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1. DISCUSS THE LONG-TERM MEASUREMENT OF MEAN ARTERIAL PRESSURE

The Kidneys play an important role in the long-term regulation of arterial blood pressure. Mean arterial pressure is given as the diastolic pressure plus one-third of the pulse pressure. When blood pressure can no longer be regulated by the short-term mechanism, the long-term mechanism kicks into gear and regulates it using two methods

- ✓ ECF volume regulation
- ✓ Renin- angiotensin mechanism.

o EXTRACELLULAR FLUID VOLUME REGULATION

Increases in blood pressure cause kidneys to excrete large amounts of water and salt, particularly sodium, by means of pressure diuresis and pressure natriuresis. Pressure diuresis is the excretion of large quantity of water in urine. Even a slight increase in blood pressure doubles the water excretion. Pressure natriuresis is the excretion of large quantity of sodium in urine. Because of diuresis and natriuresis, there is a decrease in ECF volume and blood volume, which in turn brings the arterial blood pressure back to normal level. When blood pressure decreases, the reabsorption of water from renal tubules is increased. This in turn, increases ECF volume, blood volume and cardiac output, resulting in restoration of blood pressure.

o RENIN-ANGIOTENSIN MECHANISM

When blood pressure and ECF volume decrease, renin secretion from kidneys is increased. It converts angiotensinogen into angiotensin I by ACE (angiotensin converting enzyme). Angiotensin II then acts in two ways to restore the blood pressure:

- ✓ Causes constriction of arterioles in the body so that the peripheral resistance is increased and blood pressure rises. In addition, angiotensin II causes constriction of afferent arterioles in kidneys, so that glomerular filtration reduces. This results in retention of water and salts, thus increasing ECF volume and blood pressure.
- ✓ Also, angiotensin II stimulates the adrenal cortex to secrete aldosterone. This hormone increases reabsorption of sodium from renal tubules. Sodium reabsorption is followed by water reabsorption, resulting in increased ECF volume, blood volume and pressure respectively.

2. WRITE SHORT NOTES ON THE FOLLOWING:

- *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. 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 diverges 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 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.

- *CIRCLE OF WILL'S*

The circle of Willis is an important junction of arteries at the base of the brain. The structure encircles the middle area of the brain, including the stalk of the pituitary gland and other important structures. 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
- ✓ The circle of Willis is critical, as it is the meeting point of many important arteries supplying blood to the brain. The internal carotid arteries branch off from here into smaller arteries, which deliver much of the brain’s blood supply.

- FUNCTION

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. If a blockage or narrowing slows or prevents the blood flow in a connected artery, the change in pressure can cause blood to flow forward or backward in the circle of Willis to compensate. This mechanism could also help blood flow from one side of the brain to the other in a situation in which the arteries on one side have reduced blood flow. In an emergency, such as a stroke, this may reduce the damage or aftereffects of the event. 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.

- *SPLANCHNIC CIRCULATION*

Also known as visceral CIRCULATION and it constitutes of three portions:

- ✓ Mesenteric circulation supplying GIT
- ✓ Splenic circulation supplying spleen and
- ✓ Hepatic circulation supplying liver.

A notable unique feature is that the blood from mesenteric bed and spleen form a major amount of blood flowing into the liver.

- MESENTERIC CIRCULATION

Distribution of blood flow.

- ✓ Stomach: 35mL/100g/Minute
- ✓ Intestine: 50mL/100g/Minute
- ✓ Pancreas: 80mL/100g/Minute

Regulation of Mesenteric Blood Flow:

- ✓ Local autoregulation
- ✓ Activity of GIT: contraction of it reduces mesenteric blood flow while relaxation does the opposite
- ✓ Nervous factor: it is regulated by sympathetic nerves which cause constriction of mesenteric vessels and direct blood to muscle, heart and brain during emergency conditions. Parasympathetic nerves shoe no direct action on mesenteric blood vessels.
- ✓ Chemical factors: Functional hyperemia; increase in mesenteric blood flow immediately

after food intake and is mainly due to gastrin and cholecystokinin.

- SPLENIC CIRCULATION

Spleen is a major reservoir of blood due to dilation by sympathetic stimulation, it relaxes blood vessels and a lot of blood is stored in spleen. Two structures are responsible for blood storage. They are; splenic venous sinuses and splenic pulp. Contraction of blood vessels releases the blood back into Circulation.

- ✓ Regulation: it is regulated by sympathetic nerves.

