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**THESE NOTES WERE WRITTEN BY:**

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**LOOKING  
FOR  
MORE  
MEMBERS !**

**TYPED, EDITED & MODIFIED BY:**

**ABO MALEK**

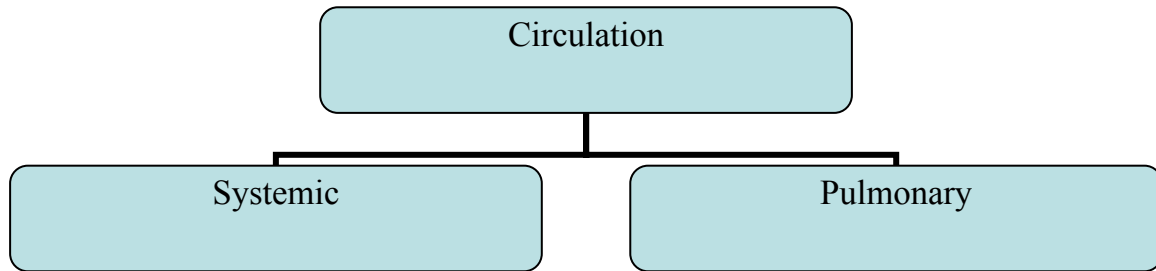
**,,, WITH REGARDS**

**PSL TEAM (A) 427**

**IMPORTANT NOTICE**

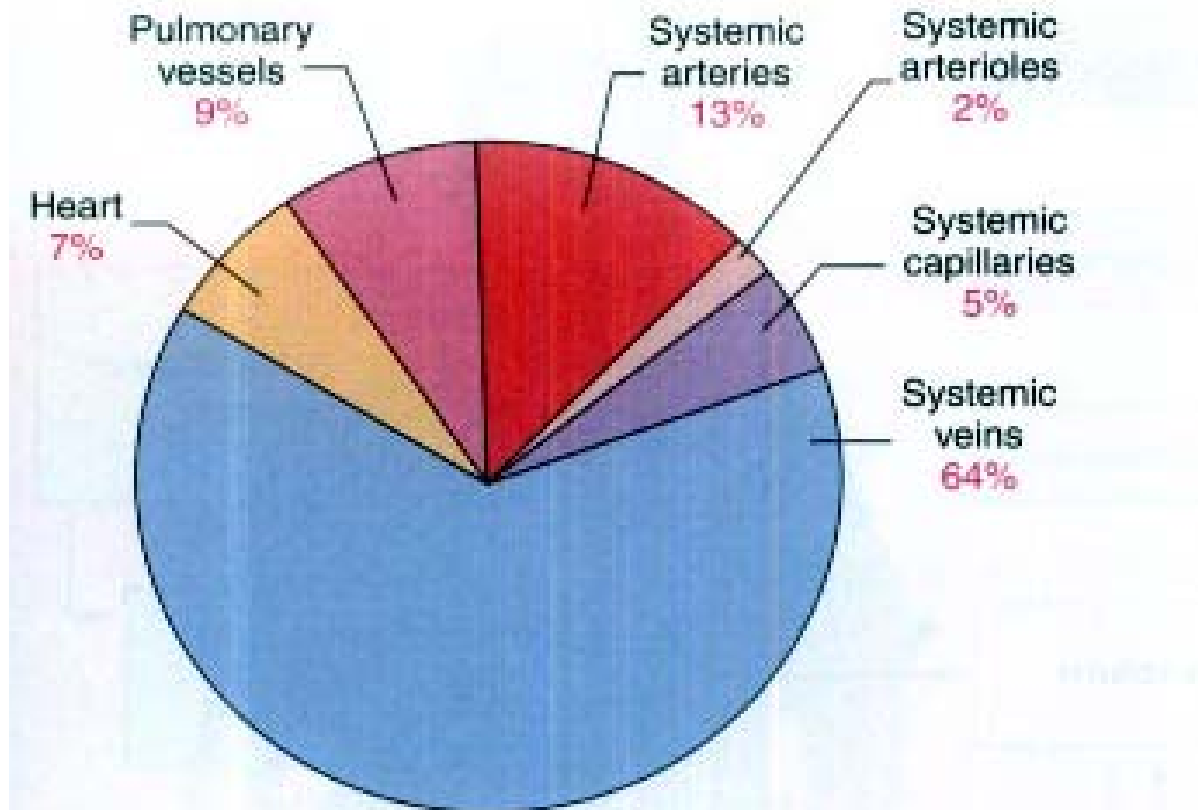
THESE NOTES ARE ONLY A **HELP** FOR STUDYING PHYSIOLOGY OF CIRCULATION AND IT **SHOULD BE REFERED TO OTHER RESOURCES FOR THE EXAM PUPROSE.**

