# *Hypertensive Heart Disease Pericarditis, Myocarditis & Cardiomyopathy*

# Hypertensive Heart Disease

***An adaptive response to pressure overload that can lead to myocardial dysfunction, cardiac dilatation, CHF and sudden death***

- ***The Framingham Study established that even mild hypertension with levels slightly above 140/90 mm of Hg if sufficiently prolonged, induces left ventricular hypertrophy***
- ***May be:***

  ***Systemic /Left sided***

  ***Pulmonary/Right-Sided (Cor Pulmonale)***

# Hypertensive Heart Disease

***Systemic (Left-Sided) Hypertensive Heart Disease***

***Minimal Pathological Criteria***

- ***For the diagnosis of systemic HHD are the following: (1) left ventricular hypertrophy (usually concentric) in the absence of other cardiovascular pathology (2) a clinical history or pathologic evidence of hypertension in other organs (e.g., kidney).***

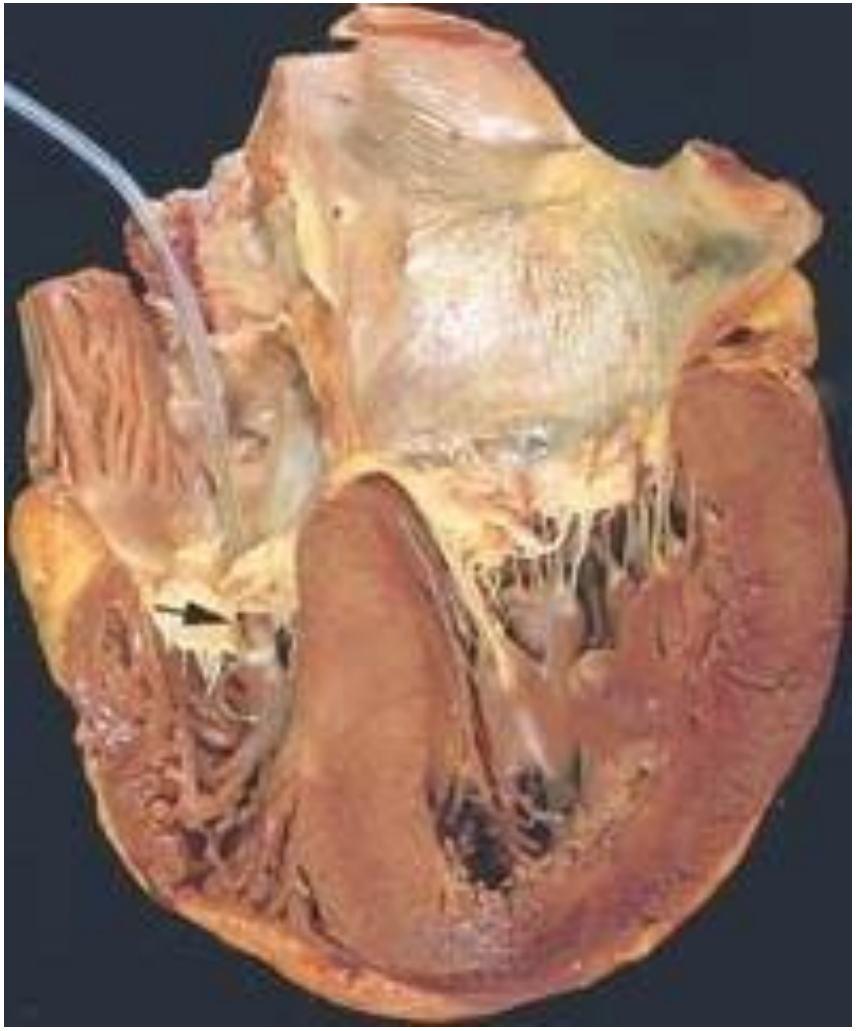
- ***Compensated systemic HHD may be asymptomatic, producing only electrocardiographic or echocardiographic evidence of left ventricular enlargement.***

# Hypertensive Heart Disease

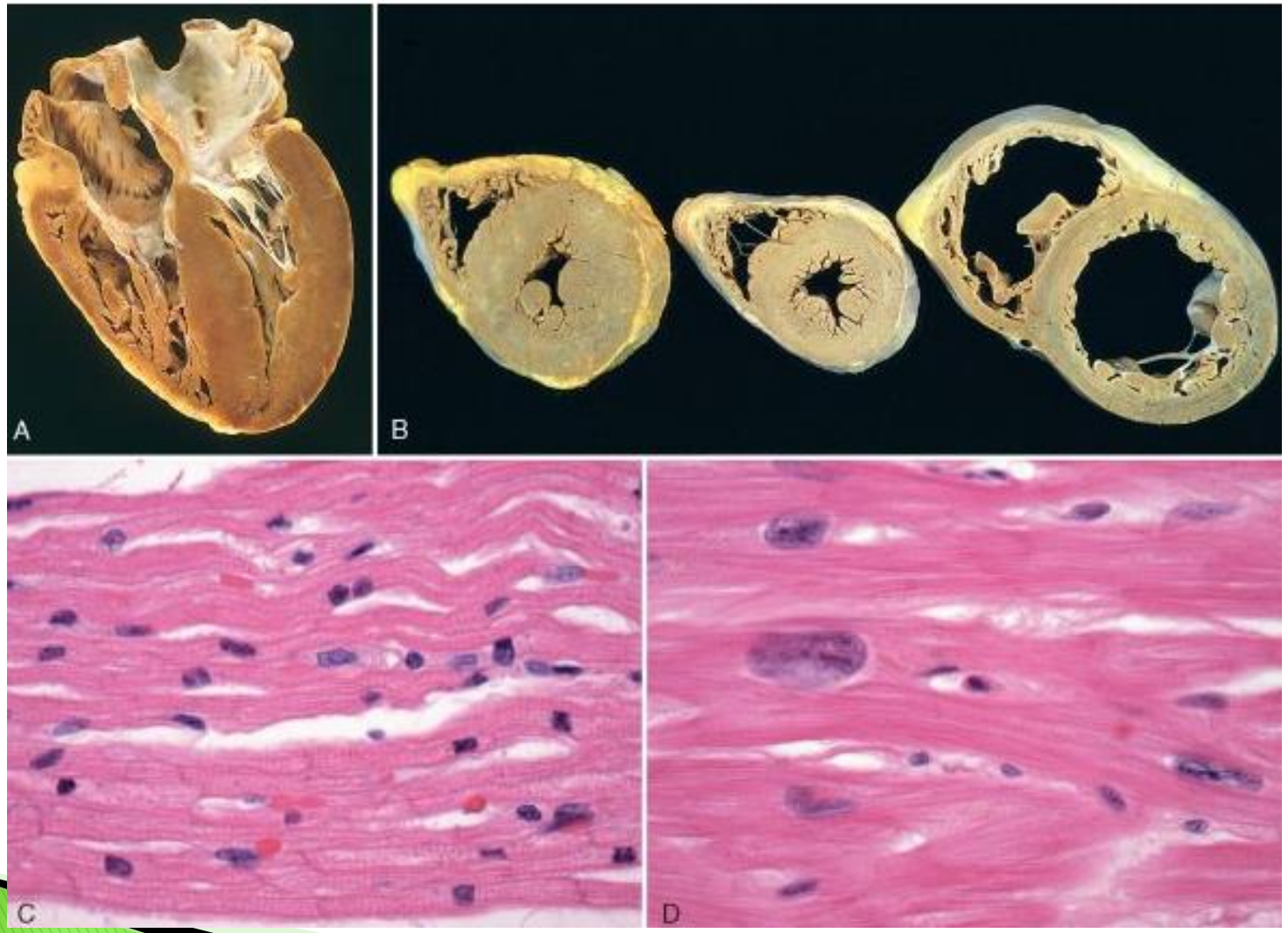
***Morphology :***

- ***Heart weight may exceed 500 gm***
- ***Left ventricular wall thickness may exceed 2.0 cm***

***Increased thickness of left ventricular wall imparts a stiffness that impairs diastolic filling reducing lumen size and inducing left atrial enlargement***



# Left Ventricular Hypertrophy



# Hypertensive Heart Disease

- ***Microscopy***
- ✓ ***Increased transverse diameter of Myocytes***
- ✓ ***In advanced cases cellular and nuclear enlargement more irregular***
- ✓ ***Variation in cell size among adjacent cells and interstitial fibrosis***
- ***Clinically***
- ✓ ***May be asymptomatic***
- ✓ ***ECG and echocardiographic indication of left ventricular enlargement.***
- ✓ ***Later atrial fibrillation or CHF with cardiac dilatation or both***

# Hypertensive Heart Disease

- ***Pulmonary(right Sided) Hypertensive Heart Disease (Cor Pulmonale)***
- ***Failure secondary to pulmonary hypertension***
- ***Due to disorders of lung or pulmonary vasculature***
- ***Right ventricular hypertrophy***

- ***Typical causes of chronic cor pulmonale are disorders of the lungs, chronic parenchymal diseases such emphysema, primary pulmonary hypertension***

