HYPERTENSIVE HEART DISEASE

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BLOOD PRESSURE REGULATION

- Blood pressure is a function of cardiac output and peripheral vascular resistance, both of which are influenced by multiple genetic and environmental factors

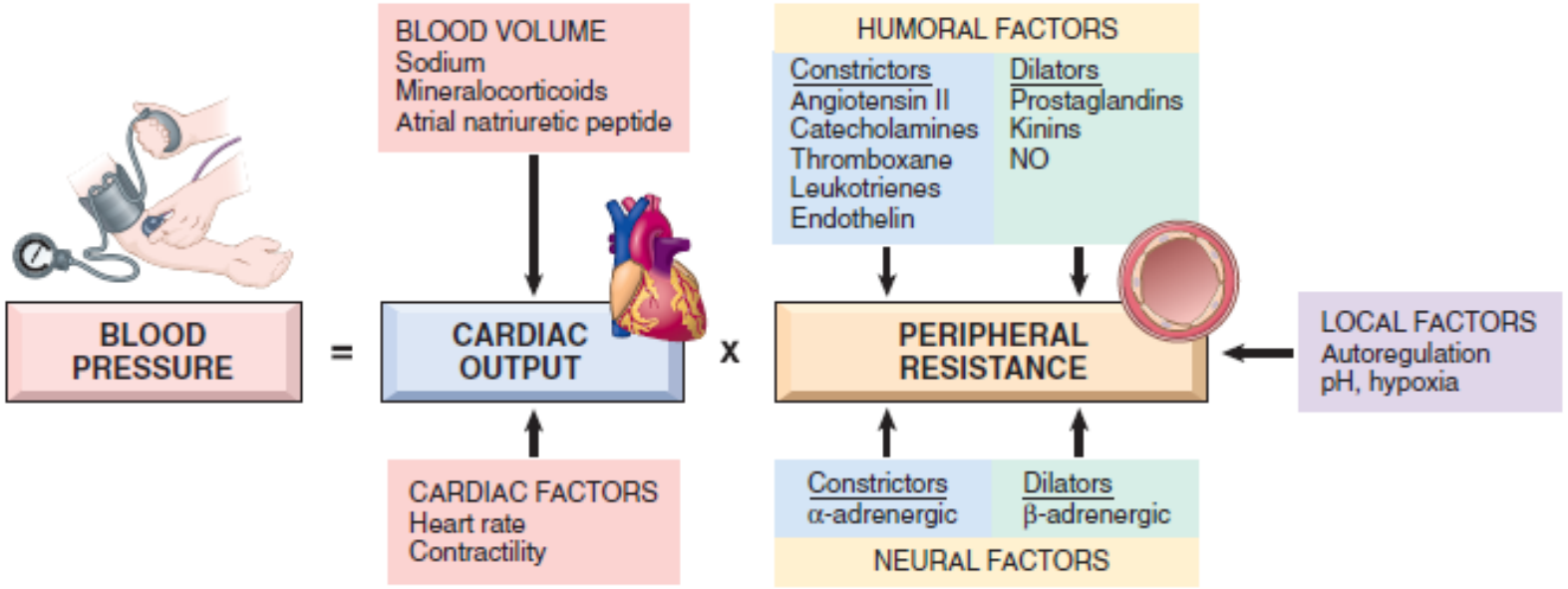


Figure 9–3 Blood pressure regulation.

- Hypertension is a common disorder associated with considerable morbidity and affecting many organs.

- Both the systolic and the diastolic blood pressure are important in determining risk; specifically, according to the newest guidelines, individuals with diastolic pressures above 80 mm Hg or systolic pressures above 120 mm Hg are considered to have clinically significant hypertension.

- 46% of persons in the general population are hypertensive. (based on these newer criteria).

- The prevalence of pathologic effects of high blood pressure increases with age and is also higher in African Americans.
- Without appropriate treatment, some 50% of hypertensive patients die of ischemic heart disease (IHD) or congestive heart failure.