**CIRCULATION**



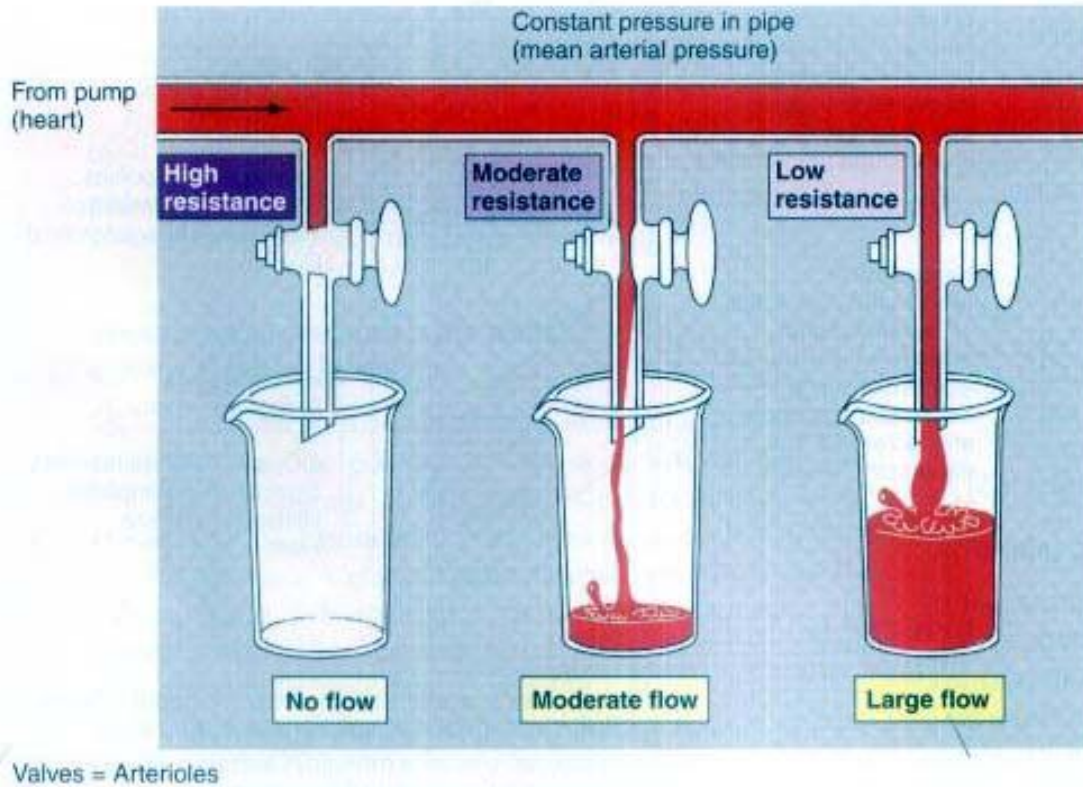
Blood circulation (Systemic) provides tissues with requirement (nutrient, O<sub>2</sub>, hormones, electrolytes) and removes CO<sub>2</sub> & wastes.

*Where is blood usually found?*



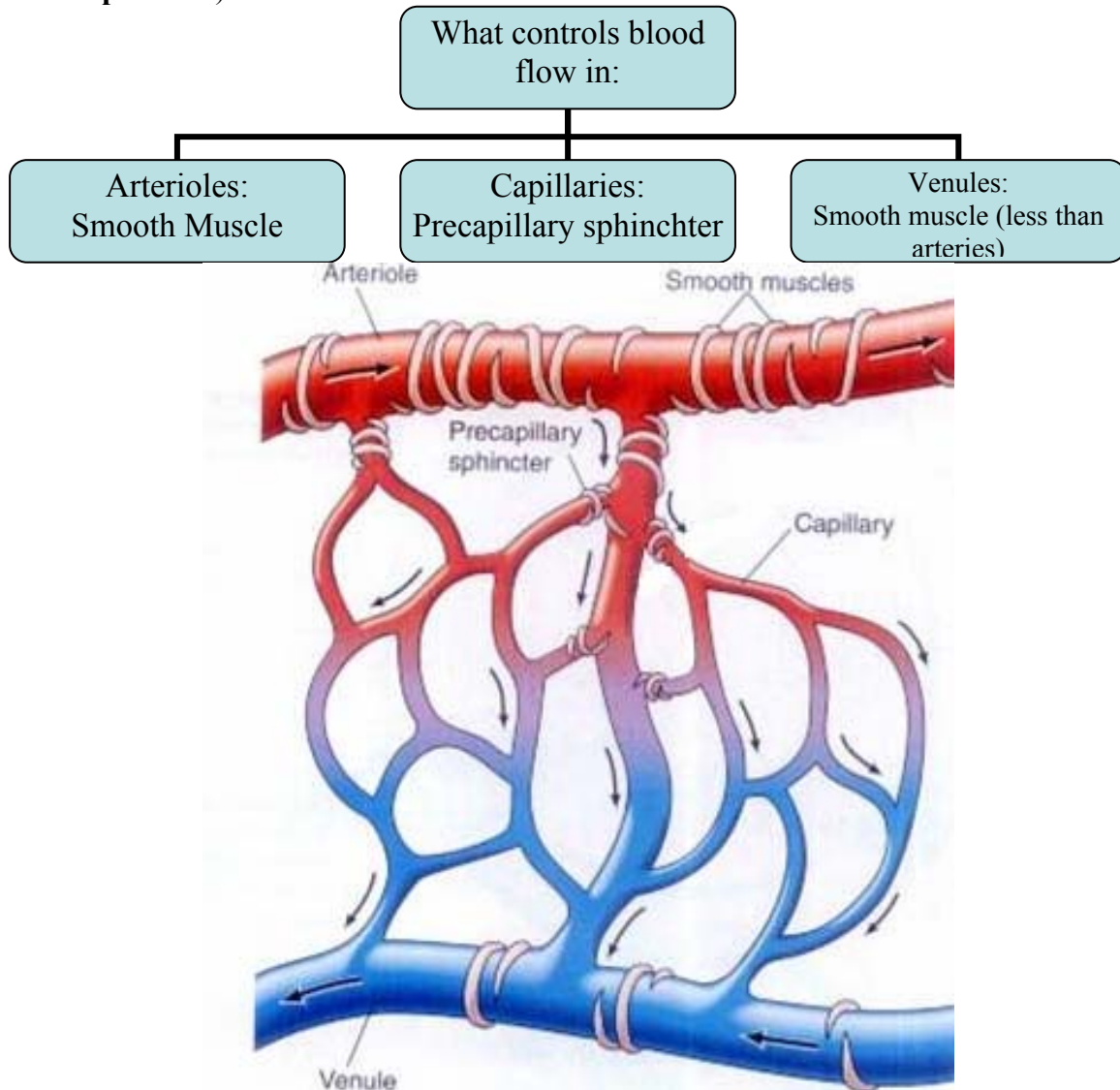
- We found that the **systemic veins** contain **most the blood**

- If there is **constriction** in small arteries & **arterioles** there will be a **↓blood flow**. They are called: resistance blood vessels.



- **Capillaries are called exchange vessels:**
  - They are the **most important** part of the circulation because **exchanging of materials** takes place there.
  - **Thin** wall (one layer of endothelial cells & no smooth muscle)
  - **Diameter** is very **small**
  - Many **pores** to allow movement of small molecular substances (exchange takes place rapidly).
  - Very **large surface are** (the largest), because every cell needs its own exchange of nutrients. (Total surface area of capillaries: 500,000 – 700,000m<sup>2</sup>).
  - There are  $\approx$  10 billion capillaries.
- **Venules & Veins: Capacitance (سعة) vessels:**
  - **Less thick.**
  - **Greater Diameter.**
  - **Largest amount of blood.**
  - In case of loss of blood (e.g. hemorrhage) it squeezes to push blood to the right atrium to compensate the loss.
  - Less smooth muscle in thinner walls.
  - It is usually under **low pressure**.

