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 Course: physiology

Level:200

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

Long-term regulation involves mainly the regulation of extracellular fluid volume by pressure natriuresis mechanisms residing in the kidney and by widespread actions of angiotensin II. 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 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.

2. Write short notes on the following:

a. Pulmonary circulation:

Pulmonary circulation transports oxygen-poor blood from the right ventricle to the lungs, where blood picks up a new blood supply. Then it returns the oxygen-rich blood to the left atrium, 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 veins.

b. Circle of Willis:

The circle of Willis is a junction of several important arteries at the bottom part of the brain. It helps blood flow from both the front and back sections of the brain. The circle of Willis gets its name from the physician Thomas Willis, who described this part of the anatomy in 1664. The circle of Willis is a part of the cerebral circulation and is composed of the following arteries: Internal carotid artery (left and right) Posterior cerebral artery (left and right) Posterior communicating artery (left and right).

c. Splanchnic circulation:

The term 'splanchnic circulation' describes the blood flow to the abdominal gastrointestinal organs including the stomach, liver, spleen, pancreas, small intestine, and large intestine. 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.

d. Coronary circulation:

Coronary circulation, 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 into the right atrium.

e. Cutaneous circulation:

The cutaneous circulation is the circulation and blood supply of the skin. Some of the circulating blood volume in the skin will flow through will flow through arteriovenous anastomoses (AVAs) instead of capillaries. Cutaneous circulation is also very important in homeostasis.

3. Discuss the cardiovascular adjustment that occurs during exercise?

During exercise, efficient delivery of oxygen to working skeletal and cardiac muscles is vital for maintenance of ATP production by aerobic mechanisms. The equine cardiovascular response to increased demand for oxygen delivery during exercise contributes largely to the over 35-fold increases in oxygen uptake that occur during submaximal exercise. Cardiac output during exercise increases greatly owing to the relatively high heart rates that are achieved during exercise. Heart rate increases proportionately with workload until heart rates close to maximal are attained. It is remarkable that exercise heart rates six to seven times resting values are not associated with a fall in stroke volume, which is maintained by splenic contraction, increased venous return, and increased myocardial contractibility. Despite the great changes in cardiac output, increases in blood pressure during exercise are maintained within relatively smaller limits, as both pulmonary and systemic vascular resistance to blood flow is reduced. Redistribution of blood flow to the working muscles during exercise also contributes greatly to the efficient delivery of oxygen to sites of greatest need. Higher work rates and oxygen uptake at submaximal heart rates after training imply an adaptation due to training that enables more efficient oxygen delivery to working muscle. Such an adaptation could be in either blood flow or arteriovenous oxygen content difference. Cardiac output during submaximal exercise does not increase after training, but studies using high-speed treadmills and measurement of cardiac output at maximal heart rates may reveal improvements in maximal oxygen uptake due to increased stroke volumes, as occurs in humans. Improvements in hemoglobin concentrations in blood during exercise after training are recognized, but at maximal exercise, hypoxemia may reduce arterial oxygen content. More effective redistribution of cardiac output to muscles by increased capillarization and more efficient oxygen diffusion to cells may also be an important means of increasing oxygen uptake after training.