- The risk of hypertension can lead to stroke, atherosclerotic coronary heart disease, cardiac hypertrophy and heart failure (hypertensive heart disease), aortic dissection, multi-infarct dementia, and renal failure.

Table 1: AHA/ACC[a] Guideline Recommendations by Blood Pressure Category

BP[b] Category	Pressure Ranges	Recommendations
Normal BP	<120/<80 mmHg	Promote healthy lifestyle; reassess BP annually.
Elevated BP	120-129/<80 mmHg	Start with nonpharmacologic therapy, reassess BP in 3-6 months.
Stage1 Hypertension	130-139/80-89 mmHg	**ASCVD[c] or 10-year CVD[d] risk ≥10%:** Start with both nonpharmacologic and pharmacologic therapy. Reassess BP in 1 month. If at goal, reassess every 3-6 months. If not at goal, assess for adherence and consider intensification of therapy.
		No ASCVD and 10-year CVD risk <10%: Start with nonpharmacologic therapy, reassess BP in 3-6 months. If not at goal, consider initiation of pharmacologic therapy.
Stage 2 Hypertension	≥140/≥90 mmHg	Start with both nonpharmacologic and pharmacologic therapy. Reassess BP in 1 month. If at goal, reassess every 3-6 months. If not at goal, assess for adherence and consider intensification of therapy.

a: AHA/ACC, American Heart Association, American College of Cardiology.
b: BP, blood pressure.
c: ASCVD, atherosclerotic cardiovascular disease.
d: CVD, cardiovascular disease

https://www.acc.org/latest-in-cardiology/articles/2021/06/21/13/05/new-guidance-on-bp-management-in-low-risk-adults-with-stage-1-htn

Malignant hypertension :

- Usually is severe (i.e., systolic pressures over 200 mm Hg or diastolic pressures over 120 mmHg)
- A small percentage of hypertensive patients (approximately 5%) present with a rapidly rising blood pressure that, if untreated, leads to death in within 1 to 2 years.
- Associated with renal failure and retinal hemorrhages, with or without papilledema.
- It can arise de novo but most commonly is superimposed on preexisting benign hypertension.

TYPES

1. **(95%) are idiopathic (essential hypertension).**

This form is compatible with long life unless a myocardial infarction, stroke, or another complication supervenes.

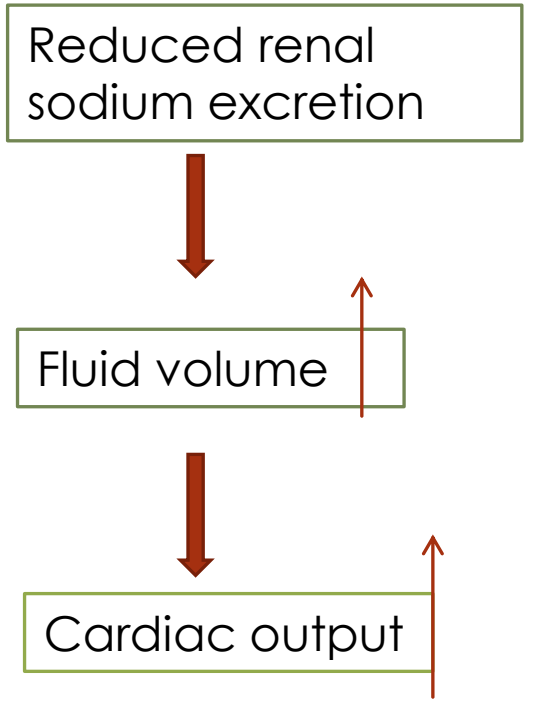
2.(5%) **(secondary hypertension)** .

