NAME:Ahuchaogu Nnaemeka Melvin

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1) LONG-TERM REGULATION OF MEAN ARTERIAL BLOOD 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 a ng I of ending on getting enzyme (ACE).

Angiotensin II is a potent vasoconstrictor. It acts directly on the kidney to increase sodium reabsorption in the proxima convoluted tubule. Sodium is reabsorped via the sodium-hydrogen exchanger.

Angiotensin II also promotes release of aldosterone.

ACE also breaks down a substance called bradkynin 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 u potassium ATP-ase, thus increasing the electrochemical gradient for movement of sodium ions. More sodium collects in the kidney tissues 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 the 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 natiuretic peptides. These includes:

-Atrial natiuretic peptide (ANP) is synthesized and stored in cardiac myocytes. It is released when the atria are stretched, indicating 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 acts as local vasidilators to increase GFR and reduce sodium reabsorption. They also act to prevent excessive ga so construction triggered by the sympathetic nervous and renin-angiotensin-aldosterone systems.Clinical Relevance -Hypertension

Hypertension is defined as a sustained increase in blood pressure. It may be primary (of an unknown cause) or secondary to another condition such as chronic renal disease or crushing's syndrome. Hypertension causes damage to the walls of blood vessels, making them weaker. This leads to a number of pathologies including atherosclerosis, thromboembolism (progressing to MI or stroke) and aneurysms. Hypertension also damages the heart itself by increasing the after load of the heart. The heart is pumping against greater resistance, leading to left ventricular hypertrophy. This increases the risk of heart failure in the future. Hypertrophy of the cardiac muscle also increases the heart's oxygen demand, predisposing to myocardial ischemia and ultimately angina.

2a) 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. 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 and inferior vena cava empty blood into the right atrium. Blood flows the tricuspid valve into the right ventricle. It then flows through the pulmonary valve into the pulmonary artery before being delivered to the lungs. While in the lungs, blood diverges into the numerous pulmonary capillaries where it releases carbondioxide and is replenished with oxygen, the blood is transported via the 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.

Pathophysiology

In some patients, fetal circulation shunts remain patent after delivery. Usually, patients with an open fetal shunt are asymptomatic and may only have a cardiac murmur upon auscultation. A patent foramen ovale conects the right and left atria and is usually found as an incidental finding on echocardiogram or after a cryptogenic stroke. In patients with a patent foramen ovale, there  is possibilitythat a thrombus from the lower extremity may bypass the lungs. This can be accomplished when the blood enters the right atrium, flows through the foramen ovale and empties into the left atrium. The thrombus would be able to travel from the left atrium into systemic circulation where it can, unfortunately, be delivered to the brain causing a thromboembolic cerebrovasscular accident (CVA).

2b) 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.

It may play a passive role in protecting a person from some health issues, such as stroke. However, it has an association with intracranial aneurysms.

Two arteries, called the carotid arteries, supply blood to the brain. They run along either side of the neck and lead directly to the Circle of Willis. Each carotid artery branches into an internal and external carotid artery. The internal carotid artery then branches into the cerebral arteries. This structure allows all of the blood from the two internal carotid arteries to pass through the Circle of Willis.

The structure of the Circle of Willis includes:

-Left and right internal carotid arteries.

-Left and right anterior cerebral arteries.

-Left and right posterior cerebral arteries.

-Left and right posterior communicating arteries.

-Basilar artery.

-Anterior communicating artery.

Functions

The Circle of Willis plays an important role, as it allows for proper blood flow from the arteries to both the front and back hemispheres of the brain. The arteries that stem off from the Circle of Willis supply much of the blood to the brain.

The Circle of Willis also serves as a sort of safety mechanism when it comes to blood flow.Importantly, the Circle of Willis does not actively carry out the function. Instead, the natural shape of the Circle and the way that pressure acts in the area simply allow for bidirectional blood flow when necessary.

2c) SPLANCHNIC CIRCULATION

The Splanchnic circulation is composed of the blood flow originating from the celiac, superior mesentric and inferior mesentric arteries and is distributed to all abdominal viscera. 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. Multiple regulatory pathways are involved in the distribution of the Splanchnic circulation. The Splanchnic circulation comprises 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 mesentric arteries. The resistance arterioles are the primary determinant of vascular resistance in the Splanchnic circulation. Neuronal control of the mesentric circulation is almost entirely sympathetic in origin. The parasympathetic fibers from the vagi have little effect on blood flow. Overall Splanchnic blood flow requires about 25% of cardiac output. The Splanchnic venous capacitance reservoir contains about one-third of the body's total blood volume. The sympathetic postganglionic fibers cause arteriolar vasoconstriction and decrease Splanchnic perfusion. Sympathetic stimulation also contracts the smooth muscle of the capacitance veins in the Splanchnic circulation, and may expel a large volume of pooled blood from the Splanchnic into the systemic circulation.

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 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 ischemia heart disease (coronary artery disease) and sometimes by embolism from other causes like obstruction in blood flow through vessels.

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 from 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.

-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 center in the hypothalamus. It regulates temperature by altering the level of sympathetic outflow to the cutaneous vessels, to return temperature to its normal range.

3) THE CARDIOVASCULAR ADJUSTMENT THAT OCCURS DURING EXERCISE

The integrated response to severe exercise involves fourfold to fivefold increases in cardiac output, which are due primarily to increases in cardiac rate and to a lesser extent to augmentation of stroke volume. The increase in stroke volume is partly due to an increase in end-diastolic cardiac size (Frank-starling mechanism) and secondarily due to a reduction in end-systolic cardiac size. The full role of the Frank-starling mechanism is masked by the concomitant tachycardia. The reduction in end-systolic dimensions can be related to increased contractility, madiated by beta adrenergic stimulation. Beta adrenergic blockage prevent the inotropic response, the decrease in end-systolic dimensions, and approximately 50% of the tachycardia of exercise. The enhanced cardiac output is distributed preferentially to the exercising muscles including the heart. Blood flow to the heart increases fourfold to fivefold as well, mainly reflecting the augmented metabolic requirements of the myocardium due to near maximal increases in cardiac rate and contractility. Blood flow to the inactive viscera (e.g, kidney and gastrointestinal tract) is maintained during severe exercise in the normal dog. It is suggested that local autoregulatory mechanisms are responsible for maintained visceral flow in the face of neural and hormonal autonomic drive, which acts to constrict renal and mesentric vessels and to reduce blood flow.However, in the presence of circulatory impairment, where oxygen delivery to the exercising muscles is impaired as occurs to complete heart block where normal heart rate increases during exercise are prevented, or in congestive right heart failure, where normal stroke volume increases during exercise are impaired, or in the presence of severe anemia, where oxygen-carrying capacity of the blood is limited, visceral blood flows are reduced drastically and blood is diverted to the exercising musculature. Thus, visceral flow is normally maintained during severe exercise as long as all other compensatory mechanism is disrupted (even the elimination of splenic reserve in the dog), reduction and diversion of visceral flow occur.