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Department: Medicine and surgery

Level: 200 level

Course: Physiology

1.Long term regulation of mean arterial blood pressure.

Blood pressure is a measure of how well our cardiovascular system is functioning. There is a range of normal blood pressures that we consider as acceptable (120/80mmHg). When blood pressure is outside of this normal range of values, people have problems and it can be either short or long term.

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

Renin is a peptide hormone released by the granular cells of the juxtaglomerular apparatus in the kidney. It is released in response to:

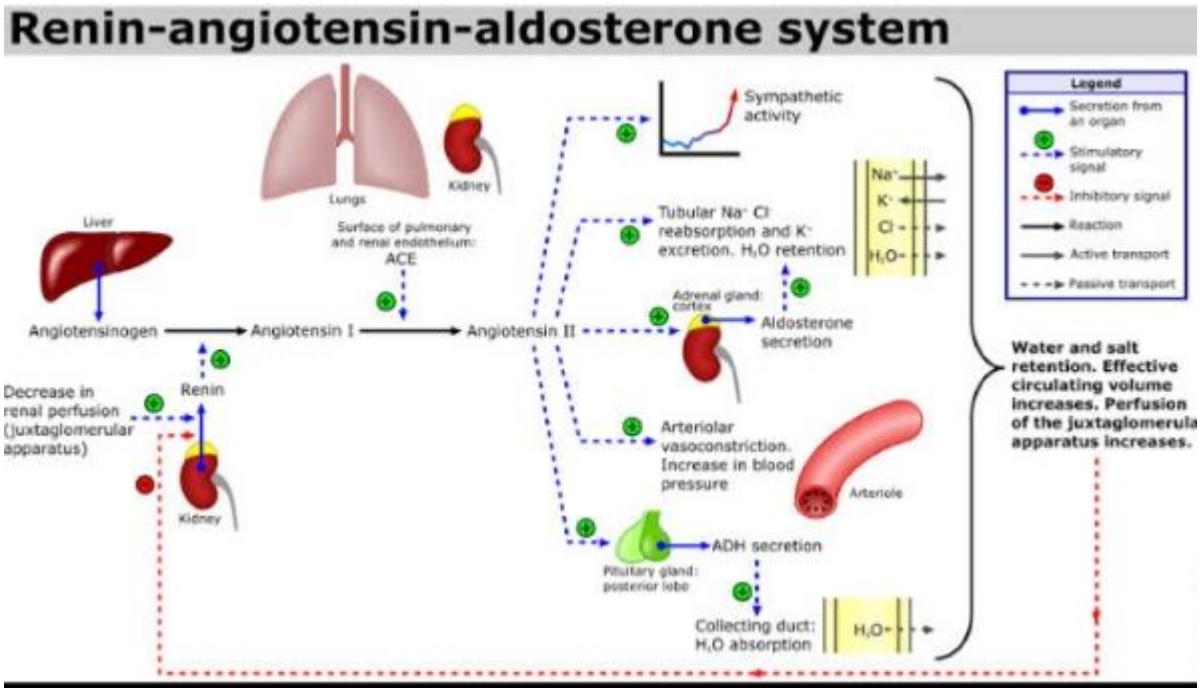
-Sympathetic stimulation

-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:

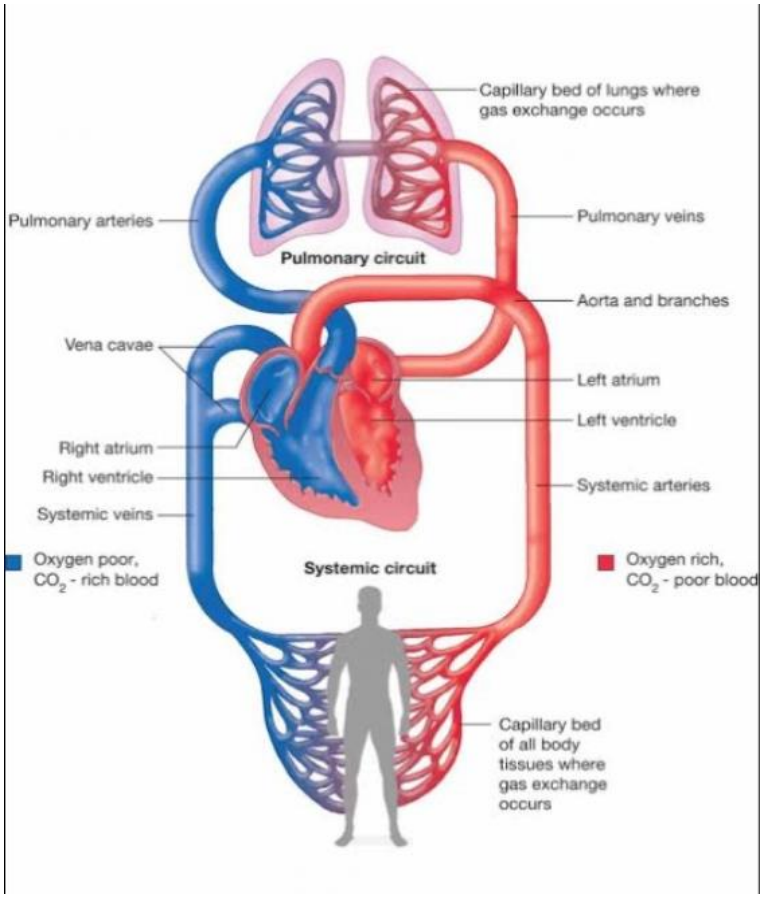
Atrial natriuretic peptide (ANP) is synthesized and stored in cardiac myocytes. It is released when the atria are stretched, indicating of high blood pressure. ANP acts to promote sodium excretion. It dilates the afferent arteriole of the glomerulus, increasing blood flow (GFR).

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.Short notes on the following;

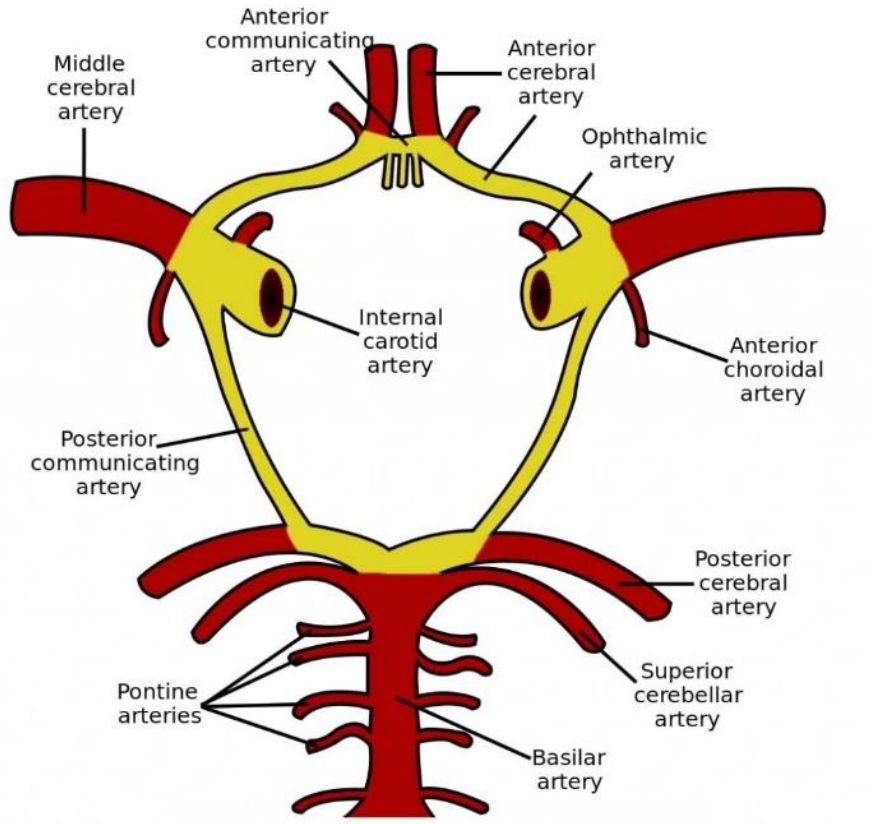
1.**Pulmonary circulation**

Pulmonary circulation refers to the portion of the cardiovascular system which carries deoxygenated blood away from the heart, towards the alveoli of the lungs to undergo gas exchange, and then returns oxygenated blood back to the heart. Deoxygenated blood leaves the right ventricle of the heart through the pulmonary valve and enters



the pulmonary trunk. This divides into the right and left pulmonary arteries. In the lungs the arteries divide further into very fine capillaries at the alveoli, allowing gas exchange to take place. Oxygen diffuses from the alveoli into the pulmonary capillaries while carbon dioxide diffuses from the capillaries into the alveoli. This newly oxygenated blood leaves the lungs through the pulmonary veins to the left atrium of the heart, completing the pulmonary cycle. The blood is then distributed around the body via the systemic circulation.

