Cardiovascular system block

Pathology team 431

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Pathology of hypertension

Objective:

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

Know the etiology, 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.

Definition of hypertension (HTN):

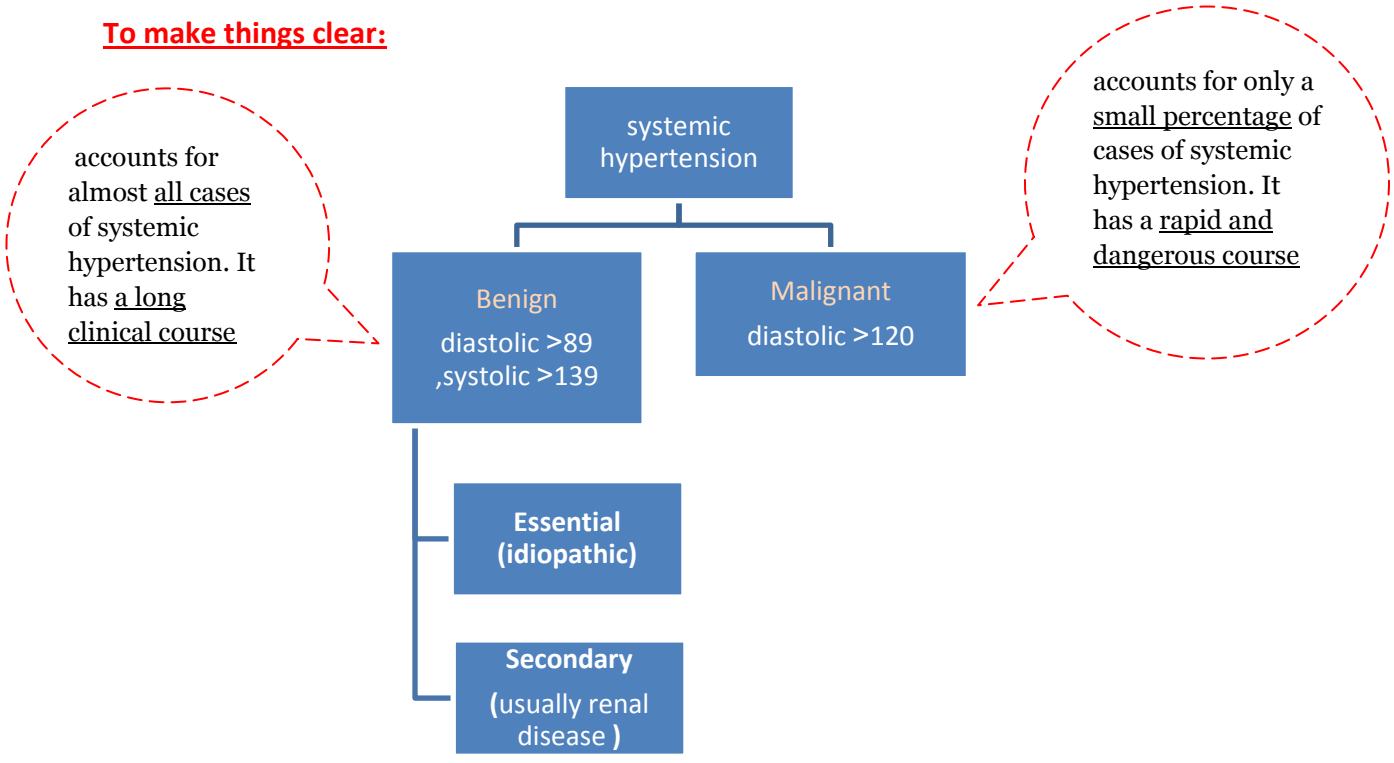
Blood pressure is a function of cardiac output and peripheral vascular resistance.

