

## Fourth Lecture **Hypertension**



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*Cardio vascular Block*



**Objectives:**

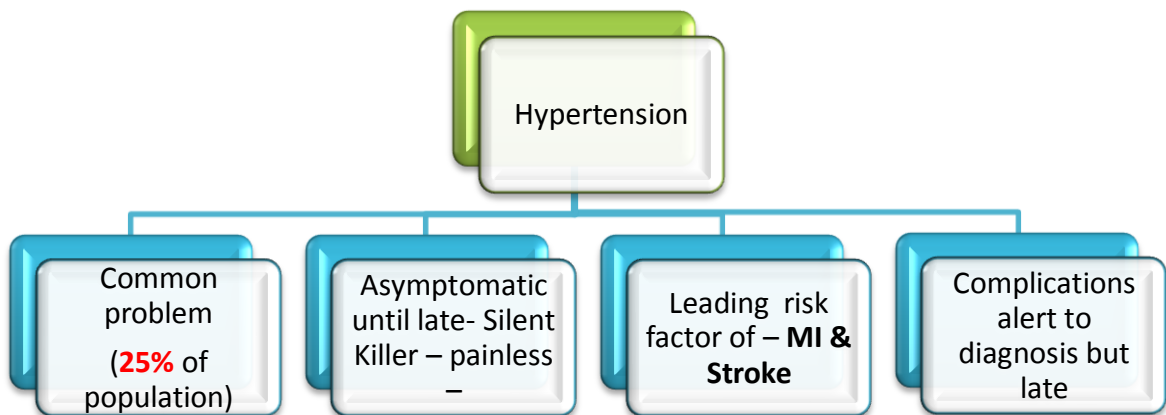
At the end of this lecture, the students should be able to:

- (1) Know the aetiology, risk factors and complications of hypertension, so as to be able to identify patient risk factors amenable to treatment by lifestyle modification, and to investigate patients appropriately for causes of secondary hypertension.

- Blood pressure is the pressure exerted by circulating blood upon the walls of blood vessels

Systemic and local tissue blood pressures must be maintained within a narrow range to prevent untoward consequences. Low pressures (*hypotension*) result in inadequate organ perfusion and can lead to dysfunction or tissue death. Conversely, high pressures (*hypertension*) can cause vessel and end-organ damage.

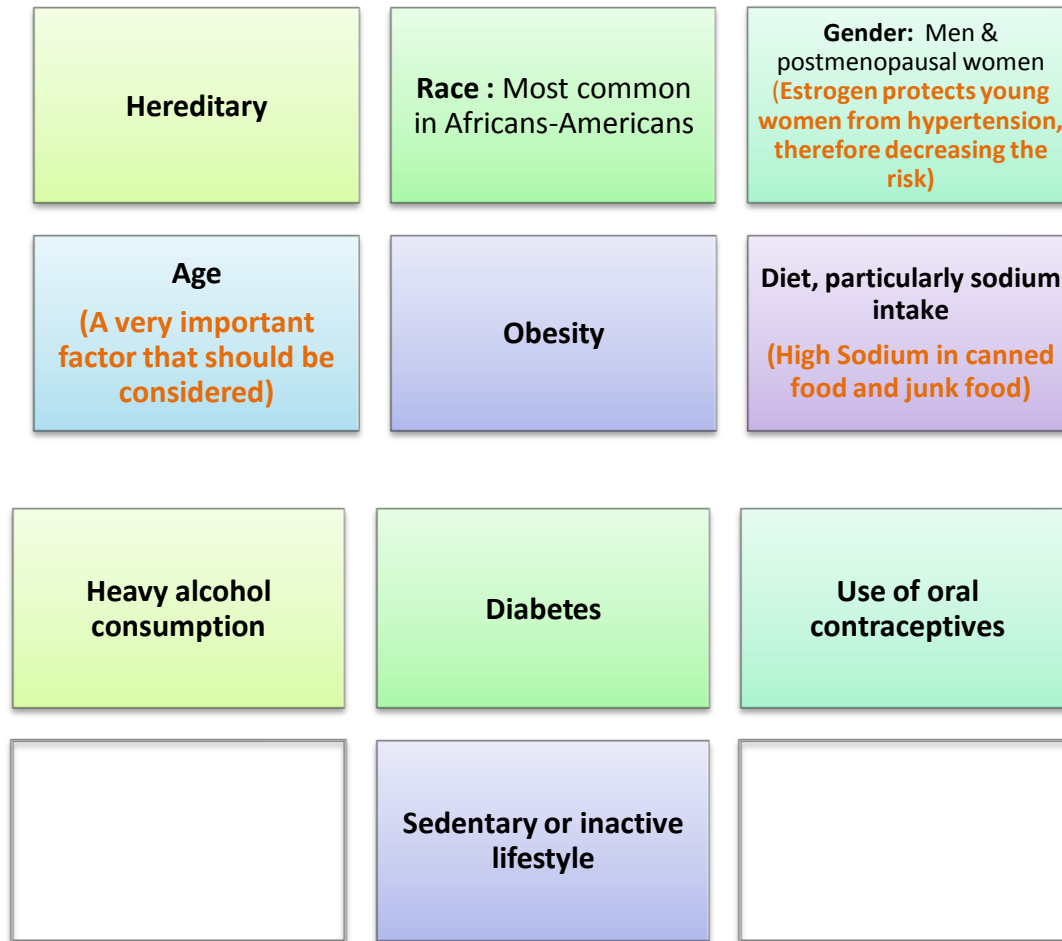
- It is a function of cardiac output and peripheral vascular resistance



