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Matric no: 18/mhs01/145

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Course: physiology

**ASSIGNMENT ON CARDIOVASCULAR PHYSIOLOGY**

1. Long term regulation of mean arterial pressure

 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 (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.

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 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.

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.

1. 2a. Pulmonary circulation

 The pulmonary circulation is the portion of the circulatory system which carries deoxygenated blood away from the right ventricle, to the lungs, and returns oxygenated blood to the left atrium and ventricle of the heart.[1] 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.

A separate system known as the bronchial circulation supplies oxygenated blood to the tissue of the larger airways of the lungs.

Deoxygenated blood leaves the heart, goes to the lungs, and then re-enters the heart; Deoxygenated blood leaves through the right ventricle through the pulmonary artery. From the right atrium, the blood is pumped through the tricuspid valve (or right atrioventricular valve), into the right ventricle. Blood is then pumped from the right ventricle through the pulmonary valve and into the main pulmonary artery.

2b. Circle of willis

 The circle of Willis (also called Willis' circle, loop of Willis, cerebral arterial circle, and Willis polygon) is a circulatory anastomosis that supplies blood to the brain and surrounding structures. It is named after Thomas Willis (1621–1675), an English physician.

The circle of Willis is a part of the cerebral circulation and is composed of the following arteries:

* Anterior cerebral artery (left and right)
* Anterior communicating artery
* Internal carotid artery (left and right)
* Posterior cerebral artery (left and right)
* Posterior communicating artery (left and right)

The middle cerebral arteries, supplying the brain, are not considered part of the circle of Willis.

Origin of arteries

The left and right internal carotid arteries arise from the left and right common carotid arteries.

The posterior communicating artery is given off as a branch of the internal carotid artery just before it divides into its terminal branches - the anterior and middle cerebral arteries. The anterior cerebral artery forms the anterolateral portion of the circle of Willis, while the middle cerebral artery does not contribute to the circle.

The right and left posterior cerebral arteries arise from the basilar artery, which is formed by the left and right vertebral arteries. The vertebral arteries arise from the subclavian arteries.

The anterior communicating artery connects the two anterior cerebral arteries and could be said to arise from either the left or right side.

All arteries involved give off cortical and central branches. The central branches supply the interior of the circle of Willis, more specifically, the Interpeduncular fossa. The cortical branches are named for the area they supply. Since they do not directly affect the circle of Willis, they are not dealt with here.

2c. Splanchnic circulation

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

Coeliac artery

The coeliac artery is the first major division of the abdominal aorta, branching at T12 in a horizontal direction ∼1.25 cm in length. It shows three main divisions such as the left gastric artery, common hepatic artery, and splenic artery and is the primary blood supply to the stomach, upper duodenum, spleen, and pancreas.

Superior mesenteric artery

The SMA arises from the abdominal aorta anteriorly at L1, usually 1 cm inferior to the coeliac artery. The five major divisions of the SMA are the inferior pancreaticoduodenal artery, intestinal arteries, ileocolic, right colic, and middle colic arteries. The SMA supplies the lower part of the duodenum, jejunum, ileum, caecum, appendix, ascending colon, and two-thirds of the transverse colon. It is the largest of the splanchnic arterial vessels delivering >10% of the cardiac output and therefore has significant implications for embolic mesenteric ischaemia.

Inferior mesenteric artery

The IMA branches anteriorly from the abdominal aorta at L3, midway between the renal arteries and the iliac bifurcation. The main branches of the IMA are the left colic artery, the sigmoid branches, and the superior rectal artery. It forms a watershed with the middle colic artery and supplies blood to the final third of the transverse colon, descending colon, and upper rectum.

Physiology

Resting splanchnic blood flow (SBF) is typically 30 ml min−1 100 g−1 of tissue, which equates to 25–30% of the cardiac output. This may decrease to <10 ml min−1 100 g−1 in low cardiac output states or peak locally at 250 ml min−1 100 g−1 after a meal. The splanchnic circulation must therefore be highly adaptive. The mechanisms of physiological regulation of SBF are complex but the academic debate focuses primarily on three circulatory determinants: intrinsic (local metabolic vs myogenic), extrinsic (autonomic nervous system), and humoral (local or circulating vasoactive substances).