- The pressure is **decreasing** when the blood flows **from the left ventricle to aorta** → arteries → capillaries → venules → veins → (right atrium having the least pressure)



**Vasomotor tone:-**

- Only **sympathetic innervations** play a role in the **vasomotor** (vaso=vessel) tone:
  - Normal sympathetic supply → Normal Diameter.
  - ↑ sympathetic supply → Vasoconstriction (↓diameter)
  - ↓sympathetic supply → Vasodilatation (↑ diameter)

**Factors affecting arteriole diameter:**

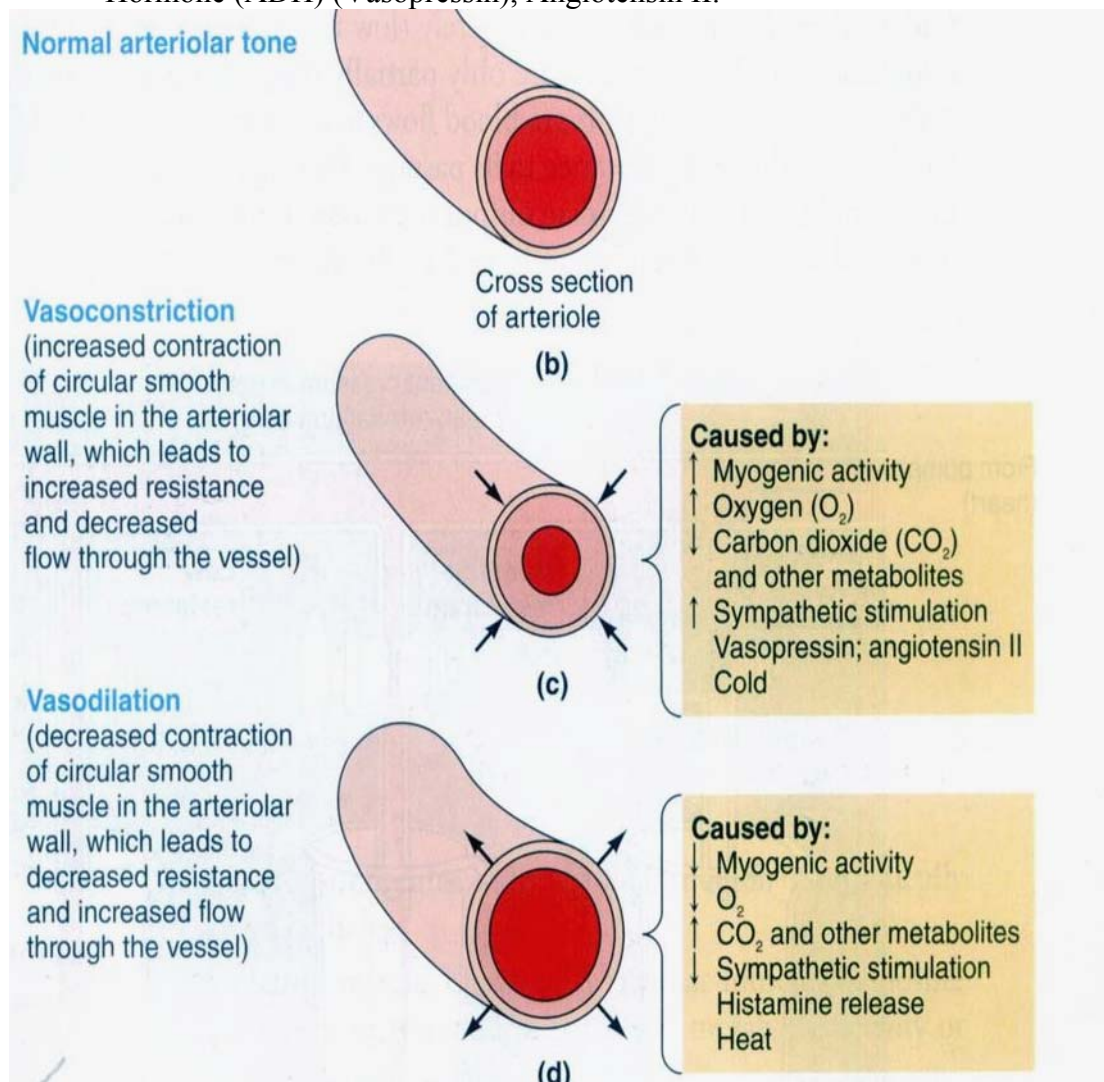
**Vasodilation**

- ↓O<sub>2</sub> , ↑CO<sub>2</sub> , ↑H<sup>+</sup>
- ↑Adenosine,

- ↑heat
- ↑Endothelial Derived Releasing Factor (EDRF) e.g. Nitric Oxide (NO).
- ↓ympathetic stimulation (below normal)
- Myogenic Activity
- **Hormonal Factors (not local)** e.g. histamine, Bradykinin

**vasoconstriction:-**

- ↑O<sub>2</sub> , ↓CO<sub>2</sub> , ↓H<sup>+</sup>
- ↓Adenosine
- Cold
- ↓EDRF e.g ↓NO
- ↑ympathetic stimulation
- **Hormonal Factors (not local)** e.g. Norepinephrine, epinephrine, AntiDiuretic Hormone (ADH) (Vasopressin), Angiotensin II.

