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1)Discuss the long term regulation of mean arterial blood pressure

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. 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

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. 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.

Lungs: The pulmonary arteries carry deoxygenated blood to the lungs, where carbon dioxide is released and oxygen is picked up during respiration. Arteries are further divided into very fine capillaries which are extremely thin-walled. The pulmonary vein returns oxygenated blood to the left atrium of the heart.

Veins: Pulmonary vein. The oxygenated blood then leaves the lungs through pulmonary veins, which return it to the left part of the heart, completing the pulmonary cycle. This blood then enters the left atrium, which pumps it through the mitral valve into the left ventricle. From the left ventricle, the blood passes through the aortic valve to the aorta. The blood is then distributed to the body through the systemic circulation before returning again to the pulmonary circulation.

Arteries: Pulmonary artery. From the right ventricle, blood is pumped through the semilunar pulmonary valve into the left and right main pulmonary arteries (one for each lung), which branch into smaller pulmonary arteries that spread throughout the lungs.

Clinical significance

- Cardiac shunt is an unnatural connection between parts of the heart that leads to blood flow that bypasses the lungs.
- Vascular resistance
- Pulmonary shunt

b) 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

Structure: 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.

Variation: Considerable anatomic variation exists in the circle of Willis. Based on a study of 1413 brains, the classic anatomy of the circle is only seen in 34.5% of cases. In one common variation the proximal part of the posterior cerebral artery is narrow and its ipsilateral

posterior communicating artery is large, so the internal carotid artery supplies the posterior cerebrum; this is known as a fetal posterior communicating cerebral artery.

Clinical significances: Subclavian steal syndrome-The redundancies that the circle of Willis introduce can also lead to reduced cerebral perfusion. In subclavian steal syndrome, blood is "stolen" from the circle of Willis to preserve blood flow to the upper limb.

c) 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).

Intrinsic control: The splanchnic vascular bed demonstrates an autoregulatory capacity similar to that seen in other vascular beds such as the renal and cerebral circulations. This ensures that a constant blood flow can be maintained across a wide variety of perfusion pressures. There are two proposed mechanisms: metabolic and myogenic control.

d) 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. 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 and right coronary arteries occasionally arise by a common trunk, or their number may be increased to three; the additional branch being the posterior coronary artery (which is smaller in size). In rare cases, a person will have the third coronary artery run around the root of the aorta. Occasionally, a coronary artery will exist as a double structure (i.e. there are two arteries, parallel to each other, where ordinarily there would be one).

e) 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. In high core temperatures: Sympathetic innervation is decreased, reducing the vasomotor tone in the AVAs.

3) 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, mediated by beta adrenergic stimulation. Beta adrenergic blockade prevents 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 mesenteric 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 anaemia, 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 mechanisms remain intact. However, when any other compensatory mechanism is disrupted (even the elimination of splenic reserve in the dog), reduction and diversion of visceral flow occur.