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1. Systemic Arterial Pressure - Long-term Regulation:

While changes to the Systemic Vascular Resistance (SVR) can transiently affect the systemic arterial pressure, arterial pressures tend to return to their original baseline within hours even if the changes to SVR are maintained. A variety of empirical studies have demonstrated that long-term control of the systemic arterial pressure over timescales of days, weeks, and months is principally regulated by the kidneys and is not dependent on changes to the systemic vasculature. The capacity of the kidneys to control arterial pressure depends on their ability to modify the extracellular fluid (ECF) volume which in a healthy individual determines the total blood volume. We first discuss how kidneys can control the extracellular fluid volume and subsequently describe how ECF volume is connected to systemic arterial pressures in healthy and pathologic contexts. The concepts discussed here heavily depend on a sound understanding of renal physiology; consequently, it may be worthwhile to revisit this page after reading through the Renal Physiology section.

Renal Volume Regulation

The kidneys respond to changes in systemic arterial pressure by modifying their urinary excretion of sodium and water. When arterial pressures are elevated, renal urinary excretion of sodium and water increases; conversely, when arterial pressures are deficient, renal urinary excretion of sodium and water decreases. The mechanisms which connect changes in arterial pressures to renal urinary excretion of salt and water are described more fully in ECF volume regulation and principally rely on mechanisms pressure natriuresis and the RAAS System. Nevertheless, this relationship between systemic arterial pressures and renal urinary excretion is largely independent of the SVR; consequently, whether or not the SVR is high or low, the kidneys will respond as described above by matching their urinary excretion to the effective systemic arterial pressure

As described more fully in ECF volume regulation the capacity of the kidneys to regulate urinary salt and water excretion allows these organs to regulate the total ECF volume which, as discussed below, is a major determinant of systemic arterial pressure in healthy individuals. Taken together, the relationship between arterial pressure, renal salt and water excretion, and ECF volume resembles a negative feedback control circuit in which changes to arterial pressure modulate renal sodium and water excretion which in turn affect ECF volume and thus modulate arterial pressure. Once again, this negative feedback appears to act completely independently of the SVR and explains why changes to the SVR can only affect arterial pressures transiently.

While a rapid increase in SVR will immediately boost the blood pressure, the kidneys will respond by progressively excreting salt and water, thus reducing the ECF volume and thus causing a slow decline in arterial pressure. Conversely, while a rapid decrease in SVR will immediately reduce the blood pressure, the kidneys will respond by retaining more salt and water than that ingested, thus increasing the ECF volume and thus causing a slow increase in the arterial pressure

ECF Volume and Arterial Pressure

Overview

In healthy individuals the total volume of the extracellular fluid is proportional to the systemic arterial pressure. Although this may appear to be an intuitive relationship, the physiological mechanism by which these two variables are linked is rather complex and is described below. However, in a variety of disease states the connection between ECF Volume and arterial pressure is deranged which can lead to increases in ECF volume without proportional enhancements of systemic arterial pressure.

Healthy State

In healthy individuals, increases in ECF volume result in a proportional increase in the total blood volume. As described in our discussion of the vascular function curve, an increase in total blood volume will enhance the "Mean Systemic Pressure" which in turn increases the venous return and thus the cardiac preload. Courtesy of the Frank-Starling Relationship, increased preload on the heart will enhance the cardiac output. Finally, as discussed in systemic arterial pressure regulation, an increased cardiac output will boost the systemic arterial pressure so long as the SVR remains constant. In this way, an increase in ECF volume results in an increase in the arterial pressure; conversely, a decrease in ECF volume will yield a decline in arterial pressure.

Diseased State

In certain disease states, increases in ECF volume does not result in proportional increases in the total blood volume. This occurs in contexts of deranged Starling Forces in which fluid leaks out of the vasculature and thus does not contribute to the total blood volume but instead contributes to states of generalized edema, peripheral edema, or ascites. In such cases, the renal mechanisms of long-term arterial pressure regulation in fact exacerbate these edematous states. This occurs because the kidneys continue to resorb salt and water in an attempt to boost the systemic arterial pressure; however, instead of contributing to correcting the deficient arterial pressure, the additional fluid volume simply ends up in the interstitial fluid and thus aggravates the edema. Frequently, such edematous states are corrected by using diuretics, which modulate renal physiology in such a way that the kidneys excrete large volumes of salt and water, thus decreasing the ineffective additional ECF volume and correcting the edema

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I. Pulmonary Circulation:

This is a system of blood vessels that forms a closed circuit between the heart and the lungs, as distinguished from the system circulation between the heart and all other body tissues. Deoxygenated blood from the lower half of the body enters the heart from the inferior vena cava while deoxygenated blood from the upper body is delivered to the heart via the superior vena cava. Both the superior vena cava and inferior vena cava empty blood into the right atrium. Blood flows through the tricuspid valve into the right ventricle. It then flows through the pulmonic valve into the pulmonary artery before being delivered to the lungs. While in the lungs, blood diverged into the numerous pulmonary capillaries where it releases carbon dioxide and is replenished with oxygen. Once fully saturated with oxygen, the blood is transported via pulmonary vein into the left atrium which pumps blood through the mitral valve and into the left ventricle. With a powerful contraction, the left ventricle expels oxygen-rich blood through the aortic valve and into the aorta: This is the beginning of systemic circulation.

II. Circle of Willis:

This is a ring of interconnecting arteries located at the base of the brain around the optic chiasm or chiasma (partial crossing of the optic nerve - CNII; this crossing is important for binocular vision), infundibulum of the pituitary stalk and the hypothalamus. This arterial ring provides blood to the brain and neighbouring structures.

III. Splanchnic Circulation:

The splanchnic circulation is composed of gastric, small intestinal, colonic, pancreatic, hepatic, and splenic circulations, arranged in parallel with one another. The three major arteries that supply the splanchnic organs, cellac and superior and inferior mesenteric, give rise to smaller arteries that anastomose extensively. The circulation of some splanchnic organs is complicated by the existence of an intramural circulation. Redistribution of total blood flow between intramural vascular circuits may be as important as total blood flow. Numerous extrinsic and intrinsic factors influence the splanchnic circulation. Extrinsic factors include general hemodynamic conditions of the cardiovascular system, autonomic nervous system, and circulating neurohumoral agents. Intrinsic mechanisms include special properties of the vasculature, local metabolites, intrinsic nerves, paracrine substances, and local hormones. The existence of a multiplicity of regulatory mechanisms provides overlapping controls and restricts radical changes in tissue perfusion.

IV. 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. This is because the rest of the body,

and most especially the brain, needs a steady supply of oxygenated blood that is free of all but the slightest interruptions, the heart is required to function continuously. Making it a circulation of major importance, not only to its tissue but to the entire body and even the level of consciousness of the brain. Any interruptions of coronary circulation quickly causes heart attack.

V. 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. Some of the circulating blood volume in the skin will flow through arteriovenous anastomosis (AVAs) instead of capillaries. AVAs serve a role in temperature regulation.

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 one, 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. Two, an increase in local blood flow to the working muscles, and three 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. Thus, 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. 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. Stroke volume increases linearly at the onset of the test, but can plateau at submaximal workloads. 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. Oxygen consumption increases linearly during a graded exercise test until VO2 max is reached. 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. 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. 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)O2 Difference, with the little a representing the arterial oxygen content, and the little v representing the venous oxygen content. 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.