### Hypertension definition

- No rigid definition
- However, hypertension (HTN) is usually considered when there is :
  - A **sustained diastolic pressure** greater than 89 (90) mmHg
  - OR
  - A **sustained systolic pressure** greater than 139 (140) mmHg

– Risk Factors:



Types and causes:

1) Essential (Primary) Hypertension (90-95%)

Postulated mechanisms of Essential Hypertension

1. Defect in sodium excretion (increased sodium resorption or decreased excretion) (Liddle Syndrome)
2. Defect in cell membrane function:
  - -Na/Ca transport
  - -Increased vasoconstrictive response
3. Increased sympathetic response

Essential hypertension ( HTN of unknown etiology):

Causes are multifactorial and results from the combined effects of multiple genetic polymorphisms & interacting environmental factors

Liddle syndrome is caused by mutation of ENaC leading to hypertension causing increased distal tubular resorption of sodium induced by aldosterone .

## 2) Secondary Hypertension

### Causes of Secondary Hypertension:

#### Renal

- Acute glomerulonephritis
- Chronic renal disease
- Polycystic disease
- Renal artery stenosis
- Renal vasculitis
- Renin-producing tumours

(this will decrease the BF to the kidneys → secretion of Renin → HTN)

#### Endocrine

- Adrenocortical hyper function: Cushing syndrome, primary aldosteronism, congenital adrenal hyperplasia
- Exogenous hormones (glucocorticoids, estrogens)
- Pheochromocytoma (tumor of adrenal gland which causes increased secretion of epinephrine and norepinephrine)
- Acromegaly (increased growth hormone release)
- Hypo or hyperthyroidism
- Pregnancy-induced

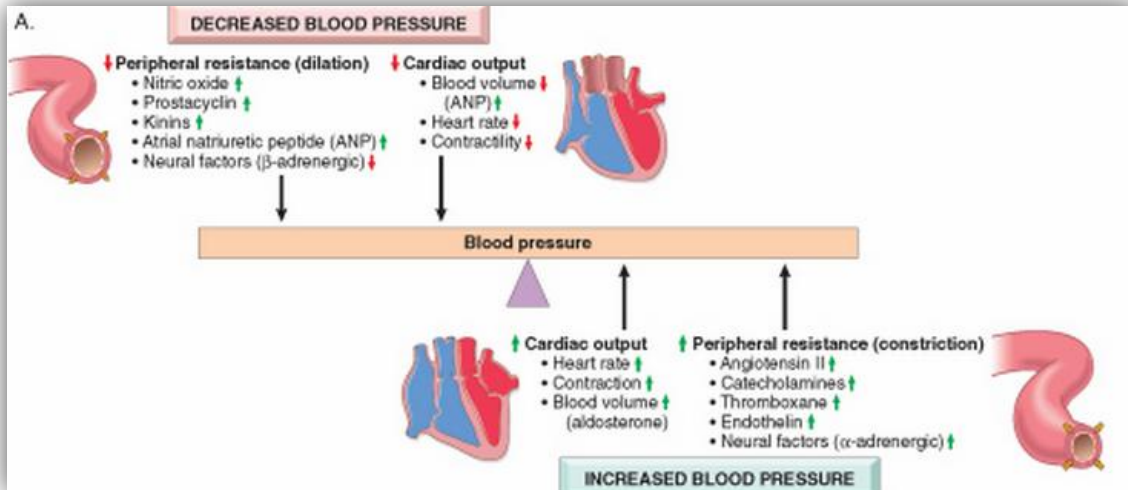
#### Cardiovascular

- Coarctation of aorta
- Polyarteritis nodosa (or other vasculitis)
- Increased intravascular volume
- Increased cardiac output
- Rigidity of the aorta

