VASCULAR PHYSIOLOGY

REGULATION OF DIAMETER OF ARTERIOLS

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Regulation of diameter of arterioles

Arterioles are the terminal branches of the arterial system

Characters of arterioles:

1)Loss of elastic elements and increase smooth ms. layer.

2)Have great resistance to blood flow (so the blood pressure drops from 80 to 30 mmHg) and act as sphincter between arterial system and venous system.

3)Have sympathetic VC fibres and some parasympathetic VD fibres.

4)They are sensitive to chemicals of blood as the blood gases, hormones, metabolites & pH.

5)The endothelium in their walls can synthesize chemical mediators → constriction or dilatation of their walls.

The only site at which arterioles can be seen is the retina.

Functions of arterioles:

1. ***Determination of the peripheral resistance*** : They are called the resistance vessels because they control the peripheral resistance and the arterial blood pressure.
2. ***They control the blood flow to the tissues*** : by changing their diameter through producing V.D. or V.C.

Factors regulate arteriolar diameter:

I. Local regulatory mechanisms

1) O_2 Tension :

The normal (PO_2) produces **partial vasoconstriction** and this is maintained by VC tone.

Decreased O_2 tension leads to **direct vasodilator** effect on the arteriolar smooth muscles **except** for the pulmonary vessels which constrict due to O_2 lack.

-When the metabolic activity of a tissue decreases the PO_2 is increased leading to vasoconstriction.

If the metabolic activity of a tissue is increased the PO_2 is decreased leading to vasodilatation.

2) Metabolites :

When the tissue becomes hyperactive, some metabolic changes occur as increase CO_2 tension ,acidosis, osmolality, K^+, and adenosine.

Active hyperemia:

The increase in blood flow at the **active tissues** by vasodilation produced by accumulation of metabolites.

Reactive hyperemia:

The increase in blood flow of a tissue produced by accumulation of metabolites due to **temporary occlusion** of its blood supply.

3) Intrinsic Mechanism (Autoregulation):

Definition :

-It is the ability of a tissue to regulate its blood flow **according to its need.**

Mechanism:

I-Myogenic theory:

When the blood flow increases to a tissue .→ stretching the vascular smooth muscles → their depolarization → vascular smooth muscle contraction → decreased the blood flow to normal.

II-Metabolic theory:

If the blood flow to an organ is decreased, vasodilator metabolites accumulate producing vasodilatation which will increase the blood flow to wash the metabolites and remove their effect.

4**) Local vasoconstrictor substances**:

Injured arteries and arterials constrict powerfully due to **serotonin** release from blood platelets

5) Local temperature:

- Drop in the tissue temperature has a direct V.C. effect.

- Increase in the tissue temperature causes V.D.

6) Substances released by the endothelium :

A-Thromboxane A2 and Prostacyclin:

Both are formed from **archadionic acid** by cyclooxygenase enzyme.

-**Prostacyclin:** released from endothelium, causes VD and inhibition of platelets aggregation.

-**Thromboxane A2:** released from platelets and cause VC and increase platelet aggregation.

-The two substances are in balance with each other in control of vascular diameter and platelet plug formation.

B- Endothelins

-Three types are now known, each is a polypeptide (21 amino acid) formed in the endothelial cells.

-Released by stretching the vessels.

-It is a potent V.C.

Actions:

•Positive inotropic effect on cardiac muscle and Vasoconstriction of the coronaries.

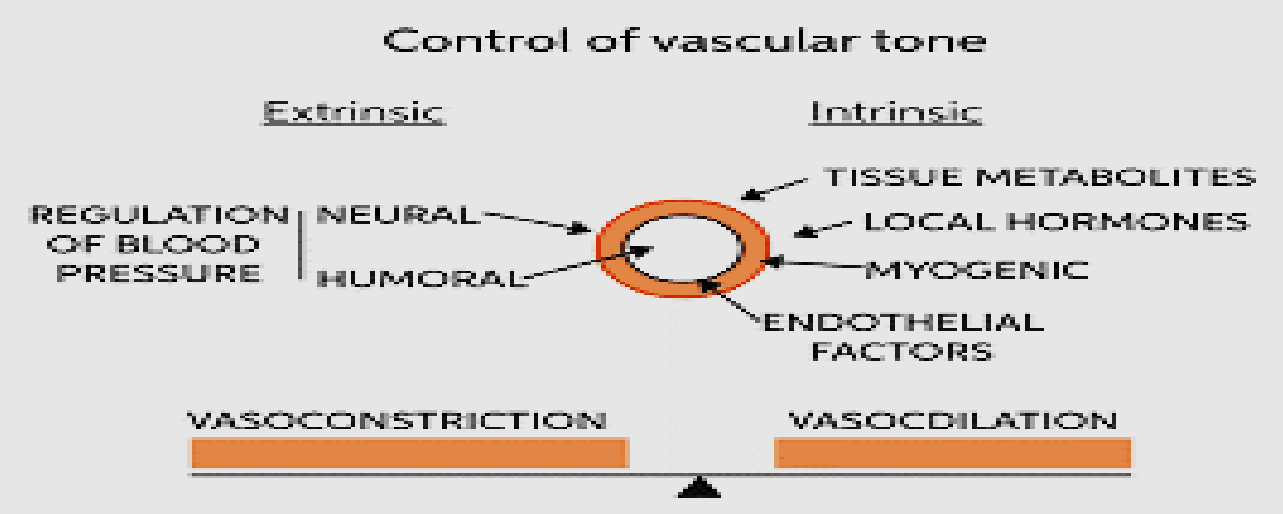
C- Endothelium-derived-relaxing factor (EDRF):

• It is identified as nitric oxide (NO) and synthesized from arginine amino acid.

