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# ARTERIOLES

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L31



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لجنة الطب والجراحة

## Continuation of the previous lecture

### Arterial pulse

The blood forced in the aorta during systole sets a pressure wave which travels down the arteries expanding the arterial wall and felt as a pulse.

- The velocity of the pressure is about 7 meters / sec.
- The rate of transmission of the pulse wave depends on the elasticity of the wall of the arteries.
- In atherosclerosis the arteries become rigid and the rate of transmission becomes faster than the normal elastic arteries.

Site: The arterial pulse can be felt in superficial arteries as radial, ulnar & femoral. The carotid pulse is most important and should be searched whenever cardiac arrest is suspected.

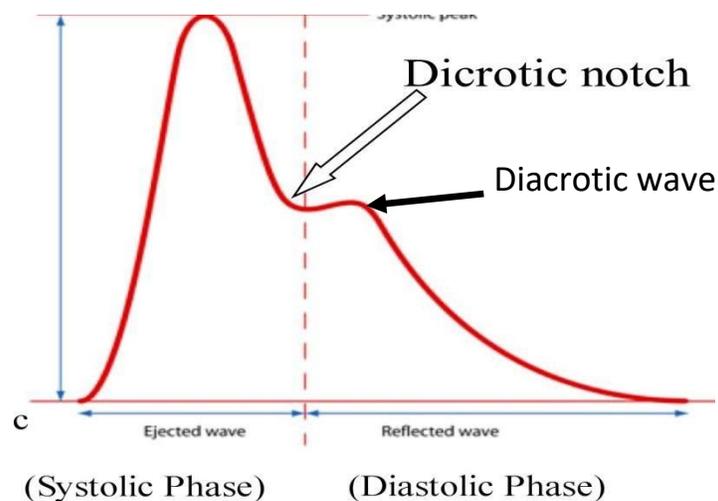
Physiological importance of arterial pulse: It is important for:

- a. Determining the heart rate.
- b. Rhythm (regular or not ) **(is the time between beats is rhythm or not)**
- c. Force of blood ejection: as when the stroke volume is increased the pulse becomes full e.g. in muscular exercise, when it is decreased as in hemorrhage the pulse becomes weak.
- d. To detect the elasticity of the artery as normally the wall of the artery is not felt because it is soft. In atherosclerosis this elasticity is lost and the artery is felt as a cord like.

Arterial pulse wave (viiip) مهمه جداااا

- The arterial pulse wave is formed of an ascending limb or (anacrotic limb) and a descending limb (Catacrotic limb).

- The ascending limb is caused by ejection of blood from the left ventricle to the aorta and the pressure in the aorta rises to its systolic level.
- While the descending limb is caused by gradual decline of pressure to the diastolic level.
- The Catacrotic limb contain the dicrotic notch or incisura
- Dicrotic notch or incisura: it is caused by sudden closure of the aortic valve in the beginning of left ventricle relaxation which causes back flow of the blood and reduction of pressure.
- Dicrotic wave: it follows the incisura and is produced by elastic recoil of the aortic wall over the moving blood.



At the rising limb → isovolumetric contraction

At the top of the slope → max ejection phase

The dropping limb of the systolic phase → reduced ejection

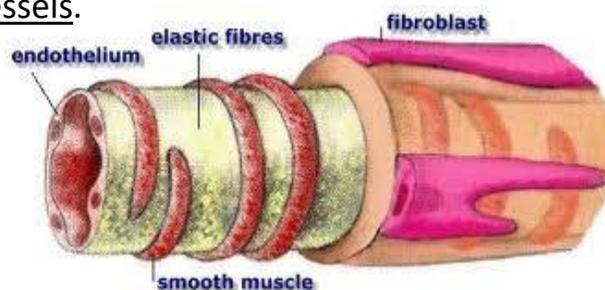
Note: To measure the elasticity of the artery first we must know it's location then we press and roll if we felt the wall of the artery that's indicates a atherosclerosis