Table 9–2 Types and Causes of Hypertension (Systolic and Diastolic)

Essential Hypertension
Accounts for 90% to 95% of all cases
Secondary Hypertension
Renal
Acute glomerulonephritis Chronic renal disease Polycystic disease Renal artery stenosis Renal vasculitis Renin-producing tumors
Endocrine
Adrenocortical hyperfunction (Cushing syndrome, primary aldosteronism, congenital adrenal hyperplasia, licorice ingestion) Exogenous hormones (glucocorticoids, estrogen [including pregnancy-induced and oral contraceptives], sympathomimetics and tyramine-containing foods, monoamine oxidase inhibitors) Pheochromocytoma Acromegaly Hypothyroidism (myxedema) Hyperthyroidism (thyrotoxicosis) Pregnancy-induced (pre-eclampsia)
Cardiovascular
Coarctation of aorta Polyarteritis nodosa Increased intravascular volume Increased cardiac output Rigidity of the aorta
Neurologic
Psychogenic Increased intracranial pressure Sleep apnea Acute stress, including surgery

Mechanism

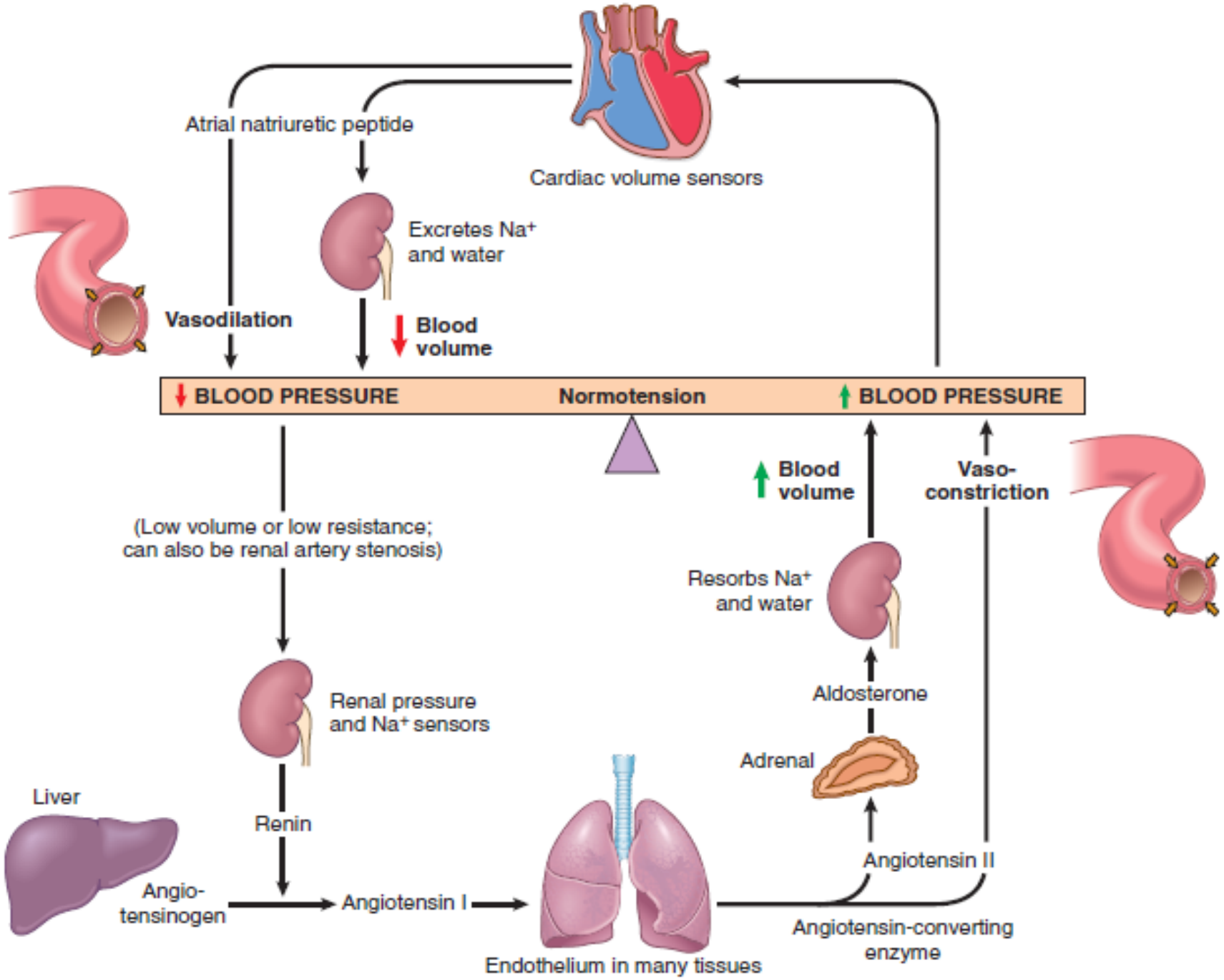
Essential Hypertension Although the specific triggers are unknown, it appears that both altered renal sodium handling and increased vascular resistance contribute to essential hypertension.



