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1) **Long Term Regulation Of Blood Pressure**

There are several physiological mechanisms that regulate blood pressure in the long-term and they are as follows:

I. 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.

II. 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.

III. Atrial natriuretic peptide (ANP)

- 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.

2)

a) Pulmonary Circulation: Pulmonary circulation is the system of transportation that shunts de-oxygenated blood from the heart to the

lungs to be re-saturated with oxygen before being dispersed into systemic circulation. The term pulmonary circulation is readily paired and contrasted with the systemic circulation. The vessels of the pulmonary circulation are the pulmonary arteries and the pulmonary veins.

b) Circle of Willis: The Circle of Willis is the joining area of several arteries at the bottom (inferior) side of the brain. The circle of Willis encircles the stalk of the pituitary gland and provides important communications between the blood supply of the forebrain and hindbrain (i.e. between the internal carotid and vertebro-basilar systems following obliteration of primitive embryonic connections). Although a complete circle of Willis is present in some individuals, it is rarely seen radiographically in its entirety; anatomical variations are very common and a well-developed communication between each of its parts is identified in less than half of the population.

c) Splanchnic Circulation: The splanchnic circulation receives over 25% of the cardiac output and contains a similar percentage of the total blood volume under normal conditions. Thus the splanchnic circulation can act as a site of regulation of distribution of cardiac output and also as a blood reservoir. .The splanchnic circulation consists of the blood supply to the gastrointestinal tract, liver, spleen, and pancreas. It consists of two large capillary beds partially in series. The small splanchnic arterial branches supply the capillary beds, and then the efferent venous blood flows into the PV.

d) 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.

e) Cutaneous Circulation: 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.

3) **Cardiovascular Adjustment That Occurs During Exercise**

Clearly, adjustments in the cardiovascular system are critical when engaging in aerobic activities but they are also required for strength training as well. The three major adjustments made by the cardiovascular system during exercise include:

I. An increase in cardiac output or the pumping capacity of the heart, designed to enhance the delivery of oxygen and fuel to the working muscles.
II. An increase in local blood flow to the working muscles,
III. A decrease in blood flow to other organs such as the kidneys, liver and stomach, thereby redirecting blood flow to the working muscles.

Cardiac output is the amount of blood pumped from the heart in one minute, generally measured in liters per minute. It's simply calculated by heart rate, in beats per minute, times stroke volume, or the amount of blood ejected by the heart with each beat. Thus in order to increase cardiac output, we can increase

heart rate, stroke volume, or as it is the case during exercise, we increase both. Let's examine the basic ways in which we can increase heart rate during exercise. First, there is a reduction or withdrawal of the parasympathetic nerve activity to the heart. As parasympathetic nerve activity causes a lowering of heart rate, its withdrawal will actually result in an increase in heart rate. Second, an increase in sympathetic nerve activity to the heart will directly cause an increase in heart rate. This increase in sympathetic nerve activity will be a function of the exercise intensity. Lastly, an increase in the hormone epinephrine or adrenaline circulating in the blood will also stimulate an increase in heart rate. These adjustments are also part of the fight or flight response which you experience when nervous or frightened. You may actually feel your heart pounding in your chest. This response is preparing the body for movement. This figure demonstrates how densely the heart is innervated with sympathetic nerve fibers. Thus, heart rate can be rapidly increased during exercise as a result of an increase in sympathetic nerve activity. Shown here is the typical heart rate response during a graded exercise test to max. Heart rate increases linearly until approaching one's maximal heart rate. This will contribute to an increase in cardiac output during the course of the test. Notice that endurance training results in lower, resting, and submaximal heart rates with no change in maximal heart rate. I will discuss this in more detail in the next video. An increase in stroke volume also contributes to an increase in cardiac output during exercise. A more forceful contraction of the ventricles of the heart, resulting in more blood being pumped per beat, can be accomplished by both increasing sympathetic nerve activity and circulating epinephrine. Shown here is the clear effect that an increase in sympathetic

nerve stimulation has on stroke volume. For a given amount of blood in the ventricles, sympathetic stimulation results in a more forceful contraction, you'll get a significant increase in stroke volume. Here is the typical stroke volume response during a graded exercise test to max. Stroke volume increases linearly at the onset of the test, but can plateau at submaximal workloads. Again, please notice that endurance training produces significantly greater stroke volumes both at rest and throughout the duration of the test. Including a large increase in maximal stroke volume. The heart becomes a more forceful pump after endurance training, this will be discussed in more detail in the next video. Taken together, the increases in both heart rate and stroke volume result in a linear increase in cardiac output during the course of a graded exercise test to exhausture. As mentioned in the calorimetry video, oxygen consumption increases linearly during a graded exercise test until VO2 max is reached. Now let's break down the cardiovascular factors responsible for this observation. The place to begin is with the Fick equation which defines the the relationship between oxygen consumption with that for cardiac output and the arterial venous oxygen difference. As indicated here, whether measured at rest or during submaximal and maximal exercise, oxygen consumption is equal to one's cardiac output times their arteriovenous oxygen difference. As we have already discussed the cardiac output component here today, let's turn our attention to the arteriovenous oxygen difference. Basically, the arteriovenous oxygen difference is the measure of oxygen uptake and utilization by a cell, in our case a muscle cell. If we know the content of oxygen in an artery delivering oxygen to a muscle and we know the content of oxygen leaving the muscle on the venous side, the difference must

be the amount of oxygen taken up and utilized by muscle for ATP production in mitochondria. This measurement is abbreviated as (a-v) $O_2$ Difference, with the little a representing the arterial oxygen content, and the little v representing the venous oxygen content. Shown here is the arteriovenous oxygen difference during a graded exercise test of VO2 max. As can be seen, the arteriovenous oxygen difference increases progressively with increasing exercise intensity. This indicates that the greater the exercise intensity, the greater extraction of oxygen from the blood and utilization by muscle mitochondria. The two main factors responsible for the increase in arteriovenous oxygen difference are a greater rate of oxygen delivery, accomplished by in an increase in local muscle blood flow, and a greater rate of oxygen utilization, as mitochondria consumed greater amounts of oxygen for ATP production at higher workloads. Thus, as per the Fick equation, oxygen consumption can increase linearly as a function of exercise intensity due to the contributions of both an increasing cardiac output as well as an increasing arteriovenous oxygen difference until VO2 max is achieved. In summary, cardiac output is a function of heart rate and stroke volume. Both factors increase in relation to exercise intensity and are regulated by both the sympathetic nervous system as well as circulating epinephrine. Oxygen consumption is the function of cardiac output and the arterial venous oxygen difference. The arteriovenous oxygen difference is dependent upon both the rate of oxygen delivery as well as the rate of mitochondrial oxygen utilization.