



# **PATHOLOGY TEAM 430**

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Note

The important notes with

**ORANGE COLOR**

## 4th Lecture:

# Hypertension

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### Introduction

- ▶ Common problem (25% of population)
- ▶ Asymptomatic until late- Silent Killer – painless –
- ▶ Leading risk factor – MI & Stroke
- ▶ Complications alert to diagnosis but late...
- ▶ Hypertension and Hypertensive Vascular Disease

**Hypertension definition:** a sustained diastolic pressure more than 90 mm Hg or a sustained systolic pressure in excess of 140 mm Hg (>140/90).

- ▶ Hypertension is considered as an important risk factor of:
  - Coronary heart disease.
  - Cerebrovascular accidents (stroke)
  - cardiac hypertrophy
  - Congestive heart failure.
  - Aortic dissection.
  - Renal failure.
  - Retinopathy

## Some of risk factors which can lead to Hypertension:

- Hereditary
- Race. African-Americans
- Gender. Men & postmenopausal women
- Age
- Obesity
- Diet, particularly sodium intake

## Other factors associated with Hypertension include:

- Heavy alcohol consumption
- Diabetes
- Use of oral contraceptives
- Sedentary or inactive lifestyle

## Types of hypertension based on the etiology:

- I. **Primary/Essential Hypertension (95%)** : Mechanisms largely unknown. It is idiopathic.
- II. **Secondary Hypertension (5-10%)**
  - ▶ **Renal**
    - Glomerulonephritis
    - Renal artery stenosis
    - Adult polycystic disease
    - Chronic renal disease, renin producing tumors.

## ► Endocrine

- Adrenocortical hyperfunction  
(Cushing syndrome, primary aldosteronism, congenital adrenal hyperplasia which is an example of gene defect affecting aldosterone metabolism)
- Exogenous hormones  
(glucocorticoids, estrogen [including pregnancy-induced and oral contraceptives] and sympathomimetics)
- Hyperthyroidism "Thyrotoxicosis"
- Hypothyroidism "Myxedema"
- Pheochromocytoma  
"damage to adrenal gland which can lead to catecholamine in the blood"
- Acromegaly

## ► Cardiovascular

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

## ► Neurogenic

Psychogenic

Intracranial pressure

Sleep apnea, acute stress "including surgery"

## Types of hypertension based on the clinical features:

Benign (90 to 95%)	Malignant (5%)
<ul style="list-style-type: none"><li>◦ Modest level.</li><li>◦ Fairly stable over years to decades.</li><li>◦ Compatible with long life.</li></ul>	<ul style="list-style-type: none"><li>◦ Diastolic pressure over 120mmHg</li><li>◦ Rapidly rising blood pressure.</li><li>◦ Severe hypertension (diastolic&gt;120)</li><li>◦ Renal failure.</li><li>◦ Retinal hemorrhages and exudates (w/wo papilledema).</li><li>◦ Leads to death in 1 or 2 years if untreated.</li></ul>

## Pathogenesis of Hypertension

Blood pressure (BP) = Cardiac Output x Peripheral Resistance

*Blood pressure is a function of cardiac output and peripheral vascular resistance*

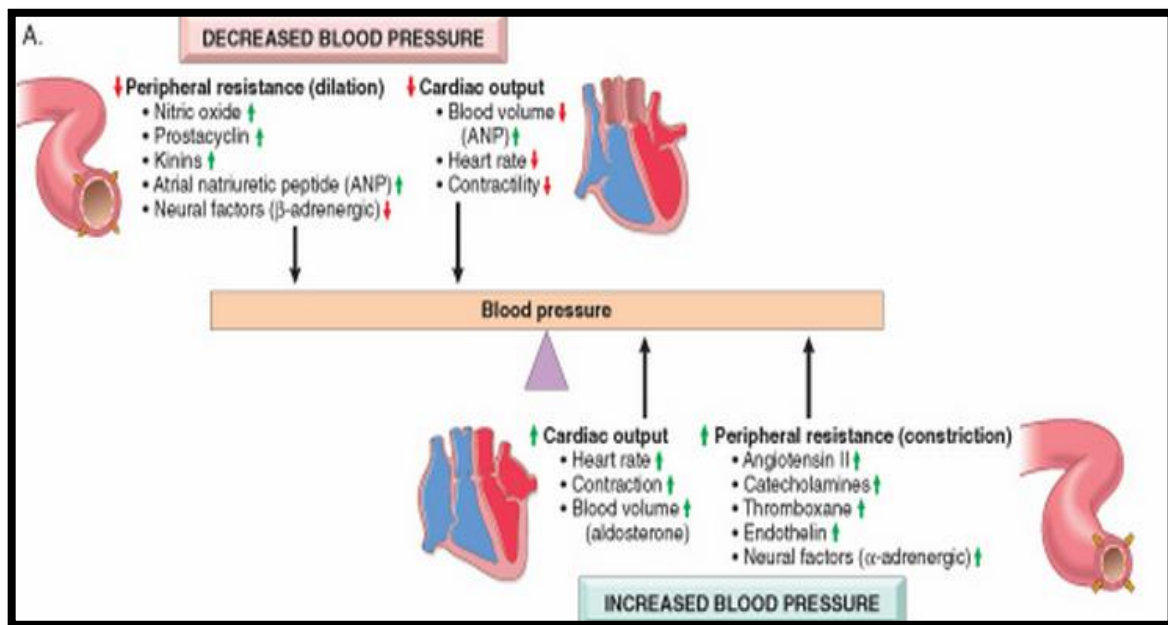
*→ two hemodynamic variables that are influenced by multiple genetic, environmental, and demographic factors.*