- Genetic factors play an important role in determining blood pressure, as shown by familial clustering of hypertension.

- Environmental factors, such as stress, obesity, smoking, physical inactivity, and high levels of salt consumption.

- Evidence linking dietary sodium intake with the prevalence of hypertension in different population groups is particularly strong.


Atrial natriuretic peptide
Cardiac volume sensors
Excretes Na+ and water
Vasodilation
Blood volume
BLOOD PRESSURE
Normotension
BLOOD PRESSURE
Blood volume
Vaso-constriction
(Low volume or low resistance; can also be renal artery stenosis)
Resorbs Na+ and water
Aldosterone
Adrenal
Renal pressure and Na+ sensors
Liver
Renin
Angio-tensinogen
Angiotensin I
Angiotensin II
Angiotensin-converting enzyme
Endothelium in many tissues

Two forms of small blood vessel disease are hypertension-related:

1- Hyperplastic arteriolosclerosis.

2- Hyaline arteriolosclerosis

1-Hyaline arteriolosclerosis

- is associated with benign hypertension.
- It is marked by homogeneous, pink hyaline thickening of the arteriolar walls, with loss of underlying structural detail, and luminal narrowing.

2- Hyperplastic arteriolosclerosis

- Is more typical of severe hypertension.
- Vessels exhibit “onionskin,” concentric, laminated thickening of arteriolar walls and luminal narrowing.
- The laminations consist of smooth muscle cells and thickened, reduplicated basement membrane.