- Blood pressure BP = CO x TPR

- Hypertension (HTN) is usually considered when there is :
  - A sustained diastolic pressure **greater than 89 mm** Hg

  OR
  - A sustained systolic pressure in **excess of 139 mm Hg**

To make things clear:



Classification of Blood Pressure:

Category	Systolic		Diastolic
Normal	< 120	And	<80
Pre hypertension	120-139	Or	80-90
Hypertension, Stage 1	140-159	Or	90-99
Hypertension, Stage 2	>160	Or	> =100

Risk Factors:

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

Other factors associated with HTN include:

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

Types and Causes :

Primary (Essential) Hypertension

- 85 - 90% of hypertensives have essential hypertension.
- Common in blacks or with positive family history.
- Worsened by:
  - Obesity.
  - Increased Na intake.
  - Stress.
  - Oral contraceptive use or
  - Tobacco use.

Causes are multifactorial and results from the combined effects of :

- multiple genetic polymorphisms
- interacting environmental factors

Secondary Hypertension

- 10 - 15% of hypertensives .
- Increased BP secondary to another disease process.

Causes

- **Renal:**
  - Acute glomerulonephritis
  - Chronic renal disease
  - Polycystic disease
  - Renal artery stenosis
  - Renal vasculitis
  - Renin-producing tumors

- **Endocrine:**
  - Adrenocortical hyper function (Cushing syndrome, primary aldosteronism, congenital adrenal hyperplasia)
  - Exogenous hormones (glucocorticoids, estrogens)
  - Pheochromocytoma
  - Acromegaly
  - Hypothyroidism (myxedema)
  - Hyperthyroidism (thyrotoxicosis)
  - 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

Signs and Symptoms:

- Primary hypertension is asymptomatic until complications develop
- Signs/Symptoms are non-specific
    - Result from target organ involvement
- Dizziness, flushed face, headache, fatigue, epistaxis, nervousness

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 for the pathogenesis of most forms of hypertension.

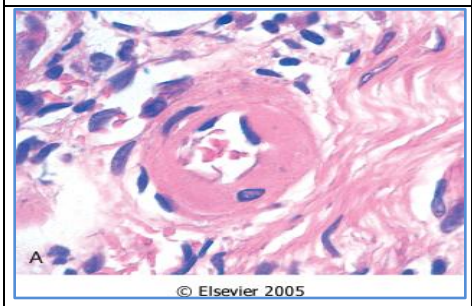
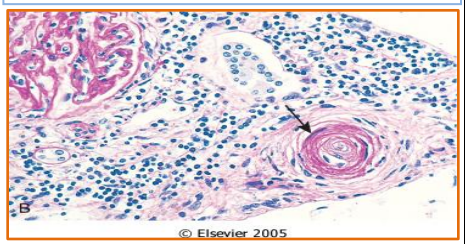
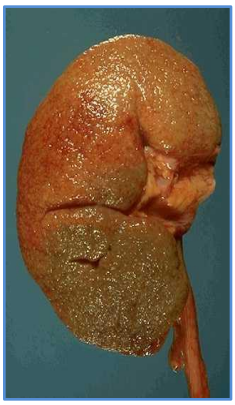
Hypertension Pathology:

- Increased BP → inflammation, sclerosis of arteriolar walls → narrowing of vessels → decreased blood flow to major organs.
- Left ventricular overwork → hypertrophy, CHF
- Nephrosclerosis → renal insufficiency, failure

