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MEDICINE AND HEALTH SCIENCES.

MEDICINE AND SURGERY.

PHYSIOLOGY.

1. LONG-TERM REGULATION OF MEAN ARTERIAL BLOOD PRESSURE (RENAL MECHANISM).

Kidney plays important role in the long term regulation of arterial pressure. When blood pressure alters slowly in several days, months or years, the nervous mechanism adapts to the altered pressure and loses the sensitivity for the changes. It cannot regulate the pressure any more. In such conditions, the renal mechanism operates efficiently to regulate the blood pressure. Therefore, it is called long-term regulation. Kidneys regulate arterial blood pressure by two ways:

1. By regulation of ECF volume
2. Through renin-angiotensin mechanism.

BY REGULATION OF EXTRACELLULAR FLUID VOLUME:

When the blood pressure increases, kidneys excrete large amounts of water and salt, particularly sodium through pressure diuresis and pressure natriuresis. Pressure diuresis is the excretion of large quantity of water in urine because of increased blood pressure. Even a slight increase in blood pressure doubles the water excretion. Pressure natriuresis is the excretion of large quantity of sodium in urine. Because of diuresis and natriuresis, there is a decrease in ECF volume and blood volume, which in turn brings the arterial blood pressure back to normal level. When blood pressure decreases, the reabsorption of water from renal tubules is increased. This in turn, increases ECF volume, blood volume and cardiac output, resulting in restoration of blood pressure.

THROUGH RENIN-ANGIOTENSIN:

Actions of Angiotensin II: When blood pressure and ECF volume decrease, kidneys' secretion of renin increases. Renin converts angiotensinogen which is produced in the liver into angiotensin I. Angiotensin-converting enzyme (ACE) found in the lungs metabolizes angiotensin I into angiotensin II. Angiotensin II acts in two ways to restore the blood pressure:

i. It causes constriction of arterioles in the body so that the peripheral resistance is increased and blood pressure rises. It also causes constriction of afferent arterioles in kidneys, so that glomerular filtration reduces. This results in retention of water and salts, increasing ECF volume to normal level. This in turn increases the blood pressure to normal level.

ii. Simultaneously, angiotensin II stimulates the adrenal cortex to secrete aldosterone. This hormone increases 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 normal level.

Together, angiotensin II and aldosterone work to raise blood pressure and blood volume.

Actions of Angiotensin III and Angiotensin IV

Like angiotensin II, the angiotensins III and IV also increase the blood pressure and stimulate adrenal cortex to secrete aldosterone.

2. (a). Pulmonary circulation: It is also called lesser circulation. Deoxygenated or venous blood leaves the right ventricles through the pulmonary artery to the lungs. Exchange of gas occurs between the blood and alveoli of the lungs at the pulmonary capillaries. Oxygenated or arterial blood then returns to the left atrium through the pulmonary vein.

(b). Circle of Willis: Circle of Willis or cerebral arterial circle or polygon of Willis is a ring of interconnecting arteries which are branches of basilar and internal carotid artery. They are located at the base of the brain around the optic chiasm and they supply the brain and surrounding structures. It is important because they offer possibility of alternate pathways for blood flow in case of occlusion in the normal route of blood supply to an area. It creates a redundancy for collateral circulation in cerebral circulation.

(c). Splanchnic circulation: or visceral circulation is composed of gastric, small intestinal, colonic, pancreatic, hepatic and splenic circulations arranged in parallel with one another. They are divided into three major portions:

i). Mesenteric circulation supplying blood to the gastro-intestinal tract.

ii). Splenic circulation supplying blood to the spleen.

iii). Hepatic circulation supplying blood to the liver.

The three major arteries that supply the splanchnic organs are the abdominal aorta, the coeliac artery and the superior and inferior mesenteric arteries. Unique feature of splanchnic circulation is that the blood from mesenteric bed and spleen forms a major amount of blood flowing to liver. Blood flows to liver from GI tract and spleen through portal system.

d). Coronary circulation: This is the circulation of blood in the blood vessels that supply the heart muscle (myocardium). The heart is highly metabolically active and has the highest oxygen consumption by mass of any organ. The demand for oxygen is met by the coronary circulation, which is responsible for delivering blood to the myocardium and represents approximately 5% of cardiac output. Coronary arteries supply oxygenated blood to the heart muscle, and the cardiac veins drain away the blood once it has been deoxygenated.

e). Cutaneous circulation: The cutaneous circulation is the circulation and blood supply to 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 in the skin will flow through arteriovenous anastomoses (AVAs) instead of capillaries. AVAs serve a role in temperature regulation. AVAs are low-resistance connections between the small arteries and small veins that supply and drain the skin. These allow shunt of blood directly in to the venous plexus of the skin, without passing through the capillaries. Since AVAs contain no capillary section, they are not involved in transport of nutrients to and from the tissues, but instead play a major role in 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 outflow to the cutaneous vessels, return temperature to its normal range.

