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1. **Discuss the long term regulation of mean arterial blood pressure.**

Kidneys play an important role in the long term regulation of arterial blood pressure. When blood pressure alters slowly in several days/months/years, the nervous mechanism adapts to the altered pressure and loses the sensitivity for the changes. It cannot regulate the pressure any more. In such conditions, the renal mechanism operates efficiently to regulate the blood pressure. Therefore, it is called long term regulation. Kidneys regulate arterial blood pressure by two ways:

1. By regulation of ECF volume

2. Through renin angiotensin mechanism.

BY REGULATION OF EXTRACELLULAR FLUID VOLUME

When the blood pressure increases, kidneys excrete large amounts of water and salt, particularly sodium, by means of pressure diuresis and pressure natriuresis. Pressure diuresis is the excretion of large quantity of water in urine because of increased blood pressure. Even a slight increase in blood pressure doubles the water excretion. Pressure natriuresis is the excretion of large quantity of sodium in urine. Because of diuresis and natriuresis, there is a decrease in ECF volume and blood volume, which in turn brings the arterial blood pressure back to normal level. When blood pressure decreases, the reabsorption of water from renal tubules is increased. This in turn, increases ECF volume, blood volume and cardiac output, resulting in restoration of blood pressure.

THROUGH RENIN-ANGIOTENSIN MECHANISM

Actions of Angiotensin II When blood pressure and ECF volume decrease, renin secretion from kidneys is increased. It converts angiotensinogen into angiotensin I. This is converted into angiotensin II by ACE (angiotensin converting enzyme). Angiotensin II acts in two ways to restore the blood pressure:

i. It causes constriction of arterioles in the body so that the peripheral resistance is increased and blood pressure rises. In addition, angiotensin II causes constriction of afferent arterioles in kidneys, so that glomerular filtration reduces. This results in retention of water and salts, increases ECF volume to normal level. This in turn increases the blood pressure to normal level.

ii. Simultaneously, angiotensin II stimulates the adrenal cortex to secrete aldosterone. This hormone increases reabsorption of sodium from renal tubules.

Sodium reabsorption is followed by water reabsorption, resulting in increased ECF volume and blood volume. It increases the blood pressure to normal level.

Actions of Angiotensin III and Angiotensin IV Like angiotensin II; the angiotensins III and IV also increase the blood pressure and stimulate adrenal cortex to secrete aldosterone.

HORMONAL MECHANISM FOR REGULATION OF BLOOD PRESSURE

Hormones which increase arterial blood pressure	**Hormones which decrease arterial blood pressure**
1. Adrenaline	1. Vasoactive intestinal polypeptide (VIP)
2. Noradrenaline	2. Bradykinin
3. Thyroxine	3. Prostaglandin
4. Aldosterone	4. Histamine
5. Vasopressin	5. Acetylcholine
6. Angiotensin	6. Atrial natriuretic peptide
7. Serotonin	7. Brain natriuretic peptide

2. Write short notes on the following;

A. PULMONARY CIRCULATION- The pulmonary circulation is the portion of the circulatory system which carries deoxygenated blood away from the right ventricle, to the lungs, and returns oxygenated blood to the left atrium and ventricle of the heart. Pulmonary blood vessels include pulmonary artery, which carries deoxygenated blood to alveoli of lungs and bronchial artery, which supply oxygenated blood to other structures of lungs.

Following are the characteristic features of pulmonary blood vessels:

1. Pulmonary artery has a thin wall. Its thickness is only about one third of thickness of the systemic aortic wall. Wall of other pulmonary blood vessels is also thin.

2. Pulmonary blood vessels are highly elastic and more distensible

3. Smooth muscle coat is not well developed in the pulmonary blood vessels

4. True arterioles have less smooth muscle fibers

5. Pulmonary capillaries are larger than systemic capillaries. Pulmonary capillaries are also dense and have multiple anastomosis, so, each alveolus occupies a capillary basket.

6. Vascular resistance in pulmonary circulation is very less; it is only one tenth of systemic circulation

7. Pulmonary vascular system is a low-pressure system.

B. CIRCLE OF WILLIS-The circle of Willis is a junction of several important arteries at the bottom part of the brain. It helps blood flow from both the front and back sections of the brain. The circle of Willis gets its name from the physician Thomas Willis, who described this part of the anatomy in 1664. The circle of Willis, or the circulus arteriosus, is formed by the anastomosis of the two internal carotid arteries with the two vertebral arteries. At the Circle of Willis, the internal carotid arteries branch into smaller arteries that supply oxygenated blood to over 80% of the cerebrum, the circle of Willis allows blood to flow across the midline of the brain if an artery on one side is occluded. The circle of Willis thereby serves a safety valve function for the brain, allowing collateral circulation (or flow of blood through an alternate route) to take place if the flow is reduced to one area. The most common anomaly of the circle of Willis in normal brains was hypoplasia of one or other components of the circle. Arteries of less than 1 mm in external diameter were considered hypoplastic, except for the communicating arteries, where less than 0.5 mm was considered hypoplastic.

