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1. **Long-Term Regulation of Blood Pressure**

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.

1. **Pulmonary Circulation**: The pulmonary circulation is the portion of the circulatory system which carries deoxygenated blood away from the right ventricle, to the lungs, and returns oxygenated blood to the left atrium and ventricle of the heart. Deoxygenated blood leaves the heart, goes to the lungs, and then re-enters the heart; Deoxygenated blood leaves through the right ventricle through the pulmonary artery. From the right atrium, the blood is pumped through the tricuspid valve (or right atrioventricular valve), into the right ventricle. Blood is then pumped from the right ventricle through the pulmonary valve and into the main pulmonary artery.
2. **Circle of Willis**: The Circle of Willis is the joining area of several arteries at the bottom (inferior) side of the brain. At the Circle of Willis, the internal carotid arteries branch into smaller arteries that supply oxygenated blood to over 80% of the cerebrum
3. **Splanchnic Circulation**: The Splanchnic circulation describes the blood flow to the abdominal gastrointestinal organs including the stomach, liver, spleen, pancreas, small intestine, and large intestine. It comprises three major branches of the abdominal aorta; the coeliac artery; superior mesenteric artery (SMA); and inferior mesenteric artery (IMA) .
4. **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.
5. **Cutaneous Circulation**:The 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 to that of other tissues. Some of the circulating blood volume in the skin will flow through arteriovenous anastomoses (AVAs) instead of capillaries. AVAs serve a role in temperature regulation

3. **Cardiovascular Adjustment that occurs during Exercise**

1. **On Blood**: Mild hypoxia developed during exercise stimulates the secretion erythropoietin. It stimulates the bone marrow and causes release of red blood cells. Increased carbon dioxide content in blood decreases the pH of blood.
2. **On the Blood Volume**: More heat is produced during exercise and the thermo-regulatory sytem is activated. This in turn, causes secrection of large amount of sweat leading to:  
   a. Fluid loss  
   b. Reduces blood volume  
   c. Hemoconcentration  
   d. Severe exercise sometimes, lead to even dehydration
3. **On the Heart Rate**: Heart Rate increases during exercise. Even the thought of exercise or preparartion of exercise increases the heart rate. It is because of impulses from crebral cortex to medullary centers, which reduces vagal tone. In moderate exercise, the heart rate increases to 180 beats per minute. In severe muscular exercise, it reaches 240 to 260 beats per 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, which are present in the exercising muscles; these impulses act through higher centers and increase the heart rate. ii. Increased carbon dioxide tension, which acts through medullary centers. iii. Rise in body temperature, which acts on cardiac centers via hypothalamus, increased temperature, also stimulates SA node directly.
4. **On Cardiac Output**: Cardiac output increases up to 20L/min in moderate exercise and up to 35L/min during severe exercise. Increase in cardiac output is directly proportional to the increase in the amount of oxygen consumed during exercise. During exercise, the 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, symapathetic activity increases leading to increases in rate and force of contraction.
5. **On Venous Return**: Venous Return increases remarkedly during because of muscle pump; respiratory pump and splanchnic vasoconstriction.
6. **On 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 4mL/100g of the muscle per minute. It increases up to 60 to 80 mL in moderate exercise and up to 90 to 120 mL in severe exercise. During the muscular activity, stoppage of the blood flowoccurs when the muscles contract. It is because of compression of the blood vessels during contraction. And in between contractions, the blood flow increases. Sometimes, the blood supply to muscles starts increasing even during the preparation for rexercise. It is due to the sympathetic activity. Sympathetic nerves cause vasodilation in muscles. The sympathetic nerve fibers causing vasodilation in skeletal muscle are called sympathetic cholinergic fibers since these secrete acetylcholine instead of noradrenaline.
7. **On Blood Pressure**: During moderate isotonic exercise, the systolic pressure is increased. It 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 enormously increases but the diastolic pressure decreases. Decrease in diastolic pressure is because of the decreases in peripheral resistance. Decrease in peripheral resistance is due to vasodilation caused by metabolites. During exercise involving isometric contraction, the peripheral resistance increases. So, the diastolic pressure also increases along the systolic pressure.