3. CARDIOVASCULAR ADJUSTMENT THAT OCCURS DURING EXERCISE.

a). Blood: There is mild hypoxia which develops during exercise and it stimulates the juxtaglomerular apparatus to secrete erythropoietin. Erythropoietin stimulates the bone marrow which causes release of red blood cells. Increased carbon dioxide content in blood decreases the pH of blood.

b). Blood volume: more heat is being produced during exercise which leads to activation of thermoregulatory system. This then causes secretion of large amount of sweat which leads to fluid loss, reduced blood volume, hemoconcentration. Sometimes, severe exercise can lead to dehydration.

c). Heart rate: Heart rate increases linearly with severity and duration of exercise. It is slightly increased even before the onset of exercise due to the influence of cerebral cortex on the medullary cardiac center. This reduces the vagal tone. In moderate exercise, the heart rate increases to 180 beats/minute. In severe muscular exercise, it reaches 240 to 260 beats/minute. Increased heart rate during exercise is mainly because of vagal withdrawal. Increase in sympathetic tone also plays some role.

Increased heart rate during exercise is due to four factors:

i). Impulses from proprioceptors present in the exercising muscles act through higher centers, increasing the heart rate.

ii). Increase in carbon dioxide tension which acts through medullary centers.

iii). Rise in body temperature which acts on cardiac center through the hypothalamus. Increased temperature also stimulates SA Node directly.

iv). Circulating catecholamine which are secreted in large quantities during exercise also increase heart rate.

d). Cardiac output: Cardiac output increases up to 20L/minutes in moderate exercise and 35L/minutes in severe exercise. During exercise, cardiac output increases because of increase in heart rate and stroke volume. Heart rate increases because of vagal withdrawal. Stroke volume increases due to increased force of contraction. Because of vagal withdrawal, sympathetic activity increases leading to increase in rate and force of contraction. So, Increase in cardiac output is directly proportional to the increase in the amount of oxygen consumed during exercise.

e). Venous return: Venous return increases remarkably during exercise because of muscle pump, respiratory pump and splanchnic vasoconstriction.

f). Blood flow to skeletal muscles: There is a great increase in the amount of blood flowing to skeletal muscles during exercise. In resting condition, the blood supply to the skeletal muscles is 3 to 4 mL/100 g of the muscle/minute. It increases up to 60 to 80 mL in moderate exercise and up to 90 to 120 mL in severe exercise.

During muscular activity, when the muscles contract, stoppage of blood flow occurs. This is because of compression of blood vessels during contraction. And in between the contractions, the blood flow increases. Sometimes the blood supply to muscles starts increasing even during the preparation for exercise. It is due to the sympathetic activity. Sympathetic nerves cause vasodilatation in muscles. The sympathetic nerve fibers causing vasodilatation in skeletal muscle are called sympathetic cholinergic fibers since these fibers secrete acetylcholine instead of noradrenaline.

Several other factors also are responsible for the increase in blood flow to muscles during exercise. All such factors increase the amount of blood flow to muscles by means of dilatation of blood vessels of the

muscles. They include Hypercapnea, hypoxia, potassium ions, metabolites like lactic acid, rise in temperature, adrenaline secreted from adrenal medulla, increased sympathetic cholinergic activity.

g. Blood pressure: During moderate isotonic exercise, systolic pressure is increased. This is due to increase in heart rate and stroke volume. Diastolic pressure is not altered because peripheral resistance is not affected during moderate isotonic exercise.

In severe exercise involving isotonic muscular contraction, the systolic pressure increases enormously but the diastolic pressure decreases. Decrease in diastolic pressure is because of the decrease in peripheral resistance. Decrease in peripheral resistance is due to vasodilatation caused by metabolites. During exercise involving isometric contraction, the peripheral resistance increases. So, the diastolic pressure also increases along with systolic pressure.