C. SPLANCHNIC CIRCULATION- The splanchnic circulation is composed of gastric, small intestinal, colonic, pancreatic, hepatic, and splenic circulations, arranged in parallel with one another. The three major arteries that supply the splanchnic organs, cellac and superior and inferior mesenteric, give rise to smaller arteries that anastomose extensively. The circulation of some splanchnic organs is complicated by the existence of an intramural circulation. Redistribution of total blood flow between intramural vascular circuits may be as important as total blood flow. Numerous extrinsic and intrinsic factors influence the splanchnic circulation. Extrinsic factors include general hemodynamic conditions of the cardiovascular system, autonomic nervous system, and circulating neurohumoral agents. Intrinsic mechanisms include special properties of the vasculature, local metabolites,

intrinsic nerves, paracrine substances, and local hormones. The existence of a multiplicity of regulatory mechanisms provides overlapping controls and restricts radical changes in tissue perfusion. The splanchnic bed forms an important circulatory reservoir, which can be mobilized during periods of physiological stress. Disorders of the splanchnic circulation may contribute to the multi-organ dysfunction syndrome and vice versa. A number of techniques used in anesthesia and critical care influence the distribution of blood flow in the splanchnic circulation.

D. CORONARY CIRCULATION- the coronary circulation can be divided into two compartments, the large epicardial conduit vessels and the resistance vessels, typically less than 300 μm in diameter. Whereas the conduit vessels exert little if any resistance to flow, resistance to flow progressively rises as the vessel diameter of the resistance vessels declines from about 300 μm in the small arteries to less than 100 μm in the arteriolar vessels. Exchange of substrates between blood and tissue occurs at the level of the capillaries. Flow across the myocardium largely depends on the pressure gradient between the aortic root (the "coronary driving pressure") and the right atrium. Under normal conditions, the driving pressure is fully maintained along the epicardial conduit vessels with little if any pressure loss in the distal epicardial arteries. However, intra-coronary pressures decline along the microvasculature (with most of the pressure dissipating in the 300-100 μm diameter vessels) until reaching a pressure of 20-30 mmHg, still adequate to ascertain a gradient across the capillaries. Additional determinants of the resistance to flow include extravascular resistive forces that are directly related to the left ventricular systolic pressure, the contractile state of the myocardium and the heart rate.

Auto-regulatory mechanisms coordinate the interaction between intra-coronary driving pressure and microvascular resistance in order to maintain adequate flow across the capillaries for substrate delivery and removal. Through this mechanism (also defined as "coronary autoregulation"), decreases in driving pressure are compensated for by decreases in resistance and conversely, increases in driving pressure by increases in resistance so that flow remains constant for a given cardiac workload. This regulatory mechanism operates within the range of physiologic arterial pressures but fails during hypotension when flows become strongly dependent on the driving pressure.

Changes in myocardial work and, thus, in energy demand, are accompanied by proportionate changes in coronary and, thus, myocardial blood flow. For example, as shown with noninvasive PET measurements, a 2.8-fold increase in cardiac work

(estimated from the rate pressure product) with supine bicycle exercise was matched by a 2.2-fold increase in flow. Similarly, dobutamine infusion in patients with coronary artery disease raised the rate pressure product by a factor of 2.2 that was paralleled by a 2.4-fold increase in flow in myocardial territories subtended by normal coronary arteries.

E. CUTANEOUS CIRCULATION- The cutaneous circulation is the circulation and blood supply of the skin. The skin is not a very metabolically active tissue and has relatively small energy requirements, so its blood supply is different to that of other tissues. Some of the circulating blood volume in the skin will flow through will flow through **arteriovenous anastomoses (AVAs)** instead of capillaries. AVAs serve a role in temperature regulation. In this article we shall consider the different adaptations of the cutaneous circulation, and its role in body temperature control.

Arteriovenous Anastomoses

AVAs are low-resistance connections between the small arteries and small veins that supply and drain the skin. These allow the shunt of blood directly into the **venous plexus** of the skin, without it passing through capillaries. Since AVAs contain no capillary section, they are not involved in transport of nutrients to/from the tissues, but instead play a major role in temperature regulation.

Temperature Regulation

The skin is the body's main heat dissipating surface: the amount of blood flow to the skin determines the degree of heat loss and therefore the core body temperature. The blood flow through AVAs is heavily influenced by the **sympathetic nervous system.** At rest, the sympathetic nervous system dominates and acts to constrict AVAs.