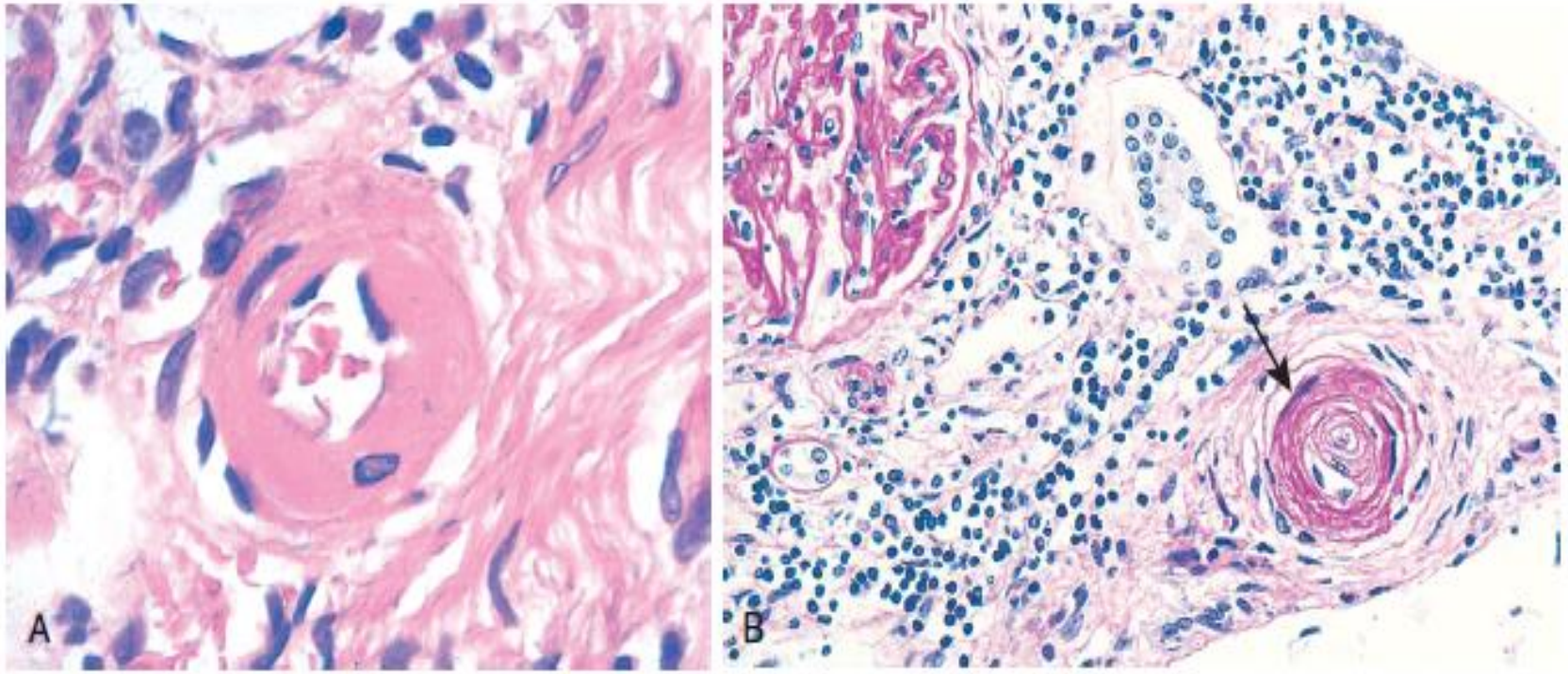


Figure 9–5 Hypertensive vascular disease. **A,** Hyaline arteriolosclerosis. The arteriolar wall is thickened with the deposition of amorphous proteinaceous material (hyalinized), and the lumen is markedly narrowed. **B,** Hyperplastic arteriolosclerosis ("onion-skinning") (*arrow*) causing luminal obliteration (periodic acid–Schiff stain).

(Courtesy of Helmut Rennke, MD, Brigham and Women's Hospital, Boston, Massachusetts.)

HYPERTENSIVE HEART DISEASE

- Hypertensive heart disease (HHD) is a consequence of the increased demands placed on the heart by hypertension, causing pressure overload and ventricular hypertrophy.

1. Systemic (Left-Sided) Hypertensive Heart Disease.
2. Pulmonary Hypertensive Heart Disease—Cor Pulmonale.

Systemic (Left-Sided) Hypertensive Heart Disease

*The criteria for the diagnosis of systemic hypertensive heart disease are:

(1) left ventricular hypertrophy in the absence of other cardiovascular pathology (e.g., valvular stenosis).

(2) A history or pathologic evidence of hypertension.

*It was established that even mild hypertension , if sufficiently prolonged, induces left ventricular hypertrophy.

The mechanisms

The mechanisms by which hypertension leads to heart failure are incompletely understood; presumably the hypertrophic myocytes fail to

contract efficiently, possibly due to :

