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QUESTIONS

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

2. Write short notes on the following:

a. Pulmonary circulation

b. Circle of willis

c. Splanchnic circulation

d. Coronary circulation

e. Cutaneous circulation

3. Discuss the cardiovascular adjustment that occurs during exercise?

1. THE LONG TERM REGULATION OF MEAN ARTERIAL BLOOD PRESSURE

The definition of mean arterial pressure (MAP) is the average arterial pressure throughout one cardiac cycle, systole, and diastole. The mean arterial blood pressure is given as the cardiac output multiplied by the heart rate. MAP is influenced by cardiac output and systemic vascular resistance, each of which is under the influence of several variables. Therefore, the MAP cannot have a constant value. There could be an increase or decrease. Albeit, the value of the MAP is kept at a close range due to the regulation by the body's homeostasis. Regulation can be brought about for a long term or short term.

Here's a light on the **LONG TERM REGULATION.**

There are several physiological mechanisms that regulate blood pressure in the long-term, the first of which is the renin-angiotensin-aldosterone system **(RAAS)**.

LONG TERM REGULATION BY RAAS

The long term regulation by RAAS is stimulated by a decrease in blood pressure

Renin-Angiotensin-Aldosterone System (RAAS)

When there's a drop in the blood pressure, *Renin* a hormone (peptide) is released by the granular cells of the **juxtaglomerular apparatus** in **the kidney**. Asides the drop in blood pressure, it is released in response to: Sympathetic stimulation, reduced sodium-chloride delivery to the distal convoluted tubule, decreased blood flow to the kidney

The release of Renin facilitates the conversion of angiotensinogen to angiotensin I which is then converted to angiotensin II using angiotensin-converting enzyme **(ACE)**. Angiotensin II is a potent vasoconstrictor. It acts directly on the kidney to increase sodium reabsorption in the proximal convoluted tubule. Sodium is reabsorbed via the sodium-hydrogen exchanger. *Angiotensin II* also promotes release of **aldosterone**.

ACE also breaks down a substance called **bradykinin** which is a potent vasodilator. Therefore, the breakdown of bradykinin potentiates the overall constricting effect. Aldosterone promotes salt and water retention by acting at the distal convoluted tubule to increase expression of **epithelial** sodium channels. Furthermore, aldosterone increases the activity of the basolateral sodium-potassium ATP-ase, thus **i**ncreasing the electrochemical gradien**t** for movement of sodium ions. More sodium collects in the kidney tissue and water then follows by osmosis. This results in decreased water excretion and therefore a negative feedback mechanism resulting to increased blood volume and thus increased blood pressure.

Anti-Diuretic Hormone (ADH)

The second mechanism by which blood pressure is regulated is release of Anti Diuretic Hormone (ADH) from the OVLT of the hypothalamus in response to **thirst** or an increased plasma osmolarity. ADH acts to increase the permeability of the collecting duct to water by inserting **aquaporin channels** (AQP2) into the apical membrane.

It also stimulates sodium reabsorption from the thick ascending limb of the loop of Henle. This increases water reabsorption thus increasing plasma volume and decreasing osmolarity.

Further Control of Blood Pressure

Other factors that can affect long-term regulation of blood pressure are natriuretic peptides. These include:

- Atrial natriuretic peptide **(ANP)** is synthesized and stored in cardiac myocytes. It is released when the atria are stretched, indicating of high blood pressure.
- ANP acts to promote sodium excretion. It dilates the **afferent arteriole** of the glomerulus, increasing blood flow (GFR). Moreover, ANP inhibits sodium reabsorption along the nephron. Conversely, ANP secretion is low when blood pressure is low.
- **Prostaglandins** act as local vasodilators to increase GFR and reduce sodium reabsorption. They also act to prevent excessive vasoconstriction triggered by the sympathetic nervous and renin-angiotensin-aldosterone systems.

2.

A.PULMONARY CIRCULATION

The lungs have both a bronchial and a pulmonary circulation. The bronchial circulation to the lungs is the part of the systemic circulation that supplies O2 and nutrients to meet the metabolic requirements of the lungs. However the pulmonary circulation refers to the portion of the cardiovascular system which carries deoxygenated blood away from the heart, towards the alveoli of the lungs to undergo gas exchange, and then returns oxygenated blood back to the heart.

Deoxygenated blood leaves the **right ventricle** of the heart through the pulmonary valve and enters the pulmonary trunk. This divides into the right and left pulmonary arteries.

