NAME: OPARA JUDITH CHIOMA

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ASSIGNMENT

1. Discuss the long term regulation of mean arterial blood pressure?

Mean arterial pressure is regulated by changes in cardiac output and systemic vascular resistance. There are several physiological mechanisms that regulate blood pressure in the long-term, the first of which is the renin-angiotensin-aldosterone system(RASS).

*Renin* is a peptide hormone released by the granular cells of thejuxtaglomerular 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 **a**quaporinchannels (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.

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.

1. Write short note on the following:

A. Pulmonary circulation: The pulmonary circulation conducts the entire cardiac output with a remarkably low driving pressure from the pulmonary artery (mean Ppa of 15 to 20 mm Hg) to the left atrium (Pla of 7 to 12 mm Hg). As in the airways, the branching pattern of vessels leads to an increase in total cross-sectional area as the alveolar vessels are approached, but unlike in the airways, this increase is not associated with a decrease in resistance. The pulmonary circulation is a network of segmental resistors that share common upstream (i.e., Ppa) and downstream (i.e., Pla) pressures. Flow is distributed to the various segments in proportion to the reciprocal of the total serial resistance through any segment. The benefit of having the highest resistance at the microvascular level is that the control of blood flow distribution can occur at a finer level, allowing active mechanisms of flow regulation (see further on) to adjust blood flow to relatively small lung regions.

B. Circle of wills: 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. Each carotid artery branches into an internal and external carotid artery. The internal carotid artery then branches into the cerebral arteries. This structure allows all of the blood from the two internal carotid arteries to pass through the circle of Willis. left and right internal carotid arteries, left and right anterior cerebral arteries, left and right posterior cerebral arteries, left and right posterior communicating arteries, basilar artery, anterior communicating artery. The circle of Willis is critical, as it is the meeting point of many important arteries supplying blood to the brain. The internal carotid arteries branch off from here into smaller arteries, which deliver much of the brain’s blood supply. 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.

C. Splanchnic circulation: The splanchnic circulation is composed of gastric, small intestinal, colonic, pancreatic, hepatic, and splenic circulations, arranged in parallel with one another. The three major arteries that supply the splanchnic organs, cellac and superior and inferior mesenteric, give rise to smaller arteries that anastomose extensively. The circulation of some splanchnic organs is complicated by the existence of an intramural circulation. Redistribution of total blood flow between intramural vascular circuits may be as important as total blood flow. Numerous extrinsic and intrinsic factors influence the splanchnic circulation. Extrinsic factors include general hemodynamic conditions of the cardiovascular system, autonomic nervous system, and circulating neurohumoral agents. Intrinsic mechanisms include special properties of the vasculature, local metabolites, intrinsic nerves, paracrine substances, and local hormones. The existence of a multiplicity of regulatory mechanisms provides overlapping controls and restricts radical changes in tissue perfusion.

D. 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.

E.Cutaneous circulation: The cutaneous circulation ensures heat exchange between the body and the environment. Skin BF mainly reflects this key role played by the skin in thermoregulation. In rabbits, on passing from wakefulness to NREM sleep, skin vasodilation accompanies the decrease in the set point of body temperature. In humans, skin vasodilator activity is unchanged on passing from wakefulness to NREM sleep. However, skin temperature, which indirectly depends on the control of cutaneous circulation, is higher during the night than during the day, causing skin warming and dissipation of body heat during sleep. Skin temperature during sleep increases especially in the distal skin areas, which are usually cooler than the proximal ones during wakefulness. Interestingly, this pattern of skin vasomotion is impaired in narcoleptic patients, possibly contributing to their low core temperature. Narcolepsy is a rare sleep disorder associated with a selective loss of hypothalamic hypocretin (orexin)-producing neurons. A substantial body of evidence indicates that thermoregulation is impaired in REM sleep. In this state, the impairment of thermoregulatory processes entails the loss of vasomotor patterns reflexly driven by ambient temperature and, hence, the occurrence of paradoxical changes in skin BF. In particular, in a cold environment, thermoregulation physiologically causes skin vasoconstriction, which is accompanied by vasoconstriction in the splanchnic and renal beds and by vasodilation in the skeletal muscle bed. However, on passing from NREM sleep to REM sleep, skin BF increases because of the decrease in thermoregulatory neurogenic vasoconstriction combined with an increase in ABP.

Discuss the cardiovascular adjustment that occurs during exercise?

The integrated response to servere 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 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 anaemia, 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), reduction and diversion of visceral flow occur.

REFERENCES:

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