# Hypertensive Heart Disease

***Acute cor pulmonale***

- ***Sudden development***
- ***Can follow massive pulmonary embolism***

***Chronic cor pulmonale***

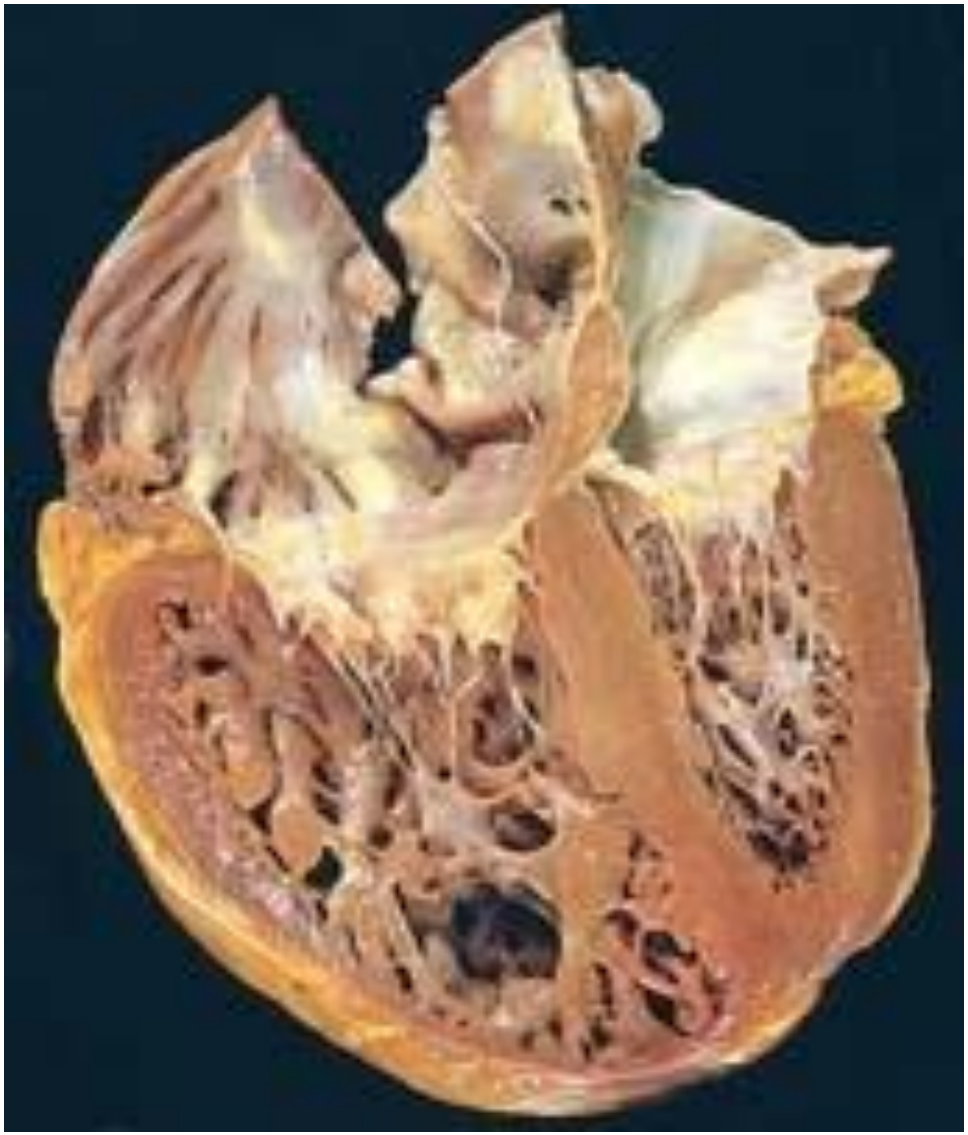
- ***Secondary to prolonged pressure overload***

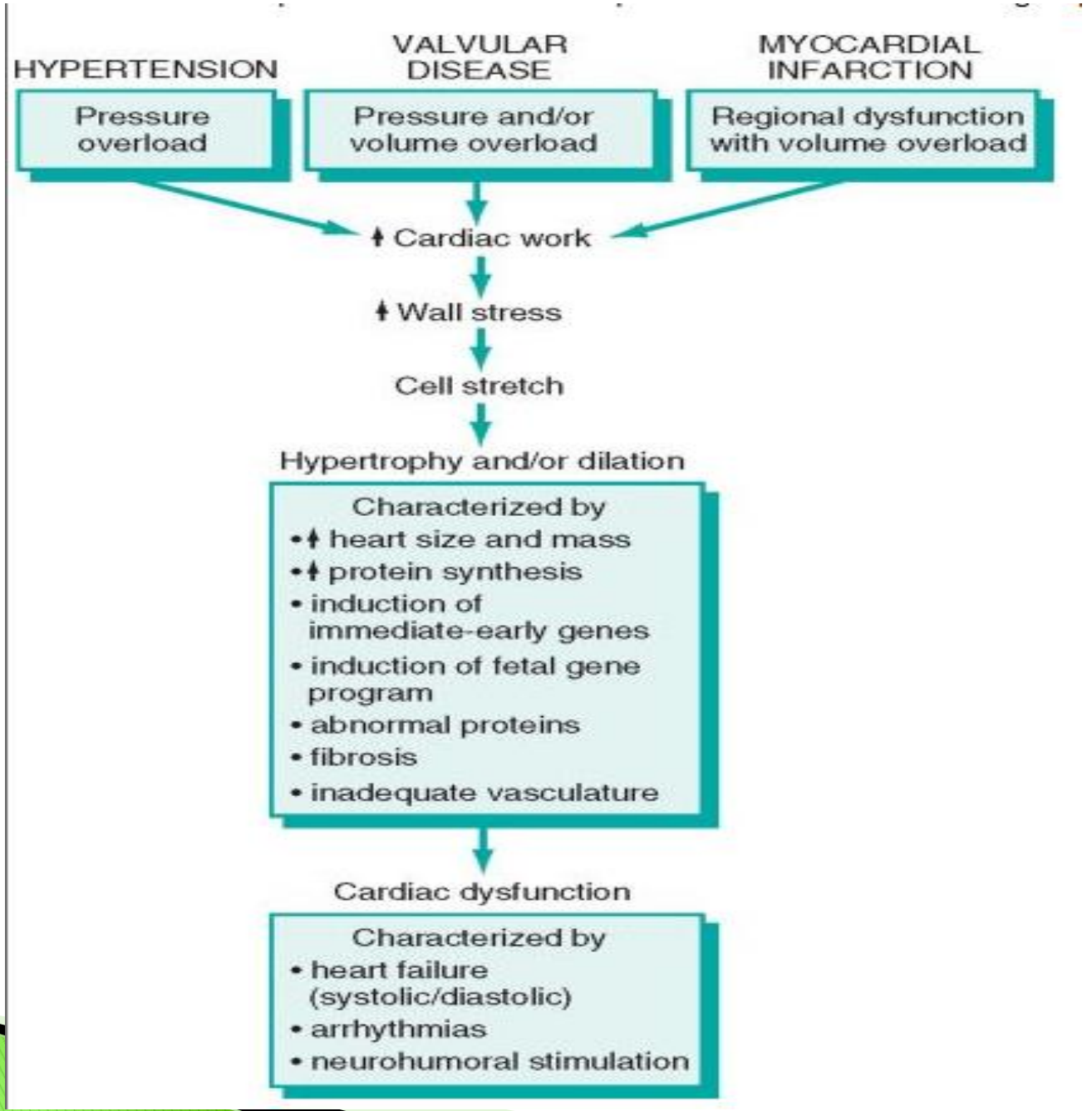
***Morphology:***

- ***Marked dilatation of rt ventricle without hypertrophy***
- ***In chronic corpulmonale right ventricular wall thickness may exceed 1 cm or more***
- ***Subtle hypertrophy take the form of thickening of the muscle bundles in the outflow tract, below the pulmonary valve***

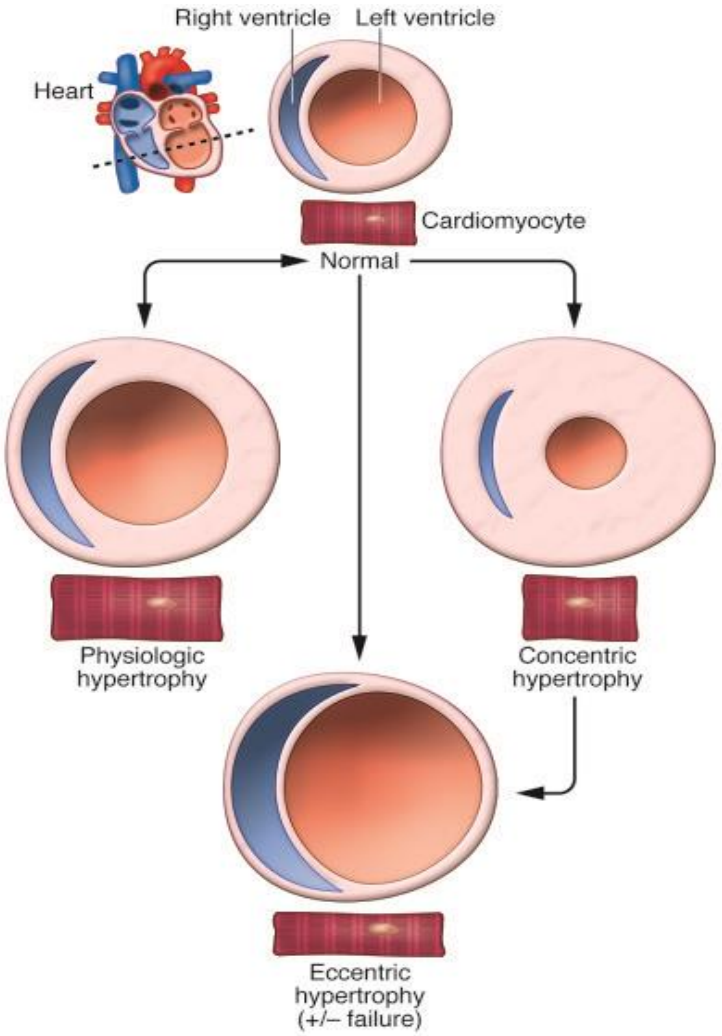
## *Cor Pulmonale*

*Dilated And Hypertrophic Right Ventricle*




HYPERTENSION
VALVULAR DISEASE
MYOCARDIAL INFARCTION
Pressure overload
Pressure and/or volume overload
Regional dysfunction with volume overload
↑ Cardiac work
↑ Wall stress
Cell stretch
Hypertrophy and/or dilation
Characterized by
• ↑ heart size and mass
• ↑ protein synthesis
• induction of immediate-early genes
• induction of fetal gene program
• abnormal proteins
• fibrosis
• inadequate vasculature
Cardiac dysfunction
Characterized by
• heart failure (systolic/diastolic)
• arrhythmias
• neurohumoral stimulation

