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PERSPECTIVE

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Genetics and the Pathophysiology of Hypertension

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Description

A branch of medicine known as pathophysiology discusses how the body works in relation to illnesses and ailments. The pathophysiology of hypertension is a field that makes an effort to mechanistically explain the origins of the chronic condition known as hypertension, which is characterized by elevated blood pressure. The two types of hypertension are essential and secondary, respectively. The majority of hypertension 90-95 percent is essential hypertension. Essential hypertension is described by some sources as having an unknown cause, while others attribute it to a diet high in sodium and low in potassium. The term "secondary hypertension" refers to hypertension that has an identifiable underlying cause and a well-established mechanism, such as chronic kidney disease, aortic or renal artery stenosis, or endocrine conditions such as elevated levels of aldosterone, cortisol, or catecholamines. A key risk factor for hypertensive heart disease, coronary artery disease, and stroke, aneurysm of the aorta, peripheral artery disease, and chronic kidney disease is persistent hypertension. The two factors that determine arterial pressure are cardiac output and peripheral resistance. Heart rate and stroke volume together determine cardiac output; stroke volume is influenced by the size of the vascular compartment and myocardial contractility. Functional and anatomical alterations in tiny arteries and arterioles impact peripheral resistance.

Genetics

Mendelian types of high blood pressure can result from a single gene mutation; 10 genes have been identified as the monogenic hypertension-causing genes. By modifying how the kidneys handle salt, these mutations have an impact on blood pressure. Greater blood pressure similarity within families than between families suggests a type of inheritance and is not the result of shared environmental factors. Genetic research methods were used to discover a statistically significant relationship between blood pressure and many chromosomal areas, including those connected to familial combination hyperlipidemia. These results imply that there are several genetic loci with modest effects on blood pressure in the overall population. However, the overall rarity of recognizable single-gene causes of hypertension is consistent with a complex origin for essential hypertension.

Autonomic nervous system

Through the use of pressure, volume, and chemoreceptor signals, the autonomic nervous system is crucial in preserving cardiovascular homeostasis. This is accomplished through controlling renal and peripheral vasculature functions, which in turn influence cardiac output, vascular resistance, and fluid retention. The sympathetic nervous system over activates, which raises blood pressure and causes hypertension. Alterations in baroreflex and chemo reflex pathways at both the peripheral and central levels are part of the factors underlying increased sympathetic nervous system activity in hypertension. In hypertensive individuals, peripheral baroreceptor resetting occurs, which elevates the pressure at which they are activated. When arterial pressure is stabilized, this peripheral resetting returns to normal. In addition, the aortic baroreflex is centrally reset in hypertension individuals, which suppresses sympathetic inhibition after aortic baroreceptor nerve activity. Renin-angiotensin-aldosterone is a system that controls peripheral resistance, extracellular fluid volume, all of which, if altered, can result in hypertension. Renin is a circulating enzyme that helps to regulate blood pressure by taking part in arterial vasoconstriction and maintaining extracellular volume. It carries out this action by

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hydrolyzing the liver-secreted angiotensinogen to produce the peptide angiotensin I. Angiotensin converting enzyme, which is mainly but not entirely present in the pulmonary circulation attached to endothelium, further cleaves angiotensin I. (ACE). The most vasoactive peptide, angiotensin II, is produced by this cleavage. All blood arteries can be effectively constricted by angiotensin II.

Endothelial dysfunction

Changes in the blood and the pressure of the blood flow have an impact on the endothelium of blood vessels, which also produces a wide variety of compounds that affect blood flow. For instance, the main regulators of vascular tone and blood pressure are local nitric oxide and Endothelin, both of which are secreted by the endothelium. The balance between the vasodilators and the vasoconstrictors is thrown in people with essential hypertension, which results in alterations to the endothelium and creates a "vicious cycle" that aids in the maintenance of high blood pressure. Changes in vascular tone, vascular reactivity, and coagulation and fibrinolytic pathways are also brought on by endothelial activation and injury in hypertensive individuals. A reliable indication of target organ damage, atherosclerotic disease, and prognosis is altered endothelial function.