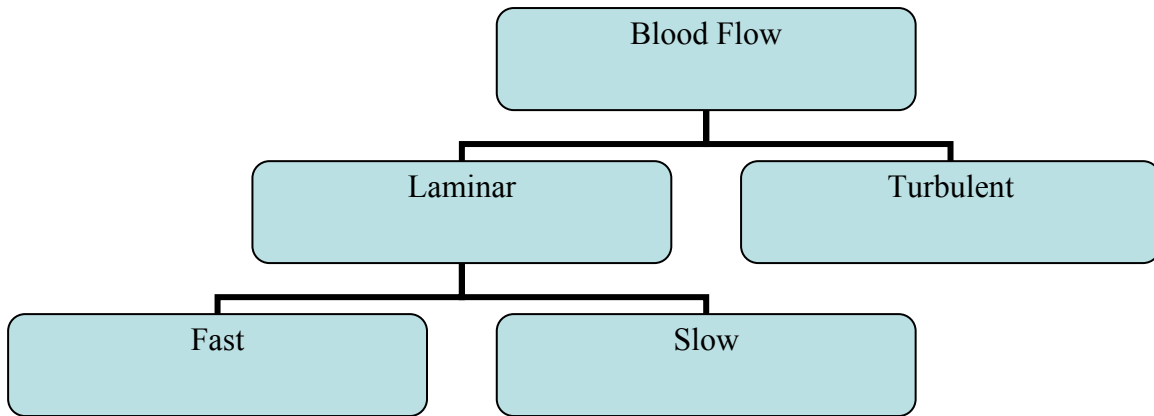


**Flow laws:**

Ohm's Law: $Q = \frac{\Delta P}{R}$	Q: Blood Flow $\Delta P$ : pressure Difference R: Resistance ( <b>inversely proportional to flow</b> ).
$R = \frac{L \times \eta}{r^4}$	R: Resistance L; Length $\eta$ : viscosity (depends mainly on plasma proteins & blood cells) r: radius
(From the 2 above equations we get:) $Q = \frac{\Delta P \times r^4}{L \times \eta}$	<b>The physiological importance of the equations:</b> $Q \propto r^4$ $R \propto \frac{1}{r^4}$ (The main determinant (محدد) of blood flow is the <b>radius</b> , which is proportional by its 4 <sup>th</sup> power).

- Blood flow in capillaries is **intermittent** (متقطع) flow

**Types of blood flow:**



**Laminar Flow:**

- **Steady**
- **No Sound**
- **Normal** Blood flow
- **Less energy** required
- Normal velocity
- **Streamline** flow

**Slow flow:**

- There is **resistance**.
- There is **friction** between RBCs and the wall of the vessel.



**Fast flow:**

- **Less resistance**
- **RBCs** are located in the **center** leading to **less friction** between RBCs and the walls.

**Turbulent Flow:**

- Happens when anything **blocks** the way of the RBCs.
- Blood goes in **different directions** (mixing of blood flow).
- There are sounds heard called **murmurs** or **Korotkoff sounds**<sup>1</sup>.
- **Less velocity.**
- More **energy loss.**
- Slow blood flow.
- Occurs by narrowing of arteries wall e.g. obstruction of blood vessels when we measure B.P.

**What happens during...****Constriction of vessel:**

- ↑B.P., ↓velocity, ↑ resistance

**Relaxation:**

- ↑ Blood flow, ↑ velocity, ↓resistance

**↑ in Hematocrit (RBC Count):**

- ↑ viscosity → More B.P. required to push blood → ↑ resistance, ↓velocity, ↓O<sub>2</sub> Delivery

So hematocrit is a determinant of the B.P. & viscosity.

**BLOOD PRESSURE (B.P.)**

**Definition:** It is pressure exerted by blood against the vessel wall.

**Systolic B.P.**

- **Definition:** the **highest** pressure recorded in the arteries during **systole**.
- **At rest:** 120 mmHg in young age, **in old age** ↑B.P. (may reach 140)
- ↑**Activity:** systolic B.P. ↑.
- **Range:** 100-140 mmHg
- **Systolic Hypertension:** caused by ↑ H.R. & ↑ contractility of the heart.

**Diastolic B.P.**

- **Definition:** The **lowest** pressure recorded in the arteries during **diastole**.

---

<sup>1</sup>Blood flow through partially open blood vessel causing turbulent blood flow and that will produce a sound called **Korotkoff** sound



- **At rest:** 80 mmHg.
- **Range:** 60-90 mmHg
- **↑Activity:** diastolic B.P. ↓.
- **Diastolic Hypertension:** caused by ↑ T.P.R.<sup>1</sup>

**Pulse Pressure**

- **Definition:** Systolic Pressure (S.P.) - Diastolic Pressure (D.P.)
- e.g. 120-80=40mmHg.
- If stroke volume ↑, pulse pressure ↑.
- If the compliance<sup>2</sup> ↑, pulse pressure ↓.
- In some cases the pulse pressure is not felt but the heart is contracting. This indicates bad contractility of the heart.

**Mean B.P.**

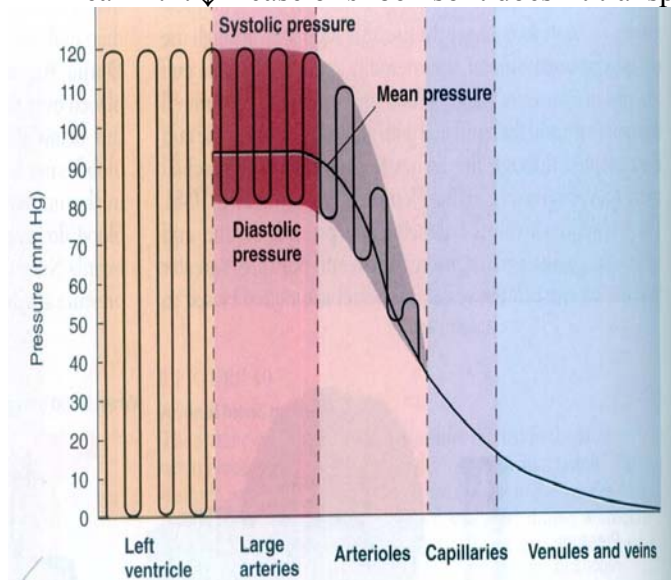
- **Definition:** It is the **average pressure** which **forces the blood to the tissues**. It is **the most important B.P.**

$$\text{Diastolic B.P.} + \frac{\text{Systolic Pressure (S.P.)} - \text{Diastolic Pressure (D.P.)}}{3}$$

$$= \text{Diastolic B.P.} + \frac{\text{Pulse Pressure}}{3} \text{ E.g. } 80 + \frac{40}{3} = 93\text{mmHg}$$

(divided by 3 because diastole is 3 times longer than systole, the number should be nearer to the diastole than the systole).

