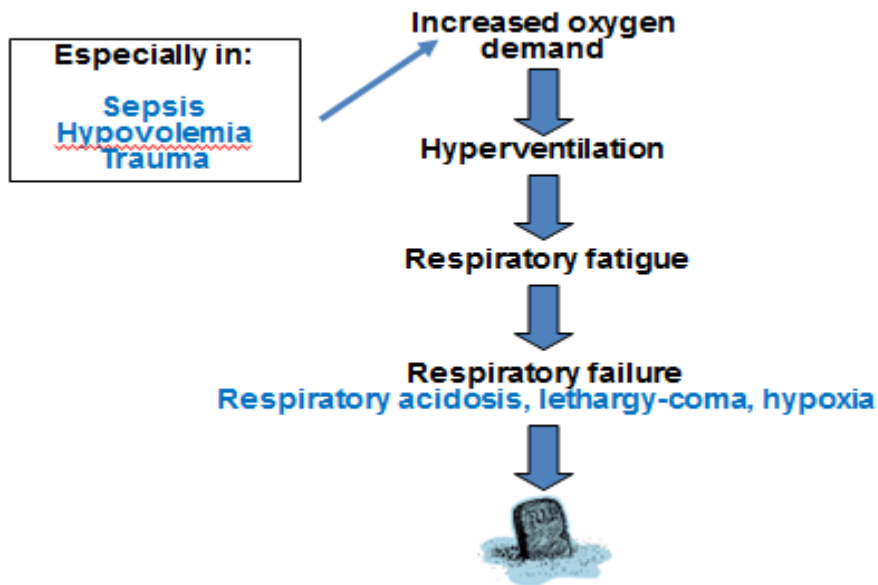


HYPOVOLEMIC SHOCK (LOSS OF FLUID INTO INTERSTITIUM)

CAPILLARY LEAK SYNDROME :

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

MAINTAIN VENTILATION



OVERVIEW :

GENERIC CLASSIFICATIONS OF SHOCK

- **Circulatory shock**

Critical reduction in tissue perfusion

Results in organ dysfunction and, if not treated, death

Usually accompanied by signs and symptoms:

- Oliguria
- Mental status changes
- Weak thready pulse
- Cool clammy limbs

- **Septic shock**

- *Hypotension*

- *Vasodilatation with warm limbs.*

The only shock we will get warm extremities is in the sepsis shock, why?

Because we will have hyperdynamic heart which will pump the whole blood which arrive to the system warm that in early stage, but later it become also cold because with the time the return blood become cold.