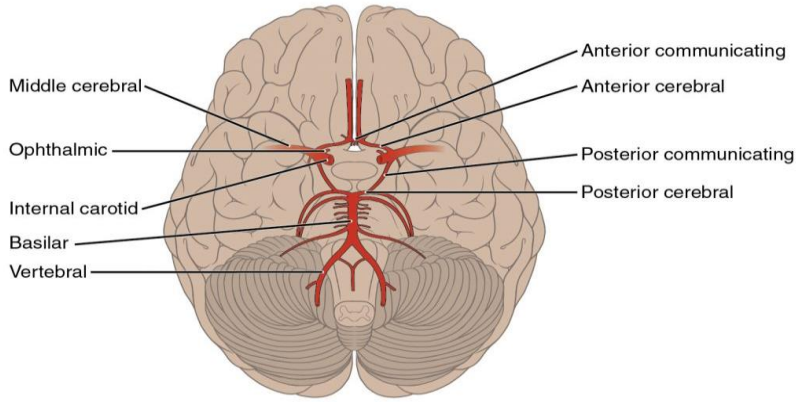
In the lungs the arteries divide further into very fine capillaries at the alveoli, allowing **gas exchange** to take place. Oxygen diffuses from the alveoli into the pulmonary capillaries while carbon dioxide diffuses from the capillaries into the alveoli.

This newly oxygenated blood leaves the lungs through the **pulmonary veins** to the left atrium of the heart, completing the pulmonary cycle. The blood is then distributed around the body via the systemic circulation.

Since the right heart cannot work independently of the left heart both the right and left ventricle must have the same cardiac output to prevent blood building up in either the systemic or pulmonary circulation. This means the pulmonary circulation must be able to accept the entire cardiac output **(5L).**

B.CIRCLE OF WILLIS

The brain requires a large amount of oxygen and glucose to meet its high metabolic demand. Therefore, its circulation has structural and functional adaptations to ensure a consistently **high blood flow** is maintained. **Circle of Willis** is a structural adaptation to meet the oxygen and glucose requirements of brain. **Circle of Willis** is the anastamoses between the basilar and internal carotid arteries. This means that even if one artery is damaged, blood flow is not compromised.



Blood-brain barrier is a highly selective barrier between the systemic circulation and the brain's extracellular fluid formed by endothelial cells. It is permeable to lipophilic molecules such as O2 and CO2. It is impermeable to lipid insoluble molecules like K+ and catecholamines. Protects the brain from potentially harmful neurotoxins and helps prevent infection from spreading to the brain (causing encephalitis).

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.

D.CORONARY CIRCULATION

The coronary circulation is the Cardiac muscle's own dedicated circulatory system: the coronary blood vessels. It constantly undergoes phases of **contraction** and relaxation to pump blood around the body from the heart. As myocardial oxygen demand increases, coronary blood flow must also increase to meet requirements.

The coronary arteries are unique, as they are perfused during **diastole.** This makes the cardiac muscle adapt. Entry of blood into the coronary arteries occurs through the aortic sinuses, which are openings found behind the flaps of the aortic valve. As the heart relaxes during diastole, blood fills the valve pockets and hence blood is able flow into the coronary arteries, allowing supply to the cardiac muscle. The coronary arteries also send branches into the myocardium, which are compressed during systole. Therefore, when the muscle relaxes, blood flow in the myocardium is increased as there is less compression on these vessels.

In order to help maintain a high basal rate of flow, there is continuous production of nitrous oxide which is a potent vasodilator. **Vasodilation** can also be caused by the accumulation of metabolites such as **a**denosine, K+ ions and H+ ions. This phenomenon is known as reactive hyperaemia, and occurs in tissues around the whole body.

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 **arteriovenous anastomoses (AVAs)** instead of capillaries. AVAs serve a role in temperature regulation.

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.

