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COURSE TITLE: PHYSIOLOGY

ASSIGNMENT:

1. Discuss the long term regulation of mean arterial pressure?
2. Write short notes on the following:

a) Pulmonary circulation
b) Circle of wills
c) Splanchnic circulation
d) Coronary circulation
e) Cutaneous circulation

3. Discuss the cardiovascular adjustment that occurs during exercise?

1. Long-Term Regulation of Blood Pressure

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

Renin-Angiotensin-Aldosterone System (RAAS)

Renin is a peptide hormone released by the granular cells of the juxtaglomerular apparatus in the kidney. It is released in response to:

i. Sympathetic stimulation
ii. Reduced sodium-chloride delivery to the distal convoluted tubule
iii. Decreased blood flow to the kidney
iv. 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 increasing the electrochemical gradient 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 increased blood volume and thus 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 synthesised 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 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. The term pulmonary circulation is readily paired and contrasted with the systemic circulation. The vessels of the pulmonary circulation are the pulmonary arteries and the pulmonary veins.A separate system known as the bronchial circulation supplies oxygenated blood to the tissue of the larger airways of the lungs.

B. The Circle of Willis: is the joining area of several arteries at the bottom (inferior) side of the brain. At the Circle of Willis, the internal carotid arteries branch into smaller arteries that supply oxygenated blood to over 80% of the cerebrum. It encircles the stalk of the pituitary gland and provides important communications between the blood supply of the forebrain and hindbrain (ie, between the internal carotid and vertebrobasilar systems following obliteration of primitive embryonic connections). Although a complete circle of Willis is present in some individuals, it is rarely seen radiographically in its entirety; anatomical variations are very common and a well-developed communication between each of its parts is identified in less than half of the population. The circle of Willis begins to form when the right and left internal carotid artery (ICA) enters the cranial cavity and each one divides into two main branches: the anterior cerebral artery (ACA) and middle cerebral artery (MCA). The anterior cerebral arteries are then united and blood can cross flow by the anterior communicating (ACOM) artery.

C. Splanchic circulation:
Splanchnic or visceral circulation constitutes three portions:
1. Mesenteric circulation supplying blood to GI tract
2. Splenic circulation supplying blood to spleen
3. Hepatic circulation supplying blood to liver.
Unique feature of splanchnic circulation is that the blood from mesenteric bed and spleen forms a major amount of blood flowing to liver. Blood flows to liver from GI tract and spleen through portal system.

D. Coronary circulation: is the circulation of blood in the blood vessels that supply the heart muscle (myocardium). Coronary arteries supply oxygenated blood to the heart muscle, and cardiac veins drain away the blood once it has been deoxygenated. Because the rest of the body, and most especially the brain, needs a steady supply of oxygenated blood that is free of all but the slightest interruptions, the heart is required to function

continuously. Therefore its circulation is of major importance not only to its own tissues but to the entire body and even the level of consciousness of the brain from moment to moment. Interruptions of coronary circulation quickly cause heart attacks (myocardial infarctions), in which the heart muscle is damaged by oxygen starvation. Such interruptions are usually caused by ischemic heart disease (coronary artery disease) and sometimes by embolism from other causes like obstruction in blood flow through vessels.

E. 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.

3.

1. ON BLOOD 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.
2. ON BLOOD VOLUME 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.

3. ON HEART RATE 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.
4. ON CARDIAC OUTPUT 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.

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

6. ON BLOOD FLOW TO SKELETAL MUSCLES 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.

7. ON BLOOD PRESSURE 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, the peripheral resistance increases. So, the diastolic pressure also increases along with systolic pressure.