• It increases cGMP → vasodilation.

• It is released by endothelium under the effect of bradykinin, substance P, vasoactive intestinal peptide (VIP) as a mediator for their vasodilator effects.

• Its deficiency as in cases of endothelium injury → loss of its vasodilator effect and the vasoconstrictors as thromboxane A2 becomes predominant causing vasoconstriction, atherosclerosis, hypertension and impotence (failure of erection by VD in male genital organs).



II. Central (or systemic) regulation

A. Neuronal Regulatory Mechanisms

(1) Vasoconstrictor fibers (V.C.):

a. Sympathetic V.C.:

-The sympathetic noradrenergic fibers arise from the lateral horn cells; of the thoraco-lumbar segments of the spinal cord .

-They discharge continuously, leading to *generalized partial vasoconstriction* which is called sympathetic *vasoconstrictor tone*.

b. Parasympathetic vasoconstrictor fibers:

-No vasoconstrictor parasympathetic fibers are known

-Parasympathetic stimulation to the heart causes **coronary vasoconstriction** by decreasing the metabolic activity and elevation of O_2 tension, together with decreasing metabolites which lead to coronary vasoconstriction.

(2) Vasodilator fibers (V.D.):

(a) Sympathetic vasodilator fibers:

-All the sympathetic innervations to blood vessels cause V.C. **except** in:

***(i) Coronary vessels*:**

This occurs indirectly by increasing heart rate and the metabolic activity of the heart $\rightarrow\downarrow O_2$, tension and accumulation of metabolites $\rightarrow$ VD (metabolic theory of autoregulation).

(ii) Skeletal muscles:

-The sympathetic (V.D.) fibers are cholinergic (secrete **acetylcholine**).

-They start to operate and dilate the skeletal muscle blood vessels even before the start of the exercise and so help to increase the skeletal muscle blood flow during exercise.

-This system can be activated by sudden strong emotions $\rightarrow$ widespread vasodilatation $\rightarrow$ severe hypotension$\rightarrow$ brain ischemia $\rightarrow$ syncope.

(iii) The splanchnic areas:

-The blood vessels of these areas are richer in (β) adrenergic receptors than (α) receptors, and (β) receptors in turn produce **vasodilatation.**

(iv) Sweat glands:

-Sympathetic cholinergic VD fibers supply blood vessels of the sweat glands.

(b) Parasympathetic vasodilator fibers:

-The only parasympathetic fibers which are definitely vasodilator are those which supply the **genital organs** (sacral out-flow).

(c) Antidromic vasodilator impulses:

-When pain receptors are stimulated by pain stimuli, (e.g. inflammation or scratching the skin with a pin) produces **dilatation of the adjacent blood vessels**.

- **Mechanism: local axon reflex:** Stimulation of pain receptors initiates impulses travel along sensory nerve fibers toward CNS until they reach a branch, they travel a long it (anti-dromically). Local axon reflex doesn't involve the CNS.

- When they reach the arterioles, cause releasing of Substance P which has a vasodilator effect on the arterioles thus the area of inflammation become red (flare).

B- Hormonal Regulation

1. Circulating **Vasoconstrictor** Substances:

a. Catecholamines:

- The sympathetic stimulation causing release of **adrenaline and nor-adrenaline** which circulate in the blood and cause the same effects of the sympathetic nervous system on the arterioles.

b-Renin- angiotensin-system:

Decreased blood pressure (dehydration, salt restriction, haemorrhage) →↓renal blood flow→ renal ischemia → juxtaglomerular apparatus secret **renin** which act on **angiotensinogen** producing **angiotensin -I** which is converted to **angiotesin -II** by the angiotensin convertase enzyme in the lung.

Angiotesin II has the following effects:

1-Strong arteriolar **VC** (50 times as noradrenaline) leading to increased peripheral resistance and blood pressure.

2-Stimulate NA release from postganglionic sympathetic fibres.

c. Vasopressin (Antidiuretic Hormone) (ADH):

- This is a potent V.C. hormone secreted by the posterior pituitary gland.

2-Circulating Vasodilator Substances:

a- Kinins:

-They are plasma kinin (bradykinin) and tissue kinin (kalidin)

Actions of kinins:

1)VD by release of EDRF→ marked decrease in blood pressure.

2)Contraction of smooth muscles as in respiratory system.

3)Stimulation of pain receptors. And Increase capillary permeability.

4)Act as mediator for VD in active salivary glands.

b. Atrial Natriuretic Peptide:

- ANP is secreted from the atria; (others are secreted from the brain and heart ventricles)

- They are secreted when: NaCl intake is increased and The blood volume is increased

- Immersion in water up to the neck with increase VR. And Increased CVP. And Increased intraventricular pressure.

Actions of ANP:

1)Natriuresis: (loss of Na+ in urine)

2)Vasodilators of blood vessels also ↓ response of blood vessels to vasoconstrictors → decrease ABP.

V.C	V.D
Decrease CO_2 tension ,acidosis, osmolality, K^+, and adenosine.	Increase CO_2 tension ,acidosis, osmolality, K^+, and adenosine.
High O2 Except Lung	O_2 lack Except Lung
stretching the vascular smooth muscles	
Drop in the tissue temperature	Increase in the tissue temperature
Thromboxane- A2, serotonin	**Prostacyclin, EDRF**
Sympathetic V.C Vasomotor tone	sympathetic innervations to bl. Vessels of Coronary vessels, Skeletal muscles, The splanchnic areas, Sweat glands
Parasympathetic **coronary vasoconstriction** **indirect effect**	Parasympathetic to **genital organs** (sacral out-flow).
adrenaline and nor-adrenaline, Angiotensin-II , ADH	**Antidromic local axon reflex**
	Kinins, ANP

Thank You