

PHYSIO-PATHOLOGY OF DIASTOLIC HYPERTENSION.†

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Introduction

In a closed circulatory system the Pressure is the product of the amount of Flow of the circulating medium and the Resistance of the conducting system. Likewise, in the Cardiovascular System blood pressure is the product of blood flow, viz., Cardiac Output, and the the Peripheral Resistance.

In the majority of Diastolic Hypertension it is found that the Cardiac output does not change to a significant degree. The factor which changes significantly is the Peripheral Resistance.

Peripheral Resistance.

The most important site of the Peripheral Resistance is primarily the Arterioles, the terminals of the outflowing tract of the Arterial System. It may be mentioned here for the sake of completion that the major vessels such as Aorta and its main branches also do play some part, but not to a significant degree. The arterioles always remain in a state of partial contraction because of nerve supply, which is regulated by a centre in the Medulla Oblongata. If, therefore, one changes the Peripheral Resistance, one also changes the Diastolic blood pressure.

Factors Modifying the Peripheral Resistance

They are mainly the following in order of importance: i) the size of the lumen of the arterioles, ii) the elasticity of the blood vessels, and iii) viscosity of the blood.

1. The size of the lumen of the arterioles.

This depends upon two mechanism: i) Nervous and ii) Chemical or Hormonal.

The Nervous Mechanism.

The Vasomotor Centre in the Medulla regulates the tone of the arterioles through the nervous pathways of Vasoconstrictor nerves. If, therefore, the Vasomotor Centre be stimula-

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ted the Vasoconstrictors are inhibited so that the tone viz., the peripheral resistance of the arterioles is diminished resulting in their dilatation causing the fall of blood pressure. On the other hand, if the Vasomotor Centre be depressed it releases the Vasoconstrictors so that the tone viz., the peripheral resistance of the arterioles increases resulting in their constriction causing the rise of blood pressure. In this context may be mentioned various organs like Carotid Sinus—Aortic Arch Nerves (so-called Buffer Nerves) or the Baroreceptors. In Clinical Medicine one comes across interesting examples such as Stiff-collar Syndrome in which the stiff collar stimulates the Carotid Baroreceptors which send volleys of impulses to stimulate the Vasomotor Centre which inhibits the Vasoconstrictors so that the peripheral resistance falls causing the fall of blood pressure. The same mechanism holds true for Carotid Sinus Syncope induced by Carotid massage, a procedure much used in Clinical Medicine to decrease the heart rate and the blood pressure. On the other hand, in such conditions as Haemorrhage and Shock of moderate degree the Baroreceptors are not stimulated so that the number and frequency of impulses arising there decrease, thereby the Vasomotor Centre releases the Vasoconstrictors resulting in the peripheral arteriolar constriction giving rise to the rise of blood pressure.

The Reflex Arc of Baroreceptors, however, is rather extensive: The afferent limb consists of the Sinus Nerves, (IX&X Cranial nerves), Atrial and Ventricular Stretch Receptors, Pulmonary Stretch Receptors; Pulmonary Arterial Stretch Receptors, and Mesenteric Stretch Receptors; the Centre is the Vasomotor Centre; and the efferent limb being the Vasoconstrictors.

Hypertension also occurs due to Vasomotor Centre dysfunction as a result of Intracranial Hypertension due to any cause, e.g. Meningitis, Subarachnoid haemorrhage; or in Poliomyelitis; or by such experimental procedures as injection of kaolin in the Cisterna Magna; section of Arterial Baroreceptors, tying of major arteries above the Carotid Sinus; by stimulating the limbic pole of the Temporal Lobe; and in experimental animals even by inducing Psychosis.

The Chemical or Hormonal mechanism

The Chemical or hormonal substances which alter the blood pressure are: $p\text{CO}_2$ and $p\text{O}_2$ of blood, Renal factor viz., Renin-Angiotensin System, Adrenocortical factor viz., Gluco-Mineralo-Corticosteroids or Aldosterone, Adrenal Medullary factor viz., Catecholamines such as Adrenaline, Noradrenaline and Serotonin, and Pituitary factor viz. Adrenocorticotrophic hormone and Antidiuretic hormone. Thus increased $p\text{CO}_2$ and decreased $p\text{O}_2$ cause rise of blood pressure either by stimulating directly the Vasomotor Centre or indirectly the Chemoreceptor System viz., Carotid Body or Aortic Body or both.

Renal Hypertension

Various experimental workers such as Goldblatt found that Renin-Angiotensin System and Aldosterone increased in the circulation as a result of renal ischaemia produced by

clamping one renal artery in man and rat and clamping both renal arteries in dog. This was also found to be so in such a congenital abnormality as Renal Artery Stenosis and probably in Coarctation of Aorta. This also applies to the parenchymatous diseases of the kidneys such as Chronic Pyelonephritis, Polycystic disease of the kidney and kidney neoplasm such as Grawitz tumour. On further analysis, however, this is not always true; for Renin-Angiotensin System does not increase in the great majority of Renal Artery Stenosis, nor does one know why at all in Renoprival Hypertension the blood pressure should rise even if both kidneys are removed. An attempt has been made to partly explain away this paradox by the theory of the absence of Vasodilator substance (which causes peripheral arteriolar dilatation) secreted by the kidneys.

In ADRENOCORTICAL HYPERTENSION rise of blood pressure is produced primarily by increased blood volume because of the retention of Sodium and Water as a consequence of the large amount of corticosteroids in the circulation, and secondarily by causing the kidney lesion viz., Hypokalaemic Nephropathy. In HYPERALDOSTERONISM or Conn's Syndrome similar mechanism holds true as a result of the large amount of Aldosterone in the circulation. In ADRENAL MEDULLARY HYPERTENSION (eg. Pheochromocytoma, Ganglioneuroma) rise of blood pressure occurs due to widespread arteriolar and venous constriction because of increased production of Catecholamines.

2. Change in the blood vessel.

Such changes as intimal thickening or fibrinoid necrosis of the arterioles which occur in collagen diseases viz., Systemic Lupus Erythematosus, Polyarteritis nodosa and Scleroderma give rise to increase of peripheral resistance resulting in the rise of blood pressure.

3. Increase in the Viscosity of blood.

Which is seen in such a condition as Polycythaemia rubra vera, also causes rise of blood pressure by increasing the peripheral resistance.

Almost all the above mentioned causes or mechanisms apply to the group of Hypertension known as Secondary Hypertension. On the contrary one knows very little of the causes of the vast majority of Hypertension—more than 90% known as Primary or Essential or Idiopathic Hypertension.

In this condition the Barostat is reset at a higher level so that the Baroreceptor mechanism fails to respond to the usual rise of blood pressure. The predisposing factors are Heredity, Age, Sex, Hypercholesteromic states such as Diabetes mellitus, Myxoedema and sometimes Chronic Biliary diseases. The pathological lesion consists of diffuse arteriolar narrowing characterised—in the benign stage—by hyalinisation of intima or splitting of the internal elastic lamina, and—in the malignant stage—by cellular hyperplasia (so-called onion skin appearance) in the subintimal region and/or arteriolonecrosis—all leading to increase of peripheral resistance.

In the balance of evidences—clinical, pathological and experimental—the aetiology of Primary Hypertension is probably multifactorial in nature, arteriosclerosis being one of the manifestation of some enigmatic disorder probably primarily involving lipid metabolism.