- **Mean B.P.** is directly **proportional** to **Cardiac Output & Total Peripheral Resistance**
- Mean B.P. ↓ in case of **shock** so it doesn't transport blood to tissues.



<sup>1</sup> To know more about TPR look at Linda Physiology, 3<sup>rd</sup> Ed, P.151

<sup>2</sup> Compliance = distensible = قابل للتوسع

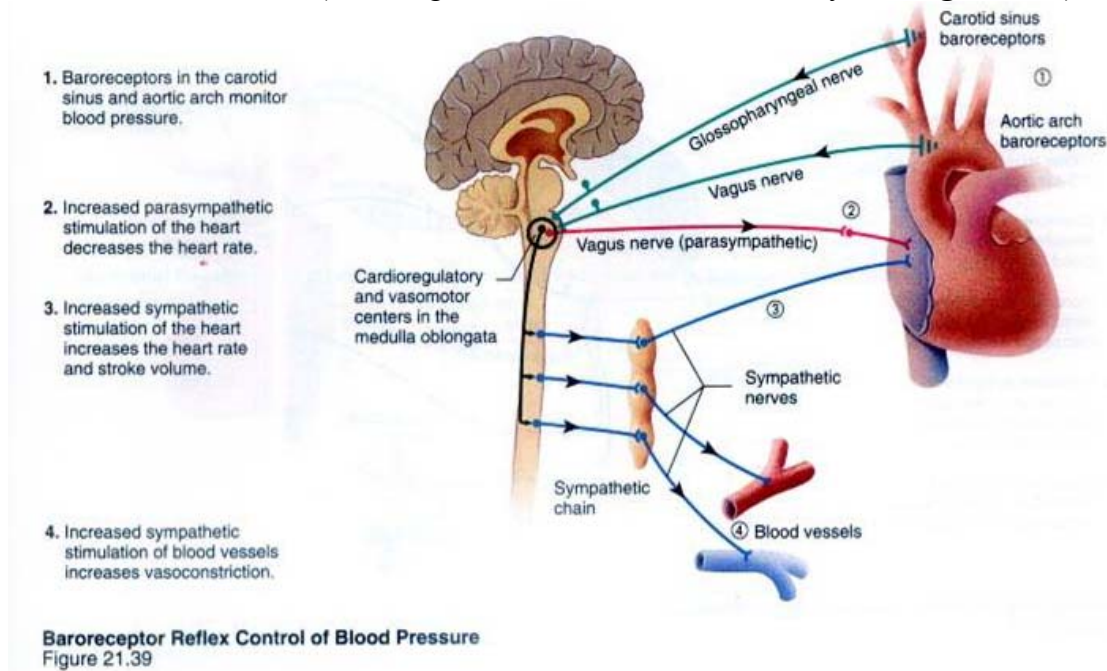
**Vasomotor Center:**

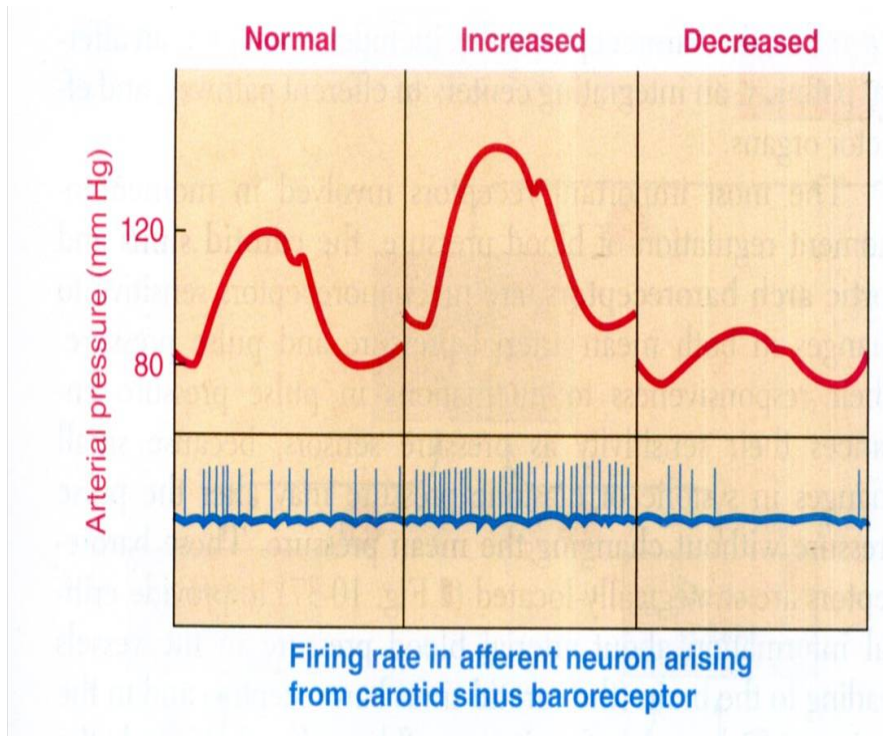
- The center that controls H.R. & circulation.
- It is found in the medulla of the brain.
- It keeps the vasomotor tone (blood vessels are partially contracted) by sending impulses continuously to sympathetic system.
- It does the action of:
  - Excitation of sympathetic → Constriction of blood vessels
  - Inhibition of sympathetic → Dilatation
- Vasomotor center regulates the B.P. in ms (**rapid control**)
- **Cardioregulatory center:**
  - ↑ heart rate → sympathetic
  - ↓ heart rate → parasympathetic