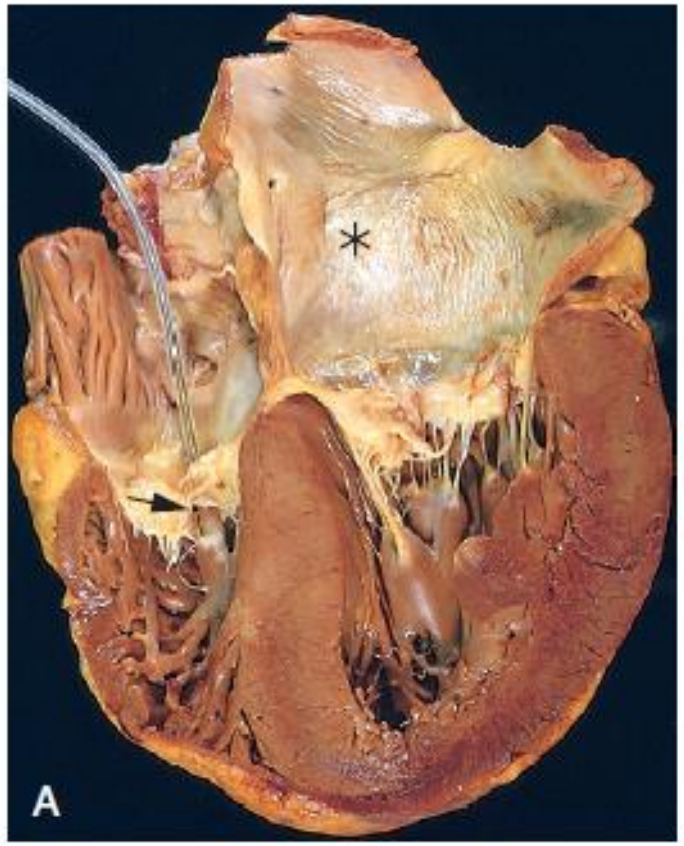
1- Structural abnormalities in newly assembled sarcomeres.

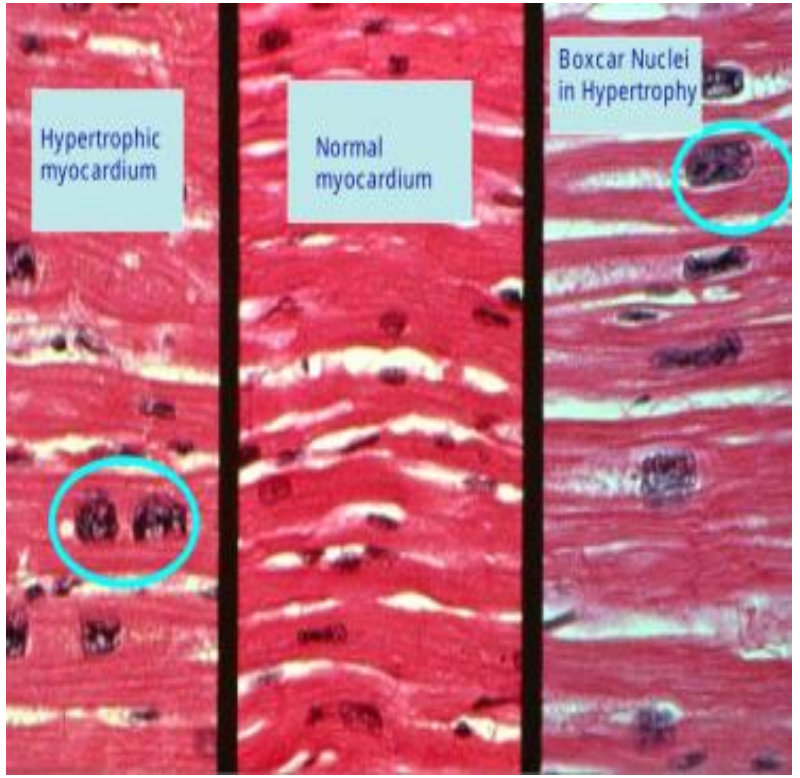
2- The vascular supply is inadequate to meet the demands of the increased muscle mass.

