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Cardiovascular Physiology Assignment

1. Discuss the long-term regulation of mean arterial blood pressure
There are several physiological mechanisms that regulate the mean arterial pressure in the long-term.

i. Renin-Angiotensin-Aldosterone System (RAAS): Renin is a peptide hormone that is released upon decreased blood flow to the kidney, and fascilitates the conversion of angiotensinogen to angiotensin I, which is the converted to angiotensin II by the Angiotensin-Converting Enzyme (ACE). Angiotensin II is a potent vasoconstricto. It acts directly on the kidney to increase Na+ reabsorption, and it induces the release of aldosterone. ACE also activates bradykinin which is a potent vasodilator that suppresses the overall vasoconstriction. Aldosterone promotes salt and water retention by increasing the expression of Na+ channels and Na+/K+ ATPase, thus increasing the reabsorption of Na+. More Na+ collects in the kidney and then water follows by osmosis. This results in decreased water excretion and increased blood volume which therfore increases the blood pressure.

ii. Vasopressin/Anti-Diuretic Hormone (ADH): Vasopressin is a hormone released from the hypothalamus in response to thirst and increased plasma osmolarity. It stimulates water reabsorption by increasing the expresion of aquaporin channels in the collecting ducts. It also stimulates Na+ reabsorption, thus increasing water reapsortion and blood volume, and decreasing plasma osmolarity, which is followed by increase in blood pressure

Other long-term regulation factors include

- Atrial and Ventricular/Brain Natruiretic Peptides: They are released in response to atrial and ventricular wall stretching, which stimulates the stretch receptors, indicating high blood volume and therfore high blood pressure. They induce vasodilation of glomerular arterioles, thus increasing glomerular blood flow (i.e glomerular filtration rate) and they supress Na+ reabsorption, thus reducing blood volume and blood pressure.
- Prostaglandins: They act aslocal vasodilators to increase glomerular fitration rate and reduce Na+ reabsorption. They also act locally to prevent excessive vasoconstriction trigerred by sympathetic stimulations or the renin-angiotensin-aldosterone system.

2. Write short notes on the following

a. Pulmonary circulation: In the pulmonary circulation, deoxygenated blood from the right ventricle flows into the pulmonary trunk. The pulmonary trunk splits into the right and left pulmonary arteries. These arteries transport the deoxygenated blood to the capillary bed of the adjacent lung. There, carbon dioxide is exchanged for oxygen oxygenated blood then flows into the right (from the capillary bed of the right lung) and left (from the capillary bed of theleft lung) pulmonary veins. The pulmonary veins transport the blood

to the left atrium of the heart. The pulmonary arteries are the only arteries that transport deoxygenated blood from the heart, and also the pulmonary veins are the only veins that transport oxygenated blood back to the heart. Pulmonary hypertension is a type of high blood pressure in the pulmonary arteries, of which the cause may be unknown, or may be caused by certain disease or drugs. This elevated blood pressure can lead to severe shortness of breath and death.

b. Circle of Willis: the circle of Willis, also called cereberal arterial circle is a circulating anastomosis that supplies blood to the brain and surrounding structures. It is composed of the following arteries:
   - Anterior cerebral artery (left and right)
   - Anterior communicating artery
   - Internal carotid artery (left and right)
   - Posterior cerebral artery (left and right)
   - Posterior communicating artery (left and right)

   The arrangement of the brain's arteries into the circle of Willis creates redundancy for collateral circulation in the cerebral circulation. If one part becomes blocked or stenosed, or one of the arteries supplying the circle is blocked or narrowed, blood flow from the other branched often preserve the cerebral perfusion well enough to prevent ischemia.

