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**ASSIGNMENT**

1. **LONG TERM REGULATION OF MEAN ATRIAL 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 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 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. **PULMONARY CIRCULATION**

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: This is the beginning of systemic circulation.

**CIRCLE OF WILLIS**

The circle of Willis is a ring of interconnecting arteries located at the base of the brain around the optic chiasma or chiasma (partial crossing of the optic nerve – CNII; this crossing is important for binocular vision), infundibulum of the pituitary stalk and the hypothalamus.

This arterial ring provides blood to the brain and neighboring structures. Polygonal anastomotic shape offers the possibility of alternate pathways for the blood flow, which is essential for the brain functioning, since it is the structure that is mostly sensitive to hypoxia. Hypoxia of the brain tissue that lasts longer than 6 minutes results with the irreversible changes in the brain parenchyma, and depending on the location of the lesion, the functional damages vary widely.

**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.

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. Overall splanchnic blood flow requires about 25% of cardiac output. The splanchnic venous capacitance reservoir contains about one-third of the body's total blood volume. 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. Auto-regulation 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 follow to areas suffering hypo perfusion 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. For example, in hypovolemic shock perfusion usually favors the mucosa of the gut at the expense of the muscular is mucosa.

The liver is unique in that it has both an arterial and a venous afferent blood supply. In the resting adult the liver receives approximately 500 mL min of blood via the hepatic artery and a further 1300 mL min from the portal circulation.

**CORONARY CIRCULATION**

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

**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

**CARDIOVASCULAR ADJUSTMENTS DURING EXERCISE**

The integrated response 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 (Frank-Starling mechanism) 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 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 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. However, when any other compensatory mechanism is disrupted (even the elimination of splenic reserve in the dog),