## Arterioles

This sheet Contains:

- ❖ Arterioles histology
  - Unitary smooth muscles
  - Multiunit smooth muscles
- ❖ Function of arterioles
  - Control peripheral resistance
  - Redistribution of blood flow
- ❖ Regulation of the diameter of arterioles
  - Central regulation
    - Neural
    - Hormonal
  - Local regulation
    - Local oxygen tension
    - Metabolites
    - Intrinsic myogenic response
    - Role of epithelium

The wall of the arterioles contains *a little amount of elastic fibers* and *an increased amount of smooth muscle fibers*. So, they are called resistance vessels.



### Histology

	UNITARY SMOOTH MUSCLES (INNER CIRUCLAR)	MULTIUNIT SMOOTH MUSCLES (OUTER LONGTUIDENAL)
<b>SITE</b>	<ol style="list-style-type: none"> <li>1. Pre-capillary sphincters</li> <li>2. The inner sheath of vascular smooth muscles in arterioles</li> </ol>	They predominate in the outer sheath of smooth muscles in arterioles, veins and arteriovenous shunts
<b>CONTRACTION ACTIVITY</b>	Spontaneous myogenic activity that is initiated in pacemaker areas	It does not contract spontaneously
<b>REGULATION</b>	They are regulated by <b>local mechanisms</b> and they have a few or no direct nerve supply They regulate local blood flow	<ol style="list-style-type: none"> <li>1. They are regulated by <b>central mechanisms</b></li> <li>2. It does not react to stretch</li> </ol>

## Functions of arterioles

### 1. Control peripheral resistance

- As  $A.B.P = C.O.P \times P.R$ . So, arteriolar V.C  $\rightarrow \uparrow P.R$  and accordingly increased ABP.

### 2. Redistribution of blood flow

- The arterioles play an important role in distribution of blood flow to various organs of the body. The blood flow to a given organ is changed by variation in the diameter of its arterioles i.e. arteriolar V.D in a given organ leads to increased flow, and arteriolar V.C leads to decrease its blood flow.

- The blood flow to a given organ is not determined by the size of the organ but by its role in homeostasis and by its metabolic activity.

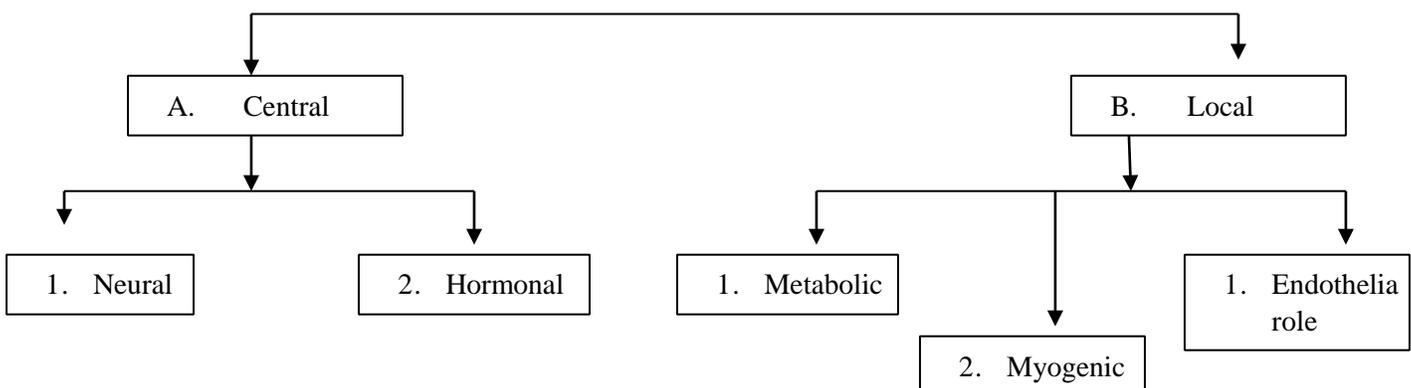
- Moving along the vasculature away from the heart, the largest pressure drop in the circulatory system occurs along the **arterioles**