Any changes in core temperature are detected by the thermoregulatory centre in the **hypothalamus**. It regulates temperature by altering the level of sympathetic outflow to the cutaneous vessels, to return temperature to its normal range:

In high core temperatures:

- Sympathetic innervation is decreased, reducing the vasomotor tone in the AVAs.
- More blood flows through the AVAs and reaches the venous plexus (close to the surface of the skin), increasing heat loss to reduce core temperature.

In low core temperatures:

- Sympathetic innervation is increased, increasing the vasomotor tone in the AVAs.
- Less blood flows to the apical skin (of nose, lips, ears, hands and feet), reducing heat loss to increase the core temperature.

3. **Discuss the cardiovascular adjustment that occurs during exercise.**

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Mild hypoxia developed during exercise stimulates the juxtaglomerular apparatus to secrete erythropoietin. It stimulates the bone marrow and causes release of red blood cells. Increased carbon dioxide content in blood decreases the pH of blood. **

More heat is produced during exercise and the thermoregulatory system is activated. This in turn, causes secretion of large amount of sweat leading to: i. Fluid loss ii. Reduced blood volume iii. Hemoconcentration iv. Sometimes, severe exercise leads to even dehydration.

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Heart rate increases during exercise. Even the thought of exercise or preparation for exercise increases the heart rate. It is because of impulses from cerebral cortex to medullary centers, which reduces vagal tone. In moderate exercise, the heart rate increases to 180 beats/minute. In severe muscular exercise, it reaches 240 to 260 beats/minute. Increased heart rate during exercise is mainly because of vagal withdrawal. Increase in sympathetic tone also plays some role. Increased heart rate during exercise is due to four factors: i. Impulses from proprioceptors, which are present in the exercising muscles; these impulses act through higher centers and increase the heart rate ii. Increased carbon dioxide tension, which acts through medullary centers iii. Rise in body temperature, which acts on car diac centers via hypothalamus, increased temperature also stimulates SA node directly iv. Circulating catecholamines, which are secreted in large quantities during exercise.

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Cardiac output increases up to 20 L/minute in moderate exercise and up to 35 L/minute during severe exercise. Increase in cardiac output is directly proportional to the increase in the amount of oxygen consumed during exercise. During exercise, the cardiac output increases because of increase in heart rate and stroke volume. Heart rate increases because of vagal withdrawal. Stroke volume increases due to

increased force of contraction. Because of vagal withdrawal, sympathetic activity increases leading to increase in rate and force of contraction.

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Venous return increases remarkably during exercise because of muscle pump, respiratory pump and splanchnic vasoconstriction.

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There is a great increase in the amount of blood flowing to skeletal muscles during exercise. In resting condition, the blood supply to the skeletal muscles is 3 to 4 mL/100 g of the muscle/minute. It increases up to 60 to 80 mL in moderate exercise and up to 90 to 120 mL in severe exercise. During the muscular activity, stoppage of blood flow occurs when the muscles contract. It is because of compression of blood vessels during contraction. And in between the contractions, the blood flow increases. Sometimes the blood supply to muscles starts increasing even during the preparation for exercise. It is due to the sympathetic activity. Sympathetic nerves cause vasodilatation in muscles. The sympathetic nerve fibers causing vasodilatation in skeletal muscle are called sympathetic cholinergic fibers since these fibers secrete acetylcholine instead of noradrenaline. Several other factors also are responsible for the increase in blood flow to muscles during exercise. All such factors increase the amount of blood flow to muscles by means of dilatation of blood vessels of the muscles. Such factors are: i. Hypercapnea ii. Hypoxia iii. Potassium ions iv. Metabolites like lactic acid v. Rise in temperature vi. Adrenaline secreted from adrenal medulla vii. Increased sympathetic cholinergic activity.

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During moderate isotonic exercise, the systolic pressure is increased. It is due to increase in heart rate and stroke volume. Diastolic pressure is not altered because peripheral resistance is not affected during moderate isotonic exercise. In severe exercise involving isotonic muscular contraction, the systolic pressure enormously increases but the diastolic pressure decreases. Decrease in diastolic pressure is because of the decrease in peripheral resistance. Decrease in peripheral resistance is due to vasodilatation caused by metabolites. During exercise involving isometric contraction he peripheral resistance increases. So, the diastolic pressure also increases along with systolic pressure. Blood Pressure after Exercise Large quantities of metabolic end products are produced during exercise. These substances accumulate in the tissues, particularly the skeletal muscle. Metabolic end products cause vasodilatation. So, the blood pressure falls slightly below the resting level after

the exercise. However, the pressure returns to resting level quickly as soon as the metabolic end products are removed from muscles.