Hypertension

Persistently **elevated arterial blood pressure** [BP]

Associated with both functional and morphologic alteration of blood vessels

- Arterial BP - generated by the interplay between blood flow and the resistance to blood flow
- Measured in mmHg

2 types of arterial blood pressure

- Systolic BP (SBP)- achieved during cardiac contraction
- Diastolic BP (DBP)- achieved after contraction when the cardiac chambers are filling

SBP – DBP = pulse pressure (measure of arterial wall tension)

  - Cardiac output - major determinant of SBP
  - Total peripheral resistance determines DBP

- Mean arterial pressure [MAP] - Average pressure throughout the cardiac cycle of contraction
- During cardiac cycle 2/3rd time spent in diastole and 1/3rd time in systole

MAP= [SBP (1/3)] +[DBP (2/3)]

BP= Cardiac output × Total peripheral resistance

Clinical classification of hypertension

Category	Systolic (mm Hg)	Diastolic (mm Hg)
Normal	< 130	<85
High normal	130-139	85-89
Hypertension		
• Mild (Stage 1)	140-159	90-99
• Moderate (Stage 2)	160-179	100-109
• Severe (Stage 3)	180-209	110-119
• Very severe (Stage 4)	≥ 210	≥ 120
Malignant hypertension	> 200	≥ 140

Etiological classification of hypertension

A. Primary essential hypertension

- Genetic factors
- Racial and environmental factors
- Risk factors modifying the course of HT

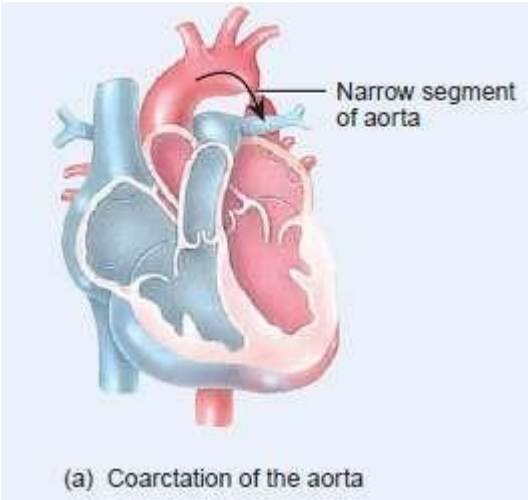
B. Secondary hypertension

- Renal – Renovascular

  Renal parenchymal disease
- Endocrine - Adrenocortical hyperfunction

  Hyperparathyroidism

  Oral contraceptives
- Coarctation of aorta
- Neurogenic



(a) Coarctation of the aorta

Clinical classification of primary and secondary hypertension

Benign hypertension

- Observed in 95% of patients
- Slow rise in BP taking years to develop

Malignant/ accelerated hypertension

- Observed in 5-10% of patients
- Rapid rise in BP to 200/140 mm Hg or more
- If left untreated, patient's life expectancy decreases

Symptoms

When BP is severe, following symptoms are observed

- Nose bleeding
- Irregular heart beat
- Head ache
- Dizziness
- Fatigue
- Flushed face
- Breathing difficulties
- Strong tendency to uinate
- Vertigo, tinnitus, etc.,

Malignant hypertension is characterized by

- Pulsating headache beneath the eye
- Visual disturbance
- Nausea and vomiting
- Disturbed sleep

Pathogenesis

BP is the product of

- Cardiac output
- Total peripheral vascular resistance
- **Cardiac output**

- Volume of blood that circulates through systemic blood vessels each minute
    - Dependent on stroke volume (SV)
    - SV - Volume of blood ejected by the left ventricle during each contraction
- Peripheral resistance depends on
    - Viscosity of blood
    - Diameter of the blood vessel
    - Compliance
- High viscosity - high pressure to pass through vascular bed
- High pressure to pass through constricted and non-complaint blood vessels

BP is controlled by

- Neural component
- Peripheral auto regulatory mechanism
- Humoral mechanism
- Vascular endothelial mechanism

Neural component

- Both CNS & ANS controls BP

Centers in CNS are

  - Vasomotor center in Medulla
  - Vagal nucleus
  - Area postrema
  - Nuclues tractus solitarii

– Maintenance of BP by sympathetic nervous system through α and β adrenergic receptors

++ post synaptic α_1 receptors — vasoconstriction — ↑ BP

++ pre synaptic α_2 receptors — negative feedback on NA release

++ β_1 in heart — ↑ HR and contractility

++ β_2 in arterioles and venules — vasodilation

Change in BP senses by barro receptors in carotid artery and aortic arch

- Respond to change in arterial pressure
- Transmitted to brain through IX cranial nerve and vagus nerve
- ↑ discharge from barroreceptors − depression of vasomotor center − excitation of nucleus ambiguus − reverts change in BP

