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1. **Long term regulation of mean arterial blood pressure**

Kidneys play an important role in the long-term regulation of arterial blood pressure. When blood pressure alters slowly in several days/months/years, the nervous mechanism adapts to the altered pressure and looses the sensitivity for the changes. It cannot regulate the pressure any more. In such conditions, the renal mechanism operates efficiently to regulate the blood pressure. Therefore, it is called long-term regulation.

Kidneys regulate arterial blood pressure by two ways:

1. By regulation of ECF volume
2. Through renin-angiotensin mechanism.

BY REGULATION OF EXTRACELLULAR FLUID VOLUME

When the blood pressure increases, kidneys 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 because of increased blood pressure. 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.

THROUGH RENIN-ANGIOTENSIN MECHANISM

Actions of Angiotensin II

When blood pressure and ECF volume decrease, renin secretion from kidneys is increased. It converts angiotensinogen into angiotensin I. This is converted into angiotensin II by ACE (angiotensin-converting enzyme).

Angiotensin II acts in two ways to restore the blood pressure:

i. It 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, increases ECF volume to normal level. This in turn increases the blood pressure to normal level.

ii. Simultaneously, 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 and blood volume. It increases the blood pressure to normal level (Fig. 103.4).

Actions of Angiotensin III and Angiotensin IV

Like angiotensin II, the angiotensins III and IV also increase the blood pressure and stimulate adrenal cortex to secrete aldosterone

2. **A)Pulmonary circulation**

Pulmonary circulation, system of blood vessels that forms a closed circuit between the heart and the lungs, as distinguished from the systemic circulation between the heart and all other body tissues. On the evolutionary cycle, pulmonary circulation first occurs in lungfishes and amphibians, the first animals to acquire a three-chambered heart. The pulmonary circulation becomes totally separate in crocodilians, birds, and mammals, when the ventricle is divided into two chambers, producing a four-chambered heart. In these forms the pulmonary circuit begins with the right ventricle, which pumps deoxygenated blood through the pulmonary artery. This artery divides above the heart into two branches, to the right and left lungs, where the arteries further subdivide into smaller and smaller branches until the capillaries in the pulmonary air sacs (alveoli) are reached. In the capillaries the blood takes up oxygen from the air breathed into the air sacs and releases carbon dioxide. It then flows into larger and larger vessels until the pulmonary veins (usually four in number, each serving a whole lobe of the lung) are reached. The pulmonary veins open into the left atrium of the heart.

B) **Circle of Willis**

The circle of Willis 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. Polygonal anastomotic shape offers the possibility of alternate pathways for the blood flow, which is essential for the brain functioning, since it is the structure that is mostly sensitive to hypoxia. Hypoxia of the brain tissue that lasts longer than 6

minutes results with the irreversible changes in the brain parenchyma, and depending on the location of the lesion, the functional damages vary widely.

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.

Clinical significance

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. Subclavian steal syndrome results from a proximal stenosis (narrowing) of the subclavian artery, an artery supplied by the aorta, which is also the same blood vessel that eventually feeds the circle of Willis via the vertebral and internal carotid arteries.

C) **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.

D) **Coronary circulation:**

Coronary circulation, part of the systemic circulatory system that supplies blood to and provides drainage from the tissues of the heart. In the human heart, two coronary arteries arise from the aorta just beyond the semilunar valves; during diastole, the increased aortic pressure above the valves forces blood into the coronary arteries and thence into the musculature of the heart. Deoxygenated blood is returned to the chambers of the heart via coronary veins; most of these converge to form the coronary venous sinus, which drains into the right atrium. The heart normally extracts 70 to 75 percent of the available oxygen from the blood in coronary circulation, which is much more than the amount extracted by other organs from their circulations.e.g., 40 percent by resting skeletal muscle and 20 percent by the liver. Obstruction of a coronary artery, depriving the heart tissue of oxygen-rich blood, leads to death of part of the heart muscle (myocardial infarction) in severe cases, and total heart failure and death may ensue. 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.

E) **Cutaneous circulation:**

Control of the cutaneous circulation by reflexes aimed at body temperature regulation, blood pressure regulation, and the reflexes attending muscular exercise was discussed in detail, as were the similarities and differences between control of cutaneous arterioles and arteriovenous anastomoses. A mechanistic treatment of interaction between adrenergic control of cutaneous blood vessels and their temperature brought physical factors and pharmacological approaches to the consideration of reflex control. Finally, the more slowly developing changes in the control of the skin circulation that accompany circadian rhythms, changes in blood volume or its distribution, physical training, and acclimatization were discussed. Because the cutaneous circulation has potentially large vascular conductance, blood flow, and blood volume, control of the resistance and compliance vessels within the skin has an importance well beyond that of tissue nutrition. Indeed, overall hemodynamics are dependent on how much blood flow and how much blood volume are distributed to skin. Consequently, reflex factors, physical factors, and their interaction all have roles

of importance with respect to exchange of heat with environment as well as maintenance of blood pressure, cardiac output, and blood flow to other tissues.

3. **Cardiovascular adjustment 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, 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 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 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.