- During muscular exercise the C.O.P. is increased and redistribution of the blood flow to various organs occurs as flow:

- The blood flow to the skeletal muscles increases 10 times.
- The blood flow to the heart increases 4 times.
- The blood flow to the skin increases 4 times to help heat loss from the body.
- The blood flow to the brain is not changed. (Constant all the time)
- The blood flow to the kidney and abdominal viscera is decreased.

## REGULATION OF THE DIAMETER OF ARTERIOLES

The diameter of arterioles is regulated by 2 mechanisms:



## A. Central regulation

(Regulates multiunit smooth muscles)

### 1. Neural mechanism

- ❖ There is a balance between both sympathetic and parasympathetic innervations
- ❖ Innervations of arterioles:

#### a. Sympathetic Innervations.

##### i. Sympathetic vasoconstrictor fibers.

- These fibers are adrenergic fibers.
- They discharge continuously to the arterioles forming the basal V.C tone.
- When **the rate of discharge increases** above the basal level it leads to V.C of arterioles &  $\uparrow$  ABP.
- When **the rate of discharge is decreased** below the basal level the tone is decreased and V.D occurs leading to decreased P.R and  $\downarrow$  ABP
- The most important function of sympathetic adrenergic fibers is to maintain the normal arterial blood pressure to ensure adequate blood flow to the organs all the time especially the heart and the brain.

- their tract is: Hypothalamus  $\rightarrow$  Lateral horn cells  $\rightarrow$  Ganglia  $\rightarrow$  Effectors

##### ii. Sympathetic Vasodilator fibers: These fibers supply.

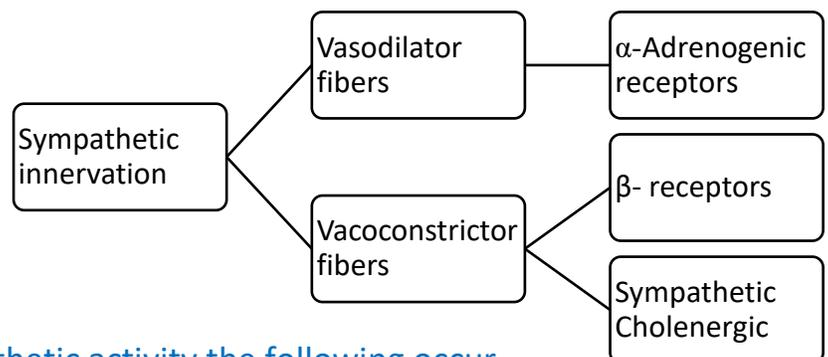
#### a) **Coronary blood vessels:** V.D occurs by two mechanisms.

- **Indirect mechanism** by the effect of metabolites.
- **Direct mechanism** by adrenaline which act on  $\beta$ - receptors which are more frequent than  $\alpha$ - receptors.

#### b) **Arterioles of skeletal muscles.**

- Sympathetic Cholinergic V.D fibers which produce V.D of skeletal muscle blood vessels before the beginning of exercise and before the accumulation of metabolites that occur during the exercise and producing V.D .These fibers does not play an important role in regulation of arterial blood pressure .

N.B: Also, skeletal muscles receive sympathetic adrenergic fibers which produce V.C. **in low blood volume like hypovolemic shock**



**Note:** During fight & flight sympathetic activity the following occur

1. V.C of splanchnic blood vessels ( $\alpha$ -receptors)
2. V.D of coronary blood vessels ( $\beta$ -receptors)
3. V.D of skeletal muscles blood vessels (cholinergic muscarinic receptors)

## b. Parasympathetic innervations

Usually act indirect by reducing V.C tone

but sometimes act directly:

Parasympathetic V.D fibers to:

i. The parasympathetic fibers to supply the genital tract

- these are the pure V.D fibers

ii. Parasympathetic fibers to G.I.T:

- The vasodilator action is mainly due to metabolites.