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. The terminal branches of the vertebral and internal carotid arteries all anastomose to form a circular blood vessel, called the Circle of Willis. There are three main (paired) constituents of the Circle of Willis:



Anterior cerebral arteries – terminal branches of the internal carotid arteries.

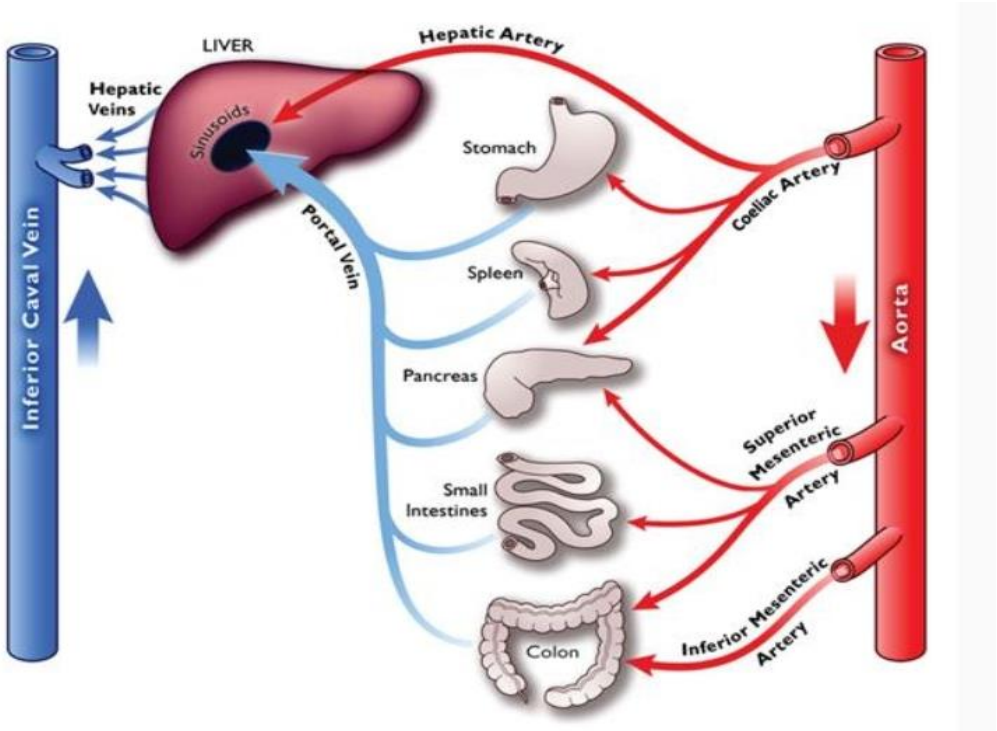
Internal carotid arteries – located immediately proximal to the origin of the middle cerebral arteries.

Posterior cerebral arteries – terminal branches of the vertebral arteries. To complete the circle, two 'connecting vessels' are also present:

Anterior communicating artery – connects the two anterior cerebral arteries.

Posterior communicating artery – branch of the internal carotid, this artery connects the ICA to the posterior cerebral artery.

3. **Splanchnic circulation**: 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 term