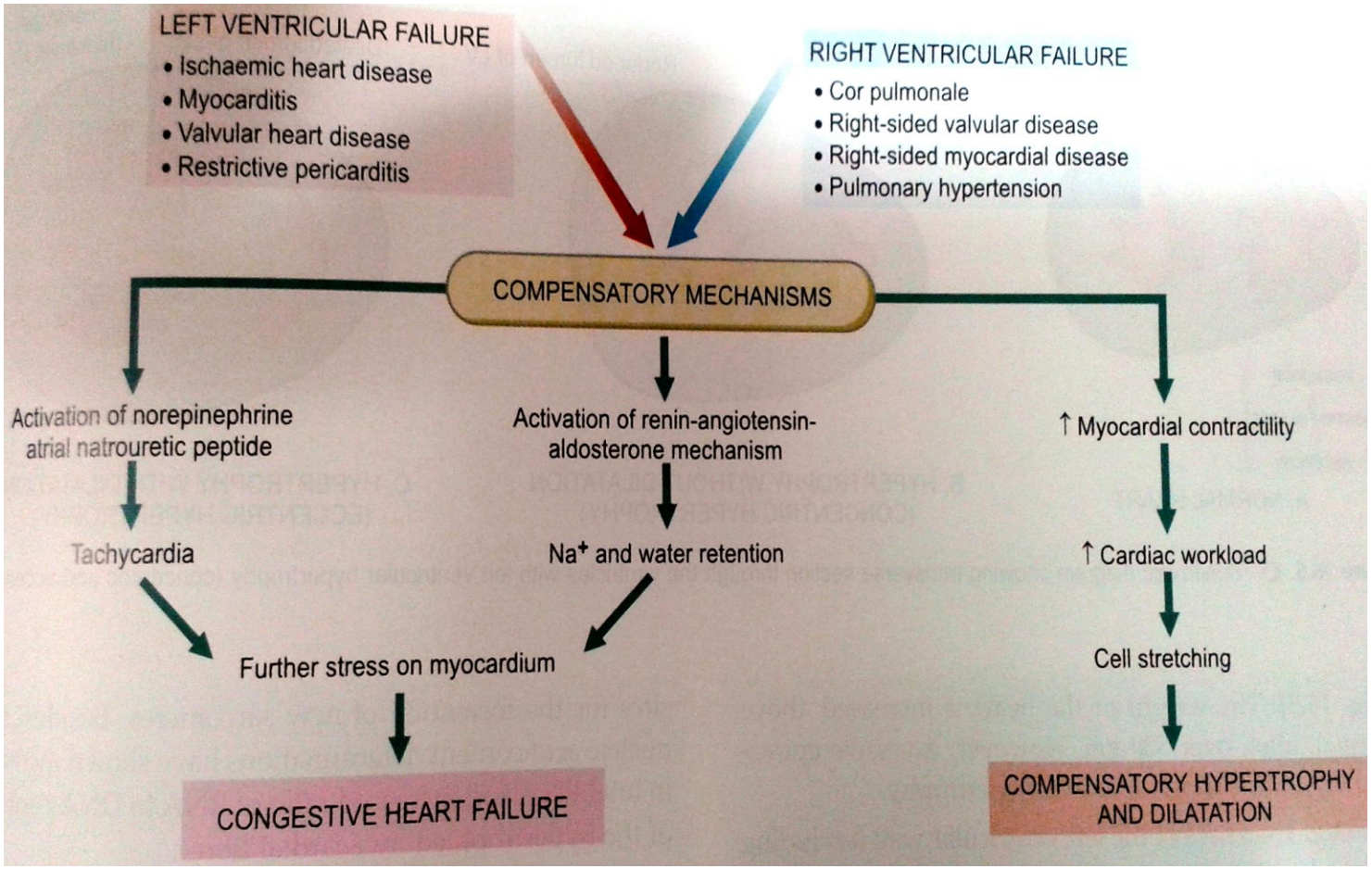
# Types Of Hypertrophy



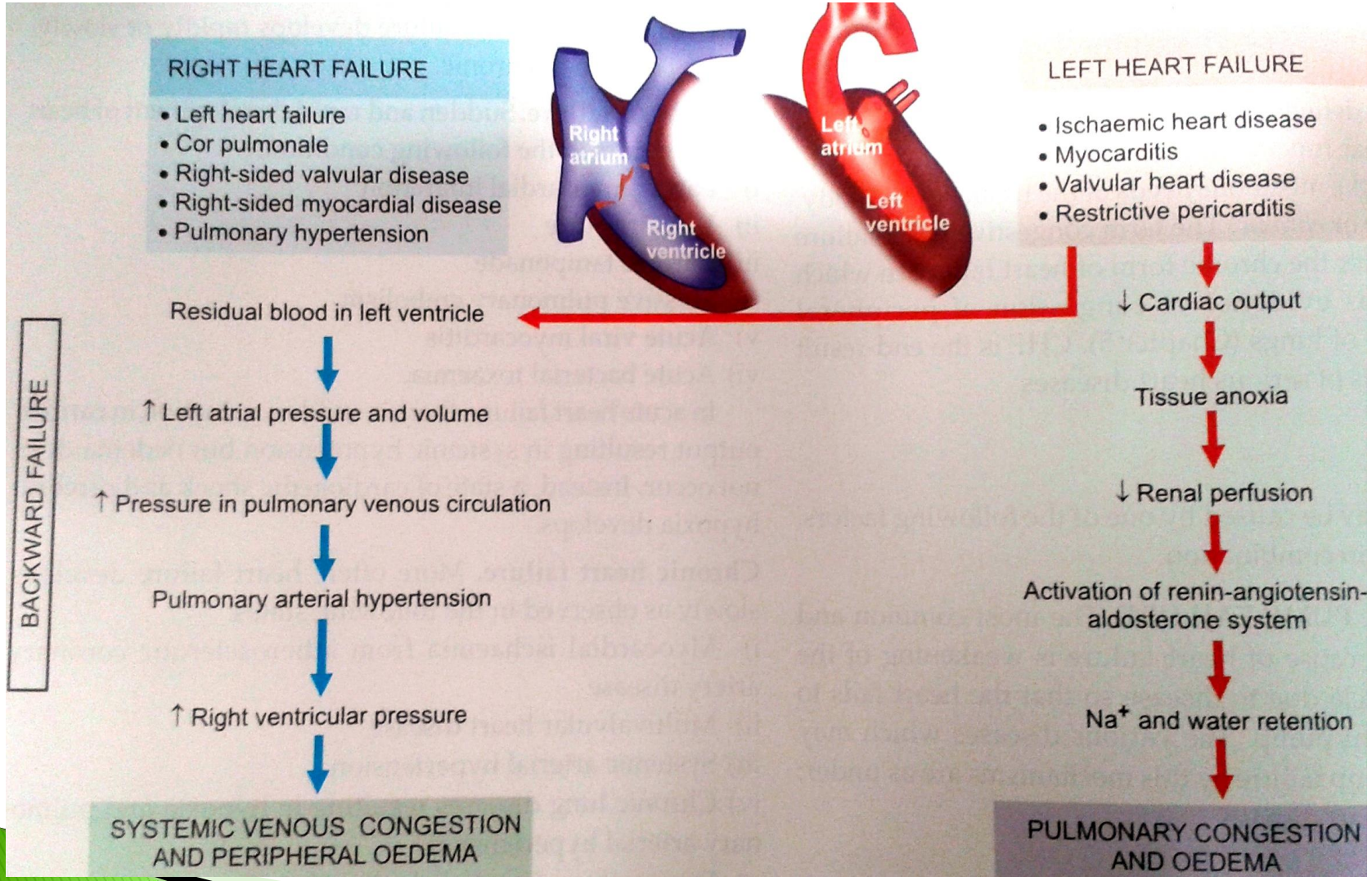

***Exercise and pregnancy result in physiologic hypertrophy, in which individual cardiomyocytes increase in length and width and the heart undergoes a balanced type of eccentric hypertrophy (chambers, walls, and septum enlarge in unison).***

***Pathologic stress/hypertrophic cardiomyopathy activates neuroendocrine factors that stimulate cardiac hypertrophy, resulting in concentric remodeling, in which cardiomyocytes mostly increase in width compared with length, resulting in wall and septal thickening and a loss of chamber area.***

# Heart Failure-types, Causes, Pathogenesis



# HEART FAILURE



# Cardiomyopathy

- *"Heterogeneous group of diseases of the myocardium associated with mechanical and/or electrical dysfunction that usually exhibit inappropriate ventricular hypertrophy or dilatation.*

- *Due to a variety of causes that frequently are genetic.*
- *Cardiomyopathies either are confined to the heart or are part of generalized systemic disorders, often leading to cardio- vascular death or progressive heart failure-related disability."*

# Cardiomyopathy

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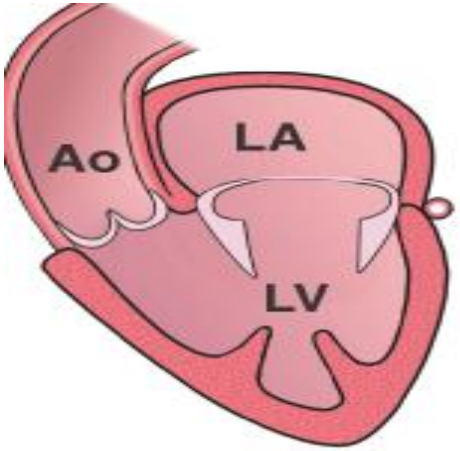
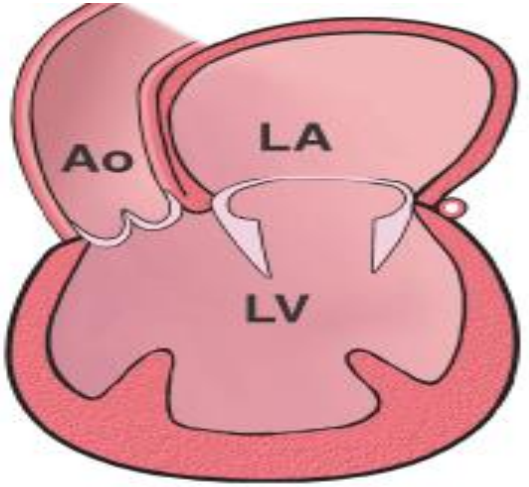
- ***Primary cardiomyopathies** can be genetic or acquired diseases of myocardium.*
- ***Secondary cardiomyopathies** have myocardial involvement as a component of a systemic or multiorgan disorder.*
- *Idiopathic in most cases*
- *Three clinical, functional, pathological patterns*
  - *Dilated cardiomyopathy*
  - *Hypertrophic cardiomyopathy*
  - *restrictive cardiomyopathy*