- HEPATIC CIRCULATION

Liver receives blood from two sources which includes:

- ✓ Hepatic artery which carries 400mL of total blood and 75% of the oxygen.
- ✓ Portal vein which carries 1100mL of total blood and 25% of the oxygen.

Thus, in total the liver receives 30% of the cardiac output (1500mL) and it is about 100mL/100g/minute.

Regulation of Blood Flow:

- ✓ Systemic blood pressure: it increases as it increases
- ✓ Splenic Contraction: increases blood flow
- ✓ Movement of intestine: increases blood flow
- ✓ Chemical factors: hypoxia, increase in Carbon dioxide conc and excess hydrogen ions increase blood flow to the liver.
- ✓ Nervous: sympathetic nerves through splanchnic nerve decreases blood flow.

- *CORONARY CIRCULATION*

The heart is highly metabolically active and has the highest oxygen consumption by mass of any organ. This demand for oxygen is met by the coronary circulation, which is responsible for delivering blood to the myocardium and represents approximately 5% of cardiac output.

The coronary arteries arise from the sinuses of Valsalva, just past the origin of the aortic root. The right coronary artery (RCA), arising from the anterior aortic sinus, supplies blood to the right atrium, right ventricle, sinoatrial node, atrioventricular (AV) node, and select portions of the left ventricle. The left coronary artery (LCA) arises from the left posterior aortic sinus and quickly bifurcates into the left circumflex artery (LCX) and left anterior descending artery (LAD), which supply blood to the left atrium and left ventricle. The coronary arteries can broadly classify as

epicardial vessels and intramuscular vessels. The former is larger and more superficial, and they serve as conductors for blood flow. The latter are smaller and course within the myocardium; their various branches and arterioles provide higher resistance but more fine-tuned control of blood flow. In most tissues, blood flow peaks during ventricular systole due to increased pressure in the aorta and its distal branches. Blood flow through the coronary vessels, however, is seemingly paradoxical and peaks during ventricular diastole. When the ventricles relax during diastole, the coronary vessels are no longer compressed, and normal blood flow resumes. Due to this pattern of blood flow, tachycardia - and the resultant decrease of time spent in diastole - can decrease the efficiency of myocardial perfusion.

Regulation:

- ✓ At rest, approximately 60% to 70% of oxygen is extracted from blood in the coronary arteries. This degree of oxygen extraction is a testament to the high metabolic activity of the myocardium. It also highlights the importance of increasing overall coronary flow during times of increased myocardial oxygen demand.
- ✓ At the most basic level, local hypoxemia and hypercarbia have shown to correlate with coronary vasodilation. Measurements of coronary venous pO_2 and pCO_2, however, show little, if any, change during states of physiologically increased demand. This situation suggests that alternative factors must contribute to coronary regulation under normal conditions that prevent hypoxemia and hypercarbia. Indeed, multiple studies have demonstrated that the concentrations of both oxygen and carbon dioxide are insufficient in explaining the majority of the total extent of coronary vasodilation in response to increased oxygen demand. While it is likely that localized hypoxemia and hypercarbia have a role in coronary regulation during pathophysiologic states, it is not yet clear whether an intermediary molecule is involved in the process.
- ✓ ATP-dependent potassium (K) channels in vascular smooth muscle and other tissues. These channels likely contribute to baseline vascular tone, as their inhibition results in a slight decrease in coronary flow.
- ✓ Other mediators of coronary flow also have been elucidated. As coronary flow increases secondary to other factors, increased endovascular shear stress stimulates nitric oxide synthesis. The release of nitric oxide results in vasodilation at both rest and states of increased myocardial oxygen consumption. However, inhibition of nitric oxide synthesis has shown in multiple studies that nitric oxide is not necessary for physiologic coronary vasodilation. Prostacyclin, an arachidonic acid metabolite, has also demonstrated some vasodilatory effect on the coronary vessels likely through interaction with nitric oxide. Adenosine has also shown effect on regulation on coronary blood flow.
- ✓ Endothelin is an extremely potent vasoconstrictor, and the coronary circulation is highly sensitive to it. Studies have shown increased plasma concentrations of endothelin with coronary related pathology.
- ✓ Neurohormonal factors also have demonstrated to regulate coronary flow, though this

effect appears to be relatively minor. Adrenergic receptors are distributed in a non-uniform manner along the coronary vessels; alpha receptors are found in greater concentration in epicardial vessels, whereas a preponderance of beta-2 receptors exists in intramuscular and subendocardial vessels. This distribution appears to minimize coronary "steal," by constricting proximal vessels and shifting the dependence of coronary flow to dilated distal vessels. Additionally, this decrease in large coronary vessel diameter also may serve to reduce the oscillations in coronary flow caused by ventricular compression of intramuscular vessels. Adrenergic control has demonstrated to contribute to physiologic vasodilation; blockade of alpha and beta-adrenergic receptors results in substantially lower coronary venous oxygen tension.