Peripheral auto regulatory mechanism

- Normal case – volume and pressure adaptive mechanism of kidney maintains BP

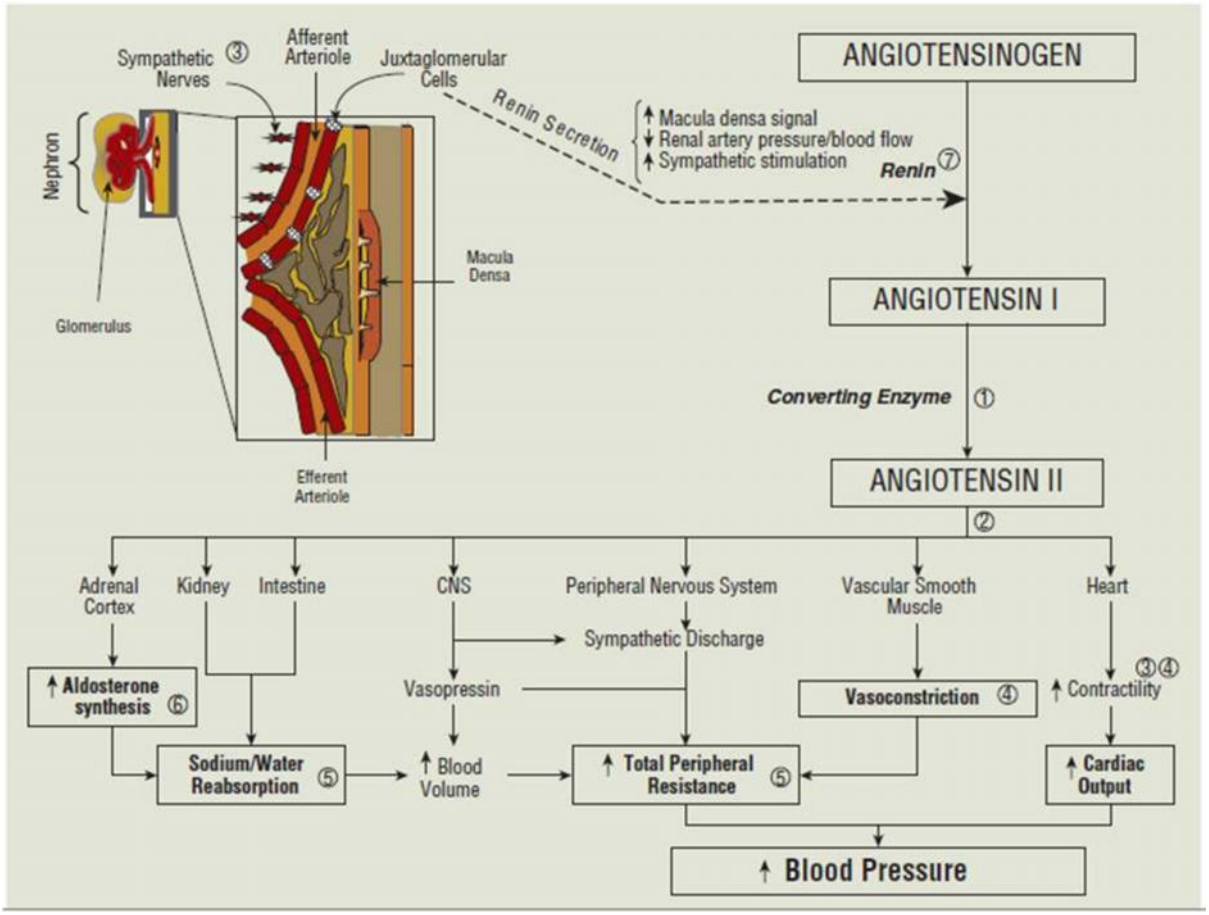
↓ BP – adaptation of kidney–more Na^+ and H_2O retention

↑ BP – adaptation of kidney – Na^+ and H_2O excretion – ↓ blood volume & cardiac output

Humoral mechanism

- Renin Angiotensin Aldosterone system
- Natriuretic hormone
- Insulin resistance and hyperinsulinemia

Renin-Angiotensin-aldosterone system



Natriuretic hormone

- Inhibits Na^+/K^+ ATP ase
- Interferes with Na^+ transport across cell membrane
- ↑ Na^+ in body fluids - ↑ Natriuretic hormone - ↑ urinary excretion of Na^+ and H_2O
- Blocks active transport of Na^+ out of the walls of arterioles - ↑ vascular tone and BP

Insulin resistance and hyper insulinemia

- Causes Na^+ retention
- Increases sympathetic activity
- Increases vascular resistance
- Increases BP

Vascular endothelial mechanism

- Regulates blood vessel tone
- Vasodilating substances – Nitric oxide, Prostacyclin (PI_2) and bradykinin – Hypotension
- Vasoconstrictors – Angiotensin II and Endothelin I - ↑ BP

Effect of dietary Na^+ Ca^{2+} K^+ on BP

- ↑ intracellular Ca^{2+} - alters smooth muscle function on blood vessels - ↑ Peripheral vascular resistance
- K^+ depletion - ↑ Peripheral vascular resistance
- ↑ Na^+ in body fluids & in arterial wall - ↑ BP

Complications of HT

- Blood vessels - Large arterioles dialatess
  - Smaller arterioles get damaged
- Eye – Arterial narrowing, haemmorhage
- Heart - Hypertropy of left ventricles, heart failure
- Kidney – Nephrosclerosis, renal damage, death in uremia
- Brain – Rupture of damaged blood vessels, encephalopathy, cerebral edema

Summary

- Persistently elevated arterial blood pressure is called hypertension
- Hypertension can be classified as benign or malignant or accelerated hypertension
- HT can also be classifies as primary and secondary HT based on etiology
- BP is controlled by neuronal component, humoral mechanism, peripheral autoregultory mechanism and vascular endothelial mechanism
- Any defects in the functioning of these mechanisms leads to the development of HT
- HT is affects kidneys, blood vessels, brain and predisposed to many cardiovascular diseases