c. Splanchnic circulation: The splanchnic circulation consists of the blood flow originating from the celiac, superior mesenteric and inferior mesenteric arteries and is distributed to all abdominal viscera. The splanchnic circulation receives over 25% of the cardiac output with a relatively constant blood volume. Thus, the splanchnic circulation can act as a site of regulation of distribution of cardiac output and also as a blood reservoir. Multiple regulatory pathways are involved in the distribution of the splanchnic circulation. Food digestion (catabolism) automatically increases the blood flow of the splanchnic circulation (i.e. autoregulation), which increases the gastrointestinal blood supply. Certain gastrointestinal hormones such as gastrin and cholecystokinin also augment the blood flow.

d. Coronary circulation: The coronary circulation is the means of blood supply to the myocardium of the heart. Coronary arteries (which originate from a small branch of the aorta) supply oxygenated blood to the heart muscles, and coronary veins (which originate from the coronary sinus) drain away the deoxygenated blood. Because the entire system, most especially the brain needs a steady flow and supply of oxygenated blood, the heart is required to function continuously, and in order for that to occur and prevent heart failure, the heart muscles need enough blood supply. Interruptions of the coronary circulation cause heart attack (myocardial infarcation), in which the heart muscles are damaged by ischemia and oxygen starvation. Causes of such interruptions include coronary embolism and coronary heart disease. High blood pressure in the coronary circulation can lead to hypertensive heart disease which includes heart failure, ischemic heart disease and ventricular hypertrophy. It is the number on cause of death in individuals with high blood pressure.

e. Cutaneous circulation: the cutaneous circulation is the blood supply of the skin. The skin is not a very metabolically active tissue and requires relatively little energy. Some of the circulating blood in the skin flow through arteriovenous anastomoses (AVAs) instead of capillaries. This allows the shunt of blood directly into the venous plexus without passing through capillaries, and therefore there is no transport of oxygen and nutrients (which are required for energy production) to the skin. Instead the AVAs play a major role in temperature regulation. When there is a change in core temperature, the hypothalamus maintains the body temperature by altering the level of blood flow to the cutaneous circulation to increase or decrease the transfer of heat from the cutaneous circulation to the surrounding.

3. Discuss the cardiovascular adjustment that occurs during excercise

The cardiovascular adjustments in exercise consist of a combination and an integration of neural and local chemical factors. The neural factors consist of:

- central command,
- reflexes originating in the contracting muscle, and
- the baroreceptor reflex.

In humans and in trained animals, anticipation of physical activity inhibits vagus nerve impulses to the heart; this withdrawal of vagal nerve activity underlies the initial increase in heart rate. Eventually, sympathetic nerve discharge also increases. The inhibition of parasympathetic areas and activation of sympathetic areas of the medulla increase the heart rate and myocardial contractility. The tachycardia and the enhanced contractility increase the cardiac output, in turn raising the arterial pressure.

Exercise can be understood with the help of cardiac function and vascular function curves. First, along with tachycardia, there is increased contractility caused by norepinephrine released from cardiac sympathetic nerves. Second, there is a change in the total peripheral resistance that results from vasodilation. At the same time that the heart is stimulated, the sympathetic nervous system elicits vascular resistance changes in the periphery.

In skin, kidneys, splanchnic regions, and inactive muscle, sympathetically mediated vasoconstriction increases vascular resistance, diverting blood away from these areas. This greater resistance in vascular beds of inactive tissues persists throughout the period of exercise. The increase in blood flow in the active muscles at the onset of exercise may be caused by increased ATP breakdown (catabolism) and heat production. As cardiac output and blood flow to active muscles increase with progressive increments in the intensity of exercise, visceral blood flow (splanchnic and renal vasculatures) decreases. Blood flow to the myocardium increases, whereas that to the brain is unchanged. Skin blood flow initially decreases during exercise. It then increases, as body temperature rises with increments in duration and intensity of the exercise. Skin blood flow finally decreases when the skin vessels constrict, as the total body O^2 consumption nears maximum. The major circulatory adjustment to prolonged exercise involves the vasculature of the active muscles. Local formation of vasoactive metabolites induces marked dilation of the resistance vessels, which progresses as the intensity level of exercise increases.