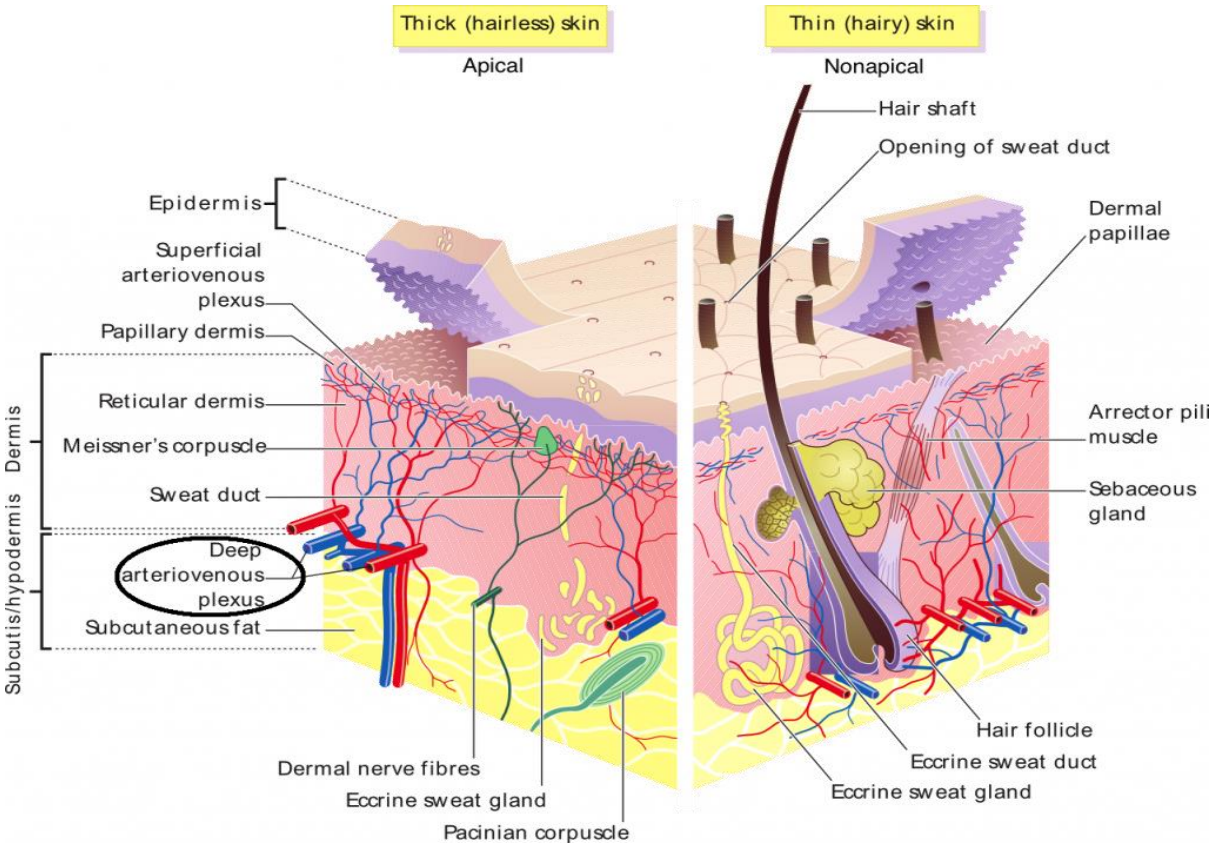
Temperature Regulation

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: The sympathetic innervation is decreased, reducing the vasomotor tone in the AVAs. Therefore, 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: The sympathetic innervation is increased, increasing the vasomotor tone in the AVAs. Therefore, less blood flows to the apical skin (of nose, lips, ears, hands and feet), reducing heat loss to increase the core temperature



Arteriovenous anastamoses (circled) near the dermal-epidermal border of apical skin

3. CARDIOVASCULAR ADJUSTMENT DURING EXERCISE

- During exercise, cardiac output is increased, fourfold, fivefold as an integrated response to severe exercise, due primarily to increases in heart rate and to a lesser extent to increase the stroke volume.
- The increase in stroke volume is partly due to an increase in end-diastolic cardiac size (Frank-Starling mechanism) and secondarily due to a reduction in end-systolic cardiac size. The full role of the Frank-Starling mechanism is masked also by **tachycardia**.
- The reduction in end-systolic dimensions can be related to increased contractility, mediated by beta adrenergic stimulation. Beta adrenergic blockade prevents the inotropic response, the decrease in end-systolic dimensions, and approximately 50% of the tachycardia of exercise.
- The enhanced cardiac output is distributed preferentially to the exercising muscles including the heart. Blood flow to the heart increases fourfold to fivefold as well, mainly reflecting the augmented metabolic requirements of the myocardium due to near maximal increases in cardiac rate and contractility.
- Blood flow to the inactive viscera (e.g., kidney and gastrointestinal tract) is maintained during severe exercise normally. It is suggested that local autoregulatory mechanisms are responsible for maintained visceral flow in the face of neural and hormonal autonomic drive, which acts to constrict renal and mesenteric vessels and to reduce blood flow. However, in the presence of circulatory impairment, where oxygen delivery to the exercising muscles is impaired as occurs to complete heart block where normal heart rate increases during exercise are prevented, or in congestive right heart failure, where normal stroke volume increases during exercise are impaired, or in the presence of severe anemia, where oxygen-carrying capacity of the blood is limited, visceral blood flows are reduced drastically and blood is diverted to the exercising musculature. Thus, visceral flow is normally maintained during severe exercise as long as all other compensatory mechanisms remain intact. However, when any other compensatory mechanism is disrupted, reduction and diversion of visceral flow occur.