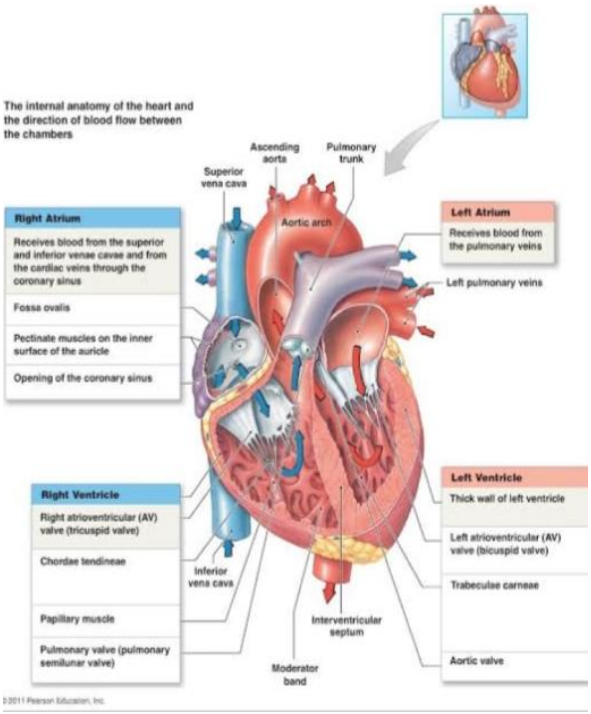


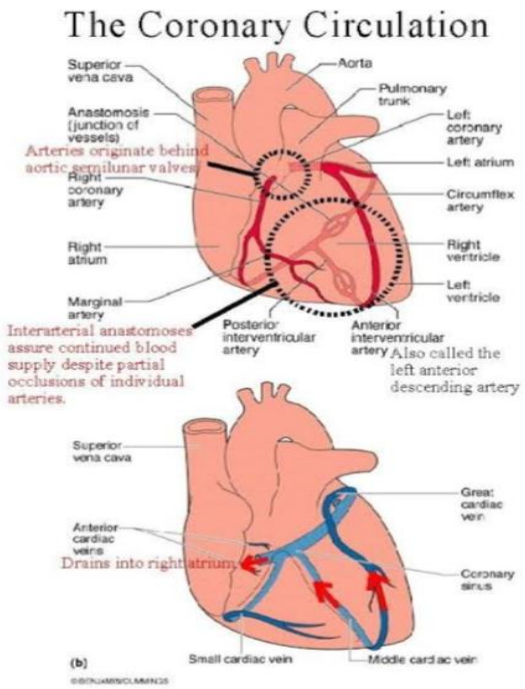
'splanchnic circulation' describes the blood flow to the abdominal gastrointestinal organs including the stomach, liver, spleen, pancreas,

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). The hepatic portal circulation delivers the majority of the blood flow to the liver.

Under physiological conditions, blood flow in the splanchnic circulation is controlled via intrinsic (myogenic and metabolic) and extrinsic (autonomic and humoral) mechanisms.
The splanchnic bed forms an important circulatory reservoir, which can be mobilized during periods of physiological stress.
Disorders of the splanchnic circulation may contribute to the multi-organ dysfunction syndrome and vice versa.
A number of techniques used in anesthesia and critical care influence the distribution of blood flow in the splanchnic circulation. The splanchnic circulation is a complex system. A number of important functions depend on its normal operation, including digestion and absorption within the gut, maintenance of the mucosal barrier, and successful healing of surgical anastomoses, but we have little quantitative information about its physiology because routine measurement in humans is so difficult.
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. 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.

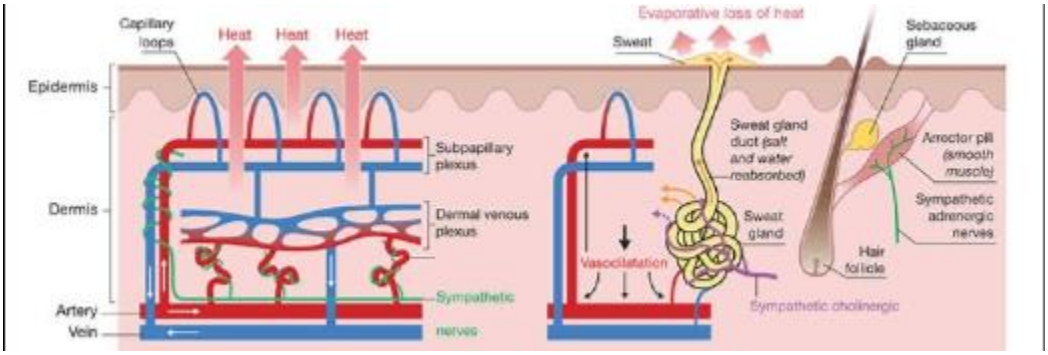




Coronary circulation, part of the systemic circulatory system that supplies blood to and provides drainage from the tissues of the heart. In the human heart, two coronary arteries arise from the aorta just beyond the semilunar valves; during diastole, the increased aortic pressure above the valves forces 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. The heart normally extracts 70 to 75 percent of the available oxygen from the blood in coronary circulation, which is much more than the amount extracted by other organs from their circulations—e.g., 40 percent by resting skeletal muscle and 20 percent by the liver. Obstruction of a coronary artery, depriving the heart tissue of oxygen-rich blood, leads

to death of part of the heart muscle (myocardial infarction) in severe cases, and total heart failure and death may ensue.