- *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.

o 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.

o 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:

- ✓ 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

3. DISCUSS THE CARDIOVASCULAR ADJUSTMENTS THAT TAKES PLACE DURING EXERCISE

During exercise, there is an increase in metabolic needs of body tissues, particularly the muscles. Various adjustments in the body during exercise are aimed at:

- ✓ Supply of various metabolic requisites like nutrients and oxygen to muscles and other tissues involved in exercise
- ✓ Prevention of increase in body temperature. These two types of exercise

Exercises classified based on contraction are of two types:

- ✓ Static exercise: here muscles contract isometrically. E.g. pushing against a heavy weight.
- ✓ Dynamic exercise where muscles contract isotonically. E.g. running, push-ups, etc.

Based on energy and oxygen utilization they are classified into two too

- ✓ Anaerobic: this is exercise done in the absence of adequate oxygen reaching muscles. It occurs during the first few minutes of exercise and exercises that require short but strong bursts of energy. Here glycogen is used.
- ✓ Aerobic: exercise done in the presence of adequate oxygen reaching muscles. It usually lasts for 30 minutes till glucose store is finished and fats are then burnt in the presence of oxygen.

And based on exercise severity, we have

- ✓ Mild exercise: little or no change occurs in cardiovascular system during mild exercise.
- ✓ Moderate exercise: doesn't involve strenuous activities thus exhaustion does not occur. the examples of this type of exercise are fast walking and slow running.
- ✓ Severe exercise: severe exercise involves strenuous muscular activity. Severity can be maintained only for short duration. E.g. Fast running for a distance of 100 or 400 meters. Complete exhaustion occurs at the end of severe exercise.

EFFECTS OF EXERCISE ON CARDIOVASCULAR SYSTEM

- ✓ On blood: Mild hypoxia developed during exercise stimulates the juxtaglomerular apparatus to secrete erythropoietin. It stimulates the bone marrow and causes release of red blood cells. Increased carbon dioxide content in blood decreases the pH of blood.
- ✓ On blood volume: More heat is produced during exercise and the thermo-regulatory system is activated. This in turn, causes secretion of large amount of sweat leading to; Fluid loss, Reduced blood volume, hemoconcentration. Sometimes, severe exercise leads to even dehydration.
- ✓ On heart rate: Heart rate increases during exercise. Even the thought of exercise or

preparation for exercise increases the heart rate. It is because of impulses from cerebral cortex to medullary centers, which reduces vagal tone. In moderate exercise, the heart rate increases to 180 beats/minute. In severe muscular exercise, it reaches 240 to 260 beats/minute. Increased heart rate during exercise is due to four factors: Impulses from proprioceptors, Increased carbon dioxide tension, which acts through medullary centers and rise in body temperature.

- ✓ On cardiac output: Cardiac output increases up to 20 L/minute in moderate exercise and up to 35 L/minute during severe exercise. Increase in cardiac output is directly proportional to the increase in the amount of oxygen consumed during exercise. During exercise, the cardiac output increases because of increase in heart rate and stroke volume.
- ✓ On venous return: It increases remarkably during exercise because of muscle pump, respiratory pump and splanchnic vasoconstriction.
- ✓ On blood flow to skeletal muscles: There is a great increase in the amount of blood flowing to skeletal muscles during exercise. During the muscular activity, stoppage of blood flow occurs when the muscles contract. It is because of compression of blood vessels during contraction. And in between the contractions, the blood flow increases. Sometimes the blood supply to muscles starts increasing even during the preparation for exercise. It is due to the sympathetic activity. Sympathetic nerves cause vasodilatation in muscles. The sympathetic nerve fibers causing vasodilatation in skeletal muscle are called sympathetic cholinergic fibers since these fibers secrete acetylcholine instead of noradrenaline.
- ✓ During moderate isotonic exercise, the systolic pressure is increased. It is due to increase in heart rate and stroke volume. Diastolic pressure is not altered because peripheral resistance is not affected during moderate isotonic exercise.