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1. The long-term level of arterial pressure is dependent on the relationship between arterial pressure and the urinary output of salt and water, which, in turn, is affected by a number of factors, including renal sympathetic nerve activity (RSNA). In the present brief review, we consider the mechanisms within the brain that can influence RSNA, focusing particularly on hypothalamic mechanisms. The paraventricular nucleus (PVN) in the hypothalamus has major direct and indirect connections with the sympathetic outflow and there is now considerable evidence that tonic activation of the PVN sympathetic pathway contributes to the sustained increased level of RSNA that occurs in conditions such as heart failure and neurogenic hypertension. The tonic activity of PVN sympathetic neurons, in turn, depends upon the balance of excitatory and inhibitory inputs. A number of neurotransmitters and neuromodulators are involved in these tonic excitatory and inhibitory effects, including glutamate, GABA, angiotensin II and nitric oxide. The dorsomedial hypothalamic nucleus (DMH) also exerts a powerful influence over sympathetic activity, including RSNA, via synapses with sympathetic nuclei in the medulla and, possibly, also other brainstem regions. The DMH sympathetic pathway is an important component of the phasic sympathoexcitatory responses associated with acute stress, but there is no evidence that it is an important component of the central pathways that produce long-term changes in arterial pressure. Nevertheless, it is possible that repeated episodic activation of this pathway could lead to vascular hypertrophy and, thus, sustained changes in vascular resistance and arterial pressure. 4. Recent studies have reactivated the old debate concerning the possible role of the baroreceptor reflex in the long-term regulation of sympathetic activity. Therefore, central resetting of the baroreceptor-sympathetic reflex may be an important component of the mechanisms causing sustained changes in RSNA. However, little is known about the cellular mechanisms that could cause such resetting.
2. a. **Pulmonary Circulation**: Pulmonary circulation is the portion of circulatory system that carries deoxygenated blood away from the right ventricle of the heart, 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. A separate system known as the bronchial circulation supplies

b. **Circle of Willis**: The circle of Willis encircles the stalk of the pituitary gland and provides important communications between the blood supply of the forebrain and hindbrain (ie, between the internal carotid a vertebro basilar systems following obliteration of primitive embryonic connections). Although a complete circle of Willis is present in some individuals, it is rarely seen radiographically in its entirety; anatomical variations are very common and a well-developed communication between each of its parts is identified in less than half of the population. The circle of Willis begins to form when the right and left internal carotid artery (ICA) enters the cranial cavity and each one divides into two main branches: the anterior cerebral artery (ACA) and middle cerebral artery (MCA). [2] The anterior cerebral arteries are then united and blood can cross flow by the anterior communicating (ACOM) artery.

c. **Splanchic Circulation**: The splanchic circulation describes the blood flow to the abdominal gastrointestinal organs including the stomach, liver, spleen, pancreas, small intestine, and large intestine. It includes three major branches of abdominal aorta, that is, the coeliac artery, superior mesenteric artery, and inferior mesenteric.

d. **Coronary Circulation**: This is the circulation of blood in the blood vessels that supply the heart muscles. 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 level if consciousness of the brain from moment to moment. Interruptions of coronary circulation quickly cause heart attacks, in which the heart muscle is damaged by oxygen starvation. Such interruptions are usually caused by ischemic heart disease and sometimes by embolism from other causes like obstruction in blood flow through vessels.

e. **Cutaneous Circulation**: This 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.

1. Exercise probably acts at various levels in the cardiovascular system. The regulation and the integration of many cardiovascular functions are modified by exercise training. For example, exercise can improve baroreflex sensitivity, blood pressure regulation, organs perfusion and vascular reactivity. 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.