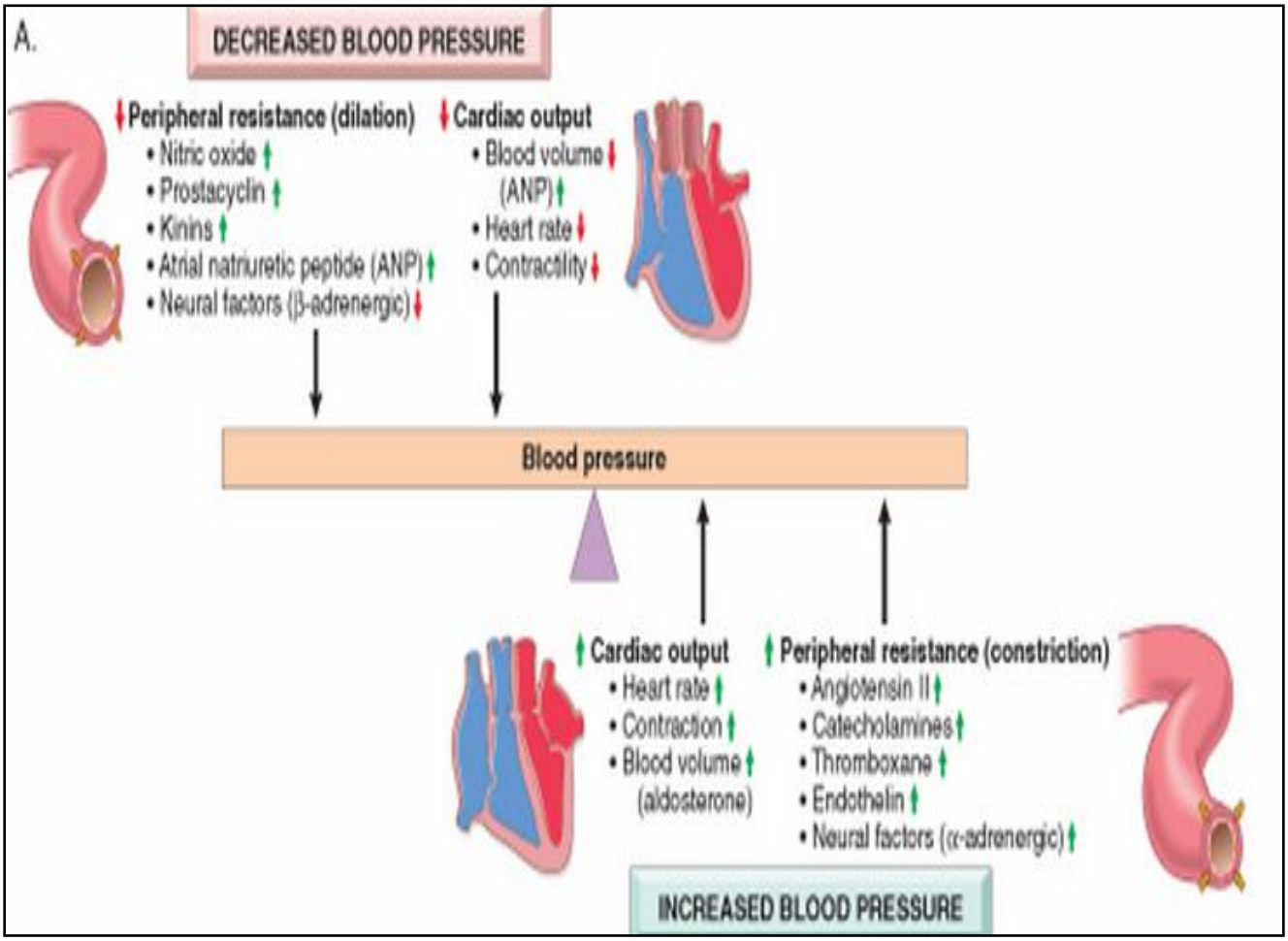
Major complications

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