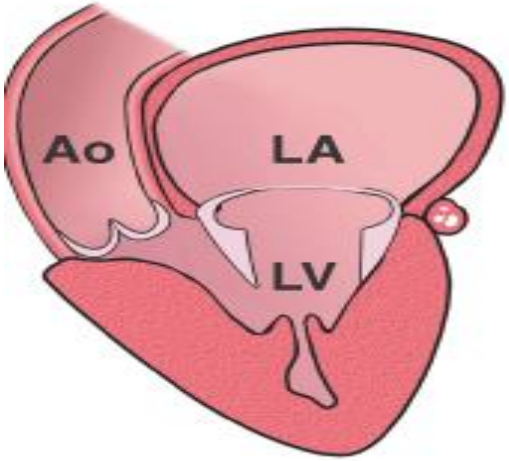
# Cardiomyopathy



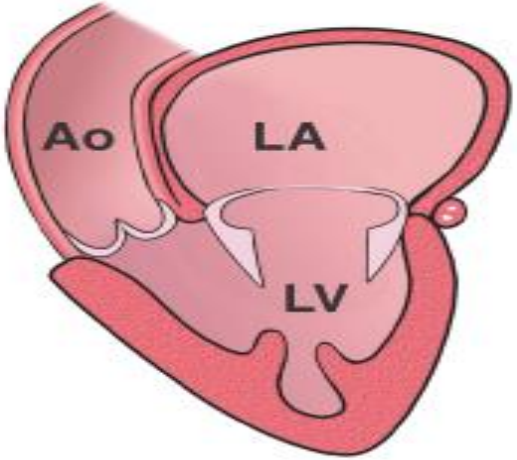

Normal




Dilated cardiomyopathy



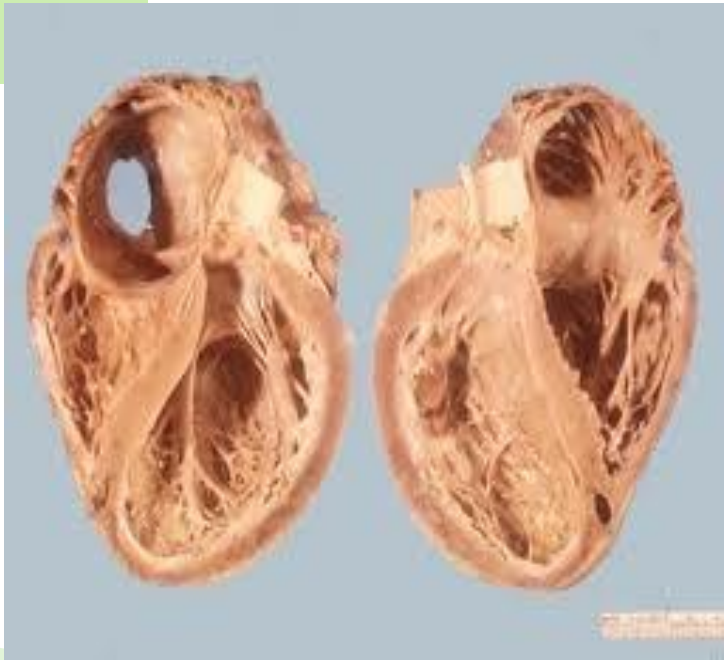

Hypertrophic cardiomyopathy




Restrictive cardiomyopathy

# Cardiomyopathy

- ***Dilated Cardiomyopathy***
- ***Most common (90%)***
- ***Progressive cardiac dilation &***
- ***Systolic dysfunction***
- ***Flabby and hypo-contracting heart***
- ***Histology nonspecific***



# Cardiomyopathy

- ***Pathogenesis.***
- ***Genetic Influences:*** ***30% to 50% of cases, caused by mutations in a diverse group genes encoding proteins involved in the cytoskeleton, sarcolemma, and nuclear envelope***
- ***Autosomal dominant inheritance is the predominant pattern***
- ***Myocarditis:*** ***Progression from myocarditis is seen. Viral myocarditis can be causal***

# Cardiomyopathy

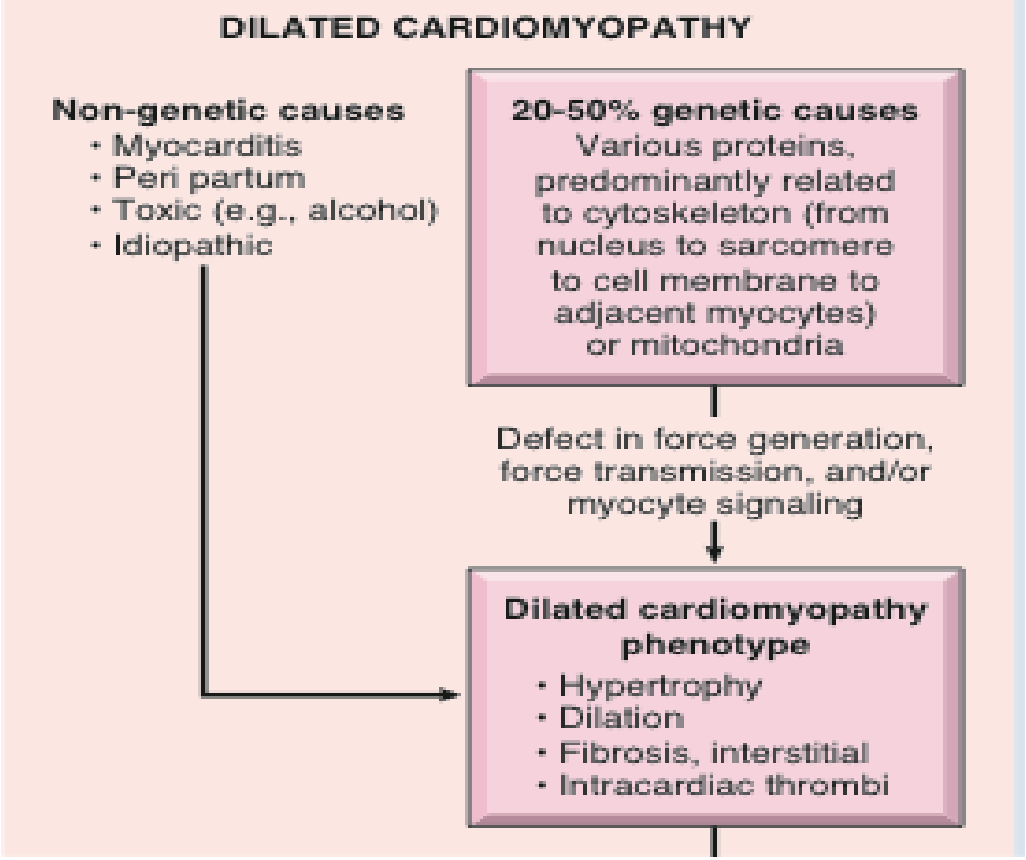
- ***Alcohol and other toxins:*** ***Alcohol or its metabolites have a direct toxic effect on the myocardium.***
- ***Chemotherapeutic agents*****, including doxorubicin (Adriamycin), and even targeted cancer therapeutics**
- ***Childbirth*****: *Peripartum cardiomyopathy*****, multifactorial. Pregnancy-associated hypertension, volume overload, nutritional deficiency, other metabolic derangements, immunological reaction have been proposed as causes.**

# Cardiomyopathy

- ***Iron overload* in the heart can result from either hereditary hemochromatosis or multiple transfusions.**
- **Could be due to interference with metal dependent enzyme system or due to reactive oxygen species.**

- ***Supraphysiologic stress:* persistent tachycardia, hyperthyroidism, or even in the fetuses of insulin-dependent diabetic mothers.**
- ***Excess catecholeamines:* result in multifocal myocardial contraction band necrosis progressing to DCM**

# Cardiomyopathy



# Cardiomyopathy

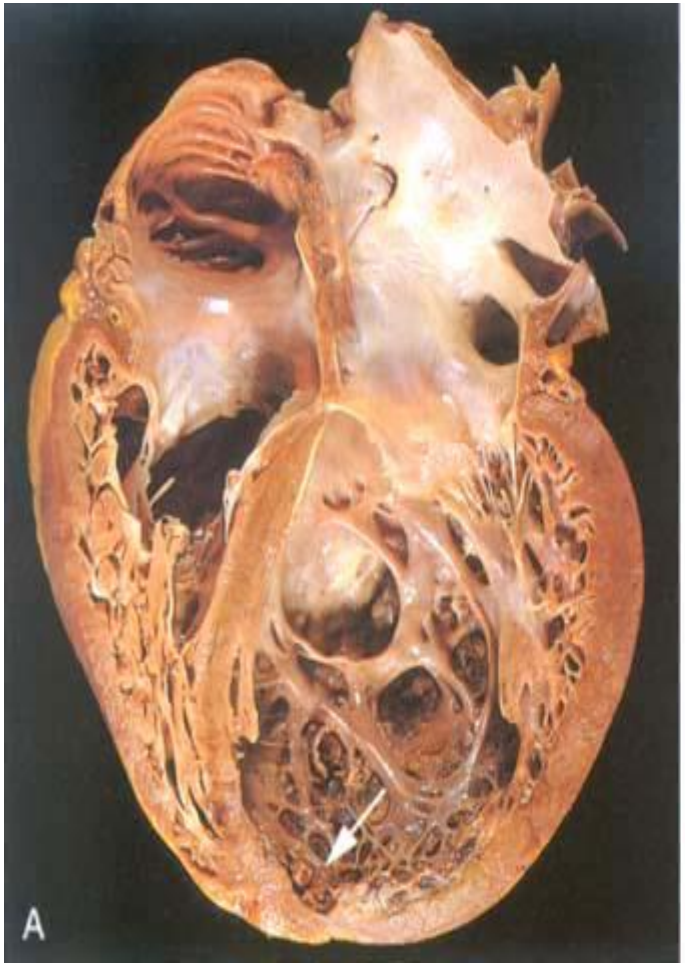
- ***Morphology:***
- ***Heart is enlarged, heavy  flabby, due to dilation of all chambers.***
- ***Mural thrombi are common and may be a source of thrombo-emboli.***
- ***Functional regurgitation.***