2d. 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. 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. Therefore its circulation is of major importance not only to its own tissues but to the entire body and even the level of consciousness of the brain from moment to moment. Interruptions of coronary circulation quickly cause heart attacks (myocardial infarctions), in which the heart muscle is damaged by oxygen starvation. Such interruptions are usually caused by ischemic heart disease (coronary artery disease) and sometimes by embolism from other causes like obstruction in blood flow through vessels.

Coronary arteries supply blood to the myocardium and other components of the heart. Two coronary arteries originate from the left side of the heart at the beginning (root) of the aorta, just after the aorta exits the left ventricle. There are three aortic sinuses (dilations) in the wall of the aorta just superior to the aortic semilunar valve. Two of these, the left posterior aortic sinus and anterior aortic sinus, give rise to the left and right coronary arteries, respectively. The third sinus, the right posterior aortic sinus, typically does not give rise to a vessel. Coronary vessel branches that remain on the surface of the heart and follow the sulci of the heart are called epicardial coronary arteries.

The left coronary artery distributes blood to the left side of the heart, the left atrium and ventricle, and the interventricular septum. The circumflex artery arises from the left coronary artery and follows the coronary sulcus to the left. Eventually, it will fuse with the small branches of the right coronary artery. The larger anterior interventricular artery, also known as the left anterior descending artery (LAD), is the second major branch arising from the left coronary artery. It follows the anterior interventricular sulcus around the pulmonary trunk. Along the way it gives rise to numerous smaller branches that interconnect with the branches of the posterior interventricular artery, forming anastomoses. An anastomosis is an area where vessels unite to form interconnections that normally allow blood to circulate to a region even if there may be partial blockage in another branch. The anastomoses in the heart are very small. Therefore, this ability is somewhat restricted in the heart so a coronary artery blockage often results in myocardial infarction causing death of the cells supplied by the particular vessel.

The right coronary artery proceeds along the coronary sulcus and distributes blood to the right atrium, portions of both ventricles, and the heart conduction system. Normally, one or more marginal arteries arise from the right coronary artery inferior to the right atrium. The marginal arteries supply blood to the superficial portions of the right ventricle. On the posterior surface of the heart, the right coronary artery gives rise to the posterior interventricular artery, also known as the posterior descending artery. It runs along the posterior portion of the interventricular sulcus toward the apex of the heart, giving rise to branches that supply the interventricular septum and portions of both ventricles.

2e. 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 will flow through arteriovenous anastomoses (AVAs) instead of capillaries. AVAs serve a role in temperature regulation. In this article we shall consider the different adaptations of the cutaneous circulation, and its role in body temperature control.

Arteriovenous Anastomoses

AVAs 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.

Temperature Regulation

The skin is the body’s main heat dissipating surface: the amount of blood flow to the skin determines the degree of heat loss and therefore the core body temperature. The blood flow through AVAs is heavily influenced by the sympathetic nervous system. At rest, the sympathetic nervous system dominates and acts to constrict AVAs.

Any changes in core temperature are detected by the thermoregulatory centre in the hypothalamus. It regulates temperature by altering the level of sympathetic outflow to the cutaneous vessels, to return temperature to its normal range:

In high core temperatures:

* Sympathetic innervation is decreased, reducing the vasomotor tone in the AVAs.
* More blood flows through the AVAs and reaches the venous plexus (close to the surface of the skin), increasing heat loss to reduce core temperature.

In low core temperatures:

* Sympathetic innervation is increased, increasing the vasomotor tone in the AVAs.
* Less blood flows to the apical skin (of nose, lips, ears, hands and feet), reducing heat loss to increase the core temperature
1. **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

1. 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.
2. An increase in local blood flow to the working muscles,
3. 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.

 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. The heart becomes a more forceful pump after endurance training. 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. 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)O2 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.