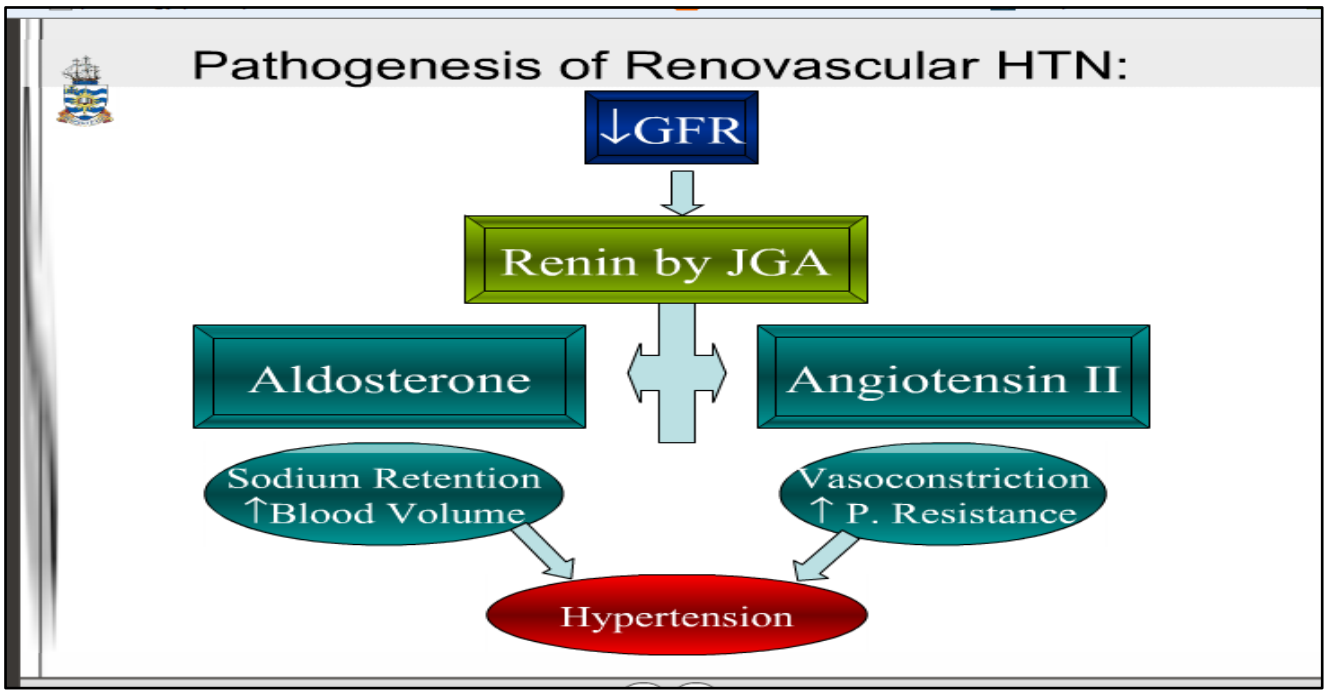
Consequences of Hypertension:

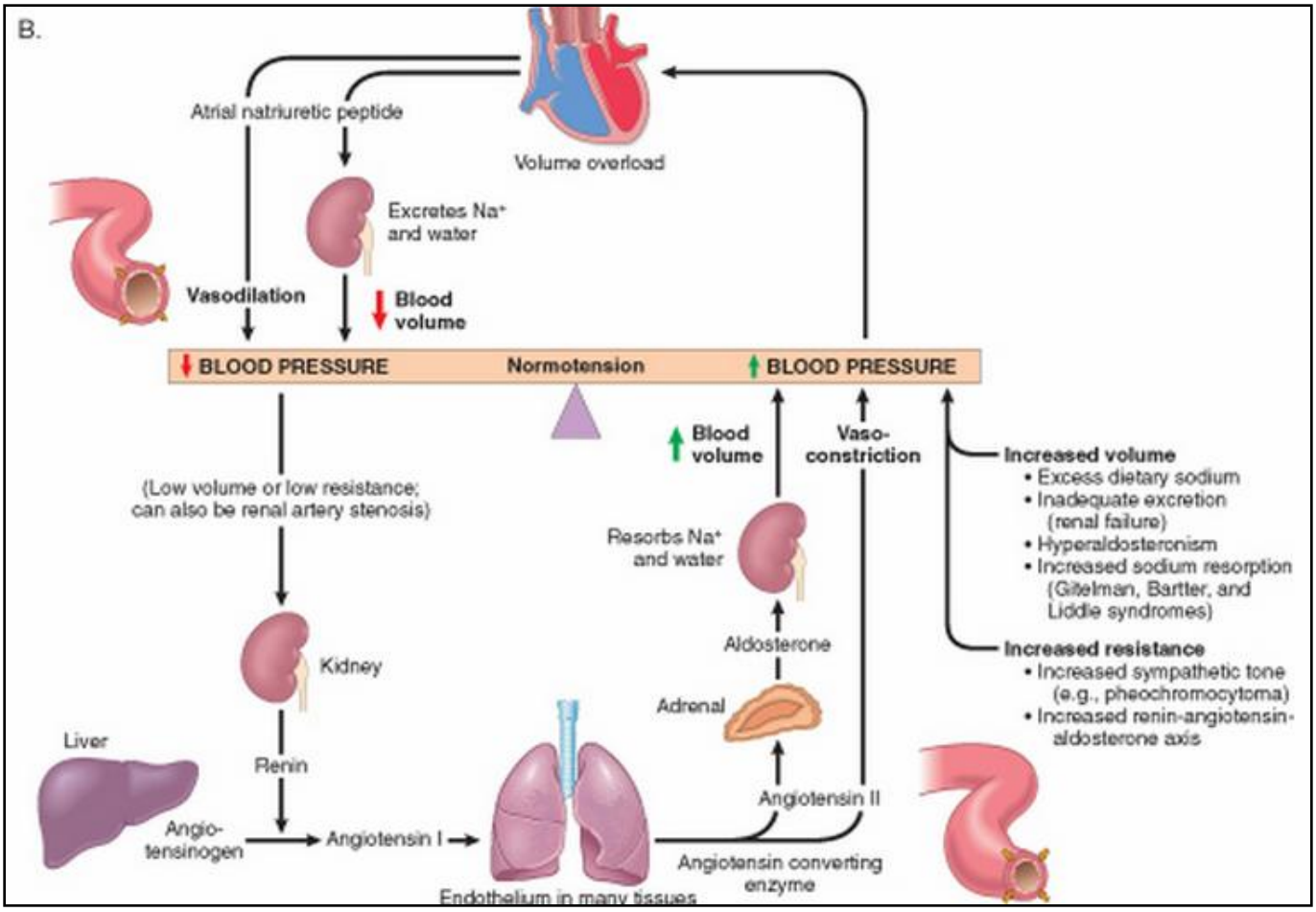
Blood Vessels	heart	Kidney	Eye	Brain
Vascular pathology • Accelerate atherogenesis • Arteriosclerosis (particularly in the kidney), lead to thick wall and narrow lumen • It can be either: – **Hyaline** (in benign HTN) – **Hyper plastic** (in malignant HTN)	Left Ventricular Hyperplasia (LVH), hypertensive cardiomyopathy → Ischemic Heart Diseases, Myocardial Infarction.	Benign/ Malignant Nephrosclerosis	Hypertensive Retinopathy – **Grade I**: Thickening of arterioles. – **Grade II**: Focal Arteriolar spasms. Vein constriction. – **Grade III:** Hemorrhages (Flame shape), dot-blot and Cotton wool and hard waxy exudates. – **Grade IV:** Papilloedema	hemorrhage, infarction, splinter hemorrhage and Lacunar infarcts.
 Hyaline arteriolosclerosis : The arteriolar wall is thickened with increased protein deposition  Hyperplastic arteriolosclerosis (onion-skinning; arrow) causing lumenal obliteration (arrow).		 Benign		 Cerebral infarction (stroke)

Blood Pressure Regulation: The Dr. said it is just for your information



The effect of cardiac output and peripheral resistance on blood pressure





Renin-angiotensin-aldosterone and atrial natriuretic peptide rule

- Cardiac output is affected by blood volume, itself strongly dependent on sodium concentrations. **Peripheral resistance is regulated predominantly at the level of the arterioles** and is influenced by neural and hormonal inputs.
- The kidneys (primarily) and adrenals (secondarily) are central players in blood pressure regulation.
- The *kidney* influences peripheral resistance and sodium homeostasis primarily through the renin-angiotensin system
- lower volumes or pressures result in a **reduced *glomerular filtration*** *rate* in the kidney with ***increased resorption*** **of sodium** by proximal tubules; these latter two effects putatively conserve sodium and **expand the blood volume.**
- **Renin** released by the kidneys catabolizes *plasma angiotensinogen* to *angiotensin I,* which in turn is converted to *angiotensin II* by angiotensin-converting enzyme in the periphery. **Angiotensin II (potent vasoconstrictor)** raises blood pressure by: **increasing peripheral**

resistance, also **increasing blood volume** by stimulating aldosterone secretion in the adrenals; increasing reabsorption of sodium.

