NSOKO-NKWOR QUEEN ESTHER

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QUESTION ONE

Mean arterial blood pressure is the average arterial pressure throughout one cardiac cycle, systole and diastole. This is influenced by cardiac output and systemic vascular resistance, each of which is under the influence of several variables. Arterial blood pressure varies even under physiological conditions, however immediately it is brought back to a normal level because of the presence of well-organized regulatory mechanisms in the body.

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 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 the 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 the expression of epithelial sodium channels. Furthermore, aldosterone increases the activity of the basolateral sodium-potassium ATP-ase, thus increasing the electrochemical gradient for the movement of sodium ions. Going further into the actions of Angiotensin II:

When blood pressure and ECF volume decrease, renin secretion from kidneys is increased. It converts angiotensinogen into angiotensin I. This is converted into angiotensin II by ACE. Angiotensin II acts in two ways to restore blood pressure:

A) It causes constriction of arterioles in the body so that the peripheral resistance is increased and blood pressure rises. In addition, angiotensin II causes constriction of afferent arterioles in kidneys, so that glomerular filtration reduces. This results in the retention of water and salts, increases ECF volume to a normal level. This in turn increases the blood pressure to a normal level.

B) Simultaneously, angiotensin II stimulates the adrenal cortex to secrete aldosterone. This hormone increases the reabsorption of sodium from renal tubules. Sodium reabsorption is followed by water reabsorption, resulting in increased ECF volume and blood volume. It increases the blood pressure to a normal level.

Anti-Diuretic Hormone (ADH)

The second mechanism by which blood pressure is regulated is the release of Anti Diuretic Hormone (ADH) from the OVLT of the hypothalamus in response to thirst or an increased plasma osmolarity. 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 synthesized and stored in cardiac myocytes. It is released when the atria are stretched, indicating high blood pressure. This hormone is secreted by the atrial musculature of the heart. It causes dilation of blood vessels and decreases blood pressure.

Prostaglandins: Acts as local vasodilators to increase GFR and reduce sodium reabsorption. They also act to prevent excessive vasoconstriction triggered by the sympathetic nervous and renin.

QUESTION TWO

A) **Pulmonary circulation** is replenishing, it is a portion of the circulatory system which carries deoxygenated blood, blood low in oxygen and high in carbon dioxide from the right side of the heart to the lungs to be re-saturated with oxygen before being dispersed into systemic circulation it begins in the right ventricle and ends on the left atrium. The steps of the pulmonary circulation are the pulmonary trunk splits into the right and left pulmonary arteries. These arteries transport the deoxygenated blood to arterioles and capillary beds in the lungs. There, carbon dioxide is released and oxygen is absorbed. Oxygenated blood then passes from the capillary beds through venules into the pulmonary vein'
B) **Circle of Willis** is the joining area of several arteries at the bottom (inferior) side of the brain. The Circle of Willis is an arterial ring sited just at the base of the brain (around eye level) and is completed by the anterior communicating artery and two posterior communicating arteries. It describes the ring of blood vessels in the base of the brain that connects the main intracerebral blood vessels. It is incomplete in most individuals, although wide variations exist.
C) **Splanchnic circulation** is composed of gastric, small intestine, colonic, pancreatic, hepatic, and splenic circulations, arranged in parallel with one another it is a very complex system. Several functions depend on its normal operation, including digestion and absorption within the gut, maintenance of the mucosal barrier, and the successful healing of surgical anastomoses.

D) **Coronary circulation** is 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 sinus, which drains the right atrium.

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, some of the circulating blood volume in the skin will flow through anteriovenous anastomoses (AVAs) instead of capillaries.

QUESTION THREE

Cardiovascular adjustment to severe exercise involves fourfold to fivefold increases in cardiac output, which are due primarily to increases in cardiac output, which are due primarily to increases in cardiac output, which are due primarily to 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. 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 is maintained during severe exercise. 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 prevented, or in congestive right heart failure, where normal stroke volume 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 blood is limited, visceral blood flows are reduced drastically and blood is diverted to the exercising musculature. Thus a 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.