#### Neurologic

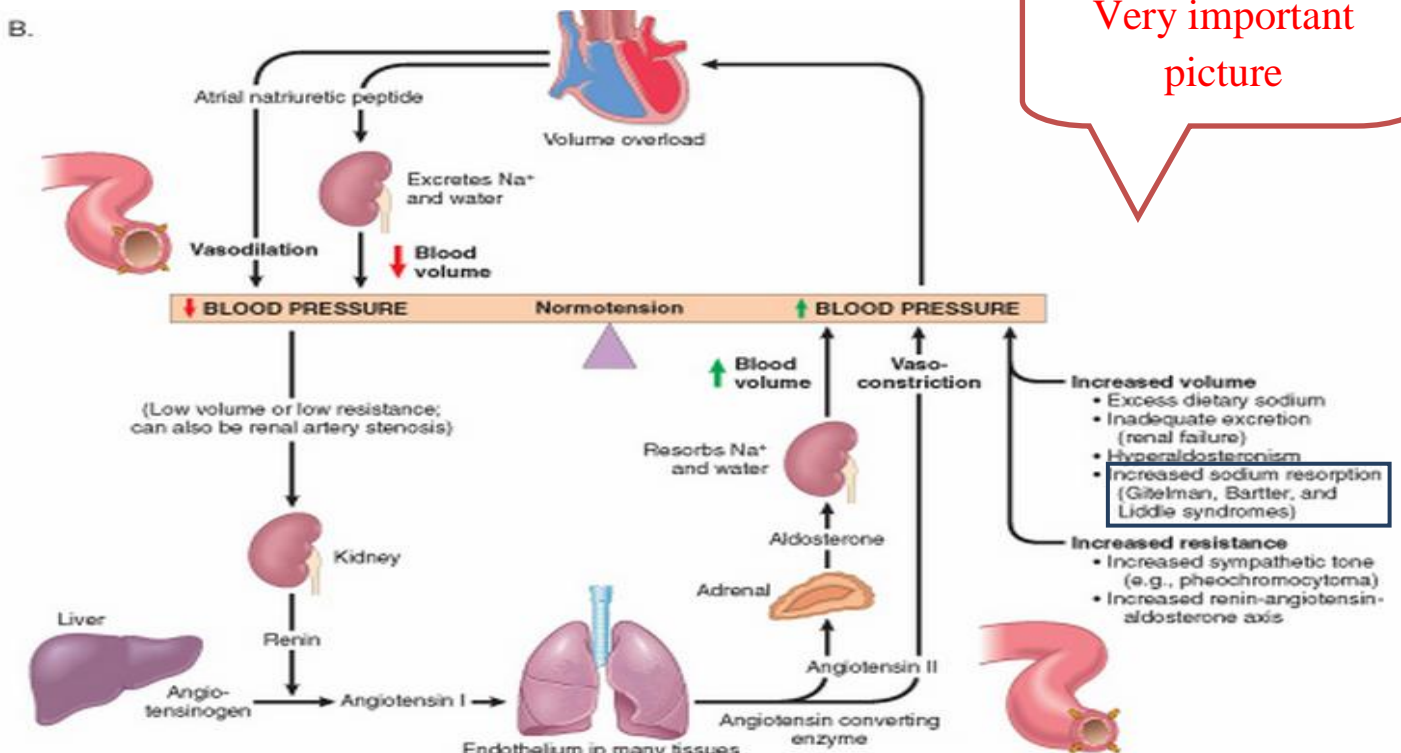
- Psychogenic
- Increased intracranial pressure
- Sleep apnoea
- Acute stress, including surgery

## Blood pressure regulation



Blood pressure is a function of cardiac output and peripheral vascular resistance → two hemodynamic variables that are influenced by multiple genetic, environmental, and demographic factors

## Renin-angiotensin-aldosterone and atrial natriuretic peptide rule

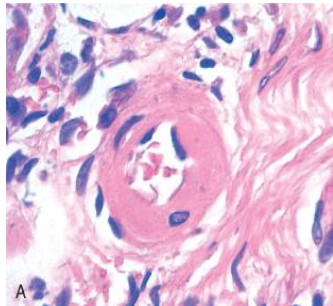


Remember:

- Peripheral resistance is regulated predominantly at the level of **arterioles**.
- **Defective sodium excretion** in the presence of **normal arterial pressure** is probably a **key** initiating event for the pathogenesis of most forms of hypertension.