**Rapid B.P. Control Mechanism**

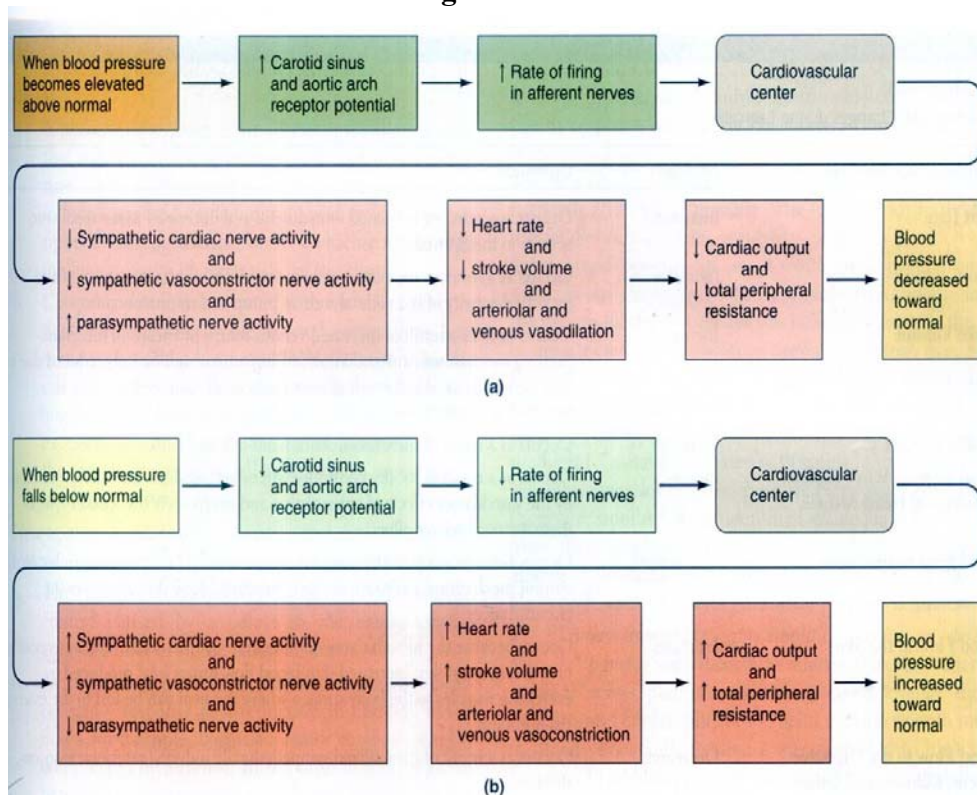
**1. Baroreceptor (pressure receptor):**

- Are **sensitive** to any change in B.P.
- Are mainly found in:
  - **Carotid sinus** (angle of the jaw) where the carotid artery bifurcates. (sends signals to the vasomotor center via the **Glossopharyngeal nerve**).
  - **Aortic arch** (sends signals to the vasomotor center by the **Vagus nerve**).





- When there is  $\uparrow$  in B.P. the baroreceptor will send **signals to vasomotor center** to  $\downarrow$ H.R. and **dilate the blood vessels**.
- **Increased signals** from baroreceptor causes:
  - **Inhibition** of the vasomotor (**sympathetic**)
  - **Stimulation** of the **Vagus** nerve



## 2. Low Pressure Receptor (stretch receptor)

- Found in **atria**, large **veins**, and **pulmonary circulation**.
- Stimulated by  $\uparrow$  **blood volume** causing stretching
- Effects of low pressure receptor stimulation
  1.  $\uparrow$  **afferent** blood flow to the **kidney**  $\rightarrow$   $\uparrow$  GFR<sup>1</sup>  $\rightarrow$   $\uparrow$  urine output.
  2.  $\downarrow$  **secretion** of **ADH** hormone.
  3.  $\downarrow$  **aldosterone** secretion.
  4.  $\uparrow$  atrial natriuretic peptide hormone (**ANP**) secreted by the atria
- **Effects** of low pressure receptor:-
  1.  $\uparrow$  urine output ( $\downarrow$  **water** in blood).
  2.  $\uparrow$  Na<sup>+</sup> excretion by the kidneys ( $\downarrow$  Na<sup>+</sup> in blood).

## 3. CNS effects

Any **stress** condition  $\rightarrow$  stimulation to **sympathetic** system  $\rightarrow$   $\uparrow$  **H.R.**  $\rightarrow$   $\uparrow$  **constriction** of blood vessel  $\rightarrow$   $\uparrow$  **B.P.**

## 4. Chemoreceptor:-

$\downarrow$  B.P. (<80 mm Hg)  $\rightarrow$   $\downarrow$  O<sub>2</sub>  $\rightarrow$  chemoreceptor stimulated  $\rightarrow$   $\uparrow$  B.P.  $\rightarrow$   $\uparrow$  O<sub>2</sub>

- It is **not as sensitive** as the baroreceptor.
- Found in the **same places as the baroreceptor**.
- They are called **carotid & aortic bodies**.

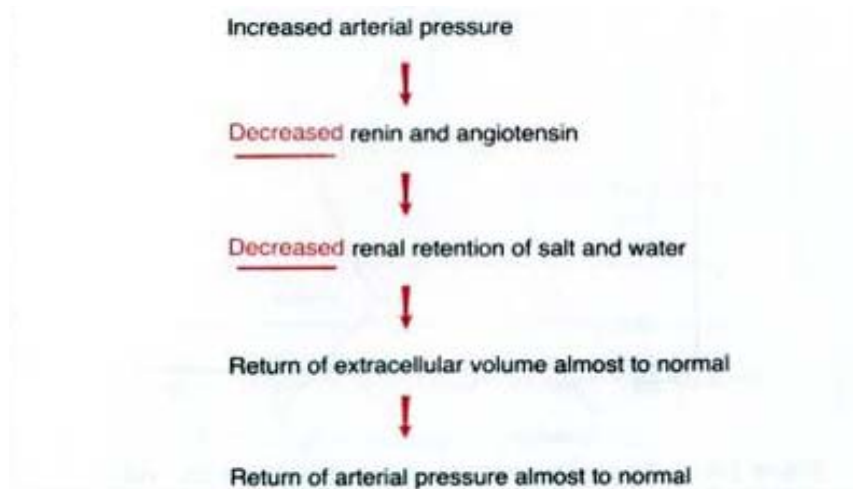
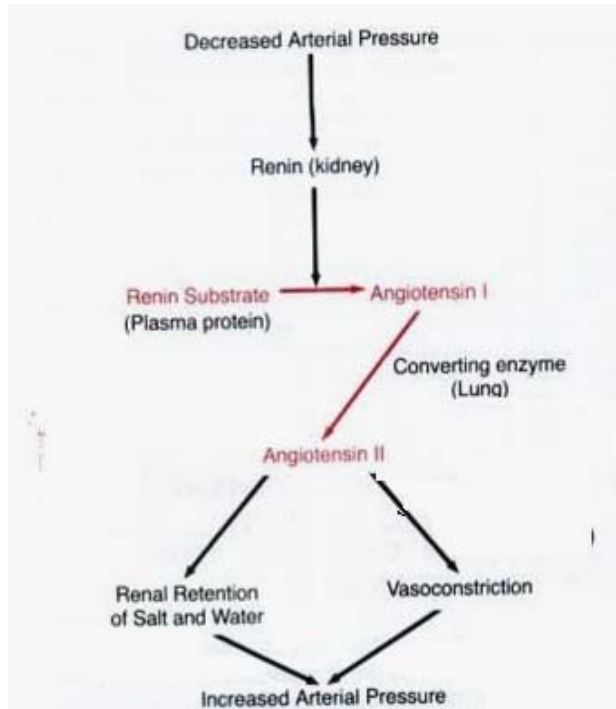
## *Long Term Regulation of B.P.*

It is used in long term and **NOT** rapidly, used when the baroreceptors (short term receptors) get used to the high B.P. so it does not affect the B.P.