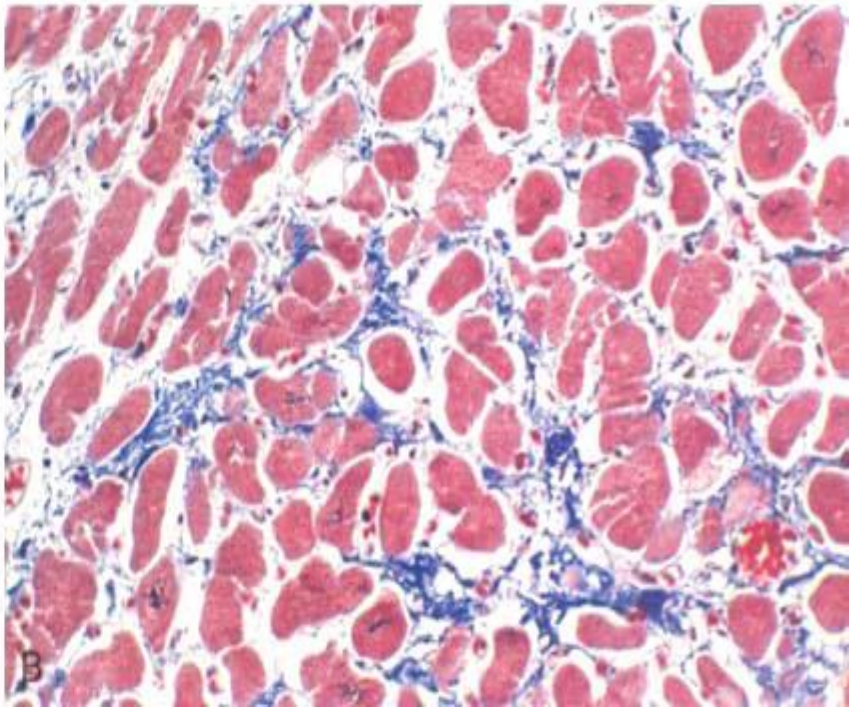
- ***Histologic abnormalities in DCM are nonspecific and usually do not point to a specific etiology.***

  ***Muscle cells are hypertrophied enlarged nuclei,***

  ***Interstitial and endocardial fibrosis***

  ***Subendocardial scars***




***Four Chamber Dilatation***

***Myocyte hypertrophy and Interstitial fibrosis(MT)***

# Cardiomyopathy

- *Clinical Features*
- *Affects individuals between the ages of 20 and 50.*
- *Slowly progressive signs and symptoms of CHF including dyspnea, easy fatigability, and poor exertional capacity.*
- *End stage, ejection fractions are typically less than 25% (normal = 50% to 65%).*
- *Secondary mitral regurgitation and abnormal cardiac rhythms are common,*
- *Embolism from intracardiac thrombi can occur.*
- *Death usually results from progressive cardiac failure or arrhythmia, and can occur suddenly.*

# Hypertrophic Cardiomyopathy

- ***Hypertrophic cardiomyopathy (HCM) is a common clinically heterogeneous, genetic disorder***
- ***Characterized by***
  - ***Myocardial hypertrophy,***
  - ***Poorly compliant left ventricular myocardium***
  - ***Abnormal diastolic filling***
  - ***Intermittent ventricular outflow obstruction.***
- ***Also k/a hypertrophic obstructive cardiomyopathy (HOCM)***
- ***Heavy, hypercontracting, thick walled heart***
- ***Leading cause of left ventricular hypertrophy unexplained by other clinical or pathologic causes***

# Hypertrophic Cardiomyopathy

- ***Heart is thick-walled, heavy, and hypercontracting,***
- ***Causes primarily diastolic dysfunction; systolic function is usually preserved.***

***Pathogenesis:***

- ***Pattern of transmission is autosomal dominant Caused by mutations in any one of several genes that encode sarcomeric proteins***
- ***HCM probably occurs secondary to exaggerated responses of the myocardial fibroblasts to primary myocardial dysfunction.***

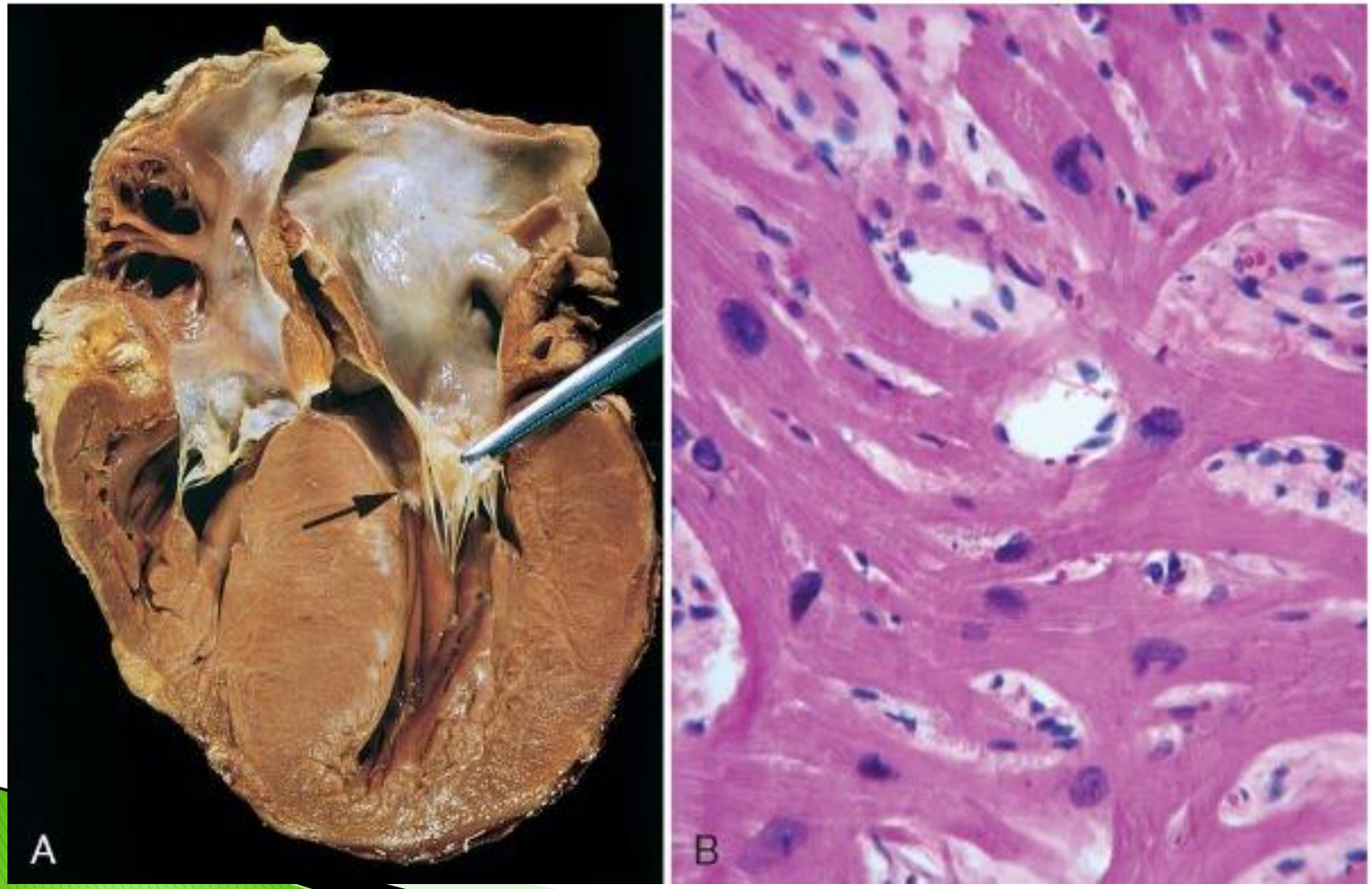
# Hypertrophic Cardiomyopathy

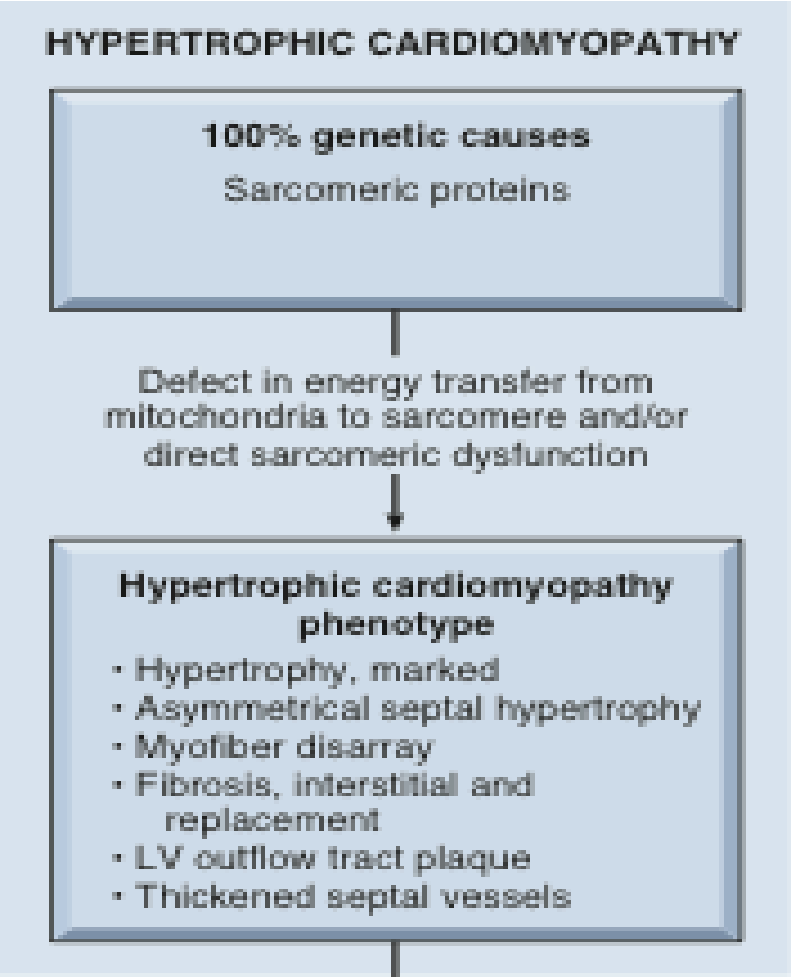
- ***Morphology:***
- ***Massive myocardial hypertrophy, usually without ventricular dilation***
- ***Classic pattern: disproportionate thickening of the ventricular septum relative to the left ventricle free wall (with a ratio of septum to free wall greater than 3:1), termed asymmetric septal hypertrophy.***
- ***Round-to-ovoid left ventricle compressed into a "banana-like" configuration by bulging of the ventricular septum into the lumen***
- ***Can involve the entire septum, usually most prominent in the subaortic region.***