**Vascular pathology (how does it affect the vessels?):**

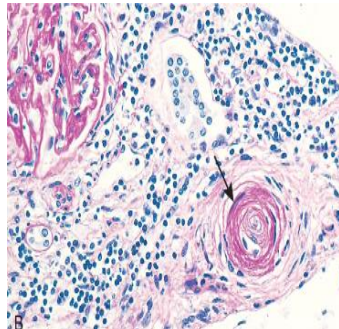
- Accelerates atherogenesis (the formation of atherosclerosis)
- Arteriosclerosis (particularly in kidney) leading to:
  - thick wall
  - narrow lumen
- Microscopically it can be either:
  - Hyaline arteriosclerosis (usually in essential HTN)



We see:

- Arteriolar wall is thickened
- Increased protein deposition (hyalinized)
- Markedly narrowed lumen

- Hyperplastic arteriosclerosis (usually in malignant HTN)



We see:

- Onion skinning (arrow) (layered) causing luminal obliteration (blocking)

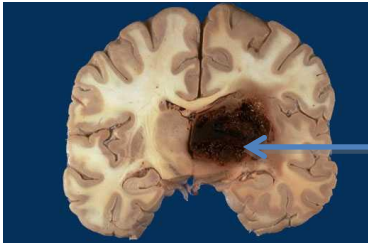
**Malignant hypertension:**

- BP > 210/120 mmHg (diastolic pressure should be more than 120)
- It is a hypertensive emergency which can complicate any type of HTN
- Leads to:
  - Rapidly progressive end organ damage
  - Renal failure
  - Hypertensive encephalopathy (increased cerebral perfusion from the loss of blood-brain barrier integrity because of hypertension which results in cerebral edema)
  - Left ventricular failure
  - Retinal hemorrhages and exudates, with papilledema (optic disc swelling)
  - Leads to death in 1 or 2 years if untreated



## Other major complications of HTN:

- Coronary heart disease
- Cerebrovascular accidents (cerebral infarction) (stroke)



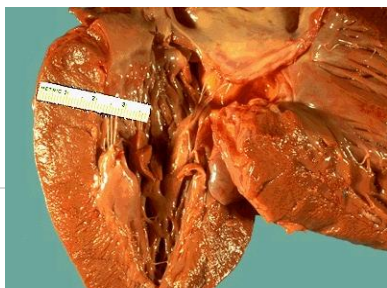
Hemorrhagic necrosis

- Cardiac hypertrophy and heart failure (hypertensive heart disease)
- Aortic dissection (a serious condition in which there is a separation of the aorta walls)
- Renal failure
- Retinopathy
- 

\*Reduction of blood pressure dramatically reduces the incidence of death rates from IHD, heart failure, and stroke.

## Systemic hypertensive heart disease:

- Pathogenesis:  
Hypertensive heart disease → increased demands placed on the heart by hypertension → pressure overload and ventricular hypertrophy
- Caused by:
  - Systemic HTN causes: Hypertensive heart disease in the left heart (most common)
  - Pulmonary HTN can cause: right-sided hypertensive heart disease or cor pulmonale
- Criteria:
  - Left ventricular hypertrophy (usually concentric) in the absence of other cardiovascular pathology
  - History or pathologic evidence of hypertension
- Findings:
  - LV wall > 2cm
  - Weight of the heart > 500g (normal weight ~300g)
  - Long-term: dilation and wall thinning
- Treatment of HTN helps recovery



### We see:

- Left ventricle very thickened (slightly greater than 2cm) but the rest of the heart is not greatly enlarged
- This is typical of hypertensive heart disease
- The hypertension creates a greater



Questions :

**What is essential hypertension?**

Elevated blood pressure with no known cause

**What is characteristic of malignant hypertension?**

An accelerated course with multiple organ damage

**What is Liddle syndrome?**

syndrome is caused by mutation of ENaC leading to hypertension causing increased distal tubular resorption of sodium induced by aldosterone .

**Which one of those people are most likely to have hypertension?**

- 1/ Young adult female
- 2/ Africans-Americans
- 3/ Young healthy male