## Sequence of events

- In case of Hypotension:
  - Angiotensinogen changes to Angiotensin I by **renin** from kidney.
  - Angiotensin I is changed to Angiotensin II by **converting enzymes** from lungs, which will cause:
    - Vasoconstriction  $\rightarrow$   $\uparrow$  B.P.
    - $\uparrow$  Aldosterone (which will  $\uparrow$  sodium and water reabsorption)  $\rightarrow$   $\uparrow$  B.P.
- In case of hypertension:
  - $\downarrow$  Renin secretion by the kidneys  $\rightarrow$   $\downarrow$  angiotensin II
  - $\rightarrow$   $\downarrow$  aldosterone,  $\downarrow$  ADH secretion,  $\uparrow$  ANP hormone secretion
  - $\rightarrow$  Na<sup>+</sup> excretion with urine (control of  $\uparrow$  B.P. by urine output),
  - $\downarrow$  Blood volume  $\rightarrow$   $\downarrow$  B.P.

<sup>1</sup> GFR stands for Glomerular Filtration Rate. It is the rate of filtration of the kidney.



**High B.P. Complications:**

- Left heart failure → pulmonary edema
- Renal failure
- Stroke in brain (brain bleeding & clotting) → paralysis.
- Impairment of vision.

**MICROCIRCULATION**

- (micro) because it is **not seen by naked eye**, it should be seen by the microscope.
- The **most important** part of the circulation.



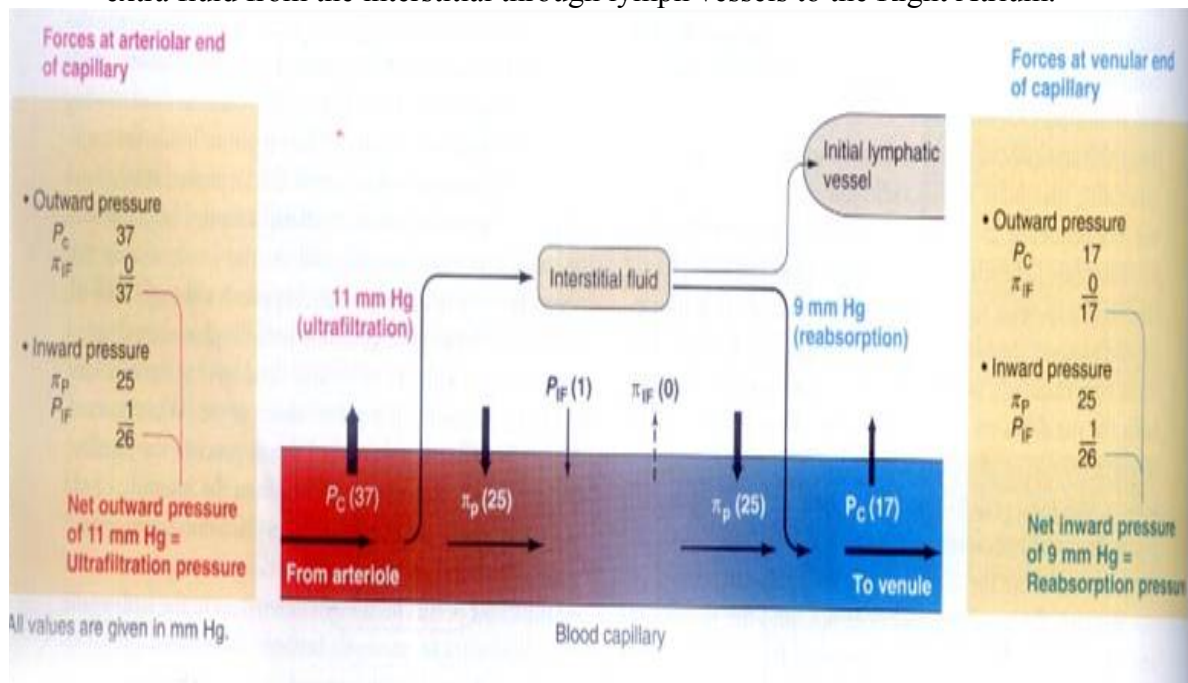
- Starts when the small arterioles enter the tissues and ends when the small venules leaves the tissue.
- Site of exchange of nutrients, electrolytes, and wastes
- Flow is slow to allow exchanging.
- It involves capillaries, arterioles, and venules.
- Very **large surface area** (the largest) in the capillaries, because every cell needs its own exchange of nutrients. (Total surface area of capillaries: 500,000 – 700,000 m<sup>2</sup>).

**Main Forces in Blood for Fluid Filtration&Reabsorption:**

1. **Hydrostatic pressure (filtration pressure):** it makes water leave the capillaries to the interstitial space:
  - When blood enters the arteriole, the B.P. is 37 mmHg (hydrostatic pressure).
  - Filtration will take place in the **arterial** end of the capillaries.
  - Filtration =  $37 - 25 - 1 = 11$  mmHg
2. **Plasma Colloid Osmotic Pressure (oncotic pressure):** it causes suction pressure or reabsorption pressure (includes inhibition of filtration).
  - When blood leaves through venules, the B.P. is 17 (oncotic pressure is **constant** and  $\approx 25$ mmHg).
  - Reabsorption will take place in the venous end of the capillaries.
  - Interstitial pressure is 1 mmHg & this pressure is hydrostatic pressure **back** to the capillaries.

