

SYSTEMIC HYPERTENSION

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SYSTEMIC HYPERTENSION.

SYSTEMIC HYPERTENSION is a disorder characterized by sustained elevation of systemic arterial blood pressure, usually above a diastolic level of 90 mm Hg and a systolic level of 140 mm Hg. This disorder is the most significant risk factor for cardiovascular disease and a major cause of heart failure, renal failure, and stroke.

A. Classification. Hypertension can be classified according to the etiology or to the course and extent of the disease.

1] *Aetiological classification*

- a] **Primary hypertension** (also known as essential hypertension) has an unknown etiology but risk factors for atherosclerosis have been implicated in the development of primary hypertension.
- b] **Secondary hypertension** has a specific underlying cause, which often is a renal disease (e.g., renal vascular or parenchymal disease, renin-secreting tumor) or an endocrine disease (e.g., primary aldosteronism, Cushing's disease, pheochromocytoma).

2] *Clinical course of hypertension*

- a] **Benign hypertension** is a chronic and relatively mild increase in systemic arterial blood pressure (to a diastolic level not higher than 110 to 120 mm Hg), which may or may not have an underlying cause. Although the secondary effects of hypertension may not be clinically evident for a long time, benign hypertension may progress to serious end-organ effects.

b] Malignant (accelerated) hypertension involves a profound and acute elevation of blood pressure (to a diastolic level higher than 130 to 140 mm Hg), which can develop de novo or as a complication of benign hypertension. It is a true emergency. In malignant hypertension, end-organ effects of hypertension develop in a brief period of time. Papilledema, retinal hemorrhage, encephalopathy, angina with cardiac and renal failure may all complicate malignant hypertension.

B. Incidence and epidemiology.

1. Primary (essential) hypertension accounts for about 90% of cases of elevated blood pressure. In about 5% to 10% patients with secondary benign hypertension, an underlying cause can be identified.
 - a] Essential hypertension is more common in adults than in adolescents and is rare in children. Men are affected more often than women.
 - b] Essential hypertension is more common among blacks than among whites.

2. Malignant hypertension is a rare form of high blood pressure that can complicate the course of both essential and secondary hypertension, rarely, it is the initial manifestation of high blood pressure. Persons at high risk include patients with renovascular hypertension, glomerulonephritis, chronic renal failure or scleroderma and pregnant women with toxemia.

C. **Pathogenesis**. Hypertension represents an imbalance in the factors that control cardiac output, peripheral resistance, and sodium homeostasis. The renal vasculature may be the primary source of kidney disease as it contributes to a renal cause for hypertension (called renovascular hypertension) as well as to the acceleration of other forms of vascular disease (e.g., thrombosis, embolization, infarction). The kidney influences blood pressure through several mechanisms.

1. The renin-angiotensin-aldosterone system is a major pressor mechanism. Activation of the system results in an increase in systemic blood pressure. The system works as follows.
 - a] Renin is released by the juxtaglomerular cells under the stress of decreased afferent arteriolar pressure, decreased sodium delivery to the distal tubule, or direct sympathetic stimulation.

- b] Renin cleaves angiotensinogen, a circulating α 2-globulin produced in the liver, to form angiotensin I, which is further modified by converting the enzyme (in a single passage through the lungs) to the potent vasoconstrictor angiotensin II. Angiotensin II is a potent stimulus for the production of aldosterone by the zona glomerulosa of the adrenal cortex. It also stimulates renin release by increasing the activity of the sympathetic nervous system.
- c] Adrenal secretion of aldosterone, in response to renin stimulation, results in increased renal absorption of sodium.

2. Atrial natriuretic factor, a hormone secreted by specialized cells in the cardiac atria, antagonizes the effect of the renin-angiotensin-aldosterone system, counteracts the vasoconstrictive action of angiotensin II, and increases the urinary excretion of sodium. Secretion of atrial natriuretic factor may be induced by atrial distention secondary to increased vascular volume.

D. Pathology. The long-term effects of sustained hypertension are most significant in the heart, kidneys and cerebral blood vessels.

1. **Macroscopic features.** The major morphologic feature of hypertensive heart disease is an enlarged heart, characterized by concentric hypertrophy of the left ventricle with hypertrophied papillary muscles. Endocardial fibrous thickening occasionally may be present.

- a] The left ventricular wall may increase from a normal thickness of 13 to 15 mm to a thickness of 25 mm or more. This hypertrophy correlates with a twofold to fourfold increase in the weight of the heart.
- b] The volume of the ventricular cavity is decreased by the cardiac hypertrophy. The degree of elevation of blood pressure often is poorly correlated with the degree of hypertrophy.
- c] Cardiac hypertrophy can be seen on echocardiogram, ECG or chest x-ray.

2. Microscopic features. No microscopic findings are unique to hypertensive heart disease, although individual myofiber hypertrophy often is seen.

E. **Clinical manifestations and complications.**

1. When the heart no longer can compensate for its increased work load by hypertrophy alone, cardiac dilatation and failure may occur (in about 40% of cases). Congestive heart failure is the most important cause of death in hypertensive patients.
2. Coronary atherosclerosis, which is exacerbated by the hypertension, increases the risk for cardiac ischaemia and infarction and for heart failure.