# Hypertrophic Cardiomyopathy

- ***The most important histologic features of HCM myocardium are***
- ***(1) Massive myocyte hypertrophy, with transverse myocyte diameters frequently greater than 40 μm (normal, approximately 15 μm)***
- ***(2) Haphazard disarray of bundles of myocytes, individual myocytes, and contractile elements in sarcomeres within cells (termed myofiber disarray)***
- ***(3) Interstitial and replacement fibrosis***

# HYPERTROPHIC CARDIOMYOPATHY






***Clinical features***

- ***Reduced stroke volume* due to *impaired diastolic filling*. Due to reduced chamber size, compliance of hypertrophied left ventricle.**
- ***Increase pulmonary venous pressure* leads to exertional dyspnea**
- **Major clinical problems are *atrial fibrillation, mural thrombus* formation leading to embolization and possible *stroke*, *intractable cardiac failure*, ventricular *arrhythmias*, and, not infrequently, *sudden death***

# RESTRICTIVE CARDIOMYOPATHY

- ***Restrictive cardiomyopathy is characterized by***
- ***Decrease ventricular compliance***
- ***Impaired diastolic filling***
- ***Ventricles approx. Normal***
- ***Can be idiopathic***
- ***Secondary to diseases or processes affecting myocardium, principally radiation fibrosis, amyloidosis, sarcoidosis, metastatic tumors, or the deposition of metabolites that accumulate due to inborn errors of metabolism.***

| FEATURE | DILATED CMP | HYPERTROPHIC CMP | RESTRICTIVE CMP |
|---|---|---|---|
| Mechanism | Systolic Dysfunction | Diastolic Dysfunction | Diastolic Dysfunction |
| Genetic factors | 20-50% | 100% | ± |
| Proteins involved | Various cytoskeletal proteins | Sarcomeric proteins | |
| Other non genetic causes | Alcohol, peripartum, idiopathic, drugs, myocarditis | - | Amyloidosis, radiation induced fibrosis, idiopathic |
| Heart | Flabby, hypocontracting heart | Heavy, Hypercontracting Heart | Normal appearance |
| Left Ventricular Ejection Fract | < 40% | 50-80% | 45-90% |
| Mural thrombi | Common | ± | ± |

# MYOCARDITIS

- ***Inflammatory Processes that cause myocardial injury***

| Infections |
| --- |
| Viruses (e.g., coxsackievirus, ECHO, influenza, HIV, cytomegalovirus) |
| Chlamydiae (e.g., *Chlamydophyla psittaci*) |
| Rickettsiae (e.g., *Rickettsia typhi*, typhus fever) |
| Bacteria (e.g., *Corynebacterium diphtheriae*, *Neisseria meningococcus*, *Borrelia* (Lyme disease) |
| Fungi (e.g., *Candida*) |
| Protozoa (e.g., *Trypanosoma cruzi* [Chagas disease], toxoplasmosis) |
| Helminths (e.g., trichinosis) |
| **Immune-Mediated Reactions** |
| Postviral |
| Poststreptococcal (rheumatic fever) |
| Systemic lupus erythematosus |
| Drug hypersensitivity (e.g., methyldopa, sulfonamides) |
| Transplant rejection |
| **Unknown** |
| Sarcoidosis |
| Giant cell myocarditis |

HIV, Human immunodeficiency virus.

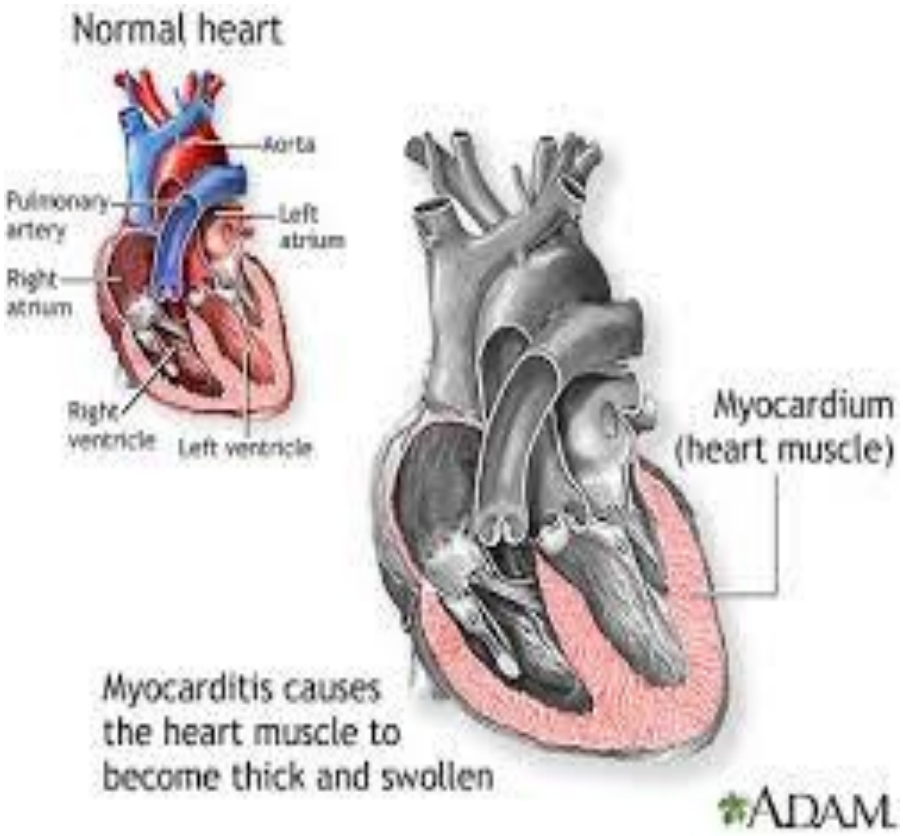
***Gross :***

***Heart may appear normal or dilated often enlarged, flabby with foci of necrosis***

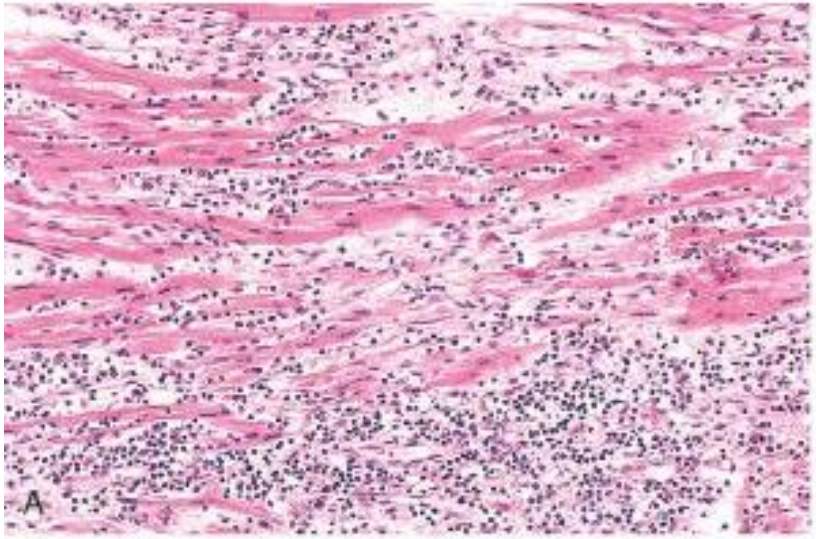
***Mural thrombi***

***M/E:***

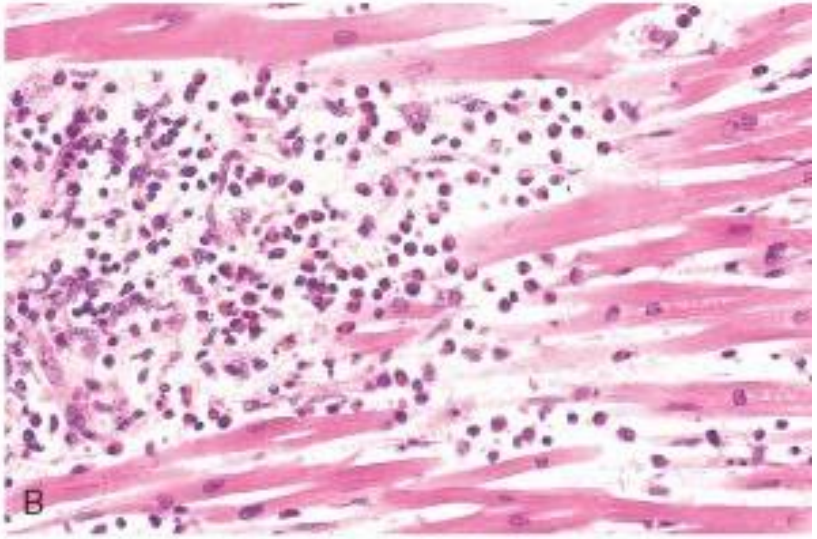
***Interstitial inflammation infilterate -lymphocytes, macrophages, plasma cells with focal myocyte necrosis.***



