NAME: OJEME DORA EBEHIREMEN

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COLLEGE: MEDICINE AND HEALTH SCIENCES

DEPARTMENT: MEDICINE AND URGERY

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ASSIGNMENT:

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) Mean arterial blood pressure is the average pressure existing in the arteries **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:

Sympathetic stimulation

Reduced sodium-chloride delivery to the distal convoluted tubule

Decreased blood flow to the kidney

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 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 osmolality.

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.

2) A) Pulmonary circulation is the system of transportation that shunts de-oxygenated blood from the heart to the lungs to be re-saturated with oxygen before being dispersed into systemic circulation. Deoxygenated blood from the lower half of the body enters the heart from the inferior vena cava while deoxygenated blood from the upper body is delivered to the heart via the superior vena cava. Both the superior vena cava and inferior vena cava empty blood into the right atrium. Blood flows through the tricuspid valve into the right ventricle. It then flows through the pulmonic valve into the pulmonary artery before being delivered to the lungs. While in the lungs, blood diverges into the numerous pulmonary capillaries where it releases carbon dioxide and is replenished with oxygen. Once fully saturated with oxygen, the blood is transported via the pulmonary vein into the left atrium which pumps blood through the mitral valve and into the left ventricle. With

a powerful contraction, the left ventricle expels oxygen-rich blood through the aortic valve and into the aorta.

b) circle of Willis: The circle of Willis is an important junction of arteries at the base of the brain. The structure encircles the middle area of the brain, including the stalk of the pituitary gland and other important structures. Two arteries, called the carotid arteries, supply blood to the brain. They run along either side of the neck and lead directly to the circle of Willis. The circle of Willis plays an important role, as it allows for proper blood flow from the arteries to both the front and back hemispheres of the brain. The arteries that stem off from the circle of Willis supply much of the blood to the brain. The circle of Willis also serves as a sort of safety mechanism when it comes to blood flow

- c) splanchnic circulation: this describes the blood flow to the abdominal gastrointestinal organs including the stomach, liver, spleen, pancreas, small intestine, and large intestine. It comprises three major branches of the abdominal aorta; the coeliac artery; superior mesenteric artery (SMA); and inferior mesenteric artery. blood flow in the splanchnic circulation is controlled via intrinsic (myogenic and metabolic) and extrinsic (autonomic and humoral) mechanisms. 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: part of the systemic circulatory system that supplies blood to and provides drainage from the tissues of the heart. In the human heart, two coronary arteries arise from the aorta just beyond the semilunar valves; during diastole, the increased aortic pressure above the valves forces blood into the coronary arteries and thence into the musculature of the heart. Deoxygenated blood is returned to the chambers of the heart via coronary veins; most of these converge to form the coronary venous sinuous, which drains into the right atrium. The heart

normally extracts 70 to 75 percent of the available oxygen from the blood in coronary circulation, which is much more than the amount extracted by other organs from their circulations—e.g., 40 percent by resting skeletal muscle and 20 percent by the liver. Obstruction of a coronary artery, depriving the heart tissue of oxygen-rich blood, leads to death of part of the heart muscle in severe cases, and total heart failure and death may ensue.

e) The cutaneous circulation is the circulation and blood supply of the skin. Vascular architecture of the skin has the general pattern of the capillary circulation. For the most part, papillae contain capillary blood vessels and nerve endings. The arteries that supply the skin, originate from richly anastomosing irregular plexus (first plexus) of the deepest part of the corium (dermis). From this cutaneous arterial plexus, the single arteriole arises and ascends through the corium and forms the second plexus just below the dermis. Capillaries arising from this plexus supply the hair follicles and papillae of the dermis. The arterioles also ascend towards the superficial layer and form the third plexus in the sub-papillary region of the dermis. Every papilla gets capillary network from this plexus.

3) The integrated response of the cardiovascular system to severe exercise involves fourfold to fivefold increases in cardiac output, which are due primarily to increases in cardiac rate and to a lesser extent to augmentation of stroke volume. The increase in stroke volume is partly due to an increase in end-diastolic cardiac size and secondarily due to a reduction in end-systolic cardiac size. The full role of the Frank-Starling mechanism is masked by the concomitant 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 four-fold 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 in the normal dog. It is suggested that local auto regulatory 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.