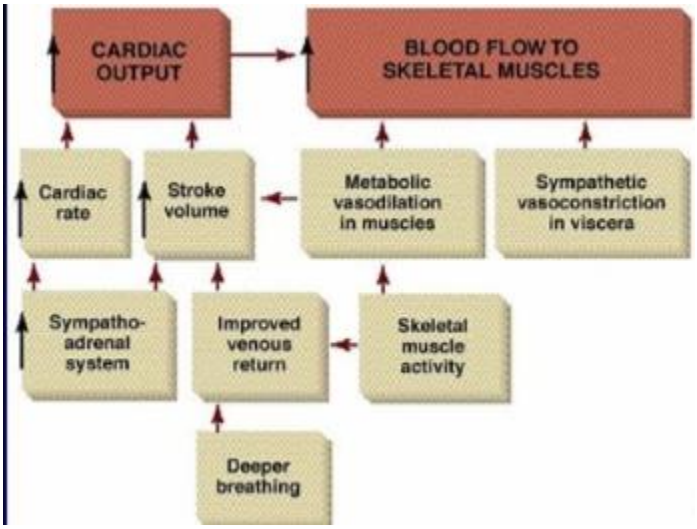
5. **Cutaneous circulation:** The cutaneous circulation is the circulation and blood supply of the skin. The skin is not a very metabolically active tissue and has relatively small energy requirements, so its blood supply is different to that of other tissues. Some of the circulating blood volume in the skin will flow through arteriovenous anastomoses (AVAs) instead of capillaries. AVAs serve a role in temperature regulation. There are different adaptations of the cutaneous circulation, and it has a role in body temperature control. Arteriovenous Anastomosis are low-resistance connections between the small arteries and small veins that supply and drain the skin. These allow the shunt of blood directly into the venous plexus of the skin, without it passing through capillaries. Since AVAs contain no capillary section, they are not involved in transport of nutrients to/from the tissues, but instead play a major role in temperature regulation.

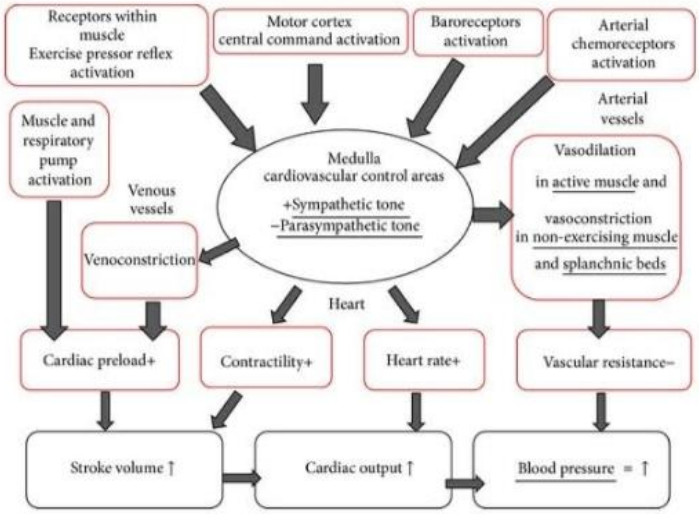


3. Discussion the cardiovascular adjustment that occur during exercise.
The three major adjustments made by the cardiovascular system during exercise include;

1. Increase in cardiac output or the pumping capacity of the heart, designed to enhance the delivery of oxygen and fuel to the working muscles.

2. Increase in local blood flow to the working muscles.
3. 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. Heart rate can be rapidly increased during exercise as a result of an increase in sympathetic nerve activity. 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. 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. 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. 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 exhauster. Oxygen consumption increases linearly during a graded exercise test until VO2 max is reached. The Fick equation defines the relationship between oxygen consumption with that for cardiac output and the arterial venous oxygen difference. Whether measured at rest or during submaximal and maximal exercise, oxygen consumption is equal to one's cardiac output times their 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. 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.