## 2. Hormonal mechanisms

### V.C Substances

Adrenaline  
Noradrenaline  
Angiotensin  
A.D.H

### V.D Substances

Histamine  
ANP

#### ❖ Adrenaline

The effect of Adrenaline depends on its concentration

- Low concentration: produce V.D
  - because it acts on  $\beta$ -receptors
- High concentration: produce V.C
  - because it acts on  $\alpha$ -receptors

#### ❖ Noradrenaline

The effect of Noradrenaline doesn't depend on its concentration

- In low and high concentration: produce V.C
  - because it acts on  $\alpha$ -receptors

## B. Local mechanisms.

(Regulates unitary smooth muscles)

✚ It is an auto regulatory mechanism which include.

## ✚ Auto regulation

It is the ability of the tissue to regulate its own blood flow according to its metabolic activity i.e. when the activity is increased the tissue produce local V.D in its arterioles to increase the blood flow **to meet it's need**, and when the activity is decreased arteriolar V.C occurs to decrease the tissue blood flow.

✚ This autoregulatory mechanism occurs by:

### 1. Local oxygen tension.

This mechanism regulates the blood flow according to the oxygen needs of the tissue and it is explained as follow:

- ❖ Decreased tissue activity leads to decreased metabolites and increased oxygen tension, which leads to local V.C.
- ❖ Increased tissue activity leads to increased oxygen utilization and decreased oxygen tension which leads to local V.D to increase the blood flow that carry more oxygen to the tissue

### 2. Metabolites (metabolic theory of Autoregulation)

- ❖ When the blood flows to the tissue decrease, metabolites accumulate and the blood vessels dilate,
- ❖ When the metabolites are washed out the vessels constrict to decrease the flow.

N.B: Vasodilator metabolites as:

- 1) ++ H<sup>+</sup> ion concentration (Acidosis).
- 2) Increased Co<sub>2</sub> tension. (Hypercapnia)
- 3) ++ Lactic acid
- 4) Histamine.

### 3. Intrinsic myogenic response (Bayliss response) – sudden stretch → V.C.

Increased pressure leads to stretch of the blood vessel wall leading to stretch of the vascular smooth muscles, leading to its contraction

### 4. Role of endothelium.

The endothelium plays a major role for regulation of local blood flow to the tissues through the following substances.

- **V.C.s:** thromboxane A<sub>2</sub>, Endothelins.
- **V.D.s:** Prostacyclin, Endothelium derived relaxing factor (NO).

#### a. Prostacyclin

- Prostacycline is produced by endothelial cells
- Inhibit platelet aggregation and cause vasodilatation.

**b. thromboxane A<sub>2</sub>:**

- It is formed by the platelets to promote platelet aggregation - Also, it causes vasoconstriction.

**c. Endothelium derived relaxing factor (NO).**

- It plays a key role in V.D
- N.O is synthesized from arginine by the enzyme nitric oxide synthase.
- N.O produces cyclic G.M.P which in turn mediates relaxation of the vascular smooth muscles.

**d. Endothelins family**

## Actions of Endothelin

- It is the most potent V.C agent yet isolated. (The most powerful)
- It has positive inotropic and chronotropic effects on the myocardium.
- It stimulates intense V.C of the coronary arteries.
- Decreased glomerular filtration rate and renal blood flow.
- Increased Na ion reabsorption through Hemodynamic actions.

## Note:

- + Adrenaline, Noradrenaline, Angiotensin, A.D.H, Histamine, ANP → Hormones which act on central regulation
- + Prostacycline, thromboxan A<sub>2</sub>, Endothelium derived relaxing factor (NO), Endothelins → Are endothelium secreted substances which act on local regulation