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ASSIGNMENT

1) Blood pressure is a measure of how well our cardiovascular system is functioning. We all require a blood pressure high enough to give our organs the blood and nutrients they need, but not so high our blood vessels become damaged. As such, our bodies must maintain control over our blood pressure to keep it at a normal level. The body's blood pressure is a measure of the pressures within the cardiovascular system during the pumping cycle of the heart. It is influenced by a vast number of variables, and can alter in either direction for various reasons. Everyone's blood pressure is slightly different and can change throughout the day depending on activity.

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 osmolarity.

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**

This is otherwise called the Lesser circulation. Blood is pumped from right ventricle to lungs through pulmonary artery. Exchange of gases occurs between the blood and alveoli of the lungs at pulmonary capillaries. Oxygenated

blood returns to the left atrium through the pulmonary veins. Hence, the left side contains oxygenated blood and the right side of the heart contains deoxygenated or venous blood.

b) **Circle of Willis**

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. The brain receives blood from the basilar artery and internal carotid artery. The circle of Willis 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 vertebro-basilar systems following obliteration of primitive embryonic connections. The circle of Willis begins to form when the right and left internal carotid artery enters the cranial cavity and each one divides into two main branches: the anterior cerebral artery and middle cerebral artery. The anterior cerebral arteries are then united and blood can cross flow by the anterior communicating (ACOM) artery.

c) **Splanchnic Circulation**

The splanchnic circulation comprises the gastric, small intestinal, colonic, pancreatic, hepatic, and splenic circulations. They are arranged in parallel and fed by the celiac artery and the superior and inferior mesenteric arteries. Overall splanchnic blood flow requires about 25% of cardiac output. The resistance arterioles are the primary determinant of vascular resistance in the splanchnic circulation. Neuronal control of the mesenteric circulation is almost entirely sympathetic in origin. The parasympathetic fibers from the vagi have little effect on blood flow. The sympathetic postganglionic fibers cause arteriolar vasoconstriction and decrease splanchnic perfusion. Sympathetic stimulation also contracts the smooth muscle of the capacitance veins in the splanchnic circulation, and may expel a large volume of pooled blood from the splanchnic into the systemic circulation. Autoregulation in the splanchnic circulation is less marked than in the cerebral, cardiac, or renal circulations. The response is present, however, and serves to restore blood flow to areas suffering hypoperfusion because of an acute reduction in perfusion pressure. The splanchnic circulation also responds to reduced perfusion pressure by the redistribution of blood flow within individual organs.

d) **Coronary Circulation**

This is the circulation of blood through blood vessels of the heart muscles (myocardium). It is responsible for functional blood supply to heart muscle itself. Blood flowing through the chambers of the heart does not nourish the myocardium. When functioning normally, blood in the coronary blood vessels supply adequate oxygen to the myocardium. Like systemic circulation and pulmonary circulation, coronary circulation is also made up of arteries, arterioles, capillaries, venules and veins. Coronary circulation is very rapid and blood flow occurs mainly during diastolic phase. Its regulation is mainly by metabolites and not neutral.

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. 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) Certain cardiovascular adjustments are made to enable the body to cope with the increased demands that occur during exercise. Muscular exercise causes the strongest physiological stress on the human cardiovascular system. It demands a huge increase in the supply of oxygen and nutrients to the exercising muscle and proportionate increase in the removal of metabolic waste product and excess heat.

The following adjustments include:

- Changes in cardiac function
- Changes in arterial blood pressure
- Redistribution of cardiac output
- Increased oxygen delivery to the tissues
- Temperature regulation

i) Changes in cardiac function: these include increase in stroke volume, heart rate and cardiac output. It is important to note the change in stroke volume alone will increase cardiac output by only 50%. Therefore, increased heart rate is the main mechanism by which increase of up to 60% in cardiac output is achieved in exercise. The changes in the force of contraction of the heart, the heart rate and cardiac output are brought about by increased sympathetic discharge to the heart, reduced parasympathetic tone to the heart, increased level of adrenaline and nonadrenaline in the blood and as exercise progresses and increase in temperature.

ii) Changes in arterial blood pressure: there can be an increase arterial blood pressure as little as 20mmHg or as great as 80mmHg depending on the type of exercise and condition under which the exercise is performed. These are the following factors needed for increased arterial pressure during exercise:

a) Vasoconstriction of arterioles and small arteries in most tissues of the body, except in exercising skeletal muscles.

b) Increased pumping activity of the heart. Both heart rate and stroke volume increase in exercise, leading to large increase in cardiac output.

c) There is mixed venoconstriction leading to great increase in venous return.

The above factors are due to increased release of adrenaline and nonadrenaline from the adrenaline medulla.

iii) Redistribution of cardiac output: blood flow to the exercising muscle increase enormously during maximal exercise. The proportion of cardiac output that goes to the exercising muscle may rise from the resting value 15% to 85%. The increase is due to the marked arteriolar dilation in the exercising muscle, cardiac output are strongly vasodilated by the local vasodilator substances such as low oxygen, lactic acid, carbon(ii)oxide in the muscles themselves when most of the arterioles of the peripheral circulation are strongly contracted. Thus, there is marked increase in blood flow to the active muscles. The redistribution of blood diverts most of the cardiac output to the exercising muscles. However, blood flow to the brain and heart are not reduced during exercise.

iv)Increased oxygen delivery to the tissues: this is achieved by the combined effects of increased pulmonary ventilation, increased cardiac output and at the tissue level there are the following adjustments ensure increased oxygen delivery to the tissue.

a) More capillaries are open and they are dilated.

b) The dilated vessels reduce the distance of diffusion between the capillary walls and the body cells.

c) Increased temperature, increased carbon(ii)oxide production, fall in pH and increase diphosphateglycerate level cause the oxygen-hemoglobin dissociation curve to shift to the right thereby increasing oxygen released to the tissues.

All the above factors operate in a synergic manner to increases oxygen delivery to the exercising muscle by up to 20 times the resting value.

v) Temperature regulation: this is achieved through increased heat loss via the lung due to increased pulmonary ventilation, and the skin. Most of the heat is generated in the deep organs and the heat is transferred from the deep organs and tissue of the skin, where the heat is lost to the air and other objects in the surrounding of the body. The rate at which heat is lost is determined by how rapidly the heat produced in the core of the body can be carried to the skin and how rapidly heat can be transferred from the skin to the surroundings.

Blood vessels are distributed profusely immediately beneath the skin. There is a continuous venus plexus beneath the skin that is supplied by inflow of blood from the skin capillaries.