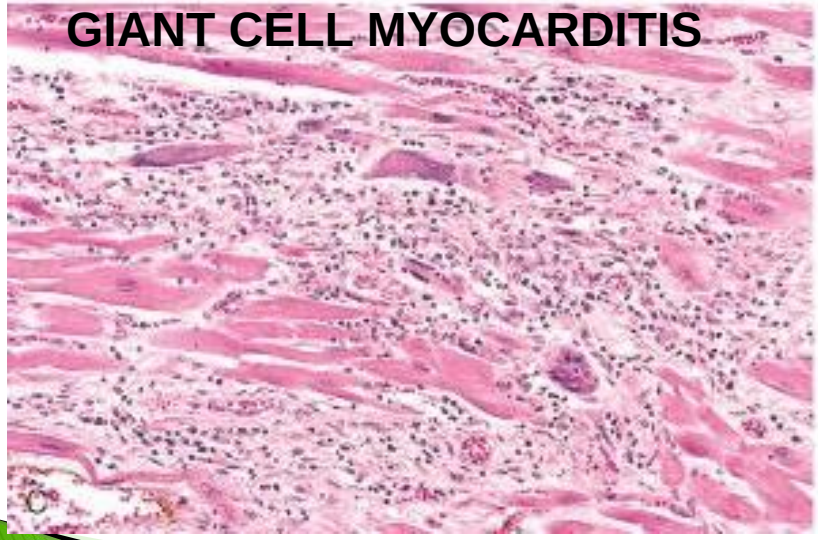
## LYMPHOCYTIC MYOCARDITIS




## HYPERSENSITIVITY MYOCARDITIS




## GIANT CELL MYOCARDITIS



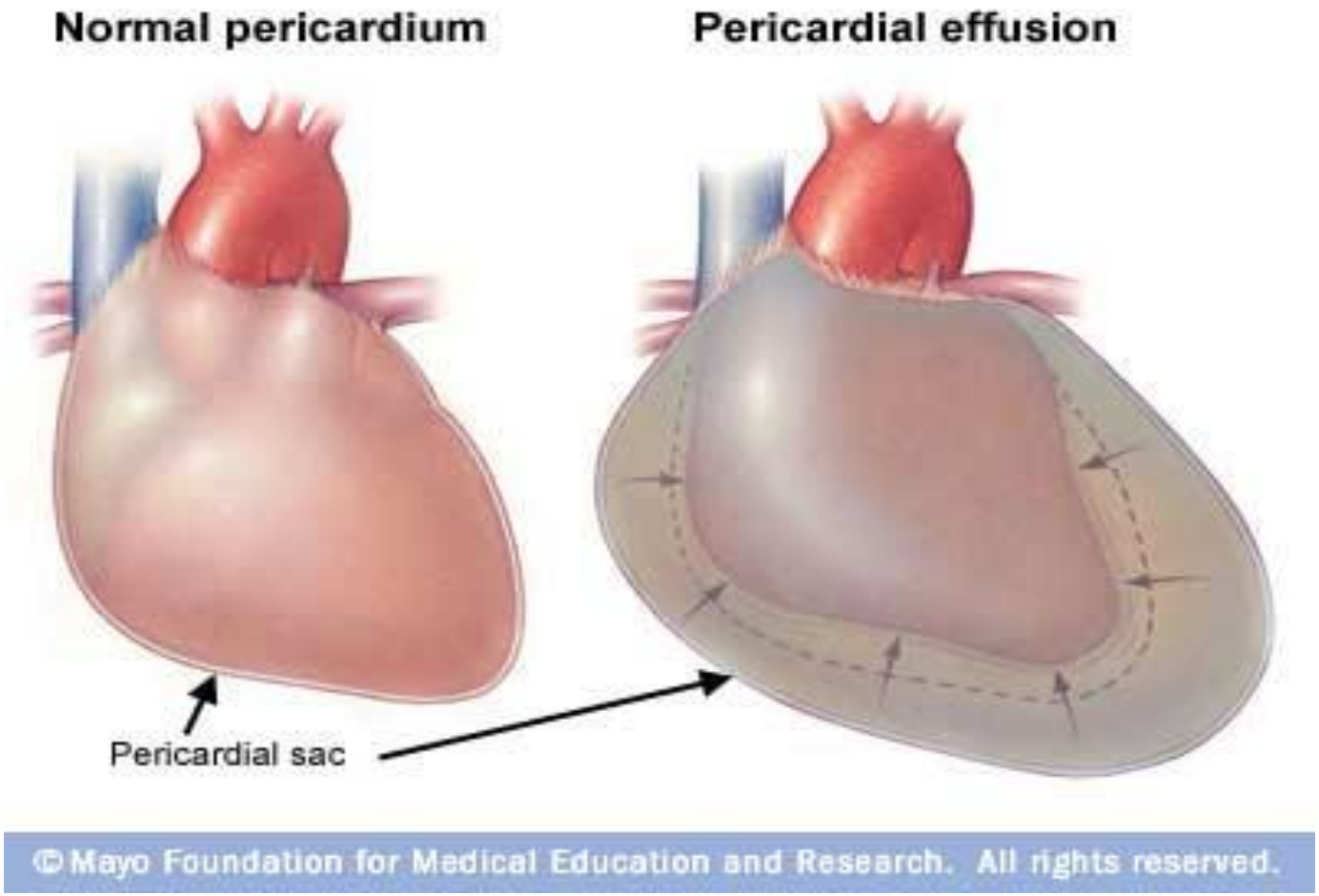

## MYOCARDITIS IN CHAGA'S DSE

# Pericardial Effusion /Hemopericardium

- ***Parietal pericardium may be distended by serous fluid (pericardial effusion), blood (hemopericardium), or pus (purulent pericarditis).***

- ***Chronic effusions < 500 mL , the only clinical significance is a characteristic globular enlargement of the heart shadow on chest radiographs.***

- ***Rapidly developing collections of 200to300 ml, due to hemopericardium caused by a ruptured MI or aortic dissection—produce clinically devastating compression of the thin-walled atria and venae cavae, or the ven- tricles.***




***Cardiac filling* is thereby restricted, producing potentially fatal cardiac tamponade.**

# PERICARDITIS

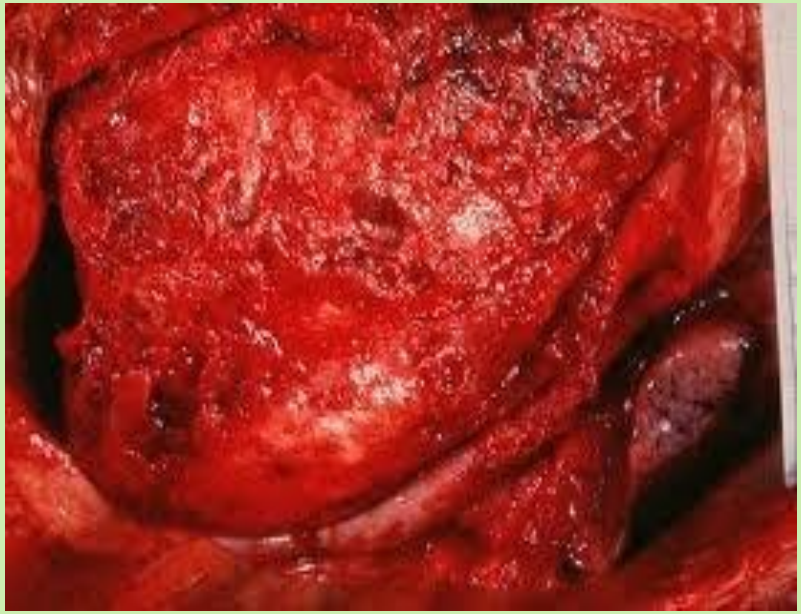
***Inflammation of pericardium with or without effusion.***

- ***primary or secondary,***
- ***acute or chronic (clinically).***

- ***Types of pericarditis :***

1. ***Serous***
2. ***Fibrinous***

- ***Sero fibrinous***
- ***Purulent or suppurative***
- ***Hemorrhagic***
- ***Caseous***
- ***Constrictive***



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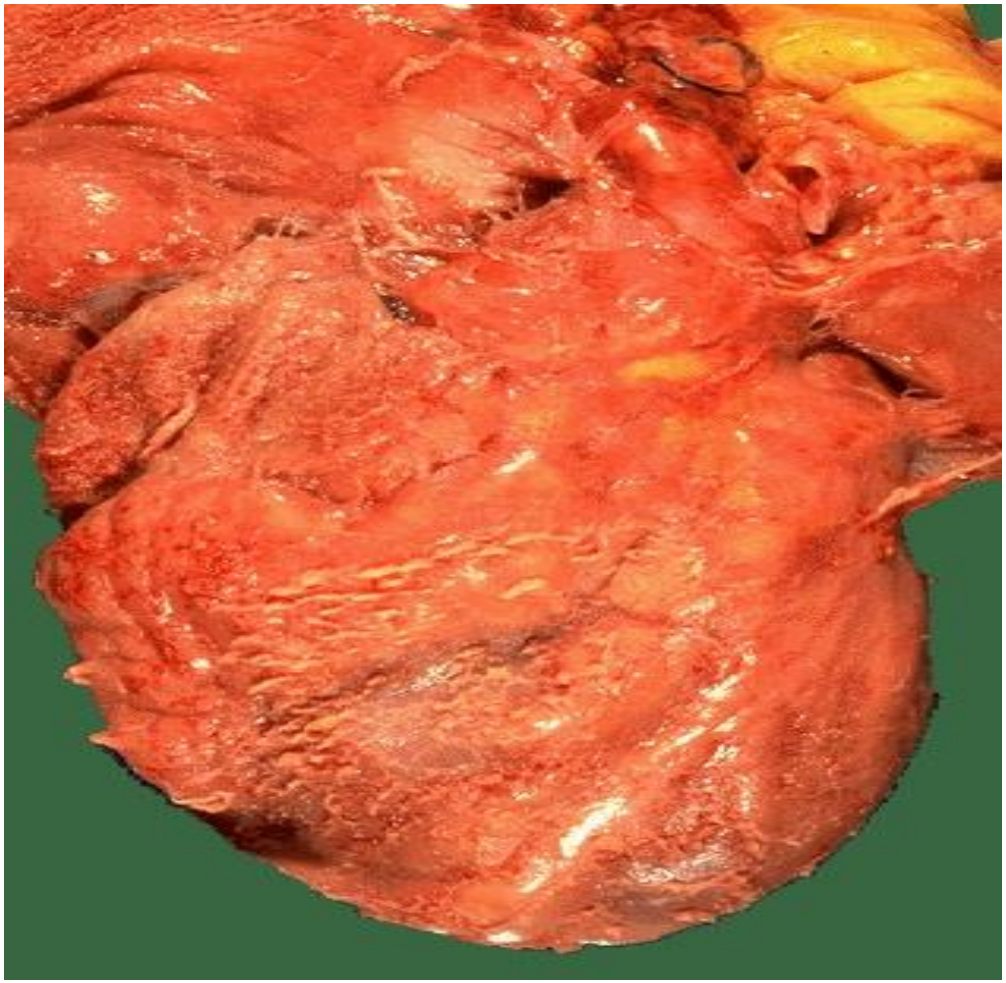
# PERICARDITIS

- ***Serous pericarditis :***
- ***Noninfectious inflammation seen in***
- ***RF,SLE***
- ***Nutritional deficiencies, uremia***
- ***Scleroderma, Tumours***
- ***Usually in young adults***
- ***Microscopically***
- ***Inflammatory reaction is in epicardial and pericardial surface with few neutrophils, lymphocytes and macrophages***
- ***Fluid accumulates ( 50-200 ml)***
- ***High SG and protein content***