### The factors:

- ▶ Endocrine Factors :Renin, Angiotensin, ADH, Aldosterone
- ▶ Neural Factors : Sympathetic & Parasympathetic
- ▶ Blood Volume :Sodium, Mineralocorticoids
- ▶ Cardiac Factors : Heart rate & Contractility

# Regulation of Blood Pressure

## "VERY IMPORTANT PICTURE"



### Note:

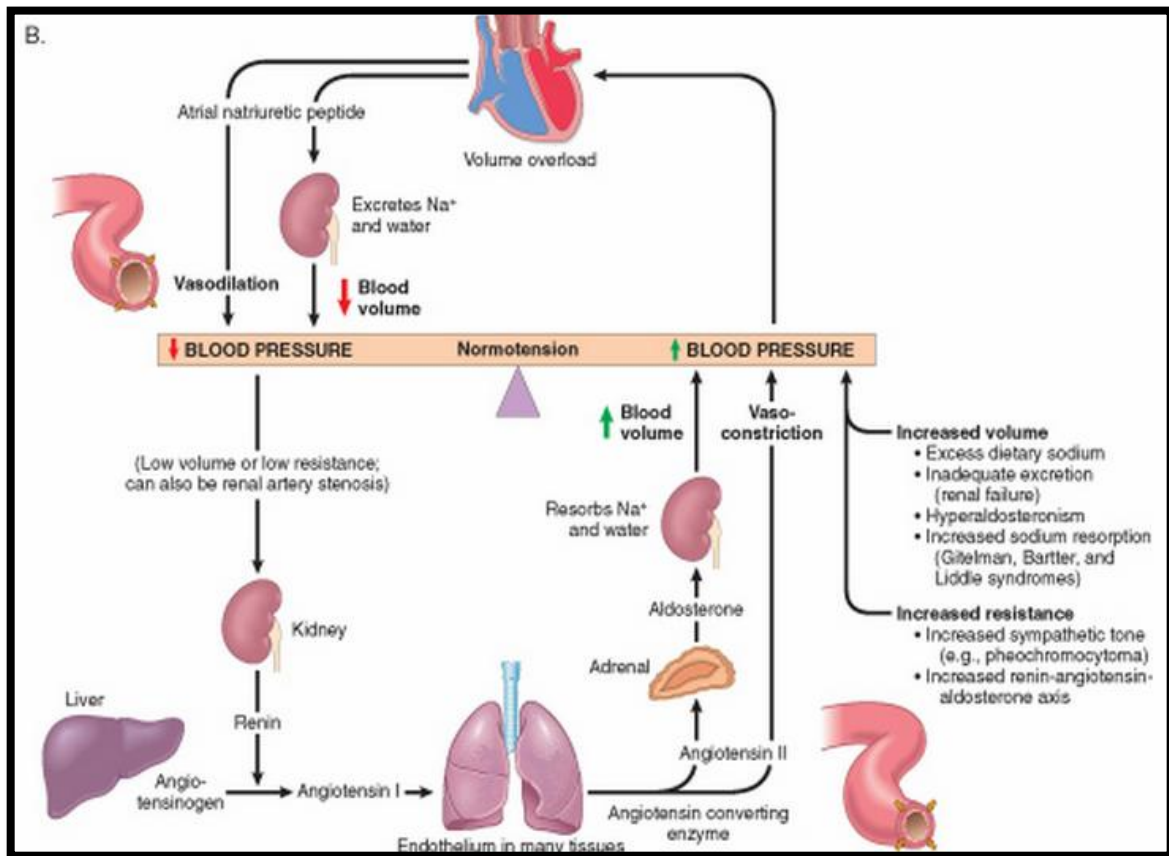
The blood pressure is influenced by the changes of cardiac output & peripheral resistance. So, any change in these factors can lead to changes in the blood pressure "either ↑ or ↓".

Peripheral resistance is regulated predominantly at the level of the arterioles. Lower volumes or pressures result in a reduced glomerular filtration rate in the kidney with increased resorption of sodium by proximal tubules.

Atrial natriuretic peptide (ANP), secreted by heart atria in response to volume expansion (e.g., in heart failure) inhibits sodium reabsorption in distal tubules and causes global vasodilation.

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

"VERY IMPORTANT PICTURE"



## Postulated mechanisms of Essential Hypertension

1. Defect in sodium excretion
2. Defect in cell membrane function:
  - Na/Ca transport
  - Increased vasoconstrictive response
3. Increased sympathetic response

### Remember !!

Peripheral resistance is regulated predominantly at the level of the arterioles

**Reduced renal sodium excretion** in the presence of normal arterial pressure is probably a **key initiating event**; it is a final common pathway for the pathogenesis of most forms of hypertension

## Vascular pathology of Hypertension

- Accelerate atherogenesis
- Arteriosclerosis (particularly in the kidney), lead to thick wall and narrow lumen
- It can be either:
  - Hyaline ( in benign HTN)
  - Hyperplastic "onionskinning" ( in malignant HTN)

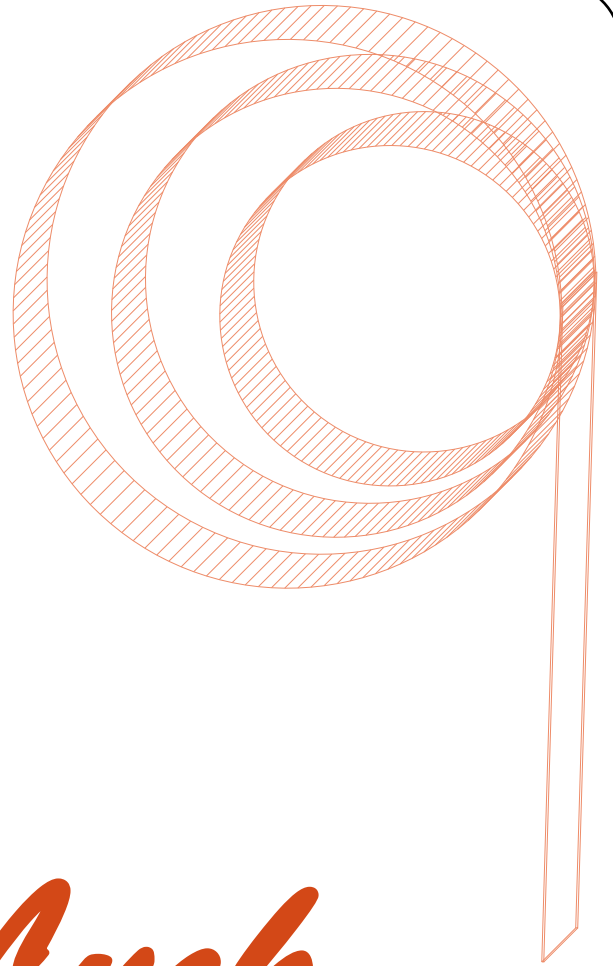
## Major complications of Hypertension

- ▶ Coronary heart disease
- ▶ Cerebrovascular accidents
- ▶ Cardiac hypertrophy and heart failure (*hypertensive heart disease*)
- ▶ Aortic dissection
- ▶ Renal failure
- ▶ Retinopathy



## Hypertension "Systemic hypertensive cardiac disease"

- History of hypertension or extracardiac anatomical evidence of HTN
- LVH "left ventricular hypertrophy": concentric with absence of other cause of LVH
- The free LV wall is  $> 2\text{cm}$  and the weight of the heart is  $> 500$  grams
- Long-term: dilatation and wall thinning
- Treatment of HTN helps recovery



*Good Luck*