**Role of lymphatics**

Lymphatics have an important role in filtration & reabsorption because it takes the extra fluid from the interstitial through lymph vessels to the Right Atrium.



**Edema**

- Edema is **excessive fluid** that accumulates in the **interstitial space**.
- When there is **high hydrostatic** or **low oncotic pressure**, edema occurs.

**Causes of Edema**

- **Retention** (احتفاظ) of salt & water by the kidneys.
  - **↑ venous pressure** → ↑ hydrostatic pressure → Heart failure:
    - If in R.V. → edema in lower limbs & congestion of the liver.
    - If in L.V. → pulmonary edema.
- And the ↑ in the venous pressure is caused by:
- Block of veins.
  - Standing for a long period because of gravity → ↓venous return → ↑pressure in venules → ↑ filtration → edema in ankle & feet.
  - Paralysis (muscle pump fails → edema).
  - Immobilization (not moving → edema).
- **Dilatation** of the **arterioles** cause:
    - Failure of the sympathetic nervous system → loss of vasomotor tone.
    - Vasodilator drug (e.g. drugs used for ↓B.P.)
  - **↓plasma proteins** → ↓oncotic pressure . It is caused by:
    - Nephrotic Syndrome: Kidney Disease (nephrosis) → protein excreted in urine (normally no protein in urine).
    - Malnutrition: not taking protein. (may be due to not eating proteins or to bad absorption of amino acids in the intestines).
    - Liver disease: Liver is the main organ that synthesizes protein (albumin). Liver disease → ↓plasma proteins → edema.
  - Increase **capillaries' permeability** → loss of proteins from blood to the interstitial space → ↓oncotic pressure → edema. It is caused by:
    - Burn or Injury.
    - Allergic reaction → ↑ histamine → dilatation of capillaries → loss of protein → ↓oncotic pressure → edema.
  - **Lymphatic obstruction** → fluid accumulates in interstitial space → edema. It may be caused by:
    - Flaviasis due to nematode (a type of worms that blocks lymphatics) → elephantiasis.





## SHOCK

(This part is NOT included in the 2<sup>nd</sup> physiology quiz on Sat.19/12/1428H)

- It is **sudden drop in B.P. & C.O.** leading to **decreased tissue perfusion** (tissue blood flow).
- Shocked person may be conscious ,unconscious, semiconscious or in coma (غيبوبة).

### Symptoms & signs;-

- Very **rapid thready** (ضعيف وسريع) **pulse**.
- ↑ H.R., ↓ pulse, ↓ contractility.
- **Skin is cold** (due to vasoconstriction on the skin), and **wet** (vasodilatation of blood vessels to sweat gland), and undergoes **cyanosis**<sup>1</sup>.
- ↓ **B.P.**
- ↓ **C.O.**
- ↑ **Respiratory rate**,
- **Oliguria**<sup>2</sup>
- **Thirst**.

### Causes of shock;

- **Hypovolumic shock** → ↓ C.O., ↓ B.P. It may be caused by:
  - **Hemorrhage** نزيف (**loss of whole blood**). This is the most common cause.
  - Vomiting, diarrhea, sweat (**loss of plasma**).
- **Cardiogenic shock** (e.g. myocardial infarction).
- **Vasogenic shock**: ↑ widespread (منتشر) **vasodilatation** of blood vessels because:
  - **Septic shock** (**bacterial infection**).
  - **Anaphylactic shock**: secretion of **histamine** due to **allergic** reaction.
- **Neurogenic shock**:
  - Due to **failure of sympathetic nervous system** → **loss of vasomotor tone** → **vasodilatation** → ↓ B.P & ↓ C.O.

### Stages of shock:

- Non-progressive (**compensated**) stage.
- **Progressive shock**.
- **Irreversible shock**.

<sup>1</sup> Blueness of the skin, and its cause is excessive amounts of deoxygenated hemoglobin in the skin blood vessels

<sup>2</sup> Diminished urine output below the level of intake of water and solutes.

**Compensatory mechanism in shock<sup>1</sup>:**

a. (The below three mechanisms happen in the first 30 seconds)

**1. Baroreceptor:**

- baroreceptor stimulated → ↑ sympathetic stimulation →:
  - → ↑ vasoconstriction of the arterioles → ↑ TPR → B.P.
  - → venoconstriction → ↑ venous return.
- ↑ C.O.

**2. Central N.S**

Ischemic response. If there are ↓O<sub>2</sub> & ↓blood flow → another sympathetic stimulation. (Last trial depending on sympathetic).

**3. Reverse stress relaxation of the circulation system**

Blood vessel will contract to contain the small amount of the blood to maintain B.P.

**b. Hormonal Mechanism (time of occurrence: 10 min – 1 hour)**

1. ↑ **renin** secretion by the kidney (its action is explained above).
2. ↑ **ADH** secretion by posterior pituitary gland. (retention of water & salt → ↑ blood volume → ↑ B.P.

**c. Factors ↑ blood volume (time of occurrence: 1 hour – 48 hours)**

1. ↑ **absorption of water** from the G.I.T.
2. ↑ **reabsorption** of fluid from the interstitial space to the capillaries.
3. ↑ **water intake**, ↑ salt desire (شهوة).

If there is ↓ of mean B.P. to 70 mmHg the **circulation of the heart & brain will not be affected** because they have minimum sympathetic effect and also excellent autoregulation.

**Progressive shock**

The situation gets much worse تدهور, and from its effects:

1. **Cardiac depression** because of ↓O<sub>2</sub>.
2. **Vasogenic failure** (loss of vasomotor tone).
3. ↑ **intravascular coagulation** (small blood vessels will be blocked because of clots)
4. ↑ **capillary permeability** because of secretion of histamine → vasodilator → ↑ interstitial fluid → may cause edema → ↓ Blood Volume.
5. **Organs Failure:** Liver, Kidney.

**Introduction to treatment,,**

- If there is **loss of plasma fluid** → **transfusing plasma expander.**
- If there is **loss of whole blood** → **whole blood transfusion.**

<sup>1</sup> If there is 10% loss of blood there is no effect on the body because it will compensate. If there is 35-45% loss of blood the C.O. & B.P. are zero (irreversible shock)