# PERICARDITIS

- ***Fibrinous & serofibrinous pericarditis***
- ***Most common***
- ***Post MI (Dressler syndrome)***
- ***Uremia, chest radiation,***
- ***RF, SLE and trauma***
- ***Exudate is largely due to fibrin, RBC & WBC. The surface shows dry finely granular roughening***
- ***M/E- pink acellular fibrinous surface deposits with inflammatory exudate & granulation tissue***
- ***Clinically: The patient presents with pain, fever and signs of cardiac failure.***
- ***A loud pericardial friction rub is the most striking feature***



***Fibrinous pericarditis***

# PERICARDITIS

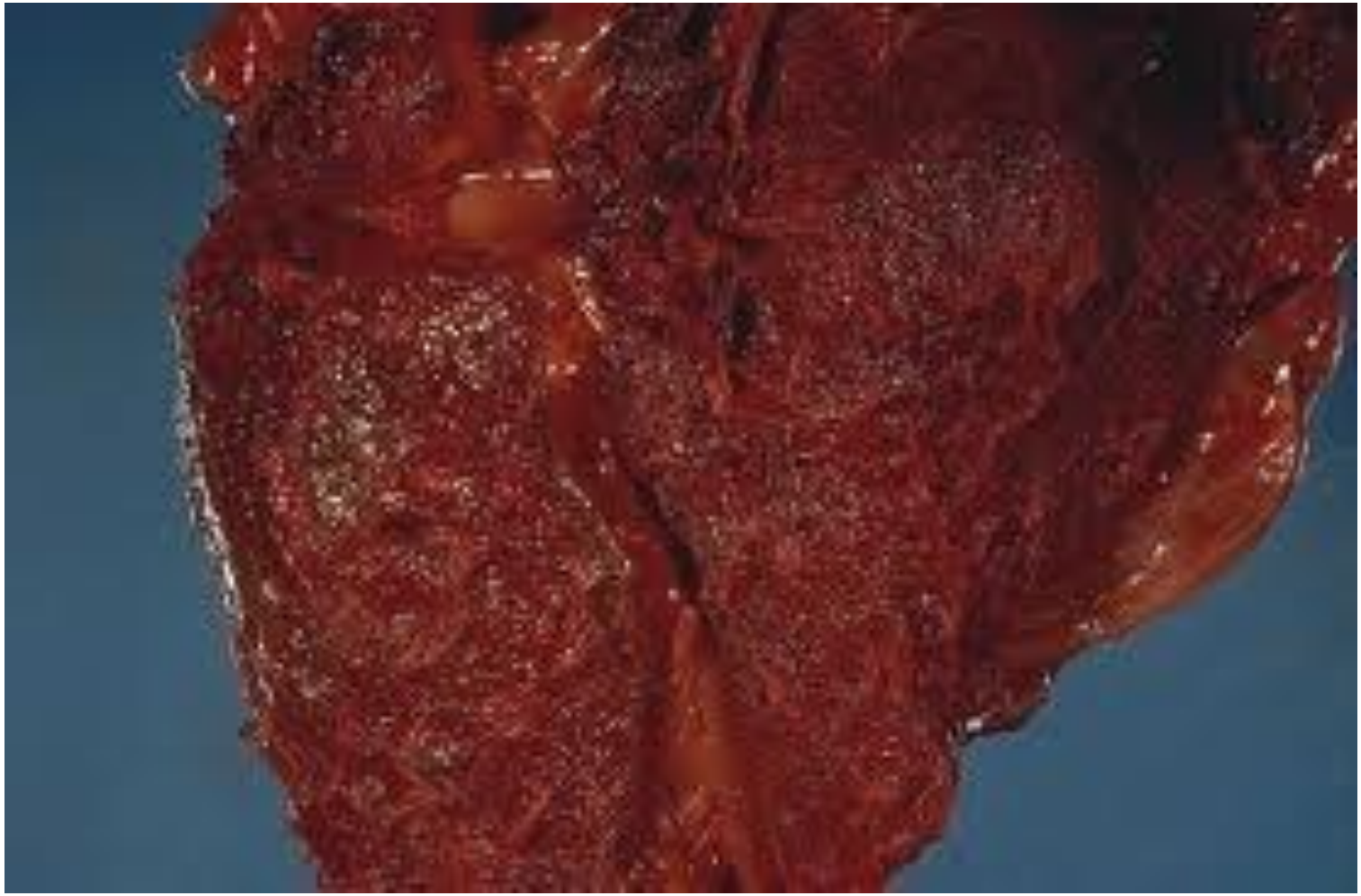
- ***Purulent Or Suppurative Pericarditis***
- ***Infective organisms invade the pericardial space***
- ✓ ***Direct extension from adjacent infection in lung, mediastinum or infective endocarditis or during surgery or by hematogenous or lymphatic route***

- ✓ ***Thin, to a creamy pus upto 500ml in volume***
- ✓ ***Serosal surfaces are red, granular, coated with exudate.***
- ***M/E :***
- ✓ ***Acute inflammatory reaction***
- ✓ ***Organisation***
- ✓ ***Frequently produces constrictive pericarditis***

***Suppurative Pericarditis***

# PERICARDITIS

- ***Hemorrhagic pericarditis***
- ✓ ***Exudate blood mixed with fibrinous/suppurative effusion***
- ✓ ***Malignant involvement of pericardiac space***
- ✓ ***Bacterial infections***
- ✓ ***Bleeding diathesis and T.B***
- ✓ ***Rarely rupture of aneurysm***
- ✓ ***Follows cardiac surgery***
- ***Caseous pericarditis :***
- ✓ ***Tb (rarely fungal infections)***
- ✓ ***direct spread from tracheobronchial ln***
- ✓ ***Fibrocalcific chronic constrictive pericarditis***



***Haemorrhagic Pericarditis***

# PERICARDITIS

- ***Chronic Or Healed Pericarditis***
- ***Organisation produces plaque like fibrous thickening of serosal membranes – SOLDIER´S PLAQUE or thin delicate adhesions are seen at autopsy and rarely impair cardiac function***

- ***Adhesive Pericarditis***
- ***Occurs when delicate stringy adhesions are seen between parietal and visceral pericardium which may restrict cardiac function***

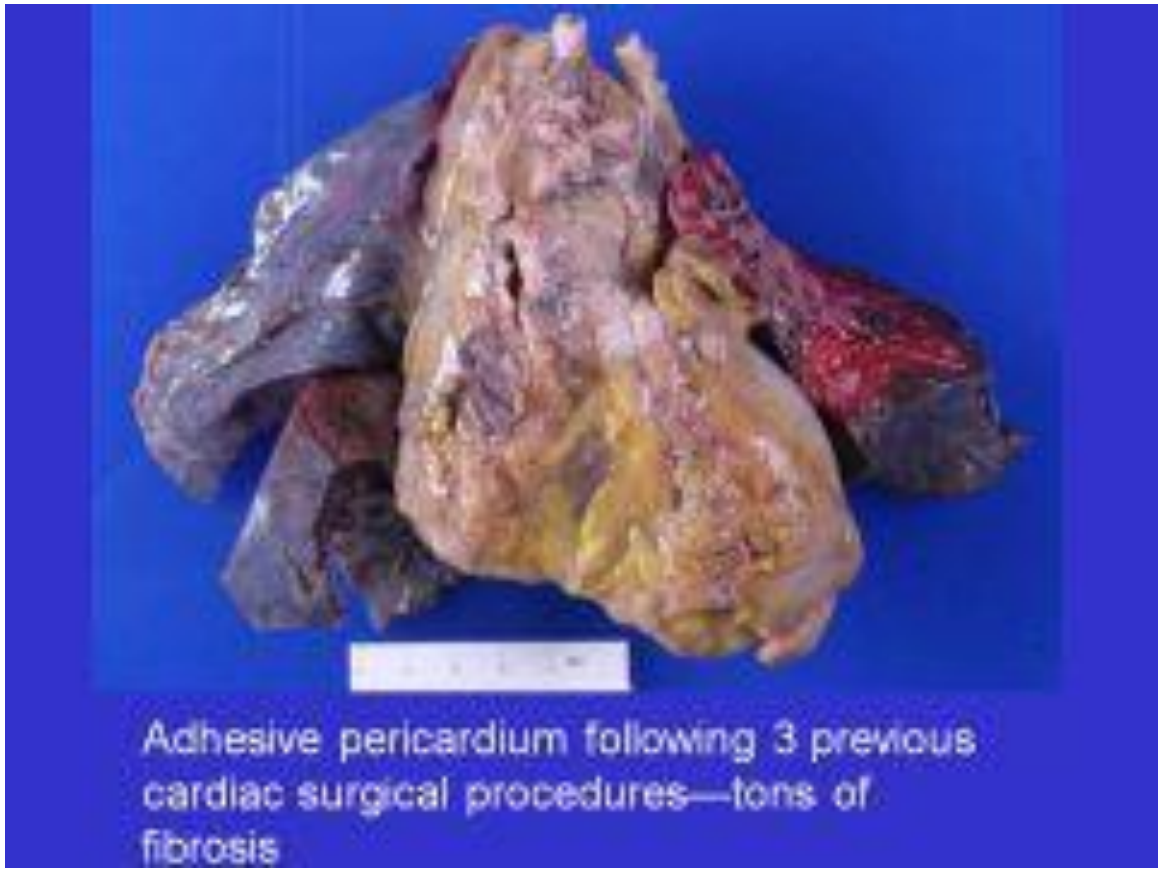
# CONSTRICTIVE PERICARDITIS

- ***Heart is encased in dense fibrous or fibrocalcific scar***
- ✓ ***Seriously restricts cardiac output, even at rest.***
- ✓ ***0.5 – 1 cm thick adherent scar with or without calcification surrounding the heart, resembles a plaster mould – CONCRETIO CORDIS***

- ***Pericardiectomy is the only treatment***

# ADHESIVE MEDIASTINOPERICARDITIS

- ***Follow suppurative or caseous pericarditis***
- ***Previous cardiac surgery***
- ***Irradiation to mediastinum***
- ***Pericardial fibrosis obliterates the pericardial sac with adherence of parietal pericardium to surrounding structures producing a great strain of cardiac function***
- ***Systolic contraction, the heart pulls against the parietal pericardium the attached surrounding structures.***
- ***Systolic retraction of the rib cage and diaphragm, pulsus paradoxus -clinical findings***



Adhesive pericardium following 3 previous cardiac surgical procedures—tons of fibrosis

***Thank you***