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LONG TERM REGULATION OF MEAN ARTERIAL BLOOD PRESSURE

There are several physiological mechanisms that regulate blood pressure in the long term, the first and major one is the renin-angiotensin-aldosterone system (RAAS).

Renin-Angiotensin-Aldosterone System (RAAS)

Renin is a polypeptide produced by juxtaglomerular cells in the juxtaglomerular apparatus in the kidney. It is released in response to:

- Sympathetic stimulation
- Reduced NaCl delivery to distal convoluted tubule
- Decreased blood flow to kidney

This renin facilitates the conversion of angiotensin to angiotensin I which is then converted II using angiotensin-converting enzyme (ACE).

Angiotensin II is a potent vasoconstrictor which acts on the kidney to increase sodium reabsorption in the proximal convoluted tubule. Sodium reabsorption is via the sodium-hydrogen exchanger. Angiotensin II also promotes the release of aldosterone.

Aldosterone promotes salt and water retention by acting at the distal convoluted tubule to increase the expression of epithelial sodium channels. It also increases the activity of basolateral sodium-potassium ATP-ase increasing the electrochemical gradient for movement of sodium ions.

The more sodium collects in the kidney and water follows by osmosis, water excretion is reduced which in turn increases blood volume and thus blood pressure.

The second mechanism is the release of Anti Diuretic Hormone (ADH) from the hypothalamus in response to thirst or an increased plasma osmolarity. ADH acts to increase the permeability of the collecting duct to water. It also stimulates sodium reabsorption from the thick ascending limb of the loop of Henle. This increases water reabsorption in total thus increasing plasma volume and decreasing osmolarity.

Other factors are;

Atrial natriuretic peptide (ANP): this is synthesized and stored in cardiac myocytes to be released when atria are stretched indicating high blood pressure. It acts to promote sodium excretion. It dilates the afferent arteriole of the glomerulus increasing blood flow (GFR).

Prostaglandins: acts as local vasodilators to increase GFR and reduce sodium reabsorption. The also act to prevent excessive vasoconstriction triggered by the sympathetic system and RAAS.

2. PULMONARY CIRCULATION

Pulmonary circulation is the portion of the circulatory system that carries deoxygenated blood from the right ventricle to the lungs to be oxygenated and returns oxygenated blood to the left atrium of the heart. The required vessels are pulmonary artery and vein.

CIRCLE OF WILLIS

This is a ring of interconnecting arteries located at the base of the brain around the optic chiasm (partial crossing of the optic nerve), infundibulum of the pituitary stalk and hypothalamus. It provides blood to the brain and neighboring structures.

SPLANCHNIC CIRCULATION

Splanchnic circulation consists of blood supply to the gastrointestinal tract, liver, spleen and pancreas. It comprises two large capillary beds partially in series. The small splanchnic arterial branches supply the capillary beds, and the venous blood flow into the PV.

CORONARY CIRCULATION

This is the circulation of blood in the blood vessels that supply the heart muscle. Coronary arteries supply oxygenated blood to the heart muscle, and cardiac veins drain away the blood once has been deoxygenated.

CUTANEOUS CIRCULATION

This is circulation and blood supply of the skin.

3. CARDIOVASCULAR ADJUSTMENT DURING EXERCISE

A series of events occur in order to increase cardiac output and oxygen supply to muscles for better performance.

First, the chemoreceptors and baroreceptors detect changes in the body. This peripheral factors send messages to the medulla which stimulate the cardiac center in the brain. This initiates sympathetic response via accelerator thus increased impulse at the sino-atrial node which in turn increases the heart rate and stroke volume which lead to increased cardiac output. There'll also be increased blood pressure, contractility etc.