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PHYSIOLOGY ASSIGNMENT

DEPARTMENT: MBBS

1. DISCUSS THE LONG -TERM REGULATION OF MEAN ARTERIAL BLOOD PRESSURE?

It may take minutes to days to go into effect, it involves kidneys, which then can regulate your blood volume, by regulating blood volume, and you can regulate mean arterial blood pressure.

HOW IT TAKES PLACE

Renin-angiotensin system, and how it controls arterial pressure. In a situation where you have a decreased mean arterial pressure, so when the mean arterial pressure is lowered, it gets detected by the kidneys and when the kidneys detect this low mean arterial pressure they produce less filtrate, meaning you urinate less and it increases the blood volume and increases the blood volume. Another pathway is Baroreceptors detecting the low mean arterial pressure, and they develop a low frequency of action potentials which winds up causing a sympathetic response, the sympathetic response will then target the kidneys, the kidneys will then synthesize and secrete renin(an enzyme),and there is a formation of a hormone called angiotensin II ,this hormone targets the adrenal cortex, specifically the zona glomerulosa ,that causes the synthetic secretion of aldosterone (it increases sodium reabsorption),and absorption of sodium is followed by increased water absorption, which brings about increase in blood volume, and then

arterial pressure. The paraventricular nucleus (PVN) in the hypothalamus has major direct and indirect connections with the sympathetic outflow and there is now considerable evidence that tonic activation of the PVN sympathetic pathway contributes to the sustained level of RSNA that occurs in conditions such as heart failure and neurogenic hypertension. The tonic activity of PVN sympathetic neurons, in turn, depends upon the balance of excitatory and inhibitory inputs. A number of neurotransmitters and neuromodulators are involved in these tonic excitatory and inhibitory effects, including glutamate, GABA, angiotensin ii and nitric oxide. The dorsomedial hypothalamic nucleus (DMH) also exerts a powerful influence over sympathetic activity, including RSNA, through synapses with sympathetic nuclei in the medulla and, possibly, also other brain stem regions. The DMH sympathetic pathway is an important component of the phasic sympathoexcitatory responses associated with acute stress, but there is no evidence that it is an important component of the central pathways that produce long-term changes in arterial pressure.

2. A SHORT NOTE ON THE FOLLOWING

A. PULMONARY CIRCULATION: This is the portion of the circulatory system that carries deoxygenated blood away from the right ventricle, to the lungs and carries oxygenated blood to the left atrium and ventricle of the heart.

B. CIRCLE OF WILLIS: This is the joining of several arteries at the bottom side of the brain. At the circle of Willis, the internal carotid arteries branch into the smaller arteries that supply oxygenated blood to over 80% of the cerebrum.

C. SPLANCHNIC CIRCULATION: This is made up of the gastric, small intestinal, colonic, pancreatic, hepatic, and splenic circulations. They are arranged in parallel and fed by the celiac artery and the superior and inferior mesenteric arteries.

D. CORONARY CIRCULATION: This 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.

E. CUTANEOUS CIRCULATION: this 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

. 3. DISCUSS THE CARDIOVASCULAR ADJUSTMENT THAT OCCURS DURING EXERCISE

1. Increased cardiac output

Increased pumping capacity of the heart enhancing delivery of oxygen and fuel to muscles that are working.

. 2. Increased muscle blood flow

Blood vessels in muscles dilate, increasing local blood flow

3. Decreased blood flow to kidney, liver and gut

Redirects/shunts blood flow to working muscles Cardiac output is the amount of blood pumped from the heart in a minute.

Cardiac output (Q) = Heart rate x stroke volume.

To increase the cardiac output you can increase either heart rate or stroke volume but in this case of exercises we increase the both heart rate and stroke volume.

Basic ways to increase heart rate during exercises

1. Reduction of parasympathetic nervous system activity

2. Increase in sympathetic Nervous system activity

3. Increase of circulating adrenaline / Epinephrine (this response also occurs when the body is frightened, it preparing the body for movement)

4. Increase in stroke volume, it increases with exercise intensity and also leads to increase in cardiac output)

Cardiovascular factors responsible include ($V_{O_2} = \text{cardiac output} \times (a-v) O_2$ difference)

Where V_{O_2} = oxygen consumption

Where a = arterial oxygen con.

Where v = venous oxygen conc.

Where (a-v) = arteriovenous oxygen difference NB: (a-v) gives the amount of oxygen taken up and utilized by muscles for ATP production in mitochondria The greater the exercise intensity, the greater the extraction of oxygen from the blood by muscle mitochondria Two major factors responsible for the increase in

arteriovenous oxygen difference are : 1 Greater rate of utilization. 2. Greater rate of oxygen delivery accomplished by increase in local muscle blood flow.