MORPHOLOGY

- ✓ The essential feature of systemic hypertensive heart disease is left ventricular hypertrophy, typically without ventricular dilation until very late in the process.
- ✓ The heart weight can exceed 500 g (normal, 320 to 360 g), and the left ventricular wall thickness can exceed 2.0 cm (normal, 1.2 to 1.4 cm).
- ✓ With time, the increased left ventricular wall thickness imparts a stiffness that impairs diastolic filling and can result in left atrial dilation.
- ✓ Microscopically, the transverse diameter of myocytes is increased and there is prominent nuclear enlargement and hyperchromasia ("boxcar nuclei"), as well as intercellular Fibrosis.

- Systemic (left-sided) hypertensive heart disease. There is marked concentric thickening of the left ventricular wall causing reduction in lumen size. The left ventricle and left atrium are on the right in this four-chamber view of the heart. A pacemaker is present incidentally in the right ventricle (arrow). Note also the left atrial dilation (asterisk) due to stiffening of the left ventricle and impaired diastolic relaxation, leading to atrial volume overload.





Clinical Features

- ✓ Compensated hypertensive heart disease typically is asymptomatic and is suspected only from discovery of elevated blood pressure on routine physical exams, or from ECG or echocardiographic findings of left ventricular hypertrophy.

- ✓ The disease comes to attention with the onset of atrial fibrillation (secondary to left atrial enlargement) and/or CHF.

- ✓ Depending on the severity and duration of the condition, the underlying cause of hypertension, and the adequacy of therapeutic control, patients Can :

(1) Enjoy normal longevity and die of unrelated causes,

(2) Develop progressive IHD owing to the effects of hypertension

in potentiating coronary atherosclerosis,

(3) Suffer progressive renal damage or cerebrovascular stroke.

(4) Experience progressive heart failure.

Pulmonary Hypertensive Heart Disease—Cor Pulmonale

- ✓ Cor pulmonale consists of right ventricular hypertrophy and dilation—frequently accompanied by right heart failure—caused by pulmonary hypertension attributable to primary disorders of the lung parenchyma or pulmonary vasculature.

- ✓ Right ventricular dilation and hypertrophy caused by left ventricular failure (or by congenital heart disease) is substantially more common but is excluded by this definition.

- ✓ Cor pulmonale can be acute in onset, as with pulmonary embolism, or can have a slow and insidious onset when due to prolonged pressure overloads in the setting of chronic lung and pulmonary vascular disease

Table 10–4 Disorders Predisposing to Cor Pulmonale

Diseases of the Pulmonary Parenchyma
Chronic obstructive pulmonary disease
Diffuse pulmonary interstitial fibrosis
Pneumoconiosis
Cystic fibrosis
Bronchiectasis
Diseases of the Pulmonary Vessels
Recurrent pulmonary thromboembolism
Primary pulmonary hypertension
Extensive pulmonary arteritis (e.g., Wegener granulomatosis)
Drug-, toxin-, or radiation-induced vascular obstruction
Extensive pulmonary tumor microembolism
Disorders Affecting Chest Movement
Kyphoscoliosis
Marked obesity (pickwickian syndrome)
Neuromuscular diseases
Disorders Inducing Pulmonary Arterial Constriction
Metabolic acidosis
Hypoxemia
Obstruction to major airways
Idiopathic alveolar hypoventilation

MORPHOLOGY

- ✓ **In acute cor pulmonale**, the right ventricle usually shows only dilation; if an embolism causes sudden death, the heart may even be of normal size.
- ✓ **Chronic cor pulmonale** is characterized by right ventricular (and often right atrial) hypertrophy.

- When ventricular failure develops, the right ventricle and atrium often are dilated.
- Because chronic cor pulmonale occurs in the setting of pulmonary hypertension, the pulmonary arteries often contain atheromatous plaques and other lesions, reflecting longstanding pressure elevations.

Chronic cor pulmonale. The right ventricle (shown on the left side of this picture) is markedly dilated and hypertrophied with a thickened free wall and hypertrophied trabeculae. The shape and volume of the left ventricle have been distorted by the enlarged right ventricle.

GOOD LUCK