- **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.

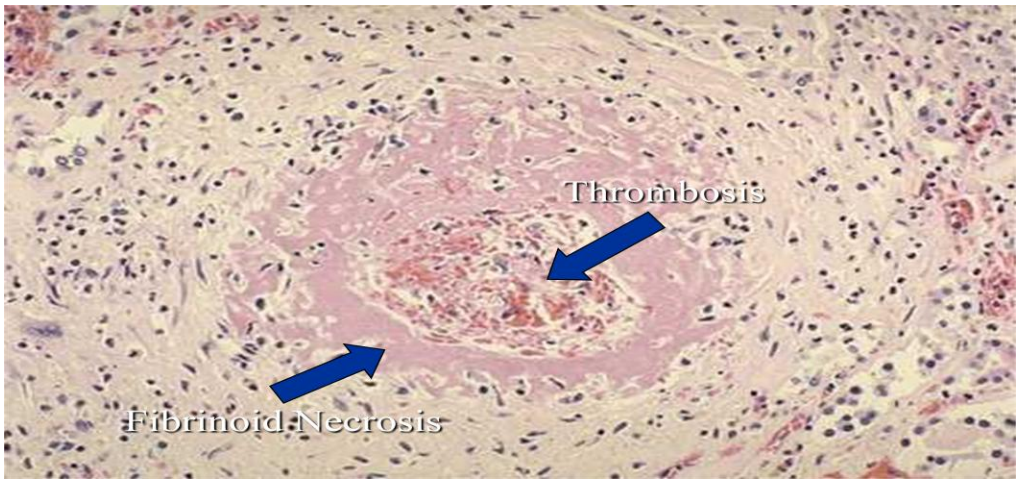
Malignant Hypertension:

-
- Very high blood pressure
- B.P > 210/120 mm Hg
- It is called malignant, because it requires immediate treatment.
- May complicate any type of HTN.
- Microscopically: Necrotizing arteriolitis, Intravascular thrombosis.

It leads to :

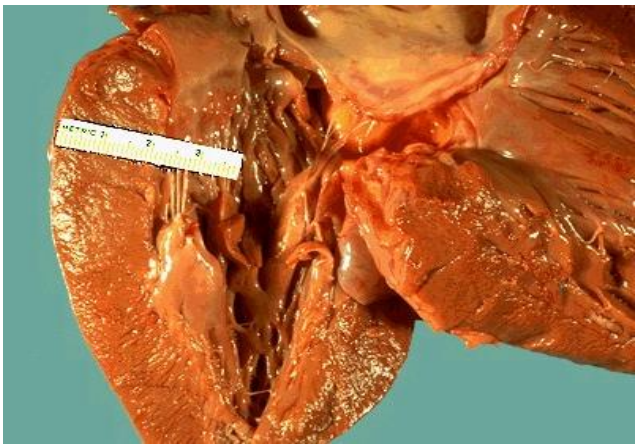
- Rapidly progressive end organ damage.
- Renal failure
- Hypertensive encephalopathy.
- Left ventricular failure.
    - Retinal hemorrhages and exudates, with or without papilledema
    - Leads to death in 1 or 2 years if untreated.

Necrotizing arteriolitis

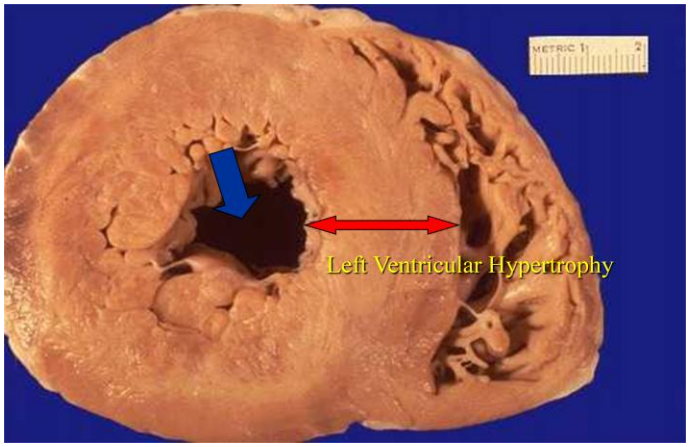


Systemic Hypertensive Heart Diseases:

- Criteria :
  - left ventricular hypertrophy (usually concentric) in the absence of other cardiovascular pathology
  - History or pathologic evidence of hypertension
- The free LV wall is > 2cm and the weight of the heart is > 500 grams
- Long-term: dilatation and wall thinning
- Treatment of HTN helps recovery



- This left ventricle is very thickened (slightly over 2 cm in thickness), but the rest of the heart is not greatly enlarged.
- This is typical for hypertensive heart disease.
- The hypertension creates a greater pressure load on the heart to induce the hypertrophy.



- The left ventricle is markedly thickened in this patient with severe hypertension that was untreated for many years.
- The myocardial fibers have undergone hypertrophy.

Table 3. Lifestyle Modifications to Manage Hypertension*

Modification	Recommendation	Approximate Systolic BP Reduction, Range
Weight reduction	Maintain normal body weight (BMI, 18.5-24.9)	5-20 mm Hg/10-kg weight loss[23,24]
Adopt DASH eating plan	Consume a diet rich in fruits, vegetables, and low-fat dairy products with a reduced content of saturated and total fat	8-14 mm Hg[25,26]
Dietary sodium reduction	Reduce dietary sodium intake to no more than 100 mEq/L (2.4 g sodium or 6 g sodium chloride)	2-8 mm Hg[25-27]
Physical activity	Engage in regular aerobic physical activity such as brisk walking (at least 30 minutes per day, most days of the week)	4-9 mm Hg[28,29]
Moderation of alcohol consumption	Limit consumption to no more than 2 drinks per day (1 oz or 30 mL ethanol [eg, 24 oz beer, 10 oz wine, or 3 oz 80-proof whiskey]) in most men and no more than 1 drink per day in women and lighter-weight persons	2-4 mm Hg[30]

Abbreviations: BMI, body mass index calculated as weight in kilograms divided by the square of height in meters; BP, blood pressure; DASH, Dietary Approaches to Stop Hypertension.
*For overall cardiovascular risk reduction, stop smoking. The effects of implementing these modifications are dose and time dependent and could be higher for some individuals.

Summary from robbins

- Blood pressure is regulated by the combined influences of cardiac output (largely related to blood volume) and vascular resistance. Blood volume is dependent on renal sodium homeostasis, and arteriolar vascular resistance is regulated by neural and hormonal inputs.

- Renin is a major regulator of normal blood pressure; it is secreted by kidneys in response to reduced afferent aretriole pressure or glomerular filtration of sodium. Renin converts angiotensinogen to angiotensin II; angiotensin II regulates blood pressure by increasing vascular SMC contraction and by increasing aldosterone secretion to increase renal sodium resorption.

- Essential hypertension represents 90% to 95% of cases of hypertension and is a complex, multifactorial disorder resulting most likely from the combined effect of mutations or polymorphisms at several gene loci (e.g., sodium resorption, renin-angiotensin system, aldosterone) in association with a variety of environmental influences.Secondary hypertension is caused by diseases of the kidneys or endocrine glands.

Questions

1. Primary hypertension is worsened by:
    a) Cancers
    b) Chemotherapy
    c) Contraceptive use
    d) Infections

2. Symptoms of Primary hypertension:
    a) Increased intravascular volume
    b) Stress
    c) Cushing syndrome
    d) Asymptomatic

3. malignant hypertension can lead to:
    a) BP > 210/120
    b) Pleural effusion
    c) Renal failure

4. A patient 80-year-old female has had several episodes of syncope, all of which have occurred while she was returning to her room after breakfast. She complains of headache and she feels cold and weak. She takes nitroglycerin in the morning for a history of chest pain, but denies recent chest pain or shortness of breath.
   The most likely method of diagnosis is:
    a) Cardiac catheterization
    b) Blood